L. S., who is aged 28 years, the "tubercles" are numerous and stand out boldly, the central giant cell being surrounded by endothelioid cells of fibroblastic tendency, for the fibrous areas mentioned above are most marked where the giant-celled formations are most numerous. In M. C., who is 65 years of age, the typical "tubercles" are few and far between, but scattered throughout the section are large numbers of "lonely," somewhat atrophic, giant cells, with no tissue reaction (fibrosis, etc.) around them. This latter appearance has been observed not infrequently by one of us in senile tuberculosis of lymphatic glands, and is, after all, what one might expect in old age where tissue reaction to infection is often half-hearted and atypical. It is further of interest from this point of view to remark that, of the sections examined, it is only in those of M. C. that necrosis of tissue was found. Although sections were diligently searched for tubercle bacilli by three competent observers, none were seen.

These two cases well illustrate the type described by Winternitz in his study of fifty-one examples. The comparatively mild symptomatology, the size of the spleen, the existence of tuberculosis in the liver and lymphatic glands leading to it, and in the lung in the one case; the super-vention of generalization in the other, despite splenectomy (performed, as Dr. Ward suggests to us, "too late"), and the microscopical findings, are all in accordance with the general picture. No inoculation experiments were performed in our cases.

Although no definite primary focus was found, and although the splenic lesion dominated the clinical picture and was the source of spread, we hesitate to accept the term "primary tuberculosis" of the spleen. From a surgical point of view the conclusion to be drawn from a study of these cases is that splenectomy should be performed early, and that, for what it is worth, irregular fever with splenomegaly should suggest tuberculosis; but we admit that though, having seen one of these cases recently, one of us (J. A. B. H.) did suggest the diagnosis in the other, yet in the presence of the splenomegaly syndrome we are still very much in the dark as to diagnosis and rational treatment.

Recent bibliography in Silvestrini, *Patologia e Chirurgia della Milza*, Capelli, Bologna, 1924.

DIFFICULTIES IN THE DIAGNOSIS OF CEREBELLAR ABSCESS.

BY

R. SCOTT STEVENSON, M.D. EDIN.,

SENIOR ASSISTANT SURGEON, METROPOLITAN EAR, NOSE, AND THROAT HOSPITAL; ASSISTANT, EAR AND THROAT DEPARTMENT, UNIVERSITY COLLEGE HOSPITAL, LONDON.

THE signs and symptoms of cerebellar abscess, in a typical case, are very definite, and it may be imagined on reading the textbook descriptions that the diagnosis of a case should present few, if any, difficulties. In practice, however, the diagnosis of a cerebellar abscess is often very difficult. So experienced a surgeon as Mr. J. S. Fraser¹ said of his own cases, not long ago, that in only four out of eight cases of cerebellar abscess was the diagnosis sufficiently certain at the time of operation to enable the surgeon to proceed to the opening of the abscess.

Intracranial abscesses are almost always the result of chronic, rather than of acute, suppuration of the middle ear, and cerebral are much more common than cerebellar abscesses. Neumann² found, in a total of 532, that 336 were cerebral and 196 cerebellar abscesses; and Hunter Tod,³ in a collection of 100 cases, found that in adults cerebral abscess occurred in 65 per cent. of cases and cerebellar in 35 per cent.

The onset of an intracranial abscess is gradual, and as the symptoms arise during the course of chronic suppuration of the middle ear, the patient is not usually seen at an early stage by the otologist. The chief symptoms, which are due to intracranial pressure, are headache (usually dull and not, as in meningitis, excruciating), insomnia, a sub-

normal temperature, a slow, bounding pulse, vomiting, and optic neuritis. It is usually taught that a subnormal temperature and a slow pulse are typical of intracranial abscess; but in intracranial abscess secondary to middle-ear suppuration a rise of temperature often occurs, and the pulse rate may not be slowed. Again, while the presence of optic neuritis may be evidence of increased intracranial pressure, its absence does not negative intracranial abscess. It is said that in cerebellar abscess the vomiting and the optic neuritis are more marked than in cerebral abscess, but, on the other hand, both of these symptoms may be absent. Headache is usually stated to be more severe, persistent, and localized in cerebellar than in cerebral abscess, but mental dullness is much less marked. A staggering gait, vertigo (which seems to be quite independent of the ataxia), and nystagmus, are all symptoms which are usually considered to point definitely to cerebellar abscess, but they may also be symptoms of labyrinthitis. In the latter, however, the ataxia and the nystagmus tend to subside, while in cerebellar abscess they are inclined to become more marked; deafness, moreover, is usually absolute in labyrinthine inflammation, but the hearing may not be affected in cerebellar disease. Sometimes in cerebellar abscess there are no disturbances of equilibrium. Nystagmus is often absent in cerebellar abscess; but, on the other hand, it would hardly be wise to make a definite diagnosis of cerebellar abscess in the continuous absence of nystagmus. Bárány has shown that in a cerebellar lesion the patient past-points outwards with the arm on the affected side, and after vestibular irritation (induced by irrigating the ear of the opposite side with cold water) the pointing deviation of the same arm continues towards the same side, whereas in a normal person the past-pointing of both arms which results is towards the opposite side; unfortunately these changes in pointing accuracy are not constantly present in cerebellar abscess, and their absence does not exclude it.

The difficulty of making a diagnosis of cerebellar abscess is well illustrated by the cases of two patients on whom I have operated recently. The first, who presented typical symptoms and signs of cerebellar abscess, proved to be a case of extradural abscess in the middle fossa, with chronic perilabyrinthitis; while the second, a case of cerebellar abscess, was diagnosed as a case of acute mastoiditis only.

CASE I.

History.—J. M., a man aged 34, came to hospital complaining of giddiness and of intense pain on the left side of the head, which had prevented him from sleeping for four nights. The pain had begun a week previously, radiating down the left side of the face and settling below the left ear. Two days after the onset of pain he had four attacks of giddiness at his work, without losing consciousness, and on his way home he vomited twice. Since then he had felt rather dazed; he had not vomited again, but had felt inclined to do so several times.

Examination.—The left ear was discharging, and the patient stated that it had discharged from time to time for the past sixteen years. There was a large perforation of the left tympanic membrane, and a considerable quantity of pus was exuding. There was tenderness over the point of the mastoid. He could hear the conversational voice at a distance of 3 feet, but not a whisper. Rinne's test was negative, and bone conduction was prolonged fifteen seconds. The fistula test was negative; he past-pointed with the left hand to the left. There was definite nystagmus to the left; the margins of the optic discs were blurred. His gait was ataxic; his temperature was 102.4°, his pulse rate 80, and his respiration rate 20.

Diagnosis.—A diagnosis was made of cerebellar abscess complicating suppurative mastoiditis on the left side, and the patient was accordingly admitted to hospital for operation.

Operation.—Next day I performed a radical mastoid operation on the left side. The mastoid cells were full of pus, and pus welled up from the antrum when it was opened up. Beyond the antrum the bone was necrotic, so I continued to clear it away, and as I opened into the middle fossa of the skull there was a gush of pus under pressure. I enlarged this opening, but there was no bulging of the dura, and there did not appear to be any intradural inflammation. The lateral sinus appeared to be healthy, and there was no track towards the cerebellum. I accordingly made a meatal flap, "bipped" the wound, and drained the cavity with a wide rubber tube in the external meatus and a gauze drain to the post-aural wound, which I closed partially.

The patient's temperature fell to normal in five days. When he got out of bed at first he complained of giddiness, but this

gradually disappeared. He remained in hospital for a month, and made an excellent recovery. When I saw him recently he was quite well, and his hearing was fair; he had had no further giddiness. The post-aural wound soon healed, and although there was a slight discharge from the ear for a month or two it is now dry.

In this case the symptoms attributed to cerebellar abscess were caused by the intracranial pressure of a large extradural abscess, and also by chronic perilabyrinthitis, which subsided as the mastoid suppuration cleared up. A more thorough examination of the vestibular apparatus would probably have helped towards a more precise diagnosis, but it is obviously impossible to carry this out in the case of a very sick man with a temperature of over 102°.

CASE II.

History.—D. B., a man aged 52, was sent into hospital by his doctor as an emergency acute mastoiditis. He complained of very severe pains in his head, which he had had for six or eight weeks, sometimes at the vertex, but mostly behind the left ear. He had been unable to sleep or to work for over a week. He had never had any ear discharge, and was not deaf. Two days before he came to hospital a swelling had appeared behind the left ear.

Examination.—The left mastoid region was swollen, and tender to the touch. There was no discharge from the left ear, and the tympanic membrane was opaque, but otherwise seemed normal. Rinne's test was positive, and the hearing was apparently unaffected; there was no vertigo, no nystagmus, no optic change apparent. His temperature was 99.6°, his pulse rate 112, and his respiration rate 24.

Diagnosis.—A diagnosis of acute mastoiditis on the left side was made, and an immediate operation was decided upon.

Operation.—I made the incision for the Schwartz mastoid operation on the left side, and pus appeared at once when I reached the bone. The mastoid cells were necrotic and full of pus, more so towards the tip of the mastoid process. I tracked the pus downwards and behind the lateral sinus to the posterior fossa of the skull, from which pus welled up as I made a wider opening into it. The dura mater over the cerebellum was bulging, and I made a crucial incision over the bulge, through which a small quantity of pus came out. I then opened up the antrum, "bipped" the wound, packed it lightly with gauze through to the cerebellum, and left the posterior wound open.

The patient was in hospital for just over a month, and left in good health, but with a post-aural sinus discharging. When I last examined him, three months after the date of the operation, this sinus had cleared up, and the posterior wound was completely healed. Rinne's test was positive; with the left ear he could hear the conversational voice at 35 feet, and a whisper at 6 feet.

This case presented no symptoms or signs of cerebellar abscess, nor, indeed, of any intracranial lesion, though perhaps the severity of the localized headache should have made me more suspicious than it did. It is also interesting as being a case of mastoiditis without any apparent middle-ear inflammation (so-called latent otitis media), which is rare in adults, but somewhat more common in children.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, November 29th, 1924, p. 993. ² *Cerebellar Abscess*, English edition, p. 1. ³ *Diseases of the Ear*, p. 259.

VACCINE TREATMENT OF SPLENOMEGALY IN CHRONIC MALARIA.

BY

R. E. INGRAM-JOHNSON, L.R.C.P.Ed.,

LABU, F.M.S.

In the tropics the presence of children with chronically enlarged spleens is always regarded as a source of danger of infection, as the enlargement is evidence of the presence of the malaria parasite in large numbers in the spleen itself, with the possibility of its appearance in the peripheral circulation at any time. The percentage of children suffering in this way is taken as an index of the health of the community so far as malaria is concerned.

Being fully convinced that some antitoxin is formed in the blood of persons who have suffered from true dengue fever, which has a destructive action upon the malaria parasite, I obtained a serum from the blood of persons just recovered from true dengue and experimented with it, by intramuscular injection, upon children with chronic splenomegaly, as I concluded that it would probably destroy the parasite, in which case the spleen would gradually resume, at any rate to some extent, its normal size. Should my conclusions be correct, the shrinkage would prove the presence of the antitoxin.

The photographs reproduced are of the three children upon whom I first tried this method of treatment. They show: (1) the line of the splenic margin before treatment; (2) the shrinkage at the date the photographs were taken; (3) the costal margin.

Case A, a boy aged 8, and Case B, a boy aged 5, each received the first injection on October 16th, 1924, the second on October

23rd, and the third on November 11th. The photographs were taken on December 25th. Case C, a boy aged 13, received the first injection on November 9th, 1924, the second on November 16th, and the third on November 30th. The photograph was taken on December 14th. I examined this patient again on March 1st, 1925, and found the spleen normal in size, but very mobile, as the ligaments were very lax.

These cases were chosen as having enormous spleens, a deficient haemoglobin index, and as having been under the usual treatment for many months without effect. They had no other treatment but the serum after the first injection. I claim to have proved—

1. That an antitoxin against malaria exists in the blood of patients who have suffered from true dengue.

2. That the antitoxin is due to the presence of dead (not artificially killed) dengue germs, thus forming a true vaccine in the blood serum.

3. That this serum, being a vaccine, and having a destructive effect upon the malaria parasite, contains dead germs which must, in life, have had some close connexion with that parasite, or be a modification thereof, even as the active principle of vaccinia is a modification of that of variola, as, were there no connexion between the two diseases, malaria and dengue,

it could not possibly have the effect that it undoubtedly has.

4. The vaccine having such an effect on malaria parasites present in the circulation (even though not causing acute attacks of malaria) should, by its presence in the blood, prevent the proliferation of parasites injected into the blood as sporozoites by infected anophelines, and hence should act as a prophylactic against the disease.

5. The logical sequence is, therefore, that by injections of this vaccine artificial immunity from malaria can be procured; the length of time that immunity holds good, and the quantity of vaccine needed to make it absolute, being matters for further experiment.

CASE A.

CASE B.

CASE C.