Medical Progress.

REPORT ON PEDIATRICS.

PHYSIOLOGY AND PATHOLOGY OF THE DIGESTION OF THE CARBOHYDRATES IN INFANCY.

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AND
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PHYSIOLOGY. FERMENTS.

Zwiefel, 1874,¹ and Korowin, 1875,² were unable to find a diastatic ferment in the pancreas of newly born babies, and to this fact is due most of the misconceptions concerning the power of digesting starch in infancy. The work which corrected this impression will be quoted later.

Saliva. — Zwiefel¹ found diastase in the parotid gland of the newly born but was unable to find it in the submaxillary. Ibrahim,³ after a prolonged piece of work, found it in both the parotid and submaxillary glands, its action being stronger in the former than in the latter. Diastase was found much earlier in fetal life in the parotid than in the submaxillary, traces being found in the former at the fourth and in the latter at the sixth month of fetal life. The diastase of the parotid is the earliest digestive ferment found in the embryo.

A diastatic ferment can always be found in the saliva of healthy infants.⁴ The diastatic action of saliva may continue in the stomach as long as two hours after feeding.⁵

Stomach. — Ibrahim³ is the only worker who has examined the gastric mucous membrane of the newly born for the carbohydrate splitting ferments, and he has been unable to find either lactase, maltase or invertin.

Pancreas. — Moro⁶ was able to demonstrate the presence of an amylolytic ferment in the pancreas of newly born babies when the pancreas was thoroughly extracted, and thus disproved the earlier work of Zwiefel and Korowin. Ibrahim³ never failed to get the ferment in a six months' fetus when he tested the action of the ferment on starch meal. He was, however, unable to find it when he tested soluble (i. e., cooked) starch.

Ibrahim was unable to demonstrate invertin and lactase in the pancreas of newly born or older babies, but he was usually able to demonstrate maltase in the newly born and always in older children. Maltase may also be found in the blood.

Small intestine. — The mucous membrane of the small intestine contains amylolytic ferments.

Lactase, the ferment which splits milk sugar, has been repeatedly found in the mucous membrane of the small intestine.⁷ Ibrahim always found it in the small intestine and meconium of newly born babies, but was unable to find it in premature infants. He says, however, that his method of determining lactase is not capable of demonstrating small amounts. Lactase is more abundant in the young animal than in the adult.

Pautz and Vogel found maltase, the ferment which splits malt sugar, in the small intestine of infants.

Invertin, the ferment which splits cane sugar,

was found in the secretions of the small intestine of the newly born by Miura,⁸ and Ibrahim was always able to demonstrate its presence both in the intestinal mucous membrane and in the intestinal contents of all fetuses.

Large intestine. — It is difficult to wash the large intestine free from meconium, and the results of the examinations of its mucous membrane are variable, as the tables of Miura, Pautz and Vogel show. It is, therefore, impossible to say whether it contains ferments or not.

Stools. — Pottevin⁹ found an amylolytic ferment in the meconium. Kerley, Mason and Craig¹⁰ were able to demonstrate the presence of a strong amylolytic ferment in the stools of very young babies, the possibility of the bacterial fermentation of starch being excluded. There is a larger amount of diastase in the stools of breast-fed babies than in those of the bottle-fed, which Hecht¹¹ believes to be due to the fact that the intestinal contents of the breast-fed baby pass more quickly through the intestinal canal than do those of the bottle-fed baby.

The power of digesting starch, while occasionally absent, is, therefore, almost always present both in the fetus and in the newly born.^{3,12} Hedenius' experiments show that it is less powerful in young babies than in later life. Young babies are, nevertheless, able to adapt themselves to a food rich in carbohydrates. There is, according to Moro,¹⁴ a rapid increase in the power of digesting starch during the first week of life. The baby, therefore, has a power of digesting starch at birth which gradually increases in strength as the baby grows older. According to Finizio,¹⁵ it is twice as strong at eight months as it is at birth, while at twelve months it is almost as strong as at three years.

The question whether the carbohydrate-splitting ferments are affected by disease has been answered only in part. Orban¹⁶ found by animal experimentation that an injured intestinal mucous membrane contained no lactase, and that the stools of babies ill with enteritis contained no lactase. Langstein and Steinitz,¹⁷ on the other hand, always found lactase in the stools of babies ill with enteritis, whether mild or severe, acute or chronic.

FORMS OF CARBOHYDRATES.

The carbohydrates used in infant feeding may be divided into the groups¹⁸ as shown on the following page.

There are various preparations of the starches, such as Mellin's Food, Malted Milk and Liebig's soup, which are more or less dextrinized.

Lactose, or milk sugar, finds its origin only in animals. The researches of Deniges¹⁹ and Bonamartini²⁰ established the fact that the lactose in the milk of the woman, cow, ass, rabbit, dog and horse is essentially the same. Its quantity varies the least of all the elements of human milk.

DIGESTION OF CARBOHYDRATES.

The carbohydrates are broken down during digestion into the simplest forms of sugar, the

Milk sugar group.	Cane sugar group.	Malt sugar group.	
		Starch (Amylum) ↓ Dextrin (Amylo-dextrin) ↓ Erythro- & Achro-dextrin ↓ Maltose (Malt sugar) ↓ Dextrose + Dextrose	Poly-saccharide Di-saccharide Mono-saccharide
Lactose (Milk sugar) ↓ Dextrose + Galactose	Saccharose (Cane sugar) ↓ Dextrose + levulose		

mono-saccharides, by the various ferments described above. According to Rohrmann²² a considerable amount of the disaccharides may pass into the intestinal mucous membrane and there be split into mono-saccharides. The mono-saccharides are carried by the portal vein to the liver, where they are transformed into glycogen, the only difference being that dextrose is more easily converted than levulose or galactose.* The pancreas has some influence on this process, because extirpation of the pancreas in dogs results in sugar in the urine and interferes with the formation of glycogen in the liver. The liver actually has the property of forming glycogen from sugar.†

The purpose of the splitting of the poly- and di-saccharides into mono-saccharides is to prepare them for use inside the body, because the unsplit carbohydrates are not burned up in the body, but are excreted in the urine. The transformation of sugar into glycogen, which is deposited in the liver and muscles, is of great importance, because this glycogen can be broken down again into sugar according to the needs of the body. The mono-saccharides are absorbed more quickly than the disaccharides.²³

A large part of the digestion and absorption of the carbohydrates takes place in the upper part of the small intestine,²⁴ but splitting and absorption may also take place in the large intestine.²⁵

There is normally about 1-10% of dextrose in the blood. The slightest disturbance of the regulating apparatus will cause a hyperglycemia which results in glycosuria. A deficit of sugar in the blood is made up from the glycogen deposits.²¹

Albertoni²⁶ and Hédon²⁷ found that sugars have a purgative action when they are given in large enough amounts. This action is more marked when they are taken in concentrated solution. All sugars have this action, the difference between them being only in degree. They found that glucose and cane sugar are much more quickly absorbed than lactose, and that the former has less of a purgative action than the latter.

Little or no sugar can be found in the stools under normal conditions, but when the food passes rapidly through the intestinal canal, as it does when peristalsis is rapid as the result of disease

or indigestion, sugar can be found in the stool (Hecht). Usually only the products of the decomposition of sugar can be isolated.

Hedenius²⁸ fed babies with milk mixed with wheat flour, oat gruel or Keller's malt extract and measured the amount of carbohydrate ingested, the amount in the stools, and their acidity. He found that when simple cereals were used, less carbohydrate was found in the stools than with complicated mixtures and that the more carbohydrate in the stool, the greater its acidity. He never found more than 3% of the ingested carbohydrate in the stool in any instance. Keller⁴ has shown that carbohydrates make the digestion of protein more complete.

METABOLISM OF CARBOHYDRATES.

Numerous observations²⁹ have shown that when milk sugar is injected directly into the circulation it may be completely recovered in the urine. Grosz³⁰ was never able to detect milk sugar in the urine of healthy babies, but found it in the urine of those suffering with gastro-intestinal disease, in which there was presumably an absence of lactase in the intestine. Langstein and Steinitz³¹ repeated Grosz's experiments and in certain instances found lactase in the stools at the same time that sugar was being excreted in the urine. This sugar was, moreover, not always lactose, but sometimes galactose, one of the products of the splitting of lactose. They tried to explain this as follows: That some of the sugar passes through functional or anatomical lesions of the intestinal wall before it is completely broken up and is excreted in the urine as an intermediary product of metabolism.

Mendel and Kelimer³² have shown that when cane sugar is introduced subcutaneously into dogs or cats in doses of one to two grams per kilogram of body weight it is not completely recovered in the urine. The quantity excreted amounts as a rule to more than 65% of that introduced. The excretion begins within a few minutes and is usually completed within thirty-six hours. Fisher and Moore³³ draw attention to the possibility that the sugar thus introduced may be excreted through the walls of the alimentary tract and there be digested. These views are supported by Japelli and D'Errico,³⁴ who conclude from their experiments on dogs that when cane sugar is introduced directly into the

* Abderhalden: Textbook of Physiological Chemistry, London, 1908.

† Grube: Pflüger's Arch. cvii, 1906, p. 490.

circulation the quantity eliminated in the urine is never equivalent to the amount injected. This causes both glycosuria and saccharosuria, the former disappearing first. The blood has no power of converting cane sugar. According to these writers, cane sugar introduced intravenously is eliminated into the alimentary tract through the gastric mucosa, the salivary glands and, to an insignificant degree, through the bile. The subsequent fate of this component is obvious.

According to Finkelstein³⁵ sugars may cause fever. This so-called sugar fever has been studied especially by his pupils. Leopold³⁶ found that 43% of the babies tested with lactose, 47% of those with glucose, 42% of those with saccharose (cane sugar), and 33% of those with maltose reacted with fever. This fever was always accompanied by diarrhea, and in none of the cases tested in which the stools remained normal did the sugar cause fever.

The limits of assimilation of the different sugars varies and are as follows:

Grape sugar: In babies, about 5 grams per kilogram (Langstein and Meyer).

Grape sugar: In one-month baby, 8.6 grams per kilogram (Greenfield³⁷).

Galactose: No accurate data.

Levulose: (Lower for babies than adults.) One gram per kilogram (Keller).

Maltose: Over 7.7 grams per kilogram (Reuss).

Lactose: 3.1-3.6 grams per kilogram (Grosz).

Cane sugar: Probably about the same as lactose (Reuss).

Escherich³⁸ divides the digestive disturbances of infancy into two main types: (a) Fermentation with the formation of acid products and (b) putrefaction with the formation of alkalies. These two processes are antagonistic to one another, the basis of fermentation being carbohydrates and of putrefaction protein. An excessive preponderance of one over the other may do harm. Fermentation results in an excessive formation of acids, especially of lactic acid from lactose. This may cause a large number of stools as the result of the increased peristalsis and the flow of serous fluid or cellular exudate. Razenski³⁹ has shown that in babies sick with what he calls "dyspepsia intestinalis acida lactorum" there is an increased acidity of the intestinal contents and that the utilization of fat is diminished. Meyer and Leopold⁴⁰ intimate that a sugar indigestion may cause the appearance of casein curds.

Finkelstein and Meyer⁴¹ believe that milk sugar is the primary cause of the fermentative dyspepsias of infancy and that when there is a disturbance of the utilization of fat in these conditions it is a secondary manifestation. They claim that these dyspepsias can be relieved by the long-continued diminution of the carbohydrates in the food and quickly cured by the withdrawal of milk-sugar and the administration of large amounts of casein (*Eiweissmilch*). In general, the fermentation of the sugar depends upon the relation between the casein and the sugar in the food. They advise the administration of other easily assimilable and consequently little fer-

mentable carbohydrates, such as maltose, in place of lactose, after the disappearance of the acute symptoms. Birk⁴² and Reuss and Sperk⁴³ have confirmed their observations. Braumüller⁴⁴ calls attention, however, to the danger of the sudden addition of sugar to the diet after it has been withdrawn and large amounts of casein given, believing that under these conditions the ability to form the ferments necessary to take care of sugar is practically abolished.

Kendall⁴⁵ has shown that the colon bacillus, like the diphtheria and tetanus bacilli, causes fermentation or putrefaction according to whether it is in a carbohydrate or protein medium, that it attacks the sugar in preference to the protein of the medium and that until the carbohydrate is used up the protein is shielded from attack. The products of the fermentation of carbohydrates are acid. He also showed that the Shiga bacillus produces toxin only when the medium has an alkaline reaction. He, therefore, proposed feeding babies ill with bacillary dysentery with an easily fermentable carbohydrate in order to change the character of the bacterial activity in the alimentary canal from the proteolytic to the fermentative type. The result is to stop further formation of toxin. This was done by feeding the babies a 5% lactose solution, under the influence of which the dysentery bacillus and streptococci tend to disappear.

BIBLIOGRAPHY.

- ¹ Zwiefel: Untersuchungen über den Verdauungsapparat de Neugeborenen, Berlin, 1874.
- ² Korowin: Jahrb. für Kinderh., 1875, vol. viii, p. 411.
- ³ Ibrahim: Verhandl. d. Ges. für Kinderh., Köln, 1908, p. 21.
- ⁴ Schiffer: Berl. klin. Wochenschr., 1872, vol. ix, p. 353.
- ⁵ Korowin: Loc. cit.
- ⁶ Zwiefel: Loc. cit.
- ⁷ Schlossmann and Wolfe: Jahrb. für Kinderh., 1898, vol. xlvii, p. 116.
- ⁸ Montagne: Dissertation, Leyden, 1889 quoted in Czerny-Keller: Des Kindes Ernährung, etc.
- ⁹ Schilling: Jahrb. für Kinderh., 1903, vol. xlviii, p. 518.
- ¹⁰ Shaw: Albany Med. Ann., 1904, vol. xxv, p. 148.
- ¹¹ Moro: Jahrb. für Kinderh., 1898, vol. xlvii, p. 342.
- ¹² Pautz and Vogel: Zeitschr. für Biol., 1895, vol. xxxii, p. 304.
- ¹³ Weinland: Ibid., 1899, vol. xxxviii, p. 16.
- ¹⁴ Orbán: Prag. med. Wochenschr., 1899, vol. xxiv, p. 427.
- ¹⁵ Miura: Zeitschr. für Biol., 1895, vol. xxxii, p. 266.
- ¹⁶ Pottevin: C. R. Soc. biol., 1900, vol. lli, p. 598-591.
- ¹⁷ Kerley, Mason and Craig: Arch. Pediat., 1906, vol. xxiii, p. 489.
- ¹⁸ Hecht: Die Faeces des Säuglings und des Kindes, Berlin, 1910.
- ¹⁹ Stauber: Pflüger's Archiv., 1906, vol. cxiv, p. 619.
- ²⁰ Moro: Jahrb. für Kinderh., 1898, vol. xlvii, p. 342.
- ²¹ Finizio: Rev. d. Hyg. et Méd. Inf., 1909, vol. viii, p. 224.
- ²² Orbán: Prag. med. Wochenschr., 1899, vol. xxiv, p. 427.
- ²³ Langstein and Steinitz: Hoffmeister's Beiträge, 1909, vol. vii, p. 575.
- ²⁴ Reuss: Die Bedeutung der Kohlehydrate für die Ernährungsstörungen im Säuglingsalter. Wien. med. Wochenschr., no. 28, no. 29, no. 30, 1910, vol. ix.
- ²⁵ Deniges: Contribution à l'étude des lactoses, Paris, 1892.
- ²⁶ Bonmartini: Rev. gén. du lait, ii, no. 1, 1906.
- ²⁷ Langstein-Meyer: Säuglings Ernährung und Säuglingsstoffwechsel, Wiesbaden, 1910.
- ²⁸ Röhrmann: Pflüger's Archiv., 1903, vol. xcv, p. 533.
- ²⁹ Hédon: Compt. rend. de la Soc. de Biol., 1900, p. 29-41.
- ³⁰ Nagano: Pflüger's Archiv., 1902, vol. xx, p. 389.
- ³¹ Röhrmann: Chem. Bei., 1895, vol. xxviii, p. 2506.
- ³² London and Polowzowa: Zeitschr. für physiol. Chem., 1906, vol. xlix, p. 328.
- ³³ Reach: Arch. für exp. Path. u. Pharm., 1902, vol. xlvii, p. 230.
- ³⁴ Schönborn: Dissertation, Würzburg, 1897.
- ³⁵ Pehu and Porcher: Rev. d'Hyg. et de Méd. Inf., 1910, vol. ix, p. 1.
- ³⁶ Albertoni: Arch. ital. de Biol., xv, xviii, xxx, xxxv, xxxviii, xl.
- ³⁷ Hédon: C. R. Soc. de Biol., 1899, p. 884. Ibid., 1900, p. 28 and p. 87.
- ³⁸ Hedenius: Ueber das Schicksal der Kohlehydrate im Säuglingsdarm.
- ³⁹ Voit: Deutsch. Arch. für klin. Med., 1897, vol. lviii, p. 423.
- ⁴⁰ Grosz: Jahrb. für Kinderh., vol. xxxiv, p. 83.
- ⁴¹ Langstein and Steinitz: Hoffmeister's Beiträge, 1906, vol. vii, p. 575.
- ⁴² Mendel and Kleimer: Am. Jour. Physiol., 1910, vol. xxvi, p. 396.
- ⁴³ Fisher and Moore: Ibid., 1907, vol. xix, p. 314.
- ⁴⁴ Japelli and D'Errico: Ref. Malays Jahresbericht für Thierchemie, 1905, vol. xxv, p. 79.
- ⁴⁵ Finkelstein: Jahrb. für Kinderh., 1907, vol. lxxv, 1 and 263; 1908 vol. lxxvii, 521 and 692.
- ⁴⁶ Leopold: Zeitschr. für Kinderh., 1910, vol. i, p. 217.

- ³⁷ Greenfield: *Jahrb. für Kinderh.*, 1903, vol. lviii, p. 666.
³⁸ Escherich: *Deutsche Klinik*, 1902, vol. vii, p. 126.
³⁹ Razenski: *Wien. klin. Wochenschr.*, 1903, vol. xvi, p. 342.
⁴⁰ Meyer and Leopold: *Arch. Pediat.*, 1909, vol. xxvi, p. 773.
⁴¹ Finkelstein and Meyer: *Jahrb. für Kinderh.*, 1910, vol. lxxi, p. 655.
⁴² Birk: *Monatsh. für Kinderh.*, 1910, vol. ix, p. 140.
⁴³ Reuss and Sperrk: *Wien. klin. Wochenschr.*, 1910, vol. xxiii, p. 9.
⁴⁴ Braumüller: *München. med. Wochenschr.*, 1910, vol. lvii, p. 2571.
⁴⁵ Kendall: *Boston Med. and Surg. Jour.*, 1911, vol. clxiv, p. 288.

Reports of Societies.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY.

MEETING, MARCH 28, 1911, DR. GEO. A. LELAND in the chair.

DR. EDGAR M. HOLMES read a paper on

A CASE OF VERTIGO CURED BY TREATMENT OF THE EUSTACHIAN TUBE.¹

DISCUSSION.

DR. F. P. EMERSON: I have been very much interested in what Dr. Holmes reported and I think the direct treatment of the Eustachian tube marks a step forward in the evolution of the treatment of aural disease, and after we have stopped the infection that we all have been treating in the anterior nares, by relieving minor foci, in which there is a discharge of pus directly into the tube, producing some of the conditions which Dr. Holmes has described, the further advance in the treatment of these cases is certainly helped out very much along the line which Dr. Holmes had advocated for it.

I have been very much surprised, in following up a series of cases which had become old chronic office cases, over a long time, in which the symptoms of tinnitus has been markedly relieved by the local application; first using a weak solution of cocaine and adrenaline, and directly following out the lines which Dr. Holmes has followed in this case, and it is surprising how few treatments will relieve the feeling of fullness and dullness in the ears; and the patient will remark that the head feels better, and then the tinnitus is modified. Many cases of tinnitus, which we have thought were located higher up, in the labyrinth or in the middle ear, certainly in my experience have been very markedly relieved by the local treatment, and I think the essential feature in that treatment, in these cases, is the cotton tipped applicator.

DR. HOLMES: There is not much to say. I simply spoke of this one case. I have treated, during the past two months, over two hundred cases. We have kept careful records of them and we have been able to keep tabs on each other by having, most of the time, two and sometimes three on the service, and we have tried not to hypnotize the patients. As Dr. Emerson has said, it is very surprising the relief obtained in these cases, as he said, old cases where they have been catheterized here and there for months, or years, some of them, and they would not come in and say they were very much relieved through any mental thing, I do not believe. We can see in these cases that the inflammation in the tubes is relieved in the same way we can see that we relieve the pharynx by local applications. Of course some of these cases are not relieved; in the middle ear adhesions have taken place which we cannot relieve by any treatment. In regard to these cases, there have been over twenty acute bulging middle ears where we ordinarily would have had to perform paracentesis, and out of those cases all but two, up to date, have been relieved without performing paracentesis; but that will

come out in a paper at Atlantic City so I will not go into the details here.

DR. W. H. MERRILL read a paper entitled

A CASE OF NERVE DEAFNESS DUE TO THE TOXEMIA OF DIABETES.²

DISCUSSION.

DR. A. M. AMADON: I am very glad that Dr. Merrill has reported this case, even though he has done it so modestly. It is a case that has interested me though it has embarrassed me to have shot so wide of the mark at the time he kindly allowed me to examine it.

In looking over my notes upon the case I find that the patient said his father was deaf in his old age and that two brothers are deaf. He heard better before getting up in the morning, but the hearing decreased after he was up and about. My hearing test showed some decrease in bone conduction and the upper limits were slightly below normal, but these items were not of great enough moment to convince me that it was a case of labyrinthine deafness. There was moisture in the tube, and by digital examination I found some fibrous bands in Rosenmüller's fossæ which I broke up with my finger. Though the diagnosis was not clear upon the day when I saw the case, I was inclined to consider it a middle ear affection rather than labyrinthine.

In an examination of the literature with Dr. Merrill there was one case of diabetes reported by Dr. Hegener of which the clinical history was given very completely. In that case the amount of sugar was much less than in Dr. Merrill's case. There was a peculiar loss of hearing for sibilants. Some nausea and dizziness were present. The hearing test showed an increasing loss of hearing for the forks as the ascent into the higher tones was made, and the upper limits were reduced. The clinical picture presented by that case strongly suggests the chronic labyrinthine deafness as it is described by Drs. Wittemaak and Manasse, involving degeneration of the nerve fibers primarily and of the structures in the ductus cochlearis secondarily. It is interesting to know that this case recovered hearing with the recovery from the diabetes, but the upper limits of hearing never returned quite to their normal.

There is in Pollitzer's textbook a reference to an early pathological report of a case of diabetes by Steinbrügge in which there were the remains of a slight hemorrhage into the cochlea.

It seems very likely that the pathological condition causing the deafness in these cases is that of the progressive labyrinthine deafness. The absence of dizziness in Dr. Merrill's case would mean that only the cochlear branch of the eighth nerve was affected, while in the case of Dr. Hegener both cochlear and vestibular branches were involved.

Book Reviews.

A Textbook of Surgical Anatomy. By WILLIAM FRANCIS CAMPBELL, M.D., Brooklyn, N. Y., Professor of Anatomy, Long Island College Hospital; Attending Surgeon to the Methodist Episcopal, Swedish and Bushwick Hospitals; Consulting Surgeon to the Jamaica Hospital. Second edition, revised. With 319 original illustrations. Philadelphia and London: W. B. Saunders Company. 1911.

¹ See JOURNAL, p. 848.

² See JOURNAL, p. 849.