almost certain he will answer in the affirmative, is whether the experiments, in which Rous's fowl sarcoma was produced by the inoculation of a chloroform-treated "sand-filtrate" of one of these tumours together with a "sub-culture" of the Rous "virus," were controlled by giving parallel injections of material in which uninoculated culture medium containing fresh chicken embryo was substituted for the "subculture." Another point which strikes one is the resemblance between the chloroformed sarcoma extract, which Dr. Gye does not regard as living and calls the "specific chemical factor," and some of the so-called "filter-passing viruses" which are associated with various other diseases. Some of these are remarkably resistant to adverse conditions. For example, the virus of vaccinia is well known to have a high resistance to chloroform and ether, and the virus of foot-and-mouth disease is known to resist drying over sulphuric acid, and was found by Loeffler sometimes to withstand 1 per cent.

carbolic for many weeks. If the "specific factor" is really a simple chemical substance, as Dr. Gye believes, it is remarkable that it should be able to determine the kind of cell in the new growth resulting from an inoculation and the kind of animal in which the virus can cause growth.

Dr. GYE said: The answer to Dr. Arkwright's first question is "Yes." Parallel experiments have been done many times with negative results. The second question is more difficult. The interpretation put upon the experiments has been founded upon: first, cultures from chloroformed extract fail; secondly, direct dilution experiments are all favourable to the interpretation given; and thirdly, the assumption that the effective factor is another virus again leads us to the position that strict specificity is dependent upon a virus. In short, the simplest and most direct interpretation has been put forward, and this is supported by available facts.

TWO CASES OF TUBERCULOUS SPLENOMEGALY.

BY

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AND

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THE title "Tuberculous splenomegaly" has been chosen to avoid the implications of the word "primary," sometimes used in the description of this form of tuberculosis.

CASE I.

Maria C., aged 65, was admitted under the care of Mr. W. G. Spencer—to whom we are indebted for permission to publish the notes—on November 10th, 1919.

There was but a five weeks' history of progressive wasting and muscular weakness, with no particular complaint or symptoms other than those due to the presence of the splenic tumour. She had noticed for two or three years that her abdomen was getting bigger. She had bad varicose veins in the left leg; they had ruptured three times. She was pallid; her atrophic skin had a subicteric tint. The tumour filled the whole left upper and lower quadrants and the right lower quadrant of the abdomen. The right side of the tumour, on which the notch was recognizable, was somewhat mobile, whilst the left side of it seemed to be deeply anchored.

Laparotomy had been performed in 1911 at St. Thomas's Hospital for removal of an ovarian cyst and the appendix: no enlargement of the spleen was noted at that time; a fibroma had also been removed from the left labium.

There was irregular fever on admission, the temperature ranging from normal to 102°; pulse 88 to 104.

The blood count on November 18th, 1919, was as follows:

Serum to corpuscles	...	...	...	1 to 1
Haemoglobin	...	...	...	65%
Colour index	...	...	...	0.8
White blood corpuscles	...	...	...	8,200
Polynuclear neutrophils	...	...	...	70%
Eosinophils	...	...	...	4%
Large mononuclears	...	...	...	10%
Small mononuclears	...	...	...	16%

The Wassermann reaction was negative.

Splenectomy was performed by Mr. Spencer on November 25th. There were no adhesions of the spleen, but the transverse colon was firmly attached to the anterior parietal peritoneum. In dealing with the lienorenal ligament the tip of the tail of the pancreas was removed and the ligatured pedicle was therefore fixed in the wound.

On the day after the operation the temperature was normal, but respirations were 40 to 50; on the next day the pulse was 124, temperature 101.2°, respirations 286. She died on November 28th, 1919.

The necropsy notes state: Emaciation; no peritonitis; along splenic vein five enlarged lymphatic glands; liver greatly enlarged (73 oz.), pale, and firm on section: no amyloid reaction. Small subperitoneal cyst on surface of left lobe. Kidneys pale and firm on section; right 6 oz., left 3½ oz. Left ovary absent; right small. No tubercles on peritoneum, diaphragm, or parietal pleura. On visceral pleura a few grey tubercles. Lungs: scattered grey miliary tubercles, more on right side than left; lungs otherwise well aerated; not engorged. Gastro-intestinal tract *nil*.

Microscopical:—Lungs: emphysema, pneumoconiosis, tuberculosis. The liver and lymphatic glands showed tuberculosis. Kidney: cloudy swelling, no tuberculosis.

The surface of the spleen (which weighed 72 oz.) was smooth; there was no thickening of the capsule; scattered over the surface were a number of pea-size yellow areas, each surrounded by a zone of hyperaemia. On section the spleen, which was very vascular, showed similar yellowish areas with hyperaemic zones, some one to two inches in diameter; no caseation.

CASE II.

Lizzie S., aged 28, a patient of Dr. Gordon Ward, was sent to one of us in April, 1920, complaining of weakness and constant fatigue; of the presence of a lump in the abdomen, noticed for the first time six months before, and associated with discomfort, sense of weight and dragging in the left side. She had lost weight. She had also a sero-purulent nasal discharge, due, as reported by Mr. de Santi, to advanced left-sided atrophic rhinitis with crust formation. The spleen, firm and apparently smooth to the touch, occupied the whole left side of the abdomen, to three or three and a half inches below the umbilicus; there was no enlargement of lymphatic glands to be detected; the liver was but slightly if at all larger than normal; no other abnormal physical signs could be found. There was irregular fever, the temperature reaching 99.6°, 100.8°, 100.2°, and so on in the evenings.

The blood count on April 30th, 1920, was as follows:

Serum to corpuscles	...	...	...	1 to 1
Haemoglobin	...	...	...	90%
Red blood corpuscles	...	...	...	5,820,000
White blood corpuscles	...	...	...	4,600
Polynuclear neutrophils	...	...	...	56%
Eosinophils	...	...	...	3%
Large mononuclears	...	...	...	27%
Small mononuclears	...	...	...	14%

Splenectomy was performed (E. R. C.) on May 1st. The spleen was adherent to the diaphragm and elsewhere, but presented no considerable difficulties in removal; a cigarette drain was employed. The stitches were out on May 9th and she was discharged on May 20th.

A blood count taken on May 7th, 1920, was as follows:

Serum to corpuscles	...	...	...	1 to 1
Haemoglobin	...	...	...	68%
Red blood corpuscles	...	...	...	4,430,000
Colour index	...	...	...	0.8 approx.
White blood corpuscles	...	...	...	10,500
Polynuclear neutrophils	...	...	...	69%
Eosinophils	...	...	...	1%
Large mononuclears	...	...	...	17%
Small mononuclears	...	...	...	6%
Transitionals	...	...	...	6%
Basophils	...	...	...	1%

Dr. Gordon Ward informs us that for some weeks after discharge she picked up nicely, but that she died six months later of generalized tuberculosis.

The spleen weighed 4 lb. It closely resembled a spleen from a case of miliary tuberculosis, but on a very much larger scale. The surface was studded all over by raised tubercles, surrounded by small zones of hyperaemia. These tubercles were of considerable size, being generally the size of a large pin's head. On cut section also the very free distribution of the tubercles was well seen; and the contrast between the very numerous tubercles of this spleen and the comparatively few of "Maria C." was noteworthy. Another contrast was the comparatively smooth surface of the spleen of "Maria C." and the nodular surface of this case, "Lizzie S." There were no areas of caseation.

In considering these two spleens, it was found that they had certain characteristic features in common, but presented certain well marked differences. A feature common to both is vascularity, with a change of the ordinary splenic structure into that of a moderately loose-meshed sponge, of which the interstices are for the most part filled with blood; indeed, in one spleen (L. S.) so loose is this meshwork that parts of it resemble lung tissue at the first casual glance. Another similarity is a general increase in the interstitial fibrous connective tissue; in M. C. the process is uniformly diffuse; in L. S. there are, in addition, well marked areas of local fibrosis where the tubercles are "healing" or have "healed." In both spleens, whilst some typical "giant-celled" formations are found, there are differences in others which, as a histological study, are of great interest. In

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

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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