CHRONIC DYSPEPSIA IN CHILDREN OF SCHOOL AGE.*

BY

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THE USUAL CLINICAL FEATURES.

THE term "chronic dyspepsia" in children is used to connote a group of symptoms, varying in degree and in proportion, which together form a common clinical picture. For the most part it arouses no memory of exact morbid anatomy or of precise deviations from the normal in functional activity. The picture presented to our minds is somewhat as follows. The child is ailing, dull, and listless, lacking vitality and composure. The appetite is poor and capricious, the tongue coated, and the breath heavy and perhaps foul. The complexion is muddy, while the facies, with sunken eyes, or puffy eyelids and dark orbital rings, is itself characteristic enough to suggest the nature of the malady. There may be *abdominal pains* of varying degrees of severity, from slight discomfort to attacks of colic; or pain may be absent. The pain may occur after food, but is seldom gastric in position, being more often about the umbilicus or along the course of the large intestine. It may be related to the passage of flatus or precede defaecation. The *general nutrition* is usually impaired, the child losing weight in severer forms, failing to gain weight in milder. On the other hand, the weight may be satisfactory but there is a general tonelessness of skin and muscles. The *stools* are variable, in some cases infrequent, in others too frequent, but always irregular. They may be uneven in their consistence, particoloured, offensive, and contain mucus or even blood, and food which has not been digested. There may be slight nocturnal *fever*, perhaps with bouts of higher fever, coincidently with which the odour of the breath and the abdominal symptoms become more evident. *Sleep* is restless, often disturbed by dreams or night terrors. In some cases there is wakefulness. In the morning the child is tired and drowsy, and dislikes getting up. During the day he is easily exhausted and fatigued, liable to headaches, and lacks concentration or interest in his school work, being slow in acquisition and poor in performance. *Psychical symptoms* take an important place, but are more prominent in some cases than in others, depending in their degree, in part at least, on hereditary psychical traits. The child is apt to be morose, despondent, resistant, and irritable, solitary in habit, taking no part in games, and subject to fits of crying and of temper. Examination of the abdomen may reveal no abnormal physical signs, or there may be some persistent or intermittent abdominal distension, related chiefly to flatulent distension, but in some cases in part to faecal accumulation. There may be considerable indicanuria.

SOME COMPARISONS WITH ADULT DYSPEPSIA.

When we scrutinize this motley collection of symptoms, which together we are accustomed to call "dyspepsia," we realize that they do not differ materially from those of many adult dyspeptic patients. It is customary to contrast the symptoms in adults and in children, but the difference lies chiefly, I think, in the relative infrequency of organic lesions in children, and not in any great divergence of symptoms in what is called functional dyspepsia at the two ages. There is a similar condition of the skin, of the tongue, and of the breath, and similar varying tumescence of the abdomen. Flatulence, anorexia, irregular colicky pains, irregular undigested motions, nutritional disturbance, morbid and uncontrolled mental states, are common to both, and differ in the main from those of adults only in a manner or direction which might be expected.

There appears to be a growing tendency to regard this common form of dyspepsia in children, called intestinal dyspepsia, as a disorder apart, contrasting etiologically as well as symptomatically with dyspepsia in adults. It

may be doubted whether this attitude is justified, if functional dyspepsia be considered alone and organic lesions, such as septic or alcoholic gastritis, gastric and duodenal ulcerations, or gastric or intestinal new growths, be ruled out of the argument.

It must be granted, however, that the form of dyspepsia known as gastric, in contradistinction to intestinal dyspepsia, is less prevalent in children than in adults. Pain after food, when it occurs, is more often situated about the umbilicus than in the epigastrium, hypochondrium, or back. Return of food and eructation of flatus are less usual, the sensation of distension, eerie sensations of detachment, so-called gastric angina, and cardiac phenomena such as palpitation and tachycardia are not so often met with. There is not, moreover, the ordered relation of pain to food which often pertains to dyspepsia in adults.

Although it be granted that dyspepsia with prominent gastric symptoms is relatively rare, and dyspepsia with intestinal symptoms relatively frequent in children, we are not justified in the present stage of our knowledge in disregarding the stomach in the common dyspepsia of childhood which we call intestinal and assuming that it plays no part. Our information on functional disorders of the stomach and intestine is less precise in children than in adults, and we cannot with an equal degree of assurance correlate symptoms and signs with defects in secretory, motor, or sensory function.

ETIOLOGY OF INTESTINAL DYSPEPSIA.

Analysis of the symptoms shows that they fall into three groups—abdominal, constitutional, and psychical. The etiological relationship between these groups has provoked some discussion. Does the intestinal dyspepsia, by leading to the absorption of poisons, cause the constitutional disturbance and the functional nervous phenomena, or is there some underlying cause responsible for all three groups? The former view is that generally held, and was supported in a recent discussion by Reginald Miller. H. C. Cameron, on the other hand, is of opinion that there is an antecedent acidosis, as shown by lowered alkalinity of the blood, and that this is responsible for the nervous and also the intestinal symptoms. The question is full of difficulty and cannot be settled without fuller knowledge. The whole position of toxæmia, arising in that home of lost causes the intestinal tract, is thorny and contentious. In attempting to solve the problem we should not, I hold, forget that there are many cases of dyspepsia in adults which are essentially the same as the intestinal dyspepsia of children. Nor should we overlook the commonplace knowledge that any disorder which causes loss of restful sleep and exhaustion will lead to fretfulness, resistance, apathy, and eventually to moroseness in a child, as in an adult, but that in a child they are likely to be more in evidence, being less controlled. Neither toxæmia nor acidosis need be invoked to explain defective nutrition in a child with chronic digestive disorder. No useful purpose would be served by my pursuing the argument further, for I believe that much more work is necessary before the question can be answered with conviction.

I propose to approach the etiology of dyspepsia in children from a different point of view and consider some of the clinical antecedents which appear to me to have an important bearing upon it.

1. Dietetic Causes.

Whether we regard it as the primary or merely as a contributory cause, food plays an important role. Some cases are associated with continued overfeeding; others with meals so frequent or irregular that appetite is absent. Frequently the sequence of events is as follows: appetite is lost from overfeeding, then tit-bits are given between meals to tempt it because the usual meals are not taken, until a coated tongue, listlessness, and the other attributes of dyspepsia follow. The failure to gain weight, or its loss, is followed by insistence upon still more food, with perhaps the addition of cod-liver oil until further dyspepsia and wasting occur. A child will always take its food if there is appetite, and there is no surer way to court dyspepsia than to eat when it is absent. Yet how often do we find an anxious parent forcing food upon a

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reluctant and resistant child, forgetting Macbeth's grace, "Now good digestion wait on appetite, and health on both!" Not infrequently I have been told by mothers that the dyspeptic child will not take its food except by the encouragement of a stick. The physiological importance of appetite is, of course, one of the ascertained truths of digestion. Among other means by which we find it destroyed is the giving of cod-liver oil, or chocolate, or other fatty foods, a short time before meals.

Another common error is the giving of food at or near bedtime, which leads to restless sleep, with dreams or night-terrors, to tiredness and headache in the morning, and to an antipathy to breakfast. The child then goes empty to school or with a stomach filled by food taken in the absence of appetite. Food bolting in excitable children, to whom meals are so much loss of time from games, or in children with ravenous appetites, brings its own train of dyspeptic symptoms. The meal is begun eagerly, but after a few mouthfuls have been hurriedly swallowed appetite ceases, there is abdominal uneasiness, perhaps definite colic, and that symptom popularly known as "stitch," accompanied by flatulence. This last symptom is one which should always arouse inquiry into the rate of feeding rather than into the particular ingredients of the food.

A diet containing too much "roughage," as it has been called—one leaving too large and coarse a residue—is an important factor in many cases, and one which, I think, is especially related to those with signs of mild colitis, in which mucus and occasionally blood is passed in the stools. It is a subject for argument whether the colitis is caused by the mechanical action of the undigested particles or whether these are detrimental to a colitis produced in other ways—for example, by bacterial causes. In this regard I would suggest that the finding of a particular substance in the stools is no certain proof that it is the primary cause. All those who eat nuts or strawberries, or jams with pips or seeds, or similar substances, will pass recognizable detritus in the stools, but most people do so with impunity and without dyspepsia. Too much stress, therefore, should not be laid upon the etiological significance of any undigested particles found in the faeces. They indicate what the child has been taking, and perhaps taking in excess, but they do not necessarily indicate the actual cause of the indigestion. Whether, however, they are the initial cause of the disorder, or whether they contribute to its persistence or its severity, there can be little doubt that such mechanical irritants are of moment and should be taboo when there is evidence of disturbance of the large intestine. Among the commoner irritants of this kind are nuts, especially the hard and resistant and altogether indigestible variety found in cakes and sweets (such as nut-milk chocolate), coarse oatmeal, and certain proprietary cereal foods, unripe fruits such as apples, and the artificially matured banana, excess of which is a prevalent abuse. On numerous occasions pieces or strings of undigested banana covered with blood-stained mucus have been presented to me by the mothers of dyspeptic children, either as curious objects found in the stools or under the guise of worms. A chemical irritant producing colitis is *liquid paraffin*, often administered too plentifully and too persistently for constipation.

Relative excess of one of the elementary food factors is apt to be less considered in children of school age than in infants, but there is no doubt that faults in dieting are often committed along these lines. There is in children of this age, as in infants, much variation in ability to digest carbohydrates and fats, so that carbohydrate and fat dyspepsia are more readily provoked in some than in others. Defective powers of digestion for either is often accompanied by distaste for the food which cannot be readily digested. This is especially noticeable in the case of fat dyspepsia, and there are many Jack Spratts among children who will eat no fat voluntarily. If made to do so they suffer from dyspepsia. The stools in such cases are pale, greasy, rancid in odour in those more severe, but may be misleading in appearance, and in spite of a brown colour may contain much excess of fat. The severest cases of fat dyspepsia do not survive to school age.

The fermentation, which arises from carbohydrate excess, leads to the stools being voided forcibly with flatus. They

are acid in reaction, sometimes sufficiently so to produce soreness of the anus, are often accompanied by mucus, may be partly or wholly fluid, and if formed are ragged and pitted by the gas evolved. The habit of eating biscuits and sweets at night on going to bed is a common cause of such evacuations in the morning. Protein dyspepsia of a chronic kind is to me, at any rate, somewhat ill defined and uncertain.

Protein Sensitization.—There is, however, a group of cases, with dyspeptic symptoms, which arise from protein sensitization. This disorder should be borne in mind when nausea and vomiting repeatedly follow rapidly upon the taking of food, in the absence of acute illness, or other ready explanation. It has already been stated that these symptoms are unusual in the ordinary chronic dyspepsia of childhood. Inquiry may elicit that the vomiting is always after a particular article of food, such as fish or eggs or apples, and their omission brings the attacks of vomiting to an end. Other signs of protein sensitization, such as asthma or urticaria, may exist in the same children and may be found to occur after the vomiting attacks. In a recent case an apple always produced vomiting, to be followed by urticaria, and withdrawal of this "apple a day" sufficed to prevent any recurrence of the attacks.

It may be doubted whether vitamin deficiency takes any part in the dyspepsia at school age, but the possible relationship of atony and dilatation of the stomach and intestine to deficiency of vitamin B requires further elucidation.

Defective mastication is another cause which must be considered, and in this respect the state of the teeth and gums is of some importance. Any mechanical defect due to an imperfect bite or to tender teeth or gums is, however, relatively slight, in my view, in comparison with the infection which accompanies these conditions. The need for laborious and painstaking mastication has probably been exaggerated; Mr. Warwick James tells me that observation of school children has shown that the strong and healthy ate their meals rapidly with a minimum of mastication, while the weakly took much longer over them. This observation is open to other interpretations, for it is possible that the weakly child, although taking more time, may not actually chew more effectively, and poor appetite may provide less saliva to mix with the food.

The state of general health and vigour is of first-rate importance, for while a strong open-air country child may eat raw turnips and unripe apples voraciously without harm, one feebler and more sheltered may suffer after indiscretions more venial.

2. The Role of Infection.

If we turn from diet as a factor to infections we enter upon more uncertain ground. There are certainly some few cases of entero-colitis where are encountered specific organisms such as those of dysentery, to which the symptoms should rightly be ascribed. There are also a few cases in which lamblia is probably the pathogenic agent. In a considerable number *Streptococcus*, generally *viridans*, less often *haemolyticus*, is preponderant in the stools over *Bacillus coli communis*; sometimes haemolytic coliforms are found in large numbers; sometimes other organisms—for example, the bacillus of epidemic jaundice—are found. It is not possible, however, to correlate the symptoms and course of the digestive disorder with the infection, either in degree or kind, nor can it be claimed with certainty, except in the case of dysenteric infections, and perhaps in that of lamblia, that the departure from the usual intestinal flora is primary and provocative and not merely secondary and incidental.

A difficult question, and one worthy of discussion, is the manner of production of the toxæmia. Is it to be explained by infection by pathogenic organisms of the intestine, by excessive formation of toxins due to abnormal digestive processes and the abundance of suitable pabulum for the ordinary flora of the gut, by increased permeability of the mucosa, or by weakness in protective power? Stasis alone does not seem to me to be an adequate explanation, for it is not a prominent feature in the dyspepsia of children, and the severest degrees of toxæmia often coincide with loose, frequent, and bulky motions.

F. Power and C. Sherwin have isolated the main non-nitrogenous and nitrogenous products of the action of bacteria upon the proteins of the intestine, and found that they had to give more than 50 mg. of indole or skatole to produce symptoms in man comparable to those of constipation. They suggest that the intestinal mucosa itself is an important detoxicator of putrefactive products, and that the body possesses a chemical defence mechanism more than adequate for the detoxication of putrefactive substances normally produced by the action of bacteria upon unabsorbed protein material in the intestine. It has often been shown that indicanuria is no guide to intestinal putrefaction. J. Olivet recently corroborated this, and demonstrated that there is no parallel between the amount of indicanuria and the degree of decomposition in the bowel, while in complete paralytic ileus indican is often absent from the urine. These observations seem to rule out stasis and putrefaction as sources of the intoxication, at least as long as the mucosa is healthy.

Whatever the relation between the intestinal flora and the dyspepsia or its toxic symptoms may be there are certainly cases of this disorder which only improve after treatment of focal sepsis. Dental sepsis, unhealthy tonsils and adenoids, and sepsis of the nose and accessory sinuses are not uncommon in dyspeptic children; when these have been satisfactorily dealt with the abatement of the dyspepsia is often manifest.

The frequency of the appendix as a source of infection and cause of intestinal dyspepsia is probably not great in children, but this is again a matter on which opinions differ. The issue is somewhat confused by the similarity between the symptoms caused by chronic appendicitis and tuberculous glands in the right iliac fossa. In a case which was under my care chronic appendicitis appeared to be the cause of pyloric spasm and secondary gastric dilatation. It was that of a girl aged 10, who suffered from repeated vomiting. There was a large area of gastric tympany with splashing several hours after food, and x-ray examinations proved the existence of dilatation and a varying degree of delay at the pylorus. During her stay in hospital she developed an attack of acute appendicitis. After removal of the appendix, which showed evidence of earlier inflammation as well as of the acute attack, the vomiting and delay at the pylorus ceased, and ultimately the signs of gastric dilatation disappeared. Gastric delay and dilatation with splashing under examination is an occasional occurrence at this age, but I have had no further opportunity of demonstrating any relation between it and chronic appendicitis. I believe that in children, as in adults, there are examples of dilated and splashing stomachs due to excessive ingestion of fluids.

3. Hepatic Dyspepsia.

Dyspepsia in children has been divided for descriptive purposes into gastric, intestinal, and hepatic forms. In this paper I have dwelt chiefly on the usual intestinal form, and have pointed out that the variety in which gastric symptoms predominate is relatively uncommon. The cases which I have been accustomed to call "hepatic" have been those in which fats are badly borne, in which there are from time to time bouts of toxæmia, with pale greasy stools, slight jaundice, and sometimes a palpable liver. Defective functioning of the liver may, however, be a factor in many of the cases and may be related to the toxic symptoms. Liver function tests, at best somewhat unreliable, are rendered still more so by intestinal disorder. In that unexplained malady, *coeliac disease*, in which disordered fat metabolism is coupled with such lack of development that the term "intestinal infantilism" came to be applied to it, and in which the mental features are so characteristic, the liver may play an important part. The use of bile salts certainly increases the power of these children to deal with fats.

In *cyclical vomiting* the extreme fatty change found in the liver in the fatal cases is the most noteworthy post-mortem appearance, and the close resemblance of the severe cases to those of delayed anaesthetic poisoning seems definitely to incriminate the liver. Whether this organ is primarily at fault, or whether it is damaged by toxins derived from the bowel, is a moot point. The bouts of toxæmia, accompanied by acetonaemia and the presence

of the acetone series in the urine, are not wholly explained by "ketosis"; they differ from diabetic coma and are little influenced by alkalis. There is also a well-defined group of cases in which periods of toxæmia occur, with a strong odour of acetone in the breath, and in which acetone and diacetic acid are found in the urine. These appear to resemble cyclical vomiting in every particular, except that vomiting is absent; perhaps they should also be included among the hepatic dyspepsias. Mellanby holds that the outpouring of bile salts into the duodenum is of vital importance in digestion, and that it is the bile salts and not the gastric hydrochloric acid or other substance which leads to the liberation and absorption of secretin, upon which depends the secretion of the pancreatic juice. This mechanism depends very largely upon the pH values in the duodenum, and this to a large extent upon the gastric activity. This view throws the essential upset to digestion upon impaired bile flow rather than upon alterations in gastric secretion, for without the presence of bile salts in the duodenum the pancreatic ferments are not produced except by a nervous mechanism. This work must be borne in mind when attempting to estimate the position of the liver in cases of dyspepsia.

4. The Role of the Nervous System.

Mention has already been made of the frequency of nervous symptoms in the disorder under discussion. They loom large in many of the cases and perhaps reach their acme in coeliac disease. Are they the result or the cause of intestinal symptoms in the usual intestinal dyspepsia, or are both due to some antecedent cause, such as the acidosis described by Cameron? In this connexion the high values of the fat in the blood found by Moncrieff and Payne in coeliac disease may be of importance in indicating a primary dysfunctioning of the nervous or chemical regulators of fat metabolism.

I should be glad to hear the opinions of others on this subject; my inclination is in favour of the nervous symptoms being secondary in most cases to the intestinal disorder, partly due to toxins, partly to loss of restful sleep, partly to the effect of exhaustion. Many of these children, however, are of the nervous type originally, though made worse by the dyspepsia and by the solicitude and petting of anxious parents.

There are a few examples of disordered action of the alimentary tract in children to which the name "nervous" may properly be applied in an etiological sense. Among them is that much-debated group in which there is *disordered neuro-muscular co-ordination*, leading to local spasm or failure of dilatation of segments of the tract—for example, at the cardiac end of the oesophagus, at the pylorus and in the colon, producing dilatation and hypertrophy above the situation of the zone of constriction. In another group there is *depression or exaggeration of the gastro-colic reflex*: dyspeptic symptoms in the former case being consequent upon constipation. When the gastro-colic reflex is exaggerated, intake of food is promptly followed by colic, or, in less severe cases, by discomfort about the umbilicus and along the colon, and a hurried evacuation of the bowel—the lenteric diarrhoea of the older writers. Again, *excitement or anxiety* in nervous children, as in adults, may lead to failure of the normal reaction to the taste and smell of food and complete abeyance of appetite. Lastly must be mentioned among the dyspepsias primarily nervous that which—accompanied by colitis—is consequent upon the neurosis known as *pica* or dirt-eating.

Summary.

After describing the symptoms of dyspepsia in children of school age as it is usually met with, and making a brief comparison between it and dyspepsia in adults, it is suggested that there is no fundamental difference between intestinal dyspepsia in children and many examples of dyspepsia in adults, and no reason for regarding it as a disease apart, with an individual etiology. Some views as to the interrelation of the various symptoms have been mentioned, but it has been held that our present knowledge is not sufficiently exact to argue this question with precision. The relations of dyspepsia at this age to diet, to local infections, to the liver, and to the nervous system have then been considered briefly.