In cases of urinary obstruction in which operative procedure was contemplated, an estimation of total function was made by means of blood cryoscopy and by the determination of the amount of phenolsulphonephthalein contained in the bladder-urine during a period of two hours and ten minutes after the injection of the dye.

Following these tests a note was made of the microscopic findings, particular stress being laid on the presence of leukocytes and degenerated small round epithelial cells. The amount of urea was determined by two Doremus ureometers, and the quantity of sugar by means of two Lohnstein saccharimeters. The quantitative phenolsulphonephthalein estimations were made by means of colorimetrically graded test-tubes as described by Hugh Cabot. These were found sufficiently accurate for ordinary practical purposes.

RESULTS

In normal kidneys, after the injection of phlorizin, sugar made its appearance in the urine in from nine and a half to thirty-one minutes. The average interval was $18\frac{1}{10}$ minutes on the right and $17\frac{8}{10}$ minutes on the left side. The quantity of sugar varied from 0.1 to 3.2 per cent., and averaged 1.1 on the right and 1.08 on the left side. The amount of urea averaged 0.14 per cent. on the right and 0.16 on the left side. The time of appearance of phenolsulphonephthalein varied from 3 to $9\frac{1}{2}$ minutes. The average interval was $4\frac{17}{18}$ minutes on the right and $5\frac{1}{18}$ minutes on the left. The amount of phenolsulphonephthalein varied from 2 to 30 per cent., and averaged 15.6 per cent. on the right and 16 per cent. on the left.

In cases of unilateral pathologic kidneys, sugar appeared after phlorizin injection in from 18 to 65 minutes, averaging 35 minutes, while it was excreted from the normal kidney in from 10 to 25 minutes, averaging $15\frac{7}{10}$ minutes. The amount of sugar in the same pathologic cases varied from 0.3 to 0.78 per cent., averaging 0.29 on the functionally insufficient side, while on the normal side the amount varied from 0.44 to 4 per cent., and averaged 1.91. The amount of urea varied from 0.002 to 0.011 per cent., averaging 0.007 from the functionally insufficient kidney and from 0.013 to 0.15 per cent., averaging 0.04 from the normal kidney. In unilateral as well as in bilateral pyelitis the time of appearance of phlorizin and phenolsulphonephthalein as well as the amount of phlorizin, phenolsulphonephthalein and urea was the same on both sides.

CONCLUSIONS

1. In normal cases the time of appearance of sugar and phenolsulphonephthalein is comparatively the same on both sides.

2. In normal cases the phlorizin, phenolsulphonephthalein and urea tests show almost identical values for both kidneys, their efficiency as comparative tests thus appearing to be about equal.

3. In pathologic cases all three tests show almost equally low values on the diseased as compared with the healthy side, thus demonstrating their almost equal practical value.

4. The simultaneous application of the preceding tests in the manner described tends toward greater accuracy of results, is not especially time-consuming, is not complicated and does not require more than the ordinary skill, which can be attained by an intelligent nurse. Moreover, it gives positive assurance as to which kidney is performing the greater amount of work, thus rendering unnecessary a repetition of ureteral catheterization for the purpose of gathering additional evidence regarding renal functions.

5. In pathologic cases a coincident diminished functional value on one side in all three tests points unmistakably to a marked defect in the corresponding kidney. This in the presence of a normal functional value on the opposite side, and satisfactory total functional values as ascertained by blood cryoscopy, the bladder phenolsulphonephthalein test, etc., would permit of the removal of the diseased organ.

6. A single renal functional test may lead to erroneous deductions and does not give sufficient assurance to justify a radical operation such as removal of the kidney.

7. Prior to operation, comparative functional tests should be strengthened by tests of total renal function.

8. The urea test consumes the least time and is accompanied by a minimum of discomfort to the patient. Moreover, it is not based on the elimination of a foreign substance.

9. As compared with the phlorizin test, the phenolsulphonephthalein test is subject to fewer technical errors and is less time-consuming, a factor of no little importance to the patient as well as to the physician. On the other hand, the quantitative estimation of the excreted dye even with the Duboscq colorimeter is subject to a not negligible amount of error, while following phlorizin injection the estimation of sugar by means of the Lohnstein saccharimeter is mathematically correct.

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A CASE OF TAY-SACHS AMAUROTIC IDIOCY WITH A POSITIVE WASSERMANN REACTION *

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The following case is reported not because it is a typical example of Tay-Sachs amaurotic idiocy, but on account of a positive Wassermann reaction having been obtained from the patient's blood-serum, and the possible bearing this may have on the etiology of the affection.

B. L., boy, aged 16 months, Hebrew, of Russian parentage, examined March 3, 1914, was said by the parents to have seemed normal until about 2 months old. He did not take any notice or sit up at the usual time, had never talked and had never attempted to walk. His first tooth was cut when he was seven months old. The child seemed backward in every way and had one convulsion two months prior to the examination in which he lost consciousness, became rigid, the eyes being open and staring. After the attack, he started to cry, then fell asleep. Since this convulsion, the baby had seemed dull and had not helped himself as much as formerly. He drops objects placed in his hand. Other attacks are described as follows: "He will become nervous, red in the face, hold his breath for a second or two, his arms and legs will stiffen, he will then cry."

The child was born at full term, the labor being normal. He is said to have weighed 7 pounds at birth and has contracted none of the infectious diseases of infancy. He is the only living child, the parents having had one other child which died shortly after birth from a "hemorrhage of the navel."

The father and mother are both living and in average health. A paternal aunt, one of twelve children, had convul-

* Reported at the meeting of the Philadelphia Neurological Society, March 27, 1914.

sions. A maternal uncle had spasms until 9 years of age, when they left him. There is no case of blindness in the family, nor any history of nervous or mental disease except as mentioned above.

On examination the child was found to be well-formed and well-nourished, but with general muscular weakness. The reflexes were preserved and Babinski's sign was present.

Impairment of vision was evident and it was learned that the child had been under the care of Dr. L. Webster Fox. On communication, Dr. Fox gave me a report of the ophthalmoscopic findings, which were as follows: "Double optic atrophy with a red spot in the region of each fovea centralis, surrounded by a whitish or grayish areola." Dr. Fox had seen cases of amaurotic idiocy at Tay's clinic in London and stated that the fundus changes in this case were identical.

Dr. Thomas B. Earley made a Wassermann test of the blood and reported it positive ($\frac{1}{2}$ unit). An examination of the cerebrospinal fluid of the child and of the blood of the mother was refused.

In 1881, Mr. Warren Tay¹ of London presented a case in which the baby, then 1 year old, had been noticed at the age of 2 to 3 weeks to have very little power in holding up its head or of moving the extremities. This was a weakness, not an absolute paralysis. The child seemed cerebrally deficient. With the ophthalmoscope, Tay found the optic disks apparently healthy, but in the region of the yellow spot in each eye "there was a conspicuous, tolerably defined, large white patch, more or less circular in outline, and showing at its center a brownish-red, fairly circular spot, contrasting strongly with the whitish patch surrounding it." Some months later, Tay found no change in the child's condition except that the disks were becoming atrophic.

Sachs² of New York, delivering the presidential address before the New York Neurological Society in 1896, described a family form of idiocy, associated with blindness, which he designated as amaurotic family idiocy. In this communication, he referred to an earlier paper, read in 1887 before the American Neurological Association, in which he described a similar case under the title "Arrested Cerebral Development with Special Reference to its Cortical Pathology." He concludes that Tay's case and his own are similar in type and collects altogether nineteen cases presenting the same symptom-group.

The chief symptoms as originally described by Sachs are mental impairment, a more or less general paresis or paralysis which may be either spastic or flaccid, with corresponding changes in the reflexes; a diminution or loss of vision with the characteristic eye-ground changes, marasmus; a fatal termination, usually by the end of the second year, and the occurrence of the affection in several members of the same family. Occasional symptoms were nystagmus, strabismus and hyperacuity of hearing. In a later paper, Sachs³ accepts two additional symptoms mentioned by Falkenheim: explosive laughter and disturbance of deglutition.

Since these earlier papers, cases have been accumulating fairly rapidly in the literature. As far back as 1908, Apert⁴ collected eighty-two indisputable cases with twenty-seven additional cases occurring in brothers, sisters or relatives of the eighty-two, but not actually seen by the authors recording them.

Then, as might be expected, came reports of cases more or less closely resembling the original Tay-Sachs type, yet presenting certain distinct differences. Thus Vogt in 1905 described a form of family amaurotic idiocy, beginning in early youth, with gradually oncoming blindness usually associated with paralysis. In this form the characteristic macular changes are not found. Death occurs after several years and the disease shows no predilection for the Hebrew race. Vogt contended that the juvenile form he described and the Tay-Sachs disease represented different degrees of the same process. Gifford⁵ quotes Vogt and reports a case of the same type.

Dercum⁶ reported three brothers, aged 11, 6 and 2 years, who were normal until the ages of 16 months, 6 years and 2 years, respectively; then developed paresis of the extremities, spastic in type and associated with imbecility. In these cases, although vision appeared defective, an examination of the eye-grounds could not be obtained.

Other cases, more or less resembling the Tay-Sachs syndrome, have been reported by Hirschberg, Gordon, Nettleship, Behr and others.

The pathologic findings are chiefly microscopic and consist primarily in lesions of the gray matter throughout the entire central nervous system, with secondary changes in the tracts of the white matter. The cells are swollen, the nuclei displaced, the Nissl bodies lose their regular arrangement and the neurofibrils are destroyed. According to Coriat, chromatolysis is more marked than neurofibrillar destruction. The optic nerves are atrophied and there is marked involvement of the retinal cells, especially in the region of the fovea centralis. Thickened skull, thickened and adherent dura, pallor and edema of the convolutions have been found. Changes in the blood-vessels have not been reported. I think that none of these findings would exclude the possibility of hereditary syphilis as an etiologic factor.

The chief interest in amaurotic idiocy at present centers about this question of the etiology. Sachs believed it to be a type of agenesis and accepted Gower's term of "abiogenesis" as most fitting. In Sachs' article³ of 1903 he defends his agenetic theory, but says that "the toxic nature of the degenerative process has been argued from the resemblance of the cell pictures to those found in cases of poisoning of various kinds." Turner⁷ reports two cases of amaurotic family idiocy, one occurring in a child not of Jewish extraction, and after reviewing the pathologic findings in his own and previously reported cases, says, "Can syphilis be so certainly excluded from the causation as Carlyll would have us believe?" Turner believed that in both his cases there were points suggestive of syphilis and further quoted Gordon Holmes who had recorded a case of amaurotic idiocy in which the periadventitial cellular infiltration was, in Holmes' opinion, suggestive of congenital syphilis. Turner, however, seemed to consider as equally tenable, the theory of glandular deficiency or anomaly.

Frey does not agree with Sachs that the condition is merely agenetic, but considers the changes found to be post partum and analogous to the state known as amyotrophic lateral sclerosis in adults. This view of Frey's is of especial interest because of the finding of

1. Tay, Warren: Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant, *Tr. Ophth. Soc. U. Kingdom*, i, 56.

2. Sachs: *New York Med. Jour.*, May 30, 1896.

3. Sachs: *Jour. Nerv. and Ment. Dis.*, 1903, xxx, No. 1.

4. Apert: *Semaine méd.*, Jan. 15, 1908.

5. Gifford: *Ophth. Rec.*, 1912, xxi, No. 11.

6. Dercum: *Jour. Nerv. and Ment. Dis.*, 1897, p. 396.

7. Turner: *Brit. Jour. Child. Dis.*, 1912, ix, 193.

positive Wassermann reactions in a considerable percentage of cases of amyotrophic lateral sclerosis.

Coriat⁸ holds that Tay-Sachs disease is not due to any arrest of development because of the normal myelinization, and asserts that the macroscopic and microscopic changes are not in any sense those of idiocy. Furthermore, he contends that his cases showed normal mental characteristics up to the ages of 4 and 10 months, and says, "The disease, therefore, is not really an idiocy, but a form of dementia." Coriat suggests the name of "amaurotic family dementia" as being more appropriate than "amaurotic family idiocy."

Studies in the metabolism of amaurotic idiocy by Heiman, Bookman and Crohn⁹ in 1912 revealed nothing abnormal.

Views as to the importance of hereditary syphilis as a cause of idiocy have undergone considerable change since the introduction of the Wassermann test. Dean,¹⁰ in a most instructive article, after citing the findings of numerous observers who had examined the blood-serum of idiots by the Wassermann test, gave the results of his own work in the examination of 330 inmates of an asylum for idiots at Potsdam, in which he found the reaction positive in fifty-one cases, or 15.4 per cent. He also found that the younger the patient, the higher the percentage of positive Wassermanns obtained. Thus, at 10 years or younger, 21.27 per cent. were positive; from 16 to 20 years, 6.06 per cent.; from 21 to 30 years, twenty-four idiots gave three positive reactions, and eight idiots, ranging in age from 31 to 44 years, all gave negative reactions. A striking point in Dean's paper was the fact that of the fifty-one cases giving positive reactions, only seven showed conclusive evidence of congenital syphilis from a clinical point of view.

The lack of certain knowledge regarding the etiology of Tay-Sachs disease, the varied opinions expressed, the character of the pathologic findings and the positive Wassermann reaction in the case which I report would seem to be sufficient ground to reopen the argument that hereditary syphilis may be the cause of amaurotic idiocy. I fully realize that "one swallow does not make a summer," and my chief object in reporting the case is to stimulate interest in the subject with the hope that, as opportunity presents, careful laboratory investigation will throw more light on the point in question.

I wish to express my indebtedness to Dr. Fox for his courtesy in giving me the ophthalmologic report and for references, and to Dr. Earley, who went to considerable trouble and expenditure of time in the fruitless effort to obtain a spinal puncture in the case of the child and an examination of the blood of the patient's mother.

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8. Coriat: Arch. Pediat., 1913, xxx, 404.

9. Heiman, Henry; Bookman, Samuel, and Crohn, Burrill B.: Studies in Metabolism of Amaurotic Family Idiocy, Am. Jour. Dis. Child., October, 1912, p. 234.

10. Brit. Jour. Child. Dis., 1912, ix, 385.

Food Poisoning.—Food poisoning is rarely, if ever, ptomain poisoning; that is an intoxication due to a preformed poison of alkaloidal nature, derived from a breaking down of the protein in the food. The cases which have been carefully worked out in recent years have, on the contrary, proved to be due to infection with specific bacteria, or to intoxication with the toxins or synthetic products characteristic of these microbes.—C. E. A. Winslow.

HOW DEEP SHOULD THE TUBE BE INTRODUCED IN INTRA- TRACHEAL INSUFF- FLATION?*

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NEW YORK

In my first description of the procedure of intratracheal insufflation as I developed it for practical use in intrathoracic surgery and for anesthesia, I stated that "a long catheter or stomach-tube is introduced through the larynx and pushed down the trachea until it meets an obstacle. The lower end is usually deep in the right bronchus. It is then withdrawn about an inch and a half, which brings the lower end of the tube to about the level of the bifurcation."¹

The same statement I repeated a year ago in describing insufflation.² "After the tube enters the trachea it should be pushed gently forward until it meets with a resistance; the end of the tube is then usually in a deep place in the right bronchus. The tube should then be withdrawn about 5 or 6 cm."

It ought to be clear to any intelligent reader that the resistance which the tube meets consists of the smaller bronchi, the lumen of which is larger than the diameter of the tube. In the literature on the subject, however, a misunderstanding crept in. There are writers who apparently believe that by the resistance I meant the bronchial partition at the bifurcation, and some³ are even criticizing my supposed statement, because, as they say, they did not find that the tube touches the partition wall. This misunderstanding, however, is of little significance and would not have caused me to write a special article on that subject. A fact of greater importance causes me to write the present article, namely, that practically all surgeons and anesthesiologists who employ intratracheal insufflation—and the number is now very large—do not follow my repeated advice. It has seemed to them that instead of pushing the catheter first deep into the bronchus and then withdrawing it to the bifurcation, it is a more simple procedure to introduce it from the start only as deep as the bifurcation. After finding that the average distance from the bifurcation of the trachea to the teeth is in an adult about 27 cm., a catheter is now used which has a mark about 27 cm. away from its lower end, and it is simply pushed into the trachea until the mark is at a level with the teeth. This practice, however, as I found out, deprives the surgeons of an important diagnostic point without which they are liable to be led into a serious error. I have observed this result in two cases and I have reason for my misgivings, that it occurs more often than it is suspected.

The diagnostic point in question is to make sure that the tube is indeed in the trachea and not in the esophagus. As a rule, at the time of the introduction, the patient has to be well anesthetized and does not respond with a characteristic reflex cough when the tube passes the larynx or touches the bifurcation; besides, some patients respond with a cough when merely the posterior wall of the pharynx or the entrance of the larynx is touched. The presence or

* From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.

1. Meltzer, S. J.: Med. Rec., New York, March 19, 1910, p. 481.

2. Meltzer, S. J.: Keen's Surgery, 1913, vi, 971.

3. Lautenschläger: Berl. klin. Wchnschr., 1913, p. 2093.