

THE STOOLS IN INFANCY.

By MONTAGUE MARZELS.

THE character of the stools in infancy depends to a considerable extent upon the nature of the food consumed. Any disproportion in the several constituents of the diet is, as a rule, reflected in corresponding changes in the fæces, examination of which may sometimes help to explain the failure of an infant to thrive on breast or artificial feeding.

Before the changes that occur in the stools can be understood, it is necessary to know something of the constituents of the fæces. These are: (1) Water. (2) The indigestible portion of the food. (3) The undigested residue of food, (a) fats, (b) nitrogenous material, and (c) carbohydrates. (4) Glandular and intestinal secretions and residues, (a) ferments, (b) mucus, and (c) bile and its derivatives. (5) Salts.

(1) Water constitutes 75 to 80 per cent. of the stools. It is increased when hurried peristalsis prevents adequate absorption in the colon; and diminished through sluggish peristalsis, and also if the intake of fluid by mouth is insufficient. Thus deficiency of fluid in the stools may be either the cause or the result of constipation.

(2) The indigestible portion of the stools—cellulose—which is found in the stools of adults, helps to give bulk to the motion. This substance is, of course, absent from the stools of the milk-fed infant, where the bulk is maintained to a considerable extent by insoluble soaps. Soaps, therefore, constitute the natural roughage of the infant's stools.

(3) The undigested residue of food.

(a) Fat makes up to 20 to 40 per cent. of the dried weight of the stools. 50 to 75 per cent. of this fat is in the form of soaps—mainly combinations of calcium (and magnesium) with oleic and stearic acids. The proportion of soap is higher on a diet of cow's milk.

Neutral fat supplies about 20 per cent. of

the fat of the stools; it consists of combinations of glycerol with the higher fatty acids.

Free fatty acids constitute a small and variable remainder. They are higher and lower fatty acids derived from the fat of the food. Lactic and acetic acids may also be found in the stools and it is important to note that they are derived, not from fat, but by fermentation from carbohydrates.

Excess of fat in the stools may be due to imperfect digestion or deficient absorption. If *digestion* is imperfect, either through deficiency of pancreatic juice or excessive intake of fat, excess of neutral fat will be found in the stools. As a rule, however, any excess of fat is digested, but *absorption* is deficient, in which case the fatty acids are increased, and either appear as such in the tæces, which become acid in reaction, or are excreted as soaps in combination with calcium and magnesium—if these salts are available, in which case the stools are neutral or alkaline.

The common causes of increase of fatty acids and soaps in the stools are: (1) over-feeding with fat; (2) too rapid transit of fæces through the intestine, allowing insufficient time for fat absorption; (3) atrophy of the mucous membrane, which interferes with fat absorption; (4) coeliac disease; (5) obstruction of the abdominal lymphatics, e.g., tubercle; (6) biliary obstruction.

Microscopically, neutral fat appears in the stools as globules; fatty acids and soaps as needle-shaped crystals. Various differential staining methods are available, but are not entirely satisfactory.

(b) *Nitrogenous Material*.—The nitrogen of the stools is equivalent in amount to about 8 per cent. of the nitrogen of the stools. It is not, however, directly derived from the food protein, but consists of mucus, bacteria (mainly dead), and much smaller amounts of urea, ammonia, amino-acids and putrefactive substances like indol and skatol. The nitrogenous matter of the fæces is about 25 per cent. of the dried

weight, and the major portion of this is bacterial protein.

(3) Carbohydrates as a rule are absent from the stools, but if they are present the diet is probably at fault.

(4) (a) *Ferments*.—Sugar ferments are always found; lipolytic and proteolytic less constantly.

(b) Mucus is the natural lubricant of the bowel. It becomes noticeable when present in excess, as in inflammatory conditions, or when relatively increased because of the deficiency of the other elements of the stools—as in starvation.

(c) *Bile and its Derivatives*.—In adults, the pigment of the fæcesis hydrobilirubin (stercobilin) produced by reduction of bilirubin. In infants, the bilirubin is practically unchanged and appears as such in the stools. When, however, constipation is present bilirubin is reduced to hydrobilirubin, or even to leucohydrobilirubin, which is colourless. Hence pallor of the stools may arise in one of the following ways: (1) Absence of bile, as in biliary obstruction; the addition of fuming nitric acid fails to give a green colour. (2) Excess of fat giving bulky stools, masking the presence of bile; fuming nitric acid gives a green colour. (3) Reduction of bile, colourless leucohydrobilirubin compound. The presence of the latter is shown by shaking up a portion of the stool with Schmidt's reagent (corrosive sublimate 25 gm., sodium chloride 2.5 gm., water to 500 c.c.) when a red colour develops after some hours.

Green coloration of the stools may be noted immediately after the motion is passed or it may not appear for some hours. In the latter case, the colour, which is due to oxidation in the air, need not be regarded as pathological. Green coloration of the fresh stool is due to abnormal oxidation processes acting upon bilirubin, in either an acid or an alkaline medium, the product being biliverdin. This occurs in infections of the bowel and as such it is important.

Less often, the green colour of the stool is produced by chromogenic bacteria.

It sometimes happens that a pink coloration may be noticed on the napkin surrounding a motion; this is of no importance.

(5) *Salts of the Stools*.—Calcium, magnesium, sodium and potassium salts of chlorides, phosphates, sulphates and carbonates are found. In severe diarrhoea large quantities of salts and water may be lost, in varying proportions. Excessive loss of water leads to acidosis in the following manner: The volume of blood is greatly diminished; the blood-supply to the tissues becomes deficient and lactic acid accumulates as a result of deficient oxidation. Also, a feeble circulation in the kidney leads to imperfect excretion of acids.

Occasionally the loss of salt in the stool overshadows the loss of water and may produce alkalosis with serious symptoms and collapse.

BACTERIOLOGY OF THE STOOLS.

At birth the gut is sterile, but the meconium soon becomes infected and the following organisms appear in the stools; *Bacillus bifidus* (a Gram-positive obligate anaerobe), a Gram-positive coccus and various Gram-negative bacilli; *B. coli*, *B. acidophilus* and *B. lactis aerogenes*. *B. aerogenes capsulatus* may also be found. The Gram-positive organisms predominate on a slide made from the stool of a breast-fed infant, while the Gram-negative organisms abound in the cow's milk stool. The reason for this is that the Gram-positive anaerobic organisms flourish in the acid stool associated with relative excess of carbohydrate in the diet. In a less favourable medium, however, they fail to thrive and are overwhelmed by the organisms of the coli group. Hence the breast milk stool is normally a fermentative one, while the cow's milk stool is putrefactive. With cow's milk or simple dilutions thereof, Gram-negative organisms exceed and prote-

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decomposition gives the stools an alkaline reaction. If, however, sugar is added to cow's milk, the Gram-positive acid-producing organisms will flourish, just as they do on a diet of human milk.

In conditions of gastro-enteritis various non-lactose fermenting organisms of the coli-typhoid group may be found in the stools, or *B. aerogenes capsulatus*; more often, however, no abnormal organism can be isolated.

TYPES OF STOOLS.

Meconium is dark olive-coloured and fluid. It consists of water, mucus, bile pigment, hairs, vernix caseosa and epithelial cells. Sterile at first, bacteria soon appear, and in a few days the excreta become less fluid and browner in colour.

The starvation stool closely resembles meconium. It consists of mucus, bile and bacteria. Sometimes, where fasting is enjoined for therapeutic purposes, numerous small green liquid stools may be passed, and this is sometimes regarded, incorrectly, as an indication for the further withholding of food. The continuation of such a mistaken policy, however, may result in the death of the patient.

The stools of the breast-fed infant number two, three or four a day. Under physiological conditions they are not watery, but are semi-liquid, rather of the consistency of butter. The reaction, when tested with litmus paper, is seen to be acid, a finding which arises out of the large proportion of sugar present in breast milk. The odour of the stools is slightly sour or faecal, and the colour is golden yellow. Slight variations from the normal may be noticed in the breast milk stool; it may be greenish, or contain a few soap curds. Such abnormalities, while requiring attention in the case of the infant fed on cow's milk, need not be regarded in the breast-fed, providing that the infant is putting on weight and otherwise thriving.

The stools of an infant fed on cow's milk, or on simple dilutions of cow's milk, are one or two a day in number. Four should be considered excessive. The stools are lighter in colour than those of the breast-fed infant, and firmer in consistency. The reaction is alkaline. These findings arise in the following way: First the high salt content of cow's milk gives an excess of soap; second, the low sugar content means diminished formation of acid, while the high proportion of protein in the diet gives rise to an increased production of alkaline bodies in the bowel. The last factor imparts to the faeces a faecal or cheesy odour.

A formed stool is the rule and if a marked excess of soaps is present the stools may become very hard, dry and pale or greyish in colour.

Stools of carbohydrate excess. These are looser and darker in colour than the two preceding varieties of stools. The reaction is acid, and the odour is sour, but not foul. With excessive fermentation, the stools become very acid, loose and frothy. They are very irritating, and specially liable to excoriate the buttocks.

The stools of fat excess are pale, bulky and greasy-looking. They have a foul and rancid odour. They are acid in reaction, unless the diet contains sufficient calcium and magnesium to form soaps, when the stools become neutral or alkaline. Numerous small curds, about the size of a grain of barley, are frequently found in the stools where excess of fat has been given. They consist of mucus, fat and bacteria, and are therefore almost entirely soluble when warmed with glacial acetic acid and extracted with ether.

Similar curds may be found in diarrhoea of any origin where the hurried passage of the bowel contents interferes with fat absorption.

Protein Excess.—Where the digestion is good, the stools may be normal or constipated. The colour is brown, and the odour faecal. The reaction is alkaline.

With abnormal putrefaction, the stools become very foul and loose. Casein curds may be found as large as a small bean, tough, and insoluble in acetic acid and ether. It cannot be over-emphasized, however, that such curds are exceedingly rare, and that the small curds which are found in the stools are composed not of protein but of fat.

Of course, the "mixed" type of stools, with one or other factor predominating, is that most commonly encountered.

The stools of infants fed on whey or buttermilk mixtures are greyish brown in colour and alkaline in reaction. The stools of the infant fed on Nestlé's milk are pale and look rather like broken-up curd. Dextrinized and malted foods impart a brown colour and an acid reaction to the fæces.

Bismuth salts, when given to adults, turn the stools black, owing to the formation of bismuth sulphide. In infants, this chemical change is exceptional.

In acute gastro-enteritis the stools become loose, green and very foul. It should be noted, however, that in some of the worst cases the dejecta may closely resemble serum, and be almost odourless.

THE RELATIONSHIP BETWEEN CERTAIN DISEASES OF THE KIDNEY.

A POST-GRADUATE LECTURE DELIVERED AT THE
METROPOLITAN HOSPITAL ON OCTOBER 31, 1929.

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MY object in this lecture is not merely to repeat to you all the descriptive material which can be read in any textbook, but, so far as I can, to bring the accepted morbid changes into line with the clinical symptoms and biochemical findings, and so to simplify

some of the difficulties surrounding a proper understanding of renal disease.

One of the chief of these difficulties has been the accumulation of terms, some derived from morbid anatomy, some of purely clinical meaning, which have all at one time or another been in general use. Thus the kidney which we associate with arterio-sclerosis has in its time had a dozen names or more, and "chronic interstitial nephritis" includes several pathologically distinct conditions. I hope in the course of the lecture to clear a good deal of ground in this direction, and to put before you a rational nomenclature and classification in the light of present knowledge.

Let me remind you of the chief points upon which morbid anatomists rely when they consider the classification of renal disease from their point of view. The size and weight of the kidneys; the smoothness or roughness of the surface and the degree of such roughness; whether the capsule is adherent to the surface of the organ or readily removable; after dividing the kidney the relative amounts of the cortex and medulla, whether they are shrunken, scarred or blended, so that the line which ordinarily marks the one from the other is lost; the amount of intrapelvic fat; and the condition of the edges of the arteries which have been cut through. The colour of the kidney is most misleading; a "small white kidney" may be anything but white, and in my experience the kidney of acute nephritis is seldom red and dripping blood as some of the textbooks teach. A soft and friable kidney suggests an acute condition, and toughness or hardness points to long continued disease. In making a report upon the histology the following four structures must be separately considered: the glomeruli, the tubules, the arteries and the interstitial tissue. A just assessment of these will generally lead to a reasonably accurate opinion.

The study of morbid anatomy and histology lead as a first stage in classification to