The Local Focus.

The next point is the removal of local focal infection as a factor in prevention. On June 7th, 1900, a man, age 28, came to St. Mary's Hospital suffering from a sore throat. The condition was one of acute tonsillitis and he had mitral and aortic disease, arthritis, and muscular pains. There was a previous attack of rheumatic fever four years before. Dr. Paine, by plate culture, isolated a strepto-diplococcus morphologically and culturally indistinguishable from that we had isolated from the lesions of acute rheumatism. Intravenous injections into a rabbit produced acute aortic and mitral endocarditis. Fitz-Meyer, two years later, by extensive experiments supported these results, and later with Mr. George Waugh we repeatedly verified the ability of this micrococcus obtained from unhealthy tonsils to produce cardiac and arthritic lesions without any previous injury to the valves. This was the birth of the local focus in rheumatism.

No one can be more convinced than I am that the rheumatic infection has one important site of entry into the system through diseased tonsils, and particularly those which resemble, in their deep lacunae, culture tubes. No one probably is less convinced than I that wholesale removal is indicated in the rheumatic child, or that the removal of the local focus shuts the door. Nature is not so easily managed, and I have seen severe carditis in a first attack occurring some years after a complete enucleation. Recent American statistics bear out this experience, for Ingerman and Wilson report that, of a series of cases in which tonsillectomy had been undertaken for rheumatic manifestations there occurred, in a period between one and eleven years after the operation, rheumatic relapses in 76 per cent. I look upon the tonsils as an important barrier; but as no man is more dangerous than a clever man who has lost his capacity, so are badly damaged tonsils a constant source of danger. Careful treatment of the throat, nasopharynx, and teeth in the prevention of acute rheumatism will be, in my opinion, a permanent factor. More investigation is required to determine other local foci as equally definite.

Research.

That further research is essential to advance the prevention of acute rheumatism is generally admitted, and this must mean the assistance of expert investigators trained in the modern methods of bacteriology, serology, and biochemistry. Opinions as to the best method for obtaining results will naturally differ, and I can but express my own views. I think there should be some institution in which the study of "rheumatism" should be continued uninterruptedly, and in a centre such as London I would have this institution concerned with the entire problem of rheumatic affections, for it is my experience that in research one is liable, if following a particular line, to reach a dead end; some fact or some technical detail is lacking or the brain becomes wearied. Then one needs to leave that line for a time and to work in other directions, returning later refreshed with new ideas or with new equipment. The problem of "rheumatism" would always have open spaces; now there might be fresh light thrown on malignant endocarditis, now a discovery in the bacteriology of rheumatoid arthritis, or some chemical advance bearing upon fibrositis. Thus the vitality of the research would be kept active.

I am no believer in an easy path. A keen intelligence even working on wrong lines will learn useful lessons and be the stronger, but one value in the establishment of a continuous research lies in the diminution of the likelihood that a young investigator would wander off the track for a long time. If I may give one personal illustration—Dr. Paine and I spent two years searching for a suspected bacillus and discarded the very micrococcus we eventually believed to be the causal agent of acute rheumatism. Two years is a long time; and wiser heads might have given us a hint that would have put us right earlier in this vain search. Such an institution could be organized for training investigators and putting them in touch with our great hospitals and infirmaries, and would have at hand an ample literature. I admit the scheme is ambitious, but I am looking ahead and outlining what appears to me the rational

steps to bring about a steady progress in our knowledge, remembering always the great wastage of money that at present results from the ravages of rheumatism.

Conclusion.

I think that I have shown that we have in this country at least the skeleton of a valuable organization for the prevention of acute rheumatism. Given the *will* to clothe this skeleton with living flesh, there seems to me to be great possibilities. Dr. Carey Coombs very wisely points out that any scheme must be evolved slowly, and I am in entire agreement with him. It is my belief that the best results in most great endeavours are obtained by testing the strength and weakness of the materials already at hand rather than by wholesale revolution. I have attempted to disclose weak points, and these can be cautiously strengthened. No one would suppose that I should advocate filling the land with country hospitals, but *one* such connected with the children's hospitals in London would be an experiment justified by our knowledge of disease, and in this hospital acute rheumatism could form one section, and the results be carefully tested.

Of this I am convinced, that Sir George Newman and his powerful staffs will continue on their side to develop our resources for prevention, and the hospital staffs and practitioners are prepared to take their parts. It is for an enlightened public to consider whether they will help also, for it is for them and for their children that we are working. If we are met by the answer that there is no money for such endeavours, then we can but improve to the best of our ability, and by mutual organization, our present resources; but the public should realize how many children and adults in this country are maimed by this disease, and should know our difficulties, our need for research, and the present limitations of our resources.

In conclusion I would venture to repeat a prophecy I dared to give in 1913 in an address to the Invalid Children's Aid Association. "That as surely as I stood on the platform the time would come when the care of the rheumatic child would attract much more attention in this country"; and I would add now "in all civilized countries." I wish also in this lecture room, where the great name of Harvey has been so often glorified, to express my belief that the experimental production of organic heart affections by micrococci obtained from rheumatic carditis has opened up the road for a great advance in the treatment of cardiac disease and for the relief of much suffering, by leading our thoughts to the problem of the *prevention* of acute rheumatism.

SPIROCHAETOSIS ICTERHAEMORRHAGICA.

THE PRESENCE OF THE CAUSAL ORGANISM IN
SLIME AS A SOURCE OF AN OUTBREAK
IN EAST LOTHIAN.

BY

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A PRELIMINARY note¹ recording the occurrence of this disease in Scotland was published in February, 1924. Since that time an extensive investigation has been in progress, and is still being continued.

At present it is desired to give an account of one aspect of the inquiry which led to the discovery of leptospiral organisms in a natural habitat hitherto unrecorded. The observation will no doubt be confirmed in other circumstances which are being investigated, but the existing conditions associated with the present finding alone may help to throw further light on the epidemiology of the disease, particularly in relation to its occurrence in certain coal mines.

In 1914 Inada and Ido discovered the causal organism of Weil's disease. They reported their discovery in a Japanese journal in 1915. An English communication by Inada, Ido, Hoki, Kaneko, and Ito,² which summarized the publications

in the Japanese literature on this subject, appeared in 1916. The disease was found to prevail in epidemic and endemic form in certain parts of Japan. In addition to the liability of butchers, cooks, and field workers to the disease in that country, they noted its frequency among coal-miners working in that part of a mine inundated with water. They also stated that "the spirochaetae seem to live in water and mud, and to invade the human body from there." A later communication by Ido and his associates³ announced the finding of the specific organism in a certain proportion of rats caught on the surface and in infected pits.

Since the discovery of the causal organism the disease has been more widely recognized, and infected rats have invariably been considered one of the chief sources of infection in man. Toyama⁴ later observed that the prevalence of Weil's disease was closely associated with conditions of the soil in Japan, and found that all infected districts were paddy fields and localities with swampy ground. Other observers have reported the presence of leptospiral organisms in water and mud, but the direct relationship of such water leptospiras to Weil's disease was not definitely established by animal experiment.

Nature and Result of the Investigation.

In the infected area in Scotland a considerable percentage of rats and field-mice was found to be infected with the pathogenic spirochaete, or leptospira (Noguchi). This fact having been established, the examination of surface and pit waters was indicated. This has been done on an extensive scale.

A personal visit to one of the infected coal mines was arranged by the county medical officer of health for East Lothian, Dr. G. Y. Richardson, who, by the kindness of the pit owner, received permission for an inspection in February of this year. It so happened that the mine visited was inundated with water in certain parts and was known as a "wet" pit. A large number of water samples was collected, by means of sterile glassware, from different sections of the mine. These samples included specimens from new cuttings where the miners were working practically under water, from recent workings which were very wet, and from old sections which may be termed damp in comparison with the other parts. It was in one of the old sections that the following observations were made which resulted in a finding of much significance.

Compared with other branches of the mine there was little ground water; no water was seen to drop from the roof, but the whole tunnel was very damp. Small stagnant, muddy pools were present from which samples were collected. Roof water was looked for in vain, but on holding a lamp close to the tunnel roof I noticed a slimy viscid substance, which formed a small thin layer only in certain areas. At parts it was seen to form hanging drops, which as they increased in size would no doubt fall into water or on to the ground. Other portions of the roof in the same section were merely damp with no slime, while in certain areas red sandstone drippings were present. The viscid substance was certainly not plentiful, and even the small amount collected was found only after careful search.

Wet films of the slime were first examined by dark-ground illumination, which revealed remarkable and varied forms of motile organisms, in addition to primitive forms of the vegetable kingdom. Different types of spirochaetes were distinguished by their morphology and motility. Only after prolonged examination were definite leptospiral organisms found, but they were few in number. The difficulty in recognizing this delicate organism in such a viscid substance may be emphasized.

To control the observation if possible, two guinea-pigs were inoculated intraperitoneally with 2 c.cm. of the glutinous material, thinned by the addition of a little saline to permit of its passage through a syringe needle. The result of these experiments was negative in both cases; no spirochaetosis developed during one month's observation.

A fresh sample of the roof slime from the same section of the mine was obtained at a later date. Dark-ground examination again showed the undoubted presence of typical leptospiras. Two guinea-pigs were again inoculated, but on this occasion with 4 c.cm. of the substance, as the

failure to infect the first two was attributed to the scarcity of the leptospiras in the smaller amount injected. Both animals died, one on the eleventh, the other on the thirteenth day after inoculation. They showed equally pronounced evidence of spirochaetal jaundice. Abundant leptospiral organisms were present in all the organs examined, and an appreciable number were also found in the blood and urine. Notable features were the intensity of the jaundice, the prominence of the widespread subcutaneous, muscular, and internal haemorrhages, the enlargement of the spleen, and the haemorrhages in the stomach in both animals. In fact, the lesions appeared more pronounced than in many animals infected with the human or rat strains.

It would appear, as far as can be ascertained from the relevant literature consulted, that this is the first occasion on which the direct inoculation of a water leptospira has proved pathogenic to guinea-pigs. This infected roof slime was found growing on slaty stone and wooden roof-bars in absolute darkness. It was greyish-brown in colour, and translucent when free from carbon particles. It formed a mass of considerable coherence and was not very readily detached from the stone roof. The consistency approached that of a thick gum solution, and resembled the semi-solid culture medium of Noguchi used in growing leptospira. The temperature of the slime in the mine was 11.7° C. in February. It was slightly alkaline when tested with litmus paper, and had a reaction of pH=7.5 approximately.

As mentioned above, the leptospira was recognized with difficulty in this substance teeming with other active life. A few of them were much longer than those found in man, the rat, or in cultures. Other types of spirochaetes were much more numerous, particularly one species. The characteristic motility of the leptospira in contrast to that of the other spirochaetes present was striking, and formed a feature which distinguished them from forms closely resembling the genus. Most of the other spirochaetes on escaping from the slime were actively translatory and moved rapidly in a horizontal plane; whereas the true leptospira remained in the field actively rotating for a second or two, then finally burrowed vertically into the viscid medium out of sight. The leptospira was found to exist in this semi-solid medium symbiotically with algae, fungi, cocci, bacilli, spirochaetes, flagellates, nematodes, and their ova. This slimy substance was formed by organisms with a glutinous envelope which yielded a jelly-like or zoogloea mass.

The continued existence of the leptospira and the other motile organisms was noted at different temperatures. Some tubes of slime were kept at room temperature, and others in the ice-chest for over five months. Dark-ground examination at the end of that time showed all the motile organisms, including leptospiras, quite recognizable and unimpaired in their activities. Animal inoculations were recently done to determine if the slime after keeping had maintained its infectivity. The results are not yet ready for publication.

It was previously noted that the slime was observed only in a very damp and old section of the mine; its situation on the roof appeared inaccessible to rats. I was informed by the pit manager that at the time of the outbreak of jaundice in May, 1923, practically all the miners affected were working in a particular section. This section was stated to have been the wettest part of the mine. Of much significance becomes the fact that this particular section happened to be that branch of the mine in which the infected roof slime was found. Eight miners developed symptoms of spirochaetal jaundice when engaged in this mine in question. Five of these were said to have fallen ill within a period of two weeks while working in this very wet cutting. Rats were never considered numerous in the pit, and an expert rat-catcher failed to trap any underground for examination, but caught one on the pit surface. This rat was found to be infected with pathogenic leptospira.

Discussion.

It is highly probable that such slime with leptospiras aggregated in it may infect by various routes. At the time of cutting into a new wet section existing slime in the soil

above may be dislodged by the force of movement of the ground water escaping into the pit. Miners locally, therefore, may be more exposed to infection at that time. After the flow of intruding ground water has diminished, the soil above is less likely to be disturbed. At a later period elements of slime in water, oozing from the soil, may settle and grow on the roof to form a nidus for the growth of the leptospira and other organisms. This slime may possibly drop on to the ground, into stagnant pools, and running pit water, from which sources man and rat alike, locally and at a distance, may conceivably become infected. It may be, however, that the pathogenic leptospira escaping from this rich natural organic medium into running water, or water with little organic matter, becomes attenuated after residence in the less favourable environment, as do the human and rat strains in culture medium. There will be a wider distribution of the organism in infected water than in the slime, therefore dosage may be an important factor in infection. Conditions in the mine which may possibly influence the pathogenicity of the leptospira are darkness, temperature, and depth. The temperature of the slime in the mine was 30° C. higher than that of pumped water in a cement surface pond. No cases of jaundice appeared to be associated with either the pond or an adjacent stream into which the pond water flowed. These waters both showed the presence of certain spirochaetes, but no leptospires were found in the centrifugalized deposits. Animal experiments with these and other surface waters have so far given negative results.

The opinion stated above, that the absence of favourable and adequate organic matter in the water may be a factor in the attenuation of a pathogenic leptospira, receives support at the time of writing. In an English version of Toyama's work⁵ just published it is recorded that a pathogenic strain actually became attenuated in experimental ponds with no soil, but preserved its virulence for a longer period in ponds with soil. Moreover, it is stated that "the spirochaete could preserve its poisonous nature two months in an imitation paddy field, which had entirely no self-cleaning function." The organic properties of the soil and water in which the leptospira may exist as a pathogen would therefore seem to be a factor in the preservation of its virulence. That the presence of the associated organisms in the slime and soil may lower the resistance of man and experimental animals, and so act as aggressins, requires consideration, as advised by Dr. P. P. Laidlaw. A French writer inoculated large doses of impure cultures of a water leptospira into guinea-pigs with negative results.

Spirochaetes, now considered similar to the genus *Leptospira* by Noguchi, were observed in water by Wolbach and Binger⁶ in 1914. They reported the isolation of a filtrable spirochaete from the shore of a fresh-water pond near Boston. Its pathogenicity for experimental animals was not tested. Several observers since then have recorded the occurrence of the same genus in water. Zuelzer,⁷ in 1922, discovered in tap water spirochaetes which morphologically resembled those of Weil's disease. Direct inoculation of the naturally infected water into animals, however, did not give rise to any symptoms. One strain of a water leptospira proved pathogenic to guinea-pigs after cultivation in serum medium for one and a quarter years—that is, mutation from saprophyte to pathogen had apparently occurred. Stevenson (unpublished observation) found a leptospiral organism in London tap water, but no guinea-pig inoculations were carried out. Rats injected with the water gave negative results. Both fresh and salt water have been found by Noguchi⁸ to contain leptospires indistinguishable from those producing disease in man. The organisms did not prove to be pathogenic to guinea-pigs.

Small epidemics of spirochaetal jaundice occurred last autumn in Northern France, one of which was associated with the river Vesle, at Reims. A number of young people who bathed in the river contracted the disease. Etchegoin⁹ examined the water and mud of the Vesle, and found a leptospiral organism which he considered had slightly wider elementary spirals than *Leptospira icterohaemorrhagiae*. The river water and mud, and massive doses of impure cultures of leptospira, were tested by animal inoculation, but did not produce spirochaetal jaundice.

It would thus seem that leptospiral organisms exist as saprophytes in certain waters and mud. The saprophytic and pathogenic forms are stated by most observers to be morphologically indistinguishable. Their close relationship in other directions has been demonstrated by Uhlenhuth and Zuelzer.¹⁰ Mention of mutation from saprophyte to pathogen has already been made. Biological and immunological tests carried out by them support the view of the water origin of Weil's disease. On the other hand, Akira Shiga¹¹ recently recorded serological differences between certain water strains and *Leptospira icterohaemorrhagiae*. The latter is stated to thrive in the immune serums of the former, but not in its own immune serum. The two different races of water leptospira tested were likewise only affected by their homologous immune serum, both being uninfluenced by the immune serum of the pathogenic strain. They also proved the greater power of resistance of the water organisms to ultraviolet rays and other influences, but found no great difference in the behaviour of the saprophytic and pathogenic strains towards certain parasitocides.

It has not been possible so far to carry out comparative serological and certain other tests with the slime, rat, and human strains in the present investigation. The most important property of pathogenicity, however, has been proved by animal experiment to be common to the three strains. Natural habitats of the genus have been found in wet soil and in water. It remains to determine if varying natural environments alone can influence mutation from a pathogenic to a saprophytic type and vice versa, or if immutable species exist. The change from a virulent to an avirulent organism in altered natural conditions was suspected, and the view held finds confirmation in Toyama's work already quoted. No mention is made of a return of virulence on introducing the water-attenuated leptospira into wet soil again. In the outbreak investigated by the writer it was observed that no cases were associated with the surface pond or stream, both of which received pumped water, presumably containing some leptospires, from the infected pit. The pond was lined with cement, contained little soil, and the temperature was 80° C.—factors possibly favouring attenuation. The stream was said to receive boiling water from mines near its banks. On the other hand, the change from saprophyte to pathogen in natural conditions has not been determined. A water saprophyte, however, was reported by Uhlenhuth and Zuelzer to have acquired pathogenic properties in culture medium.

The localization of the disease has been generally recognized, in spite of the presence of leptospiral organisms in many waters and rats in districts where the disease has not occurred. Epidemics have invariably been associated with wet and badly drained broken ground, as instanced by outbreaks among workers in rice fields and wet mines. During the war epidemics among soldiers were localized more or less to wet, ill drained trenches. The disease has also occurred among workers in sewers. In such environments conditions no doubt exist for the development of slime similar to that found in the mine. This slime medium apparently affords an ideal nidus for the growth and aggregation of pathogenic leptospires. Apart from environment such as favourable wet soil and slime, the organism may become attenuated as mentioned above, which suggests a possible reason for the localization of the disease.

Swimming baths in the poorer quarters of Paris have been held responsible for recent outbreaks, and certain bathing pools in Germany have been incriminated. It may be that leptospires, attenuated by sojourn in water, borne from soil, gain entrance to the ponds along with primitive vegetable elements capable of forming a zoogloea mass as a nidus. In this organic medium exaltation of virulence may possibly occur. One instance has been referred to in which a saprophytic water leptospira became pathogenic.

It is highly probable that rats inhabiting wet mines originally become infected from broken soil. They may acquire infection by feeding on slime in old disused tunnels blocked up with loose stones, as seen in the mine visited. One rat was actually observed to scamper into this ideal

hiding place. These old damp inaccessible branches no doubt afford favourable situations for the collection of much slime, the development of which will probably depend on the nature of the soil above. One means of combating the rat source of infection may perhaps be accomplished by completely walling off such conceivable breeding grounds from the main roadways.

Rats far distant from infected mines in East Lothian were also found to be infected with the leptospira, but no cases of jaundice have been reported in the particular areas. Foulerton,¹² Balfour,¹³ and Stevenson¹⁴ recorded the presence of pathogenic leptospires in rats from different counties in England where the disease was unknown. Coles,¹⁵ at Bournemouth, noted the existence of infected rats there, but pathogenicity tests were not done. These findings suggest that the leptospira is more ubiquitous in soil than at present suspected, and that rats may find a food delicacy in infected slime in soil. It may be also that they can acquire infection from water-attenuated leptospires which, by passage, become pathogenic to man in certain circumstances.

Reasonable evidence for the assumption that rats become infected from slime, soil, or water, and so act as secondary disseminators of the disease, was found on examination of their urine. In addition to the presence of leptospires, similar spirochaetes to those found in the slime and certain waters were found in the urine of many rats by dark-ground illumination.

Summary.

The discovery of an unrecorded natural habitat of the causal organism of spirochaetal jaundice has been reported. The natural habitat was found in an infected coal mine, and consisted of roof slime formed by organisms with a glutinous envelope which yielded a jelly-like or zoogloea mass.

Inoculation of the slime into guinea-pigs produced pronounced and fatal spirochaetal jaundice.

The viscid slime proved to be a favourable medium for the growth and continued life of the leptospira, which lived symbiotically with many other species of organisms.

The observation has established a possible source of infection underground, where the leptospira existed in a situation inaccessible to rats.

I desire to thank the Committee of the Royal College of Physicians' Laboratory for the facilities afforded in carrying out this research.

The Medical Research Council has defrayed the expenses of this work and given valuable assistance in other directions.

REFERENCES.

- ¹ Gulland and Buchanan: *BRITISH MEDICAL JOURNAL*, 1924, i, 313. ² Inada, Ido, Hoki, Kaneko, and Ito: *Journ. Exper. Med.*, 1916, xxiii, 377. ³ Ido, Hoki, Ito, and Wani: *Journ. Exper. Med.*, 1915, xxiv, 471. ⁴ Toyama: *Abstract Trop. Dis. Bull.*, 1924, xxi, 275. ⁵ Toyama: *Japan Medical World*, 1924, iv, 193. ⁶ Wolbach and Binger: *Journ. Med. Res.*, 1914, xxx, 23. ⁷ Zuelzer: *Cent. f. Bakt.*, 1 Abt., Orig., 1922, lxxxix, Beiheft, 171. ⁸ Noguchi: *New York State Journ. Med.*, 1922, xxii, 426. ⁹ Etchegoin: *Compt. rend. Soc. Biol.*, 1924, xc, 482. ¹⁰ Uhlenhuth and Zuelzer: *Klin. Woch.*, 1922, Jahrg. 1, 2124. ¹¹ Akira Shiga: *Zeit. f. Immunitätsforschung u. Exp. Therap.*, 1924, xl, 148. ¹² Foulerton: *Journ. Path. and Bact.*, 1919, xxiii, 78. ¹³ Balfour: *Parasitology*, 1922, xv, 282. ¹⁴ Stevenson: *Amer. Journ. Trop. Med.*, 1922, ii, 77. ¹⁵ Coles: *Lancet*, 1918, i, 468.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, BRADFORD, 1924.

SECTION OF LARYNGOLOGY AND OTOTOLOGY.

W. JOBSON HORNE, M.D., President.

CEREBELLAR ABSCESS.

BY

J. S. FRASER, M.B., F.R.C.S. EDIN.,

Surgeon, Ear and Throat Department, Royal Infirmary, Edinburgh. DURING the period 1907 to 1923 (inclusive) 2,000 cases of acute purulent otitis media were seen at the Ear and Throat Department of the Royal Infirmary, Edinburgh, under the charge of Dr. Logan Turner (1907 to 1921) or under my own charge (1921 to 1923). Among these cases, 609 came to mastoid operation. One patient had cerebellar abscess and three had abscess of the temporal lobe. During the same period 6,492 cases of chronic middle ear suppuration were seen by Dr. Logan Turner or myself; 1,305 radical or modified radical mastoid operations were performed, and among these 26 had cerebellar abscess and 27 abscess of the temporal lobe. I am deeply indebted to Dr. Logan Turner for his kindness in providing me with these statistics.

During the years 1907 to 1923 I have performed 140 Schwartze operations and 725 radical or modified radical mastoid operations, and have dealt with eight cerebellar and with ten temporal lobe abscesses. Of the eight cerebellar cases, only one followed acute middle ear suppuration, while the remaining seven resulted from chronic purulent otitis media. (Neumann states that 85 per cent. of cases are due to chronic suppurative otitis media.) Four of these eight cases recovered and four died; six were males and two females. (It is stated that males are affected twice as frequently as females.) With one exception, all cases were under 30 years of age. The cerebellar abscess was on the right side in five cases and on the left in three. (Of Milligan's twenty-seven cases, twenty were on the left side. Neumann gives the following figures: left, 304; right, 284.) Of the seven chronic cases, two were due to scarlet fever and two to measles; in the other three the cause was not known.

ROUTE OF INFECTION.

1. *Labyrinthitis*.—Of the seven chronic cases only two had labyrinth suppuration, and in these there was an extradural abscess deep in the posterior fossa in the region of the saccus endolymphaticus; in both of these cases the sinus was healthy. (Neumann, Milligan, and others state that the majority of cases of cerebellar abscess are secondary to labyrinthitis, though Neumann's actual figure is 43.75 per cent.)

2. *Perisinus Abscess, but no Sinus Thrombosis and no Labyrinthitis*.—Two cases; one of these was the acute case.

3. *Extradural Abscess in Posterior Fossa in Triangular Area, but no Sinus Thrombosis or Labyrinthitis*.—One case.

4. *Perisinus Abscess and Sinus Thrombosis, but no Labyrinthitis*.—Three cases.

My experience is, therefore, that cerebellar abscess is usually associated with extradural abscess in the posterior fossa, with or without sinus thrombosis. Labyrinth suppuration was present in only two out of eight cases.

SYMPTOMS.

We must remember the condition in which these patients are admitted to hospital. As a rule they are extremely ill and in a drowsy or semi-comatose condition, so that it is practically impossible to put them through a complete "third degree" examination in the way of functional tests of the cochlear and vestibular apparatus and of the nervous system. In the majority of cases questions were answered slowly, but it was only noted in one instance on admission that the speech had a slurring character.

(a) General Symptoms.

In at least three of the seven chronic cases symptoms of an intracranial complication had been present for two or three weeks before the patient was sent into hospital. Headache was present in all cases, and in two was not only occipital but also frontal; one patient held his head with his hands. (Okada considers fixed occipital pain the chief diagnostic symptom.) In six of the eight cases earache was noted apart from the headache; in all vomiting was a prominent symptom. Drowsiness was marked in at least five of the seven chronic cases; in the other two it was not noted. In three of the seven chronic cases shivering or rigors were present: these symptoms, however, were probably due to the presence of an extradural perisinus abscess or sinus thrombosis. In three of the chronic cases constipation was noted and the tongue was dry and furred. Emaciation was marked in four of the seven chronic cases.