If the spasms are violent and repeated the local treatment must be reinforced by the internal use of special antispasmodics. In the case of the larynx, the best of these, beyond all comparison, is the liquid extract of grindelia—a drug which has a very decided sedative action upon spasm of all muscles connected with the respiratory apparatus. It may be given in doses of ten to thirty drops, in water flavoured with the liquid extract of liquorice and well sweetened with glycerine, every four hours. In the case of young patients grindelia is an antispasmodic of the utmost value. In asthma, in whooping-cough towards the end of the spasmodic stage, and indeed in all varieties of respiratory spasm, we may turn to grindelia with full confidence in its beneficial effect.

Another instance of reflex spasm is seen in the rigidity of the muscles of the neck which not infrequently occurs in cases of acute otitis. The child lies in a state of seeming stupor, but is not entirely unconscious and shows no sign of pain. He holds his head stiffly and slightly retracted, and whimpers if it is moved, especially if an attempt is made to bend it forward. The stiffness ceases at once when the purulent contents of the tympanum are allowed to escape by puncture or rupture of the membrane.

The above are the most prominent and persistent varieties of reflex spasm, the only rational treatment of which consists in measures which produce a direct impression upon the source of the nervous trouble. Other minor examples of spasmodic muscular contraction of similar mechanism are met with in daily practice. The severe nocturnal cramps in the limbs which are a common subject of complaint by the habitual dyspeptic are due to acidity, and can be put a stop to by an alkaline draught taken at bedtime. Acid collections in the caecum and the small intestine near by may set up cramp in the bowel by causing spasmodic contraction of its muscular coat, and so penning up flatus and preventing it passing away. The painful over-distension which is apt to ensue can be satisfactorily relieved by the administration of the insoluble alkalis, such as the heavy carbonate of magnesia, combined with a quarter or a third of a grain of codeine given several times in the day. Codeina is a valuable sedative in all forms of abdominal pain, and has the advantage over opium of interfering little with the regular action of the bowels. Under certain conditions, however, belladonna is to be preferred. Spasmodic contraction of the muscular coat of the intestine may induce an obstinate form of constipation which sometimes amounts to impaction of the bowel, and resists the action of strong aperients given again and again. In most cases of this kind the difficulty can be overcome by repeated doses of the green extract of belladonna, giving a $\frac{1}{4}$ grain every three hours until the pupils begin to dilate. This, the first sign that the system is beginning to respond to the remedy, is followed quickly by relaxation of the intestinal spasm and copious relief.

In every case, then, of spasmodic contractions, either of voluntary or involuntary muscle, especially in such as occur in children, before resorting for their relief to nerve tonics and antispasmodics, we should search carefully for some sufficient signs of local trouble to which the nerve disturbance may be referred. In young people, especially in rickety infants and rapidly-growing boys and girls, our first thought should always be of reflex mechanism, and it is only when a fruitless examination has shown our suspicions to be unfounded that we can venture to lay this explanation of the symptoms aside. We are then obliged to fall back upon plain antispasmodic treatment. But even in this case every attempt should be made to keep the nervous system at rest by avoidance of undue excitement, attention to the regularity of the bowels, and in the matter of diet a careful moderation, not only in red meats, but in acid and acid-making articles of food.

The kind of antispasmodic treatment to be employed is of importance. In the case of true epileptic attacks, excessive dosing with large quantities of bromide is, I believe, a plan of treatment which is better avoided. By such means, no doubt, we may lessen the frequency of the seizures for a time, but sedatives alone have little power to put an end to them altogether. These remedies are best used as aids to the action of nerve tonics; as a means of quieting undue nervous excitement, while the more slowly acting drugs are given time to produce their bracing effect. Of the latter I rely chiefly upon ergotine given in conjunc-

tion with strychnine. To be effectual the dose of ergotine must be pushed, and this may be done without fear of ill consequences; indeed, little effect can be expected until a good dose is reached. Beginning with 3 gr. every four hours given in conjunction with $\frac{3}{4}$ grain of strychnine, the dose may be increased gradually to 10 grains, and every evening at bedtime a draught containing 20 grains or more of bromide of sodium may be given with infusion of senna and aromatics. In the case of children the most satisfactory results often follow this method of treatment, especially if it be combined with a free country life in a healthy situation. The seaside, or, at any rate, the neighbourhood of the sea is to be preferred. It is wise to occupy the child's mind with light employments which interest and amuse; and if he can share these with young people of his own age, who may be trusted not to irritate or excite him, it will be distinctly to his advantage.

Another example of universal spasm which has to be treated with general nervine sedatives is chorea. In the management of this complaint it would be difficult to name a sedative which has not been employed with more or less success. I have obtained very striking results in obstinate cases with ergotine prescribed in large doses in conjunction with strychnine in the same way as recommended for epilepsy. Recently I have been giving chloretone in doses of 3 to 5 grains three times a day and have been well satisfied with the results. The remedy is one which decomposes very quickly and should, therefore, never be prescribed in combination with another drug. It is best given in "cachet" or dissolved in water sweetened with glycerine. A dose of 5 grains three times a day often causes some drowsiness. If this be so, the dose should be reduced to 2 or 3 grains after the first twenty-four hours.

But cases, such as the above, in which we are forced to rely solely upon the action of pure antispasmodic and tonic remedies for the control of the morbid phenomena form—in early life at any rate—but a small proportion of the examples of muscular spasm which are being continually brought under our notice. As a rule in the young the phenomena are purely reflex, the impulse being derived from irritative action at some point of the periphery. In all such cases the employment of general sedatives must be made subservient to the more pressing needs of local medication; but these remedies are still useful as aids to the treatment to reduce the general nervous susceptibility while special local measures are being employed to put an end to the primary cause of the nervous distress.

A Clinical Lecture

ON

AMAUROTIC FAMILY IDIOCY.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET.

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AMAUROTIC family idiocy is such a rare disease that it may seem hardly suitable for a lecture such as this, but I have chosen it because I happen to have met with four cases in recent years, and have one of them to show to-day; and also another of them, minutely investigated after death by Dr. Gordon Holmes and Mr. Herbert Parsons, stands as one of the most complete cases on record, for it was studied by recent neurological methods. This disease is, too, a very remarkable one, and, though you may never meet with a case, a short time given to its study can hardly fail to direct one's thoughts to some interesting points in the study of medicine.

I think, to begin with, it is interesting to trace the gradual recognition of the disease, because this illustrates how the specialist may sometimes point out to the general physician some fact in medicine which has been previously overlooked—just as the general physician may sometimes throw an entirely new light upon a subject which at first sight would seem to be quite a special one. In 1881 Mr. Warren Tay directed attention to peculiar macular

changes in the eyes of an infant. The title of his contribution was "Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant," and an excellent picture is given of the condition in this paper, which was published in the first volume of the *Transactions of the Ophthalmological Society*.

Knapp of New York in 1885 recorded a necropsy, and in 1887 Sachs (B.) of New York, who has done so much to elucidate this disease, described the changes in the brain in a case under the title of "Arrested Cerebral Development." In his later contributions he named the disease amaurotic family idiocy, and defined the chief clinical features. A valuable paper by Mr. E. C. Kingdon and Dr. Risien Russell was published in the *Royal Medical and Chirurgical Society's Transactions for 1897*, under the title of "Infantile Cerebral Degeneration with Symmetrical Changes at the Macula," and since that time various writers have contributed to the literature and further elucidated a disease which was, as you see, first recognized by an ophthalmic surgeon through one of the most striking of its symptoms. American physicians have in particular made valuable contributions to the literature, and among the more recent of them Spiller has been prominent. I believe that almost every case recorded which is undoubtedly an example of the disease has occurred in a Jewish child, although Mohre described a case in an American. In all my four cases both father and mother were Jews and not related. It is a disease of infancy, and shows itself in the first year, usually before the sixth month and frequently about the third, and it is almost invariably progressive, and ends fatally somewhere in the second or third year of life.

If it were not for its rarity it would be a disease of the utmost importance, and for those who have charge of numerous Jewish patients a knowledge of its chief features must be of some value.

It is interesting that my four cases have been sent to the hospital as cases of rickets, because they have shown loss of power and general feebleness. Extreme rickets, we know, does occur among the Jewish poor; but I think we must remember that the mothers generally nurse their children, and unless they are hopelessly poor I hesitate to hurry into the diagnosis of rickets with breast-fed children. As you will see, there are probably no two diseases more unlike than rickets and amaurotic family idiocy when well developed; but the error arises, no doubt, because the medical man is not acquainted with the rarer disease, and, finding himself driven into the corner where we all in turn crouch, falls back, for the need of a name, on that comprehensive and elastic one of "rickets."

The usual history that is given by the mother is to this effect: The child was healthy for some three months after birth, and during that time there had been no illness. Then she noticed that instead of going forward the child began to go back and the muscles of the back and neck were becoming feeble. The real gravity of these signs may not be realized for some months, and then the suspicion grows that the child cannot see well, and that the expression of its face is dull and the mental state feeble. As the disease progresses there is complete inability to raise the head or sit up, and even to turn when lying down, though sometimes the arms are thrown over the head in a wild, purposeless fashion. The limbs are also enfeebled, although at first no visible wasting will be apparent. A very interesting symptom present in all my cases, but I believe not invariable, is the survival of an acute sense of hearing, which, coupled with the general nervous degeneration, causes the infant to start at any sudden sound. The result is striking, for when the child is lying motionless it starts at a sharp sound, almost as if an electric shock had been applied. The sense of taste is preserved. There may sometimes be curious vermicular movements of the fingers and hands and a general drowsiness. In the last stage the limbs may become spastic, the muscles are wasted, and the reflexes increased.

Although some writers have stated that the temperature remains normal, this is not invariable, for in one of my cases the temperature rose shortly before death to 103°. There were screaming and head retraction, ptosis, and finally coma. Yet the anterior fontanelle was not distended, and no sign of meningitis was present after death.

There is, then, in this disease a slowly progressive paralysis, unmarked at first by any startling symptoms, but relentless in its steady advance unless death be accelerated by some intercurrent malady.

There is another very important feature in amaurotic family idiocy—its tendency to appear in several members of a family. The unfortunate mother of this child lost her first from the same cause, and will, I fear, surely lose this one also.

The history of Kingdon and Risien Russell's cases is, however, far more striking. The first child died of this disease; the second was healthy; the third died; the fourth was healthy; the fifth, sixth, and seventh all fell victims. The two healthy ones were a girl and boy respectively. More cases, however, have been reported in females than in males.

Ocular Changes.

The ocular changes are most interesting and important, and were, as I have already mentioned, the means of the discovery of the disease.

In most cases there is a primary optic atrophy, although Tay detected some neuritis in the earlier stage. I have never seen a case yet in which the ocular sign of atrophy has not been already developed and distinctive. Coupled with this atrophy there is in the position of the macula lutea, a white, hazy appearance of the retina, in the middle of which area there is a cherry-red spot of colour, closely resembling the appearance found in embolism of the central artery of the retina. In the case here to-day these signs are exceedingly well marked, and their presence enabled me to clinch a diagnosis which was at first not quite obvious.

The explanation of the changes is given briefly under the pathology of the disease, and they remain constant until death, with trifling modifications. The pupils are equal, and when the atrophy is well developed become widely dilated and inactive.

Pathology.

The pathology of this disease is at once interesting and obscure, and it is only in comparatively few cases that a complete investigation has been made.

Macroscopic changes in the brain are seldom evident. Sachs, however, described in two of his cases a low type of development of the cerebral convolutions, and in three cases the brain seemed unduly hard. In one of my cases there was the same unnatural hardness of the cerebral tissues. In other cases no change from the normal has been observed at all.

Microscopical changes were demonstrated by Sachs in 1887 and 1892, and in 1897 Dr. Risien Russell with Mr. Kingdon gave a full account of the changes they had observed in one case, and that account holds good at the present date, though it can be supplemented by additional observations which result from the use of other methods than staining with the aniline dyes. In 1906 Dr. Gordon Holmes and Mr. Herbert Parsons made a very careful and extensive investigation into one of my cases, which was published in detail in *Brain*, 1906, p. 180. This confirmed many of the previous observations, emphasized the statement of Spiller in 1905 that the disease of the nerve cells is general, and also elaborated the nature of these changes. I must refer you to that paper for the details, which are too intricate for this lecture.

The great fact to grasp in the pathological anatomy is the remarkable general and extreme disease of the nerve cells. It would appear to be a primary cell disease, and the changes in the fibres are apparently secondary. We may even go a step further and say that this disease is primarily a disease of the interfibrillar protoplasm of the cells, the changes in the neurofibrils being secondary.

The cell destruction may be most extensive; thus, in my case not a single normal cell was found by Dr. Holmes in any part of the central nervous system he examined, and the same was true also of the dorsal ganglia and ganglion cells of the retina. Spiller has found peripheral nerve changes. The tough consistence of the brain was the result of a secondary proliferation of the neuroglia.

It is also important to recognize that the disease of the nerve cells is not a result of inflammation, for there are no vascular signs of either acute or chronic inflammation.

The changes in the nerve cells are: Swelling of the cell body with frequently gross alteration in shape; disappearance of Nissl bodies, excentric position of nuclei, vacuolization of the protoplasm.

It may well be expected that the explanation of the cause of such a disease as this is beyond us. The very fact that, so far as I am aware, it is almost invariably met with in the Jews points at once, I think, to some very obscure disorder of metabolism.

The inevitable progress of the disease does not suggest an arrest of development but some active process. A bacteriological cause is improbable, for there are no positive facts in its favour, and my case was entirely negative, and the absence of vascular changes, the age, and racial nature of the disorder make such an explanation unlikely.

It does not appear from the microscopical evidence to be a pure atrophy, and I can get no further than the statement made by Holmes, Parsons, and myself in our paper that it is due "to some inherent bio-chemical property of the protoplasm of the cells." The only *post-mortem* examination I have seen showed no obvious changes in any of the viscera or internal glands which even suggested that they were at fault, but you will readily appreciate that a decisive statement on this point would be impossible with such a scanty experience. If there is some internal secretion at fault, at present no one, I believe, has discovered the gland that is to blame.

The explanation of the remarkable fundus changes comes next. Treacher Collins, Holden, Mohre, Shumway, and Mary Buchanan, and more recently H. Parsons, have written upon this point. There is profound disease of the ganglion cells in the retina, and at the fovea the disease may become so extreme that there is atrophy of the retinal tissue; and possibly this atrophy may even result in an actual destruction of the retina at this spot, as seemed to have occurred in my case. Whether this be so or not, you can imagine to yourselves the condition that results. We have the retina spread out over the vascular pink choroid, and where it is thinned by disease it is evident that this coloured background when illuminated by the ophthalmoscope will shine through with particular brightness and colour, and by contrast with the surrounding less diseased parts this greatly damaged area will appear as a bright cherry-red spot. The contrast is rendered all the greater because it would appear that the retina around the fovea may become oedematous, or, more probably, through swelling of the diseased cells, thickened, thus cutting off the colour of the choroid and forming around the cherry-red spot a whitish zone. The optic atrophy is due to the extensive disease of nerve cells and nerve fibres.

There are some remarkable cases on record which only in some details resemble amaurotic family idiocy.

Spiller, whose name I have already mentioned, found changes in the nervous system which he believed to be identical with those of amaurotic family idiocy in two children, one of which was 9 years of age, the other 8 years, who were not suffering clinically from this disease. These two children were mentally defective, but were not blind, and they would appear to be quite exceptional cases, which we must bear in mind when we dwell on the entity of amaurotic family idiocy. This is the more needful, because Falkenheim mentions a case of this disease in which the ocular signs were not present.

I have never myself recognized any case of an aberrant type, all the four I have published were classical in their onset, symptoms, and course. There is one case (Peterson's) which had survived to the age of 5 years, but in the absence of a necropsy, one is obliged to suspend decision as to its exact nature.

In this country I am not aware of a single case published which has not occurred in a Jewish child, but I believe that this is not an invariable rule, although there can be no doubt that it is to a great extent a racial disease, and far more so than the condition diabetes, which at once occurs to one as also more prevalent in the Hebrew race.

When confronted with a case of this kind, which is so mysterious in its nature and so hopeless in its prognosis, it is only natural to attempt to add any fact one can, that is of the least importance. Dr. F. Mott, from his laboratory, has done this in the case you see to-day, for he kindly had the blood and cerebro-spinal fluid examined by Dr.

Candler by Wassermann's reaction, and has informed me that they both gave a negative result. This bears out so far the clinical experience of these cases that they are not a result of inherited syphilis, and is, I believe, a new observation on the disease.

Treatment.

It is quite useless for me to speak of the treatment. Russell and Kingdon tried numerous remedies of all kinds in vain in their series. I should not even follow Hirsch's suggestion that the mother should not nurse any child she might have in the future. One feels that this is but a faint hope. Further, should the child not show any sign of the disease, we must remember that not every member of a family need necessarily suffer whether nursed or not. Sachs, in fact, has already pointed out that it is of no avail.

I have tried one of the organic phosphorus compounds in this case, but I see no good result at all in consequence, and I think we need some much more subtle remedy than this.

Diagnoses.

You will readily understand that the great point in the diagnosis of these cases is really a knowledge of the disease. It is so rare that we never think of it, and perhaps have never heard of it, and then, of course, we must lose our bearings.

It has no resemblance at all to rickets, except in so far that the child is muscularly feeble, and I need not dwell on the extreme dissimilarity of the two diseases.

Cerebral diplegia is more likely to cause confusion, but the distinction is usually an easy one. For example, take such a case as this which I saw recently. A child of 7 months was brought for paralysis, which had been noticed by the parents soon after birth. The boy's head fell back from weakness of the cervical muscles, he snorted when asleep from weakness of the palate, and all four limbs were spastic, but there were no ocular changes. You will see that the spasticity was an original symptom, but it is not an early symptom in amaurotic family idiocy. Then, again, he was no worse than he was at 2 months of age; in fact, he was a little better. There was no blindness and no curious starting at a sudden noise. The reflexes were greatly exaggerated.

In another case there was double optic atrophy and consequent blindness, but the disease was not advancing; there was the same spasticity, and an entire absence of the macular changes of amaurotic family idiocy.

In some congenital syphilitic lesions of the brain there may be a progressive downhill course, or there may be a downhill course in which the steps are marked by sudden exacerbations of symptoms. In such cases epileptic attacks are common, and there may be other stigmata of congenital syphilis including choroidal changes, but not the characteristic ocular changes of amaurotic family idiocy. The history of stillbirths, miscarriages, or syphilis in the parents is often met with in such cases, but this is not the experience in the disease we are now considering.

Whether or not there are cases of progressive idiocy met with in asylums for children, which resemble this disease closely, I am not competent to state, but if they exist they must be very exceptional, I think, for even Spiller's cases did not show a clinical resemblance. Hydrocephalus can be at once excluded because that condition is not present in amaurotic family idiocy.

Post-basic meningitis, although it may give rise to blindness and paralysis, is acute in its onset and associated almost invariably from the first with such symptoms as fever, retraction of the head, convulsions, and vomiting. There are no macular changes and the cerebro-spinal fluid obtained by lumbar puncture will at once differentiate the disease.

Possibly a cerebral tumour in an infant might show some resemblance, but I have never myself met with any case in the least resembling one of these.

Cretinism could not possibly be a cause of difficulty.

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AN EPIDEMIC OF TINEA CRURIS.

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RINGWORM of the scalp and body occurring in epidemic form in schools is familiar, but ringworm of the groin, fork, and axilla is not of such common occurrence except in tropical climates; its appearance in epidemic form during the last three years at the universities and in our public schools is my reason for publishing the following notes.

Tinea cruris has also been called tinea marginata or eczema marginatum. In the majority of cases I have seen the lesion starts in the fork or inner surface of the thigh, where it is touched by the scrotum. A red patch, looking like an area of chafing, appears, and rapidly spreads down the inner side of the thigh and backwards towards or behind the anus. The spreading edge is raised, dry, scaly, or papular, whilst the portion within the ring is moist and eczematous. Gradually extending from the margin, the lesion in the lower extremity may almost reach the knee, and in the axilla may reach the level of the nipple. There does not appear to be any tendency to a spontaneous return to the normal on the part of the skin within the growing margin. The hairs are not affected and do not fall out. Beyond slight itching, there are no subjective symptoms. On the trunk and limbs of infected persons there may often be found patches which can in no way be distinguished from ordinary ringworm of the body (tinea circinata).

On March 8th, 1907, a boy presented himself, suffering from a condition I diagnosed as eczema intertrigo affecting the scrotum and inner side of the right thigh. At the end of the term, about the first week in April, the condition showed no improvement, and he went under the care of his doctor at home, Dr. McLannahan, of Stonehouse, who very kindly wrote to me, saying that he regarded the case as one of tinea marginata, a condition he had met with amongst soldiers and amongst stokers in the P. and O. service. This boy was apparently well by April 21st, and he returned to school a few days later, and had no recurrence. On May 31st, 1907, a second boy from the same house was seen with tinea marginata of the axilla. An undress parade of the boys in the house led to the discovery of 9 other cases, 2 having the disease in the axilla, and the remaining 7 in the groin or fork. A second inspection of the house a month later led to the detection of 2 more cases; the fourteenth case appeared twenty-four days later, and the 2 last cases were found thirteen and eighteen days after this. In all, there were 16 cases, all in one house of about forty-five boys.

Clinically, there was little to note about the cases. In a few, which were of old standing, the rash extended as low as the lower third of the thigh and backward behind the anus. Not uncommonly a few spots, the size of a three-penny-bit, were found on the thighs, calves, trunk, and more rarely on the arms. These isolated spots were drier and more scaly than the patches in the groin or axilla, and readily yielded to treatment. The rings gave rise to no symptoms beyond slight irritation. The average length of time under treatment was five weeks. Three cases recurred, two after an interval of six weeks, and one after the lapse of four months.

Scrapings from the growing edge of some of these cases were examined microscopically, both by myself and by a

local bacteriologist. In all, a large-spored ringworm fungus was found.

Treatment was alike in all cases. The superficial epidermis was removed first, and then an antiseptic ointment was applied. To remove the epidermis I employed a solution of 40 grains of iodine with 20 grains of potassium iodide in an ounce of methylated spirit. Two applications were generally sufficient; then the parts were treated morning and evening with an ointment containing half a drachm of precipitated sulphur and half a drachm of ammoniated mercury in an ounce of lanolin. A few boys were treated according to a suggestion of Dr. McLannahan: the affected parts were painted every other day with a camel-hair brush soaked in ol. terebinth. rect., and the following ointment rubbed in every night: Sulph. precip. 3ss, hydrarg. amm. 3ss, acid. salicyl. gr. x, lanolin and vaseline aa 3iv. In both cases the applications sometimes gave rise to severe smarting, but this was of short duration, and relieved by smearing with vaseline. Cases seen early and treated by this method were well in three weeks; old-standing cases took, of course, longer.

The factors common to all the boys who suffered in the epidemic were as follows: With one exception, they were all senior boys, members of the First Fifteen, using the same lavatory and bathroom. They did not all belong to the same dormitory, and there was no suspicion that they wore each other's football or running togs; indeed, one boy who suffered was not allowed either to run or play football on account of a chronic cardiac lesion.

The precautions taken to prevent the spread of the infection were as follows: All the boys in the infected house were stripped and inspected about every three weeks. All who were detected with the rash were made to sleep in the same dormitory, which was reserved for this purpose; they also had the exclusive use of a separate changing and bath room and water-closet. For baths and ordinary washing purposes Parke Davis's germicidal soap was used. All towels from the infected changing room were disinfected, and also the underclothing and running kit. The walls of the changing room and lavatory were washed down with disinfectant at the termination of the outbreak. The infected boys were not withdrawn from school work nor from the common dining-room. When the rash in the fork was profuse they were not permitted to join in the school runs.

These precautions were entirely successful; no other house was infected, and no cases have appeared during the subsequent fourteen months. During the period covered by this epidemic I saw in other schools several cases of body ringworm (tinea circinata); 2 isolated cases occurred in a boys' school, 1 in a girls' school, and 7 in a school for junior boys. None of these cases assumed the type of tinea cruris.

In my own mind there is no doubt that the outbreak was caused by the common use of the hot bath, or by the boys using each other's towels. Probably the growth of the parasite was encouraged by the moist, hot atmosphere of the bathroom, while the vigorous rubbing which boys give to the forks, groins, and axillae decided the points of inoculation. It has sometimes been thought that infection is carried in clothing, and that chairs and water-closet seats may harbour the parasite, but in the outbreak under consideration these factors could be eliminated.

Tinea cruris resembles tinea circinata in that it attacks patients after the period of puberty; but it is probable that there are two distinct parasites causing these conditions, for in tinea cruris the hairs are never attacked. The parasite of tinea cruris shows a larger spore and a freer growth of mycelium than tinea megalosporon; it is also very much easier to find in scrapings taken from the growing edge of a ring. These differences may be due only to the site in which the fungus has grown. In order to settle this point it is desirable that scrapings should be taken and growth obtained on Sabouraud's medium.

THE annual general meeting of the Society for Training Teachers of the Deaf and for the diffusion of the German (pure oral) system will be held at 33, Cavendish Square, W., on Wednesday, May 26th. Sir Melville Beachcroft, Chairman of the London County Council, will take the chair at 3 p.m.