TABLE II.

No.	Count on Zeiss Slide.	Count of Dried Drop.	A-B.	Percentage of Error.
	A.	B.		
1	6,825,000	6,712,000	+113,000	+1.7
2	2,430,000	2,406,000	+24,000	+1.0
3	4,662,000	4,728,000	-66,000	-1.4
4	3,737,000	3,584,000	+153,000	+4.9
5	4,459,000	4,324,000	+135,000	+2.9

In Column A are the number of red cells per c.mm. as found by counting in the ordinary way on a Zeiss slide. A dilution of 1 in 200 in the methyl violet diluting fluid was used for this purpose, eight large squares each consisting of sixteen small squares being counted.

For column B 5 c.mm. of the first dilution were taken and mixed with .495 c.mm. of the eosin solution. This gives a final dilution of 1 in 20,000. From this dilution drops were blown on to slides from one of the specially graduated 5 c.mm. pipettes. These were dried and mounted in Canada balsam. One slide was selected and counted. Since the dilution was 1 in 20,000 and the total red cells in 5 c.mm. were counted, the total count multiplied by 20,000 ÷ 5 = 4,000 gives the number of red cells per c.mm. The numbers so obtained are given in column B.

It will be seen that Columns A and B are approximately the same. On working out the difference, Column B is found on the average to be 1.1 per cent. less than A. This may be taken as proving that few or no red cells are lost while the drop is drying.

THE IODOFORM TREATMENT OF TUBERCULOUS DISEASE.

By MIDDLETON CONNOR, M.B., C.M., D.P.H.,
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THE two following cases of tuberculous joint disease, successfully treated by the injection of iodoform emulsion, are reported because such treatment is still upon its trial. The success in the cases reported has been encouraging, and such as invites further application of this method of treatment. It shows, moreover, that, even in cases of longstanding mischief, good results may be looked for.

Both cases were met in private practice. The preparation of iodoform emulsion used was according to Squire's formula and the method of injection was as follows: About a drachm of the emulsion was injected every alternate day into the joint affected, through the sinus, with an ordinary glass syringe. By keeping the nozzle of the syringe firmly against the sinus opening the emulsion was allowed to act on the joint for from three to five minutes. At first pain was complained of after the injection, but this was alleviated by the subsequent injection of very weak carbolic solution to wash out the iodoform. After a fortnight, however, this was unnecessary as the pain after injection disappeared. The sinus was subsequently covered with a pad of dry double cyanide gauze, and left untouched till the next injection, that is, two days later.

CASE I.—L. A., aged 17, message girl. Nine years ago resection of the right elbow-joint was performed for tuberculous disease, following a fall on the olecranon. The joint is now stiff, but she can make good use of hand and arm.

History.—Towards the end of summer, 1898, she sprained her left foot severely, but received no skilled treatment till the end of November, when, on account of the formation of an abscess at the joint, an incision had to be made. The ankle continued discharging a large amount of pus till May 1899, when part of the fibula was removed and the joint scraped. Further scraping was carried out on two occasions in June and July.

State on Examination.—The patient came under my notice in September 1899, when there appeared very marked wasting of the foot and leg, with distinct puffiness of the ankle. On the inner side of the joint a recent scar was visible, and on the outer side a large ulcerated surface, tuberculous-looking, with a sinus at its base, which allowed the passage of a probe for $1\frac{1}{2}$ in., when necrosed bone was felt. Pain was acute on attempting to flex the joint, and tenderness was elicited all round the articulation. A large amount of pus was being discharged. The general health was poor.

Treatment.—The injection of iodoform emulsion in the manner noted above was commenced in October, 1899, a dry dressing being placed on the wound. This form of treatment was carried out for two months, when the sinus and ulcerated surface were quite healed. The foot and ankle were then encased in plaster-of-paris for six weeks, and the patient allowed to go about and out by means of crutches, a patten being placed on the sound foot. After the removal of the plaster a thick-soled boot was worn, and at the present time the patient is able to go about without the aid of either crutches or sticks. The joint is stiff and the patient has a decided limp, but is free from pain and her general health is all that could be desired.

CASE II.—J. B., aged 9, a schoolboy. His mother and uncle died of acute phthisis, but he enjoyed excellent health till he was three years old.

History.—The present illness began in 1896 with indefinite symptoms of pain in both legs; he always desired, after walking a short distance, to be carried. In February, 1897, pain was complained of in the left knee-joint, which was swollen. The knee was thought to have been strained, and an ointment said to contain tar was applied. A few weeks later pain was complained of in the left hip-joint, and symptoms of morbus coxae became evident. The lower part of the abdomen and the left leg were encased in plaster-of-paris, the patient being permitted to get about on crutches with a patten on the sound foot. At the end of ten weeks this was removed, and a Thomas's splint was worn day and night for eleven months, when it was gradually done away with and the patient allowed to go about on crutches. No pain was at this time complained of.

State on Examination.—The patient first came under my notice in February, 1899, on account of a bulging in the left groin. This was distinctly fluctuating, and on incision a large amount of pus was evacuated. A probe passed into the joint detected a large area of necrosed bone. The discharge continued for several months. The general health was very indifferent.

Treatment.—In November, 1899, as no progress was noticeable, the iodoform emulsion was injected as noted above, and was repeated on alternate days till May, 1900, when the treatment was discontinued, as the wound was quite superficial and had no communication with the joint. For six months after the healing process was completed the boy was allowed to go about on crutches and a patten. Since then, by the aid of a stick, he can walk long distances, and is a most regular attender at school. He has a decided limp, but is in excellent health, free from pain, and shows no sign of any active disease whatever.

In both cases, immediately after commencing the injection, the discharge was materially and surprisingly lessened. Both patients, moreover, began to improve in general health and to gain weight from the time of its first use. Cod-liver oil and cream were administered, and these, no doubt, materially aided the recovery of the patients. The pain at first felt on injection of the iodoform was, in all probability, due to its irritative action on the diseased tissues, the subsequent washing out with weak carbolic solution getting rid of any excess of iodoform. The fact that the patients have remained quite well for fully eighteen months is an encouragement for further use of this method.

In conclusion, it may be mentioned that in two recent cases of tuberculous cervical glands with discharge lasting for from two to four months, the injection of the emulsion rapidly stopped the discharge with the subsequent complete healing of the sinuses. Both patients were young and followed outdoor employment.

SCIENTIFIC GRANTS COMMITTEE

OF THE

British Medical Association.

REPORT LXXVI.

THE CLINICAL DIAGNOSIS AND SURGICAL TREATMENT OF TUBERCULOSIS OF THE TEMPORAL BONE,

CONSIDERED WITH REFERENCE TO THE PATHOLOGY AND MORBID ANATOMY OF THE DISEASE.

By W. JOBSON HORNE, M.D., B.C.CANTAB., M.R.C.P.,
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TUBERCULOSIS of the ear, I venture to think, has not received the attention it deserves. The subject is beset with doubts and difficulties, and these, I trust, the discussion this evening will help to clarify. The present paper, to economize time, is given in abstract form, and does not attempt to do more than draw attention to some of the doubtful points, and to put forward theories in explanation which may elicit discussion.

In the first place, it is a question whether tuberculosis of the ear should be regarded strictly as a form of middle-ear disease in precisely the same sense as any other form of suppurative otitis, or whether it should not more properly be grouped under tuberculous disease of bone. It will be noted from the title of the paper that the more comprehensive view of the disease is for the present tentatively held.

As regards the frequency of the disease, the statistics so far published are not sufficiently uniform to allow of an opinion

being based upon them, and they present discrepancies owing to the inequality of evidence upon which diagnosis has been allowed to rest. Some observers have required positive evidence, at times too difficult to obtain, even when the disease has far progressed; whilst other observers have contented themselves with a diagnosis based upon certain clinical phenomena, which may be briefly summarized as follows: (1) Absence of pain out of all proportion to the destructive character of the disease; (2) insidious onset; (3) marked loss of hearing power; (4) extensive destruction of bone, rapid extension to labyrinth, absence of headache and dizziness; (5) progressive and destructive character, leading perhaps to facial paralysis or even severe haemorrhage; (6) absence of intracranial complications; and (7) in a particular group of cases considerable involvement of adjacent glands. These signs collectively go to make up a very strong case in favour of tuberculosis of the ear, but they all have this in common—they are only relative.

First, the absence of pain. Speaking generally, pain seldom accompanies diseases of the ear. It is, however, met with in myringitis. Those who have been fortunate in observing tuberculous disease in its earliest stages speak of it as commencing as a deposition of tubercle in the membrana tympani, unattended by pain. This absence of pain is not to be accounted for by an absence of tension, but by the absence of ordinary inflammatory phenomena. Then, again, acute middle-ear suppuration, especially in children, is commonly associated with pain, and yet in the tuberculous variety it is absent. Caries of the temporal bone in the course of its progress is commonly attended with pain, and yet there is no form of ear disease with so much destruction of bone and with so little or no pain as tuberculous disease. In one case which I had the opportunity of investigating, not only was there extensive necrosis, but a subperiosteal abscess had formed over the necrosed area; and yet it was not pain, but facial paralysis that drew attention to the ear.

I think one is therefore justified in saying that the absence of pain is not only deserving of comment, but that it cannot be entirely accounted for by the absence of ordinary physical causes. To this point I shall presently revert.

The insidious nature of the onset and progress of the disease follows upon what has been said, and I need not dwell upon this feature.

The rapid extension to the labyrinth, the absence of headache and dizziness, and the marked loss of hearing are all features of diagnostic importance.

The progressive and destructive character, with, perhaps, facial paralysis or even severe haemorrhage, makes up an important feature in the clinical diagnosis, but asserts itself too late to be of any practical utility in the clinical diagnosis.

The presence or absence of intracranial complications and the enlargement of adjacent glands I shall deal with later in discussing the differential diagnosis of the primary and secondary forms of the disease.

Here I may refer to the frequency—of course, comparatively speaking—with which the disease is secondary to pulmonary phthisis; and when I say phthisis, let me add that, in my dead-house experience, it is usually secondary to cavity formation, and not to pure miliary tubercle of the lung. I am therefore led to assume that infection in such cases is by the sputum. Observers abroad have noted that the ear infection, when it occurs secondary to phthisis, takes place not uncommonly shortly before death. In this country, in our hospital practice, we seldom have the opportunity of observing the very last stages of phthisis, as such cases go to infirmaries. But I would suggest that the bedridden condition of the subject renders the sputum more liable to remain in the neighbourhood of the Eustachian tubes, and the ear more exposed to infection; and I would mention the danger of infecting with tubercle the ear of a phthisical patient by incautious catheterizing and inflating to relieve an intercurrent aural affection. But, admitting all this, the fact remains that the suppurative disease of the middle ear occurring in a tuberculous subject is not necessarily tuberculous in nature.

The only positive proof of the disease being tuberculous is the detection of the tubercle bacillus within the affected area. Negative evidence is of no value; failure to detect the bacillus tuberculosis does not prove the disease to be non-tuberculous, nor does it disprove the possibility of it being tuberculous.

At times there is considerable difficulty in finding the bacillus, notwithstanding the presence of clinical evidence

which leaves little doubt in the mind of the aurist that the disease is tuberculous both in nature and in origin. By way of illustrating this difficulty, I would refer to a case at the necropsy of which the bacillus could not be detected in any of the numerous films made from the middle ear and accessory cavities. Animal experiment was resorted to and controlled, without evidence of tubercle being obtained. The bacillus was eventually found in the external fascia and tissues, superficial to the area of necrosed bone. I need scarcely add that such a detection of evidence does not come within the range of practical otology.

The difficulty in obtaining positive proof arises from the bacilli being broken up and disintegrated in the presence of other organisms or of leucocytes, and it is important here to discuss some points in connexion with the acid-fast property of the bacillus, a property upon which we depend for our microscopic diagnosis. At first it was held by Unna and Klebs that the acid-fast property depended upon the presence of a fat in the bacillus. Aronson, in 1898, showed that the body was not a fat, but was of the nature of a wax, and that tubercle bacilli, after being treated with alcohol and ether alone, retained their acid-fast properties; whereas, if the mixture were allowed to act in the presence of hydrochloric acid, the bacilli were no longer acid-fast. This work has been confirmed by Dr. Bulloch¹ and Mr. Macleod, and to these observers I am largely indebted. It is, therefore, proved that the acid-fast property is due to the presence of a wax in the bacillus and not to a fat. For the moment I stop at the wax stage of my argument and pass to another point.

Various bacilli are met with in the ear, and the question arises whether any of these, by their sojourn in the neighbourhood of ceruminous glands, can absorb wax and become "acid-fast," and by simulating tubercle bacilli vitiate a microscopic diagnosis. Even if that were possible—and I believe it may be—it would be undesirable to refer to them as acid-fast bacilli, or to confuse them with a well-defined group, the members of which have all been cultivated, and when injected produce pseudo-tubercle, which after all is purely a histological conception, and can be produced by other than acid-fast organisms. The presence of a member of this group in a film preparation from the ear would probably be due to faulty technique in obtaining the specimen. On the other hand, a bacillus which has acquired an acid-fast property by absorption of wax from the meatus could be differentiated by culture and animal experiments. But this is a point requiring further investigation.

Now, to return to the body of the nature of wax, one of the constituents of a tubercle bacillus, and the one which gives to the bacillus that acid-fast property that is lost when the wax is broken up. When a fat is decomposed a glycerine and an acid are produced, whereas in the case of a wax another member of the alcohol series other than glycerine is liberated. The precise nature and position in the series of this alcohol has not at present been ascertained, but it apparently represents the measure of the acid-fastness of the bacillus.

I would suggest that the difficulty in staining the bacilli and the absence of pain in tuberculosis of the ear are due to one and the same cause, namely, the disintegration of the bacillus and the decomposition of its wax-like constituent, with the resulting liberation of a product—tentatively called an alcohol—which produces an anaesthetic effect. Such a theory would explain not only the causation of two important clinical phenomena of the disease, but also their correlation. This absence of pain is not peculiar to tuberculosis when it affects the ear. I might instance cold abscesses of a tuberculous nature. Then, again, it is within our experience to have witnessed necropsies revealing tuberculous peritonitis, which, owing to the absence of pain, notwithstanding a matting together of intestines by adhesions, has not been suspected during life. Possibly an investigation into the nature of cerumen of the ear with reference to the points I have mentioned might not be altogether void of interest and of value.

I now pass to a brief consideration of some points which may guide us in the differentiation of a primary from a secondary infection of the ear. Primary tuberculosis is more commonly met with in children, I might almost say, from my own limited experience, exclusively, and I believe it to be of more frequent occurrence than is generally stated. Tuberculous disease may be added to an existing infection of the ear in an adult, but then that is not tuberculosis *de novo*. What has always appeared to me to be a feature of primary

infection is the enlargement of the adjacent lymphatic glands; and here enters a great problem, the part played by the lymphatics in arresting tuberculosis. The enlargement of the glands, although in a sense evidence of disease, is at the same time the outcome of a battle fought to resist disease. The lymphatics form not only the lines of defence, but also, where inefficient, the paths of general invasion, and we have to consider this when we come to deal surgically with a case of primary infection.

In secondary infection of the ear with tubercle there is comparatively, if not entirely, an absence of enlargement of the adjacent lymphatics. Why they are not affected in the secondary form I must leave for a paper by itself.

A second point in differential diagnosis between primary and secondary infection is that in the latter there is evidence of tubercle elsewhere, commonly in the lungs, and, as I have already said, with cavity formation. The tonsils and adenoids, when they play a part in the infection, I regard rather in the light of half-way houses than as primary foci.

A third point is the presence or absence of intracranial complications. It is hardly correct, without a qualifying clause, to say that tuberculous suppuration leads much less often than other forms of middle-ear suppuration to intracranial complications. Intracranial complications seldom occur in the secondary form, but in the primary form the child is not uncommonly killed by a meningitis, as part of a general blood infection.

The practical bearing which discrimination between primary and secondary infection has upon the surgical treatment—and by that I mean an attempt to eradicate the bone disease—may be briefly stated as follows:

In the primary form, when the adjacent glands are involved, let these be removed, first attacking the more distal and least affected, the intention being to cut off the spread of infection, and so localize the focus of disease to be removed at a later date.

When the disease is secondary to advanced pulmonary tuberculosis, a mastoid operation is seldom required; on the contrary, a mastoid operation is more likely to be what a tracheotomy is to laryngeal tuberculosis—the beginning of the end.

I am desirous of expressing my indebtedness and thanks to the British Medical Association, under whose auspices the research, of which this is the record of a part, is being conducted.

REFERENCE.

¹ The Morphological and Physiological Variations of the Bacillus Tuberculosis and its Relations. By William Bulloch, M.D. *Transactions of the British Congress on Tuberculosis*, vol. iii, p. 494.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

ON THE POSSIBILITY OF THE OCCURRENCE OF
TRYPANOSOMIASIS IN INDIA.

WITH regard to Major Leishman's contribution under the above head in the *BRITISH MEDICAL JOURNAL* of May 30th, I wish to state briefly that I have noted bodies similar to those described by him in smears taken *post mortem* from enlarged spleens of patients—natives of India—said to have died of chronic malaria. I obtained them in three consecutive cases on April 9th, 23rd, and 24th, 1903.

In the first instance, I thought I had discovered the long-sought-for resting-stage form of the malarial parasite in man, but could not compare them with any analogous stages in the sporozoa. However, on again procuring the same bodies in the two other cases, I changed my views, and considered they were probably *post-mortem* degenerations of the nuclei of the splenic pulp cells.

On reading Major Leishman's paper, I at once recognized the similarity of his so-called degenerations of the trypanosomata to those found by me in the spleens of the cadavers above mentioned.

Yesterday (June 17th) I had occasion to puncture *intra vitam* the spleen of a native boy, aged 12 years, suffering from irregular pyrexia, with no malarial parasite in his peripheral blood (careful examination of stained films on four several occasions), and found identical bodies in the blood from the

spleen, thus removing any doubt there was as to the products being due to *post-mortem* changes.

It is unwise to theorize on the insufficient grounds at present in hand. I hope to contribute something more definite on the subject after further and more prolonged study of these organisms. My films were stained by the Maurer-Romanowsky method. I am familiar with the appearance of the trypanosomata—*T. evansi*, *T. lewisi*, and a species doubtful occurring in the blood of the common Indian squirrel (*Sciurus palmarum*). There was nothing resembling trypanosomata in the peripheral blood of the boy in question.

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A CASE OF ASCITES MOST PROBABLY CAUSED BY
INFECTIVE THROMBOSIS OF THE HEPATIC
VEINS.

DURING 1902 I visited five times in consultation with her medical man a female patient, aged 47. She was well nourished, and of very healthy appearance. She was married, had had two children, the youngest aged 11 years, and two miscarriages since, the last one seven years ago. Her family history was good.

My first visit was March 3rd, 1902, when I was told that she had been in bed for two months before Christmas with cellulitis of one arm. This was dealt with by an operating surgeon, and 14 incisions were made, but no pus came out. The surgeon thought the cause was partly lymphangitis and partly thrombosis. The arm was completely restored. The patient said that she had had white leg at least once after a confinement.

At Christmas there was a sense of fullness in the abdomen. She had not menstruated since the middle of October, having been quite regular up till then. Ascites was recognized a week or so before my first visit, and was attended with pain and tenderness in both iliac regions. The ascites increased, and began to interfere with the breathing; there was also some slight swelling of the ankles. I could not feel the liver or spleen, and there were no superficial abdominal veins to be seen. By a process of exclusion I narrowed down my diagnosis to infective thrombosis of the hepatic veins. There was no likelihood at all of syphilis notwithstanding the miscarriages, although there had been a tendency to thrombosis of other veins in former illnesses. More than once we thought we must recommend paracentesis, but by rest and elaterium, which she stood well, and by inducing profuse diaphoresis and diuresis the patient after three or four months made a complete recovery. It is possible that the climacteric was intimately connected with the *fms et origo mali*.

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CASE OF PROLAPSUS UTERI TREATED BY PARAFFIN
INJECTIONS.

THE patient, aged 69, and too feeble for a radical operation, had for the last twelve years suffered from complete prolapsus uteri. The uterus was nearly always down, even when she was in bed, and was a source of much misery. The perineum was rather less than an inch in length. Pessaries had been tried but failed to keep the prolapse up.

Operation.—May 24th, 1903. The patient was anaesthetized and placed in the lithotomy position. The method followed was that suggested by Mr. Stephen Paget (*Lancet*, May 16th, 1903). Paraffin melting at about 46° C. (115° F.) was used, and injections of about 9 c.cm. (f. 3 iiss) each were made in several places under the mucous membrane of the lateral and posterior vaginal walls. Two or three injections were also made about the cervix. About 95 c.cm. (f. 3 iij 3 iij) of paraffin were used in all. A finger was kept in the rectum to guide the posterior and lateral injections: and a probe was passed into the urethra while making the more anterior injections. An ordinary antitoxin syringe was found to act well. The operation lasted about one hour and a quarter, some time being lost by the paraffin setting in the needle of the syringe.

To speak of cure at present would be, perhaps, premature, but the result has been surprisingly satisfactory. The lumen of the vagina has been very much narrowed and its walls stiffened. The uterus is now completely held up during