look upon it as an accident, and a rare accident, either due to rather stronger than usual adhesions between the peritoneal coats of the retrograde intussusception or due to a ballooning of the gut at the site of the retrograde intussusception as the direct one approaches it. I have given as my reason for thinking that the condition was produced by a swallowing process of direct by retrograde rather than by a secondary retrograde intussuscepting process of a primary direct invagination that the retrograde cuff was so tight that it was impossible for it to have been formed in this latter way. In my experience, one does not get this condition of extreme tightness in the case of the superimposition of a direct upon a direct intussusception, which condition is definitely recognized as the secondary intussusception of a primary direct intussusception *en masse*.

I have noted that the mother said the child had been ailing for five or six weeks; the greater part of this period no doubt represented a period of acute enteritis, which was the most important causative factor in the development of the intussusception.

In this case one cannot but be struck by the fact that, in spite of the size of the intussusception and the tightness of the cuff at its proximal end, the pathological changes which had taken place were only very slight. One can only attempt to explain this by surmising that the swallowing of the direct intussusception by the retrograde occurred very rapidly and recently, and that the tightness of the retrograde cuff became at once sufficient to completely obstruct both arterial supply and venous return, instead of, as in simple types of intussusceptions, the venous return becoming obliterated before the pressure was sufficient to obstruct arterial flow.

After the operation the child almost at once gave evidence of great hunger; considering that he had not retained much food for some time, this is not to be wondered at, but the fact that he called most loudly for tomatoes was certainly a clue to the cause of the original enteritis.

Regarding differential diagnosis I shall always bear in mind in the future the peculiarity of the tumour to be felt—namely, the upper firm rounded portion, representing the site of the retrograde superimposition, which was quite distinct from the larger, softer, and less defined lower portion, representing the portion which was purely the direct descending intussusception.

REFERENCES.

- ¹ D'Arcy Power: *Trans. of Path. Soc. of Lond.*, vol. 1, 1899, p. 121.
² Charles P. B. Clubbe: *The Diagnosis and Treatment of Intussusception*, p. 66. ³ D'Arcy Power: *Trans. of Path. Soc. of Lond.*, vol. xxxvii, p. 240.

KALA-AZAR IN MESOPOTAMIA AND ITS INCUBATION PERIOD.

BY

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AN interesting discussion has been taking place in the pages of this JOURNAL as to the indigenous existence of kala-azar in Mesopotamia. It started with Major Low's article on June 7th, 1919, and was continued by Dr. Hamill on July 5th, and by Lieut.-Colonel Ledingham on July 19th. To this last letter Major Low and Captain Billing replied on July 26th. That my contribution is so late is due to my continued presence in the country under discussion.

The most important point in settling the question of the presence of kala-azar in Mesopotamia is the maximum length of the incubation period of that disease.

Major Low and Dr. Hamill both consider the question of the existence of kala-azar in this country settled in the affirmative. Colonel Ledingham expresses doubts on the subject, and I am quite with him in this attitude, although I have met cases of kala-azar here where the interval between the commencement of an unbroken period of residence in Mesopotamia and first evidence of illness is much longer than in Major Low's and Dr. Hamill's cases. In Major Low's case, taking his own reading of the date of onset, the incubation period might be eight months.

The patient had been in Bangalore, a place from which I have met other cases of this disease.

Dr. Hamill's case I was acquainted with, and I think I am right in saying that repeated spleen and liver puncture by very competent observers failed to find the Leishman-Donovan body in this patient. Even, however, if the diagnosis of kala-azar were correct there was here also an interval of only eight months between the patient leaving India and the onset of his illness in Mesopotamia. In Colonel Ledingham's first case the interval appears to be the same; in his other cases the dates are uncertain.

I have met with several cases of kala-azar in Mesopotamia, and I have selected from my records of these cases five examples—four Indians and one British—where the interval between leaving India and the onset of the disease in Mesopotamia was more than double that mentioned above.

In each of these cases the diagnosis was confirmed by finding the *Leishmania donovani* in the spleen during life, and the man's movements were confirmed by reference to his service book.

1. *Follower A.* Nearly two years in Mesopotamia before admission to hospital in October, 1917.

2. *Bearer D.* Had been in Mesopotamia for seventeen months continuously previous to the first onset of the fever on August 31st, 1918.

3. *Private G.*, a British soldier. Had been in Mesopotamia (near Baghdad) for seventeen months continuously before onset of the fever on January 8th, 1919. Came to Mesopotamia from Bangalore.

4. *Follower M. M.* Had been in Mesopotamia continuously from September, 1917, up to the date of his admission to hospital for kala-azar on March 2nd, 1919—an interval of just over seventeen months. Came from Noakhale in East Bengal.

5. *Follower G.* Came from Dacca in East Bengal. Left Dacca in March, 1917, arrived in Mesopotamia, April 10th, 1917, and has not left the country since. Arrived in Baghdad in May, 1917, and remained in Northern Mesopotamia. Was in hospital for a month in the autumn of 1918 for some febrile disease (nature unknown), and was discharged as fit. Came to Basra apparently well on April 22nd, and was admitted to hospital for kala-azar on April 30th, 1919. There is here an interval of over two years between arrival in Mesopotamia and admission for the disease.

Long as these intervals are, I do not regard them as conclusive proof of the endemicity of kala-azar in Mesopotamia, though certainly they are weighty evidence. But with equal force it may be held that the disease in these cases was contracted in India and that the many months of health in Mesopotamia are evidence of a long incubation period in some cases of kala-azar. Moreover, each of the Indian cases I have seen in Mesopotamia has come from a known infested area in India.

Previous experience in India has led me to believe that kala-azar is not always the malignant disease we used to think it was, and that, independently of active treatment and independently of the occasional beneficial effects of such dangerous complications as cancerum oris, cure may sometimes spontaneously occur. I think the resemblance to malaria may perhaps go further, and that the parasite may in some cases long remain quiescent in internal organs and only begin to evince its malignant toxicity when some other factor has lowered resistance.

As Colonel Ledingham says, proof of the existence of kala-azar in Mesopotamia must come from its occurrence amongst the native population, and such civil surgeons as I have asked have not seen a case here; neither have I in a limited experience of local Arabs and Persians; nor has any native practitioner that I have met. Dr. Ramy, a Syrian physician of extensive practice, who spent three years in Khanqin and has practised for many years in Basra, and who is, moreover, familiar with kala-azar from his Syrian experience, tells me that he has never seen a case in Mesopotamia.

With regard to Dr. Hamill's reference to Arab belief in a generalized disease following Baghdad boil no more reliance can be placed on their ideas than on other oriental stories, and no such pilgrimage from Baghdad to Basra for inoculation purposes ever took place. It is true the Arabs call some boils "male," and others "female," but the distinction is fanciful and devoid of scientific support. The only connexion known between Baghdad boil and kala-azar is in the morphological resemblance of their respective parasites, and that is a matter beyond the Arab ken.

In conclusion, therefore, the evidence as to the existence

of kala-azar in Mesopotamia is negative. Perhaps when the Mosul area has been more opened up we may obtain some positive evidence from that direction; but meanwhile the matter must be considered unsettled.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TREATMENT OF CHILBLAINS.

As chilblains are now prevalent I send prescriptions for applications which I have used for many years, with or without the internal administration of calcium chloride, according to the state of the patient.

For Unbroken Chilblains.
R. Tinct. capsici ... 3 ss
Lin. camph. co. ... 3 ss

Misce.
To be painted frequently over the affected area.

For Broken Chilblains.
R. Ung. resinae ... 3 ss
Ung. acidi carbolic ... 3 ss
Zinci oxid ... 3 ij
Vasellini ... ad 3 ij

Misce. Ft. ung.

London, W.

T. MARK HOVELL.

SEVERE ANAPHYLAXIS: RECOVERY.

THE fatal case of anaphylaxis after diphtheria antitoxin recorded in the BRITISH MEDICAL JOURNAL of November 8th (page 596) prompts me to record an equally severe case with recovery.

The patient was a man suffering from compound fracture of the tibia. He had the usual doses of antitetanic serum, and had been rendered sensitive to horse serum. When seen by me on August 23rd, 1919, he was suffering from erysipelas spreading from the wound and spreading bronchopneumonia. His condition was critical, and it was decided to give him 30 c.c.m. of polyvalent antistreptococcal serum intravenously. This was diluted in saline, but the mistake made was that no effort was made to desensitize the patient before administration. Within a minute after the injection he complained that he was choking, grasped his larynx, and breathed jerkily and spasmodically. He became cyanosed at once, his lips, cheeks, and neck almost assuming a black colour, and the pulse was flickering; breathing ceased in full inspiration.

The foot of the bed was raised immediately, and artificial respiration commenced by forcibly pressing in the lower ribs, whilst the mouth was cleared of much mucus by the nurse. I decided to try and relieve the spasm by making him take in any air which could be got in through a chloroform mask. A nurse held the mask, fairly well saturated with chloroform, over his mouth and nose whilst I continued to try and force in his chest, thereby getting a little air in with a mixture of chloroform. At the same time he was given 30 minims of adrenalin solution and atropine gr. $\frac{1}{10}$ subcutaneously. Very soon chloroform began to have its effect, for in about five minutes the patient breathed, and when fully under the anaesthetic breathed normally. The cyanosis disappeared and the pulse returned; when counted it was 150 to the minute and of very low tension.

About one hour later the patient was comparatively well, and the next morning the desired effect from the serum was manifest; the general condition was much improved, and the temperature and pulse only a little above normal. He eventually made an excellent recovery.

The decision to try chloroform in a man who was apparently dying seemed a bold one, but when it is remembered that death occurs from anaphylaxis with the chest in full inspiration, and that the condition appears to be a spasm of muscle fibres in the fine bronchi, there seemed only one thing to be done, and that was to try and relieve the spasm by an anaesthetic. It should also be remembered that it is not possible to produce anaphylaxis in an animal while it is under the influence of ether.

The death recorded on November 8th was after the administration of diphtheria antitoxin given as a prophylactic. Is this advisable? (1) May not this first dose render the child sensitive to horse serum, and risk a catastrophe should it require a horse serum at any future date? (2) Diphtheria antitoxin is antitoxic, and not antimicrobial.

Colchester.

WM. T. MUNRO, M.D., D.P.H.

Reports of Societies.

WAR LESSONS IN SPINAL SURGERY.

At a meeting of the Section of Neurology of the Royal Society of Medicine, held on November 13th, the president, Dr. W. ALDREN TURNER, being in the chair, Mr. PERCY SARGENT read a paper on some lessons of the war applied to spinal surgery.

In some respects, he said, gunshot injuries of the spine differed from those usually met with in civil practice. The primary outstanding difference was that of wound infection. The gunshot wound was invariably compound whilst the fracture-dislocation was not. The majority of wounds in which the cord was directly or indirectly implicated did not lend themselves to early radical incision. Whether or not a patient with a gunshot injury of the spine died of meningitis depended almost wholly upon whether or not the dura mater had been torn, and in no circumstances was it justifiable to open the intact theca in the presence of a septic wound in order to ascertain the state of its contents. It became evident in the earliest days that the escape of cerebro-spinal fluid almost certainly meant death from meningitis unless the leak could be stopped, and this, in an otherwise favourable case, he always regarded as one of the few definite indications for early operation. In many cases the theca had been punctured by a fragment of bone which, remaining *in situ*, plugged the hole which it had made, and prevented both the escape of cerebro-spinal fluid and the ingress of micro-organisms. An operation which uncorked the theca in the presence of a septic wound enormously decreased the chances of recovery. Mild infections of the spinal membranes from which the patient recovered might produce intrathecal adhesions such as gave rise later to that loculation of cerebro spinal fluid which so closely resembled the meningitis *circumscripta serosa* described by Horsley.

The second outstanding difference concerned the manner in which the cord was damaged. Most of the injuries of civil life were produced either by the massive displacement of one part of the vertebral column upon another, or by a crushing fracture of the vertebral bodies, whereby part of the comminuted bone was squeezed backwards so as to contuse and compress the cord. Momentary displacements of the two vertebrae upon one another, with immediate rebound, might also injure the cord. In any case the damage was most commonly produced by direct violence, and the result might vary from a slight contusion up to a complete shearing across of the cord. The cases of gunshot injury comparable with these were those in which a spent bullet or shrapnel ball, impinging upon some part of the spine, entered the vertebral canal, driving fragments of bone before it. But when a missile of high velocity struck some part of a vertebra without entering, or directly violating the contents of, the vertebral canal, effects were commonly produced which were less often seen in the spinal injuries of civil life. The resulting changes in the cord were usually described as concussion phenomena, and the lesions varied from transient oedema up to total disintegration of the cord at the point of maximum intensity. Momentary or very short-lived loss of function had been observed many times, not only from actual hits, but also as the result of the bursting of a high explosive shell close to the patient.

Morbid Anatomy of Cord Lesions.

After referring to the description of the morbid anatomy and histology of the damaged spinal cord given by Dr. Gordon Holmes in his Goulstonian Lectures (1915), Mr. Sargent said that in the light of these observations the pre-war conception of haematomyelia needed revision. Minute haemorrhages were present in every cord which had been at all severely damaged, and were relatively unimportant, but in fracture-dislocation haemorrhages of any size were very rare. Similarly, whilst a slight extramedullary and extrathecal haemorrhage was commonly met with in recent examples of gunshot wounds, a haemorrhage of sufficient extent to cause symptoms of compression was rarely encountered. On the other hand, a small amount of effused blood within the theca might perhaps give rise later to adhesions and loculation of the cerebro-spinal fluid.