Cyclitis.

Cyclitis is frequently tuberculous, and I have obtained most encouraging results from tuberculin therapy.

Choroiditis.

I do not intend to say much about this subject because I recently read a paper upon tuberculous disease of the uvea to the Midland Ophthalmological Society, which was published in the *Birmingham Medical Review*, May, 1912. It is not an uncommon affection, and exists in three forms—the acute miliary form generally seen in association with tuberculous meningitis, the solitary form, and the disseminated form. The miliary form does not concern us because it is not a strumous disease—it is not attenuated tubercle, but a highly virulent form. It is, however, interesting to note that Stephenson and Marple have both stated that tubercles are present in a very large number of cases of tuberculous meningitis. Marple suggests that if only the examination be made with sufficient frequency they will be detected in every case. They may be very evanescent, appearing rapidly and disappearing within a few hours. The solitary form may advance steadily to the total loss of the eye, but I believe them to be generally benign. I have so far never seen a case which has not cleared up under tuberculin treatment, leaving a practically undamaged eye. I constantly find scars in the cornea, pigmented patches, which represent all that remains of a past tuberculous process. It is, however, a not infrequent occurrence that a fresh solitary tubercle develops close to an old quiescent one. In children obsolescent tubercle of the macula is not uncommon, and naturally their central vision is very defective. Disseminated choroiditis is more likely to be due to syphilis than to tubercle, but I believe that many cases ascribed to this disease are in reality caused by the tubercle bacillus in an attenuated form—to struma, in fact.

Lacrymal Sac.

The lacrymal sac is frequently affected with tuberculosis, especially in children, and should be extirpated at once.

TREATMENT.

We have in tuberculin a weapon which is of inestimable value in the treatment of struma or attenuated tuberculosis. The small chronic foci which originate the ocular manifestations, and the small deposits in the eye do not generally generate enough tuberculo-toxin to excite the formation of antibodies in the blood. In these cases the injection of tuberculin supplies the absent stimulus, and the circulation becomes flooded with the necessary antibodies, whatever they may be, and so the tendency to repair obtains the necessary advantage over the destructive influences at work in the small focus of disease. If, however, the local focus in the eye be but a part of a widely-spread tuberculosis, I believe that tuberculin is apt to be very harmful. The same is true when a state of anaphylaxis is present. Two cases will illustrate this point. Both were adolescents; both had florid tuberculosis of the iris, with large tuberculous masses in the anterior chamber. The first patient was a healthy lad with no other signs of tuberculous infection and a normal temperature. He failed to react to three increasing doses of old tuberculin, but reacted strongly to a small dose of bovine old tuberculin. After a year's treatment with T.R. he made a complete and absolute recovery. The second case had a similar condition in both eyes, but, in addition, had masses of palpable glands in her abdomen and a hectic temperature. After rest in bed, when the temperature curve had become normal in character, I gave her an injection of 100 mg. of T.R.; the result was disastrous, and hastened the inevitable total loss of both eyes. Probably—nay, almost certainly—the same thing happens when tuberculin is used in phthisis and other tuberculous affections where the actual tubercles are hidden from us, and it behoves us to be exceedingly cautious how we use such a potent agent for good or evil. If recent legislation leads to the indiscriminate use of tuberculin, the results can only be disastrous both to the patients and to tuberculin therapy.

Up to the present I have only used T.R. I begin with 25 mg., unless I have special reasons for extreme caution. This dose I repeat once a week, and perhaps, after eight to ten weeks, increase it to 100 mg. It may, in some

cases, be advisable to again increase the dose to 250 mg., but generally I keep to the initial dose. A focal reaction obtained during a course of tuberculin treatment always does harm in eye cases, sometimes great harm. It is necessary to be very patient. Sometimes no improvement may be manifest for weeks or even months. My successful case of tuberculous iritis did not show the slightest sign of improvement for five or six months, and then he began to improve and got quite well.

In conclusion, gentlemen, the fact which I have brought to your notice, that attenuated tuberculous or strumous disease of the eyes is exceedingly common and generally amenable to treatment with tuberculin, should lead to the obvious conclusion that the eye is not likely to be the sole organ affected by attenuated tuberculosis. The presence of chronic rhinitis and of enlarged tonsils and adenoids in children should be regarded as indicating the probability of struma, and doubtless much of the chronic rheumatism so prevalent among us has a similar origin.

ON THE RIGIDITY OF CALCIFIED ARTERIES.

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(Communicated by PROFESSOR ADAMI.)

THE ordinary conception of calcification within the organism wherever it occurs is that it is of a stone-like rigid character, and this whether it occurs in a calcified fibroid of the uterus, a calcified obsolescent tubercle in the apex of a lung, or a calcareous plaque in an artery. A familiar expression is "the pipe-stem radial." Here it is true the calcareous deposit is not in the intima, but in the media, but in support of this view our museums show beautiful pipe-stem arteries, with dense white rings of calcification.

But is this view correct or constantly correct? Are there not data which are difficult to reconcile with such a view? Were the radial artery as rigid during life as we find it sometimes after death, why is it capable of expansion with each heart beat, as is indicated by the pulse tracing; why is it compressible by the finger; or why is it not shattered by the fingers of the numerous students during the clinic or ward round? And in the case of the aorta, if the saucer-like calcareous plates and scales which we find after death exist as such during life, why do we not have formed upon them, much more frequently, parietal thrombi, which, becoming free, cause emboli and gangrene? Why, again, do surgeons experience so little difficulty in ligaturing the arteries in a case of senile gangrene, although on examining the amputated limb later the proximal ends of the severed arteries are rigid and brittle?

As a matter of fact I would lay down as a general rule that arteries are not of a stony rigidity during life. (I shall later on give exceptions to this rule.) During life the calcareous matter in the arterial wall is not present in the form of rigid plates. It is there, but in the state that may be compared with unset mortar.

Let me place before you the following observations:

CASE 1.—C. D., aged 72, was a patient in Dr. Finley's service at the Montreal General Hospital. He had a blood pressure of from 180 to 205 mm. of mercury. The radial arteries were tortuous and could be rolled under the finger. He died somewhat suddenly, and permission for autopsy was obtained immediately. The radial arteries were dissected out within one hour of the patient's death. On removal they were no doubt more resistant than the normal, but they could be compressed and could be bent and curved like a moderately stiff rubber tube. About ten minutes after removal, however, the removed lengths of the vessel were found absolutely rigid, possessing "pipe-stem" characters. They crackled on compression and broke on being bent.

CASE 2.—Was one of senile gangrene of the foot in the service of Dr. Armstrong at the Royal Victoria Hospital, Montreal. Dr. Armstrong amputated at the upper portion of the popliteal space. The artery was much sclerosed, but on being severed bled freely, and was ligatured without difficulty.

The distal portion of the artery from Case 2 was immediately dissected out and disposed of as follows: A small length was allowed to stand in the open air; another was dropped into a one-half per cent. solution of sodium hydrate; another into a similar solution of hydrochloric acid; one into a normal saline, and another into a 20 per

cent. solution of potassium sulphocyanide, which latter paralyzes the muscle, and therefore dispenses with any rigidity due to muscular contraction. (These solutions were previously boiled and cooled to drive off any gas.) The following changes were noted:

In about ten minutes the artery exposed to air had become hard and brittle, would crackle on pressure, and break in the thicker areas when bent. After four hours the artery in the alkaline solution was still comparatively soft, could be cut easily with a knife, and could be ligatured without rupture. The length of artery placed in the acid solution had become quite hard, and almost as brittle as that in the open air. The artery in the saline solution presented about equal hardness to that in the acid solution, that in the potassium sulphocyanide was of about the same hardness as that in the hydrochloric acid, showing that the contraction of the muscles had no bearing on the extreme hardening.

The lower 2 or 3 cm. of the popliteal and the upper few cm. of the anterior and posterior tibial arteries were filled with recent clot, but like the parts above were pliable on removal. Below this, however, they were hard and brittle, and, on cutting, the knife encountered hard calcareous deposits, similar to that in the artery exposed to the air. As these tibial arteries were removed immediately after the popliteal, I cannot but conclude that this dense calcification had taken place before amputation.

Other confirmatory observations I owe to Dr. Keenan and to Dr. Gruner of the Royal Victoria Hospital, Montreal. Dr. Keenan, amputating a leg for senile gangrene, took special note of the popliteal artery at operation. He found it thickened and nodular but pliable, being ligatured without difficulty. He slit the amputated portion for a few centimetres and found it to cut easily. One hour later he again observed the artery, and found that it had become quite calcified and brittle.

Dr. Gruner, who did an autopsy immediately after death on a case dying of ruptured iliac aneurysm and extensive aortic atheroma, found the aorta fairly soft and pliable, moving without resistance under the fingers. Within a few hours, however, the specimen presented a very different appearance and feel, there being many rigid calcareous disc-shaped plaques with peripheral ulceration between which were areas of atheromatous degeneration.

I have employed the simile of unset mortar, but apparently this is more than a simile—it is, in fact, an identity. On making inquiries, I find that it is generally accepted by chemists that the process of setting of mortar is one of conversion of calcium hydroxide into calcium carbonate by the absorption of carbon dioxide from the air. Kept under an alkaline solution, or kept in bulk away from the air that contains carbon dioxide, mortar remains unset. I have it on the authority of Professor MacIntosh, of McGill University, Montreal, that a mixture of calcium carbonate and calcium phosphate remains soft so long as it is in an alkaline medium; placed in an acid solution it sets.

To test the matter I have made the following observations:

A. Experiments with Plain Mixed Mortar.

1. 200 grams of mortar were mixed with about 50 c.cm. of a 2 per cent. solution of sulphuric acid. A mild reaction took place with slight effervescence and evolution of heat. In about half an hour the mass had become dried, hard, and firmly "set," and remained so for over a week.

2. 200 grams of mortar mixed with 50 c.cm. of a 2 per cent. solution of sodium hydrate remained soft until it became dried out, twenty-four hours later, and then could be rubbed to a powder between the fingers.

3. 200 grams of mortar mixed with plain water, dried and became fairly hard and "set" in twenty-four hours, similar to the mortar to which no extra water had been added.

B. Experiments with Mixtures of Calcium Oxide and Calcium Phosphate.

1. About 10 grams each of calcium oxide and calcium phosphate were mixed to a thin paste with a 2 per cent. solution of sulphuric acid, and became hard and "set" in eight hours.

2. About 10 grams each of calcium oxide and calcium phosphate were mixed to a thin paste with a 2 per cent. solution of sodium hydrate. This dried out in twenty-four hours, but could be rubbed to a powder between the fingers.

3. A mixture of calcium oxide and calcium phosphate was rubbed to a thin paste with the fluid expressed from muscle tissue (acid in reaction). This became firm and set in twelve hours.

4. A similar preparation was made, using plain water. This set in about fourteen hours, but even then was not quite so hard as the one with the muscle juice.

5. A mixture of calcium oxide and calcium phosphate was rubbed to a thin paste with muscle juice, to which was added about one-quarter its volume of acetic acid. This became hard and set in eight hours.

6. A similar preparation was made, making the muscle juice strongly alkaline with sodium hydrate. This dried out after about twenty-four hours, but could then be rubbed to a powder between the fingers.

From these observations it would seem quite evident that these mixtures of calcium oxide and phosphate and also plain mortar "set" more quickly in an acid than in an alkaline medium. Also the more strongly acid the medium the faster they set, because when I added strong acid to the mortar there was a marked reaction, which, of course, is easily explained by the equation $\text{Ca}(\text{OH})_2 + \text{H}_2\text{SO}_4 = \text{CaSO}_4 + 2\text{H}_2\text{O}$, and the preparations became dry and extremely hard in a very few minutes.

Again passing CO_2 through the mortar did not seem to immediately affect it, as I stirred for about fifteen minutes a dish of mortar with a glass tube which was attached by a rubber tube to a CO_2 generator, thus while stirring the gas was bubbling through the mortar. Professor MacIntosh thinks there must be a complete reaction going on similar to that in the magnesium compounds used in flooring preparations.

Now it is worthy of note that in the first place the reaction of the tissues and body fluids during life is alkaline but becomes acid shortly after death. It is deserving of note particularly, in the second place, in connexion with arterial calcification, that the gas which after death replaces the blood within the arteries is largely composed of carbon dioxide.

Without doubt when there are large areas of necrosis appearing in the tissues, there the production of acid is sufficiently abundant to bring about an *intra vitam* setting of the calcareous matter. For instance, it is the experience of the gynaecologist that a calcified fibroid of the uterus may be found of stony hardness even while in the body; so also our own experience is that in a case of senile gangrene the arteries of the necrosed or gangrenous area are of stony hardness, whereas the same arteries higher in their course, where the circulation is still maintained, are soft and pliable and can be ligatured with ease.

In short, the realization of this fact, that in arteries provided with an adequate circulation the calcareous matter, whether it be in the intima or the media, remains unset, is calculated to fully explain the cruces indicated at the beginning of this paper. It is easy to understand now why, after handling pipe-stem radials, there is no thrombosis and gangrene, easy to understand why rigid and tortuous splenic and other mesenteric arteries in the aged arterio-sclerotic do not undergo traumatic rupture. During life they are not rigid. It is easy also to understand the occurrence of extensive so-called atheromatous ulceration of the aorta without thrombosis commonly occurring during life. Those ulcers, in short, and the saucer-like plaques with surrounding rupture of the endothelium are *post-mortem* productions. The calcified areas become in the intima set and rigid before the muscular contraction of the aorta gives way and the tube undergoes dilatation. The result is that a tension upon the endothelium at the edges of the plaque induces rupture of this lining.

THE Report of the Inspector-General of the Insane for 1911 states that there were 844 persons certified as insane in Western Australia on December 31st, 1911, an increase of 55 from December, 1910. The total population of the State on December 31st, 1911, was 294,133 (males, 168,260; females, 125,873). The number of admissions to the Government hospitals for the insane was 215 (males, 135; females, 80), and the proportion of admissions to population was 1 to 1,368, or 7.30 per 10,000. The readmissions only numbered 22, or 10.23 of the total admissions, which is gratifying. The number of patients discharged was 99, of whom 78 were discharged recovered; the proportion of recoveries to admissions was 36.27 per cent. During the year 66 patients died; of the deaths, 21, or 31.84 per cent., were due to general paralysis. There were only 4 deaths from phthisis. In connexion with the hospitals at Claremont and Whitby, farms are worked, which, besides affording occupation for the patients, are a source of revenue.