GASTROMEGLALY FROM ARTERIO-MESENTERIC COMPRESSION OF THE DUODENUM IN THE NEW-BORN

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In this paper it is proposed to produce evidence that certain cases of obstructive vomiting in the new-born, simulating examples of intrinsic duodenal stenosis or atresia, are instances of duodenal ileus from arterio-mesenteric compression and are amenable to medical treatment; and to urge that where, in this type of case, radiological or other examination shows that the duodenal obstruction is not complete, duodenal ileus should be suspected and brief trial made of the effect of gastric lavage, in the hope of obviating the extreme risk of an abdominal operation. In support of these contentions three cases are reported; and it should be noted that, unlike the rare cases of duodenal ileus hitherto recorded in the very young, the obstruction is not associated with a gross megaduodenum although the duodenum is definitely larger than normal.

Chronic duodenal ileus (arterio-mesenteric compression), now established as a comparatively rare condition in adults, is regarded as possessing two causative factors: first, the congenital anatomical factor, the compression of the duodenum by the root of the mesentery and the superior mesenteric artery, and secondly, the accessory factor of visceral distension or ptosis whereby the compression of the duodenum is increased. The congenital anatomical factor has had to be assumed, otherwise it would be difficult to see why all cases of visceral distension or ptosis should not show signs of duodenal obstruction. The importance of the congenital factor has been emphasised in the studies of chronic duodenal ileus in young children by the present writers^{1, 2}; and it is still more evident in examples of the same condition in the new-born such as are described in the present communication. In all cases in the new-born, and in most of those seen in early childhood, the presence of an obstructive factor dating from birth can be clearly traced. Nevertheless, even in early life, the importance of the accessory factor must not be under-estimated. In babies this takes the form of a 'paralytic' distension of the stomach, and this is responsible for the occurrence of the urgent vomiting of the

obstructive type. Further, its treatment by gastric lavage and dieting promptly relieves and allows satisfactory progress to be made, even though the congenital factor in the duodenal ileus can be shown still to be operating.

Previous literature.—Several points of interest emerge from a perusal of the previous literature. Of prime importance is the observation of surgeons dealing with adult cases of duodenal ileus, that there is frequently a history of vomiting and dyspepsia in childhood, and that these symptoms tend to pass off in late childhood or adolescence, to return in later life. In a study of the same conditions in young children^{1, 2} the same tendency to spontaneous remission of symptoms in later childhood was noticed and it was claimed that the type of case then reported was that previously foretold by surgeons. But in the cases described in early childhood it was found that there was sometimes a history of severe vomiting in the first few weeks of life, giving rise in some instances to a suspicion of hypertrophic pyloric stenosis. The cases recorded in the present paper are doubtless the same as these, caught in the neonatal period. Thus, if the evidence is accepted, chronic duodenal ileus has now been recognised at all stages: in the new-born, in later infancy and early childhood, and in later middle age as the long period of freedom from symptoms passes off. The age at which examples of this condition will come under notice may be assumed to depend on the comparative severity of the congenital and accessory factors in its production.

Another point of more difficulty is found in the fact that the only examples of chronic duodenal ileus described hitherto by other authors in infants and young children have shown megaduodenum associated with the duodenal obstruction. In 1930 eight such cases between the ages of nine days and four-and-a-half years were collected from the literature¹. In them the vomiting of bile was a conspicuous feature. In none of the patients personally observed, neonatal or at a later age, could the enlargement of the duodenum be reasonably classed as megaduodenum, nor has bilious vomiting been more than exceptional. It seems clear that congenital arterio-mesenteric compression can produce in very early life two groups of cases: one showing megaduodenum with bilious vomiting and presumably requiring surgical relief, and the other, described here, which hardly shows either of these features and appears amenable to medical treatment. It is not easy to explain the difference between the two groups. If the megaduodenum is really due to the obstruction and not merely an associated anatomical abnormality, it may be supposed that in the cases with megaduodenum the congenital and unalterable pressure on the duodenum is more severe than in the type of case described in the present paper.

Lastly, extrinsic duodenal obstruction in the new-born has been described as due to peritoneal folds or bands, and success has been claimed to follow division of such bands. Such cases are exceedingly

rare, and in some instances operation has failed to relieve the symptoms. It is therefore possible that in certain instances at least the bands were really associated with arterio-mesenteric compression.

In 1927 D. P. D. Wilkie³ pointed out the occurrence of symptoms in childhood in his adult cases of chronic duodenal ileus, and in 1933 R. P. Rowlands⁴ noticed the same sequence of events.

Important papers on intrinsic duodenal obstruction were written by E. Cautley⁵ in 1919 and H. C. Cameron⁶ in 1925. Both agree that atresia is much more common than stenosis. 'In the great majority the occlusion is complete or all but complete, and unless relieved by operation life is not prolonged beyond two or three weeks' (Cameron). Bile is seen in the vomit in 80 to 90 per cent. of the cases, and the duodenum appears as a 'widely distended pouch.'

Bernheim-Karrer⁷ was the first to describe arterio-mesenteric compression as an extrinsic cause of duodenal obstruction in an infant. In 1904 he recorded the case of an infant whose symptoms dated from birth and who died at the age of eight months. At post-mortem examination megaduodenum was found associated with chronic duodenal ileus. Other cases of the same sort, all with megaduodenum, have been reported by Frank⁸ (two cases), Downes⁹, Dubose¹⁰, Jewesbury¹¹, Henske and Best¹², and Camera¹³.

R. Jewesbury and Max Page¹⁴ in 1922 reported two cases of duodenal obstruction by bands. Both were neonatal cases. Adhesions were found round the duodeno-jejunal flexure, binding the jejunum in its first two or three inches to peritoneum on the posterior abdominal wall. These were the first cases of this type to be cured by operation, but at the time of reporting the second case had relapsed and its condition was stated to be not good enough to warrant further laparotomy. Both these cases showed megaduodenum, as did the successful one reported in 1926 by T. T. Higgins and D. Paterson¹⁵. F. Braid¹⁶ in 1933 recorded an interesting case in which vomiting dated from birth, necessitating bottle-feeding until the age of two-and-a-half years. At four-and-a-half years the abdomen was opened and peritoneal bands, thought to be obstructing the duodenum, were divided. Progress was unsatisfactory, and the author suspected later an associated arterio-mesenteric compression, unrecognized at the time of the operation.

Case records.

Three cases are reported. In the first a diagnosis of incomplete duodenal obstruction was reached, but the possibility of arterio-mesenteric compression was not considered nor was its presence recognized at operation: this unsuspected condition was revealed post-mortem. In the second and third cases, seen later, this condition was suspected, and the diagnosis was taken to be confirmed by the evidence afforded by radiological examination and the successful results of medical treatment.

Case 1. (previously recorded²).—B. S., female, aged five days, was admitted to Paddington Green Children's Hospital on November 21, 1931. From birth she had vomited everything, and twice the vomit had been black or brownish.

On admission on the fifth day, she was dehydrated and on being fed she squirmed about as if in discomfort until the stomach was emptied by a copious vomit. The vomit contained much mucus, but showed no colouration by bile. Irregular gastric peristalsis was observed,

but was rather obscured by the wriggling movements of the child. No pyloric tumour was felt.

X-ray examination by an opaque meal showed the presence of incomplete duodenal obstruction (see below).

On the seventh day the child was failing, and in the hope that the obstruction might be due to some condition such as a band that could be easily dealt with, the abdomen was opened. The stomach was found to be large, the pylorus normal, the duodenum large and the jejunum

FIG. 1.—Case 1, aged 6 days; $1\frac{1}{2}$ oz. opaque meal at 15 minutes, prone.

FIG. 2.—Case 1, aged 6 days; $1\frac{1}{2}$ oz. opaque meal at 35 minutes, prone.

definitely narrow, but the cause of the duodenal obstruction was not localized. The infant died next day, aged eight days.

RADIOGRAPHIC EXAMINATION (November 21, 1935): One-and-a-half ounces of a fluid suspension of barium sulphate were introduced into the stomach by tube. On screening vigorous gastric peristaltic waves were seen.

The first film (fig. 1) was taken at fifteen minutes, prone. It shows a flocculent shadow of the barium, due to admixture with the contents of the stomach present before the meal was given. The outline of the

shadow is thus irregular and does not indicate in silhouette the true contour of the stomach, but only a puddle resting on the anterior gastric wall. It does not reach the fundus, nor does it fill the pyloric antrum as this passes to the right and backwards towards the duodenal cap. The stomach is therefore larger than the patch of barium at first sight suggests. The vigorous peristalsis seen on screening has already ceased although only a few minims of the meal have passed into the intestine,

FIG. 3.—Case 2, aged 10 days; 1 oz. opaque meal at 20 minutes, supine.

FIG. 4.—Case 2, aged 16 days; 2 oz. opaque meal on ingestion, supine

to be seen in the commencement of the jejunum as a narrow streak. No part of the duodenum is seen at this examination, but it is to be noted that the streak in the jejunum is at most not more than half the width of the part of the duodenum seen in fig. 2.

The second film (fig. 2) was taken at 35 minutes, prone. The general appearance of the stomach remains unchanged and only a few more minims of the meal have passed on, to be seen as flecks distributed

FIG. 5.—Case 2, aged 5 months; 7 oz. opaque meal at 1 hour prone.

FIG. 6.—Case 2, aged 5 months; 7 oz. opaque meal at $2\frac{1}{2}$ hours, prone.

throughout the small intestine. This film, however, shows the duodenal cap and a small quantity of the meal in the second part of the duodenum.

The emptying time of the stomach was not taken, the presence of gastric delay being already established.

AUTOPSY.—The body of a small emaciated baby. When the abdomen was opened the stomach was seen to be moderately enlarged and the duodenum, also enlarged, was proportionate to the size of the stomach. Hypertrophy of the gastric wall could not be recognized with assurance.

FIG. 7.—Case 3, aged 12 weeks; $4\frac{3}{4}$ oz. opaque meal on ingestion, oblique.

The jejunum was definitely smaller than the duodenum, between one-half and two-thirds of its width, and the change in the size of the gut came abruptly at the level of the crossing of the intestine by the root of mesentery and the superior mesenteric artery. The right kidney was completely absent. Except for the compression exercised on the duodenum, the disposition of the root of the mesentery and its vessels appeared normal.

Case 2.—A. F., female, aged ten days, was a first child, born at full term and admitted on May 18, 1934, with her mother to a private room at the Victoria Hospital for Children. She was brought by Dr. H. Brade-Birks who stated that from the first the baby had seemed quite disinclined to take the breast. For the first two days she had vomited mucus, and from the third day she had vomited all breast-feeds. The vomits had been projectile, occurring about ten minutes after the feeds. No bile had been seen in the vomit at any time. It was thought that there had been a trace of milk-curd in the motion on the sixth day, but it was by no means certain that any food had passed into the intestine.

The baby on the tenth day was well nourished, but beginning to look ill. On being fed she squirmed about as if uncomfortable until the contents of the stomach were forcibly ejected. The stomach appeared large, but no peristalsis was seen nor tumour felt. The vomit contained much mucus but showed no colouration by bile.

In order to settle whether the obstruction were complete or not, an opaque meal was given without delay. This showed (fig. 3) that the obstruction was incomplete (see below), and in the hope that the case might be one of duodenal ileus the stomach was ordered to be washed out at once and again twelve hours later. The child had vomited the remains of the barium meal, but after the first wash-out all serious vomiting ceased. The first two wash-outs contained traces of bile.

The baby was seen next morning, May 19, in consultation with Mr. Jennings Marshall, and it was decided to continue the twelve-hourly lavage for a further twenty-four hours. At the end of this time progress was so satisfactory that it was agreed to continue with the lavage and four-hourly breast feeds.

On the sixteenth day, after six days of treatment, a second opaque meal was given. Radiographic examination showed that the stomach, although of more normal shape, was clearly enlarged and obstructed. Further, duodenal stasis was well demonstrated (fig. 4).

Gastric lavage was reduced to once daily and the baby progressed so well that she was sent home to continue treatment on May 29.

At five months she weighed $15\frac{1}{2}$ lb., and was progressing in every way satisfactorily. At this time (October 11, 1934) she was examined for the third time by an opaque meal. This showed conclusively that there was still present some obstructive factor which the vigour of peristalsis was overcoming satisfactorily (fig. 5 and 6). Gastric peristalsis was well seen clinically.

She continued to progress well, and remain symptomless. At ten-and-a-half months her weight was 22 lb. $1\frac{1}{2}$ oz.

RADIOGRAPHIC EXAMINATIONS. (1) On May 18, 1934, the infant, aged ten days, was given one ounce of a fluid barium feed. Fig. 3 is from a film taken 20 minutes after ingestion, supine. It shows the lower oesophagus greatly dilated; the stomach much enlarged, ovoid, devoid of peristalsis, and protruding well over the region of the duodenal cap, which is consequently not seen. Very little food has escaped into the intestine. At 40 minutes the stomach and oesophagus were much the same, but a little more food had passed into the intestine. At 100 minutes the stomach was empty, the baby having vomited.

(2) After the stomach had been washed out twice daily for six days a second meal, consisting of two ounces of fluid barium was given on May 24, the baby being sixteen days old. Fig. 4 is from a film taken immediately after ingestion, supine. The stomach, although greatly enlarged, is more normal in shape than before, thus enabling the duodenal cap to be seen. A later portion of the duodenum is seen crossing the

vertebral column and exhibiting the classical cog-wheel formation of duodenal ileus. Much more food has passed into the intestine than at the first examination.

(3) On October 11, 1934, the baby, now aged five months and free of symptoms, was given a third opaque meal consisting of seven ounces of fluid barium. Fig. 5 is from a film taken at one hour, prone. The

FIG. 8.—Case 3, aged 12 weeks; $4\frac{3}{4}$ oz. opaque meal on ingestion, prone. (The outline of the duodenum below the stomach has been artificially emphasised.)

stomach is very large and the barium is resting on the anterior wall of the stomach. The real outline of the stomach is seen to be larger than the shadow of the barium. Its wall exhibits obvious hypertrophy and hyper-peristalsis is clearly seen. Fig. 6 is from a film taken at $2\frac{1}{2}$ hours prone. The residue of the meal is in the fundal portion of the stomach, and the duodenum is seen crossing the vertebral column obliquely, indicating duodenal stasis.

Case 3.—A. G., female, born November 9, 1934, was a first child, full-term, weighing at birth $6\frac{1}{4}$ lb., and breast fed for six weeks. From birth until she was admitted to St. Mary's Hospital, aged eight weeks, she had been under continuous medical treatment, at first for vomiting and later for vomiting and diarrhoea. Vomiting started at birth, but it was not sufficient to prevent some gain in weight during the first three weeks. Then it gradually increased and became frequent, copious and projectile, and later was accompanied by diarrhoea. By December 27 she was so ill that she was admitted to another hospital from which she was removed on January 7, 1935, having lost one pound in weight. The next day she was admitted to St. Mary's Hospital.

On admission (January 8, 1935) she was eight weeks old and weighed 8 lb. She looked ill and dehydrated, and was passing frequent small green stools. The most conspicuous symptom was the regular projectile vomiting, the vomit containing much mucus but no obvious bile. The stomach appeared large under the abdominal wall, and gastric peristalsis was once observed. No pyloric tumour was felt.

An opaque meal, given on January 9, showed obvious obstruction to the emptying of the stomach, and barium was vomited as long as ten hours after the meal. Gastric lavage was therefore ordered twice daily, and the child given small feeds of a half-cream dried milk every two hours. One vomit showed traces of bile. The vomiting was much reduced by these measures. After twelve days the wash-outs were free of mucus and were reduced to once daily. The feeds were altered to four-hourly feeds.

On January 28 a second opaque meal was given which showed that food was leaving the stomach much more quickly than before. On February 5 a third meal was given, this time immediately after gastric lavage, and the diagnosis of duodenal obstruction was confirmed (see below).

Subsequent progress was slow but satisfactory. In order to keep the vomiting entirely in abeyance gastric lavage was still necessary every third day at the end of March. Gastric peristalsis was seen several times. Massage to the limbs and back was useful to get tissue assimilation restarted.

By April she was a good colour and very contented, and at the age of six months her weight was $12\frac{1}{2}$ lb. (See postscript, p. 193.)

RADIOGRAPHIC EXAMINATIONS. (1) On January 9, 1935, at eight weeks of age, an opaque meal of $2\frac{1}{2}$ ounces of fluid barium was given. The stomach was much enlarged and ovoid in shape. A suspicion of duodenal stasis was seen in the descending duodenum in the first film. At one hour very little food had passed into the intestine, the calibre of which seemed unduly small. At two hours the amount of food in the small intestine was still very slight. Barium was vomited ten hours after the meal.

(2) On January 28 the opaque meal was repeated. Food passed much more quickly than in the previous examination and at four hours practically all the small meal had left the stomach.

(3) On February 5, immediately after gastric lavage, a third opaque meal, consisting this time of $4\frac{3}{4}$ ounces of fluid barium, was given in an attempt to display duodenal stasis. Fig. 7, taken from a film in the oblique position on ingestion, shows considerable enlargement of the stomach. The duodenal cap is also enlarged to some extent, and the descending part of the duodenum is seen. Fig. 8, from a film taken in the prone position at about the same time, shows a large ovoid stomach, devoid of peristalsis, and covering the region of the duodenal cap. Lying

below the lower border of the stomach is seen the duodenum, somewhat enlarged and showing stasis. For the purpose of reproduction the outline of the duodenum in this figure has been artificially emphasized.

Discussion.

Symptoms.—In the duodenal ileus (arterio-mesenteric compression) of infancy the factors responsible for the production of obstructive symptoms are the degree of compression of the duodenum with which the child is born, the volume of the feeds given, and the amount of distension of the stomach. On these depend the time of the first appearance of the symptoms and the severity of the vomiting. The influence of the congenital anatomical fault is obvious, yet it is not necessarily paramount. This is well illustrated in case 2 above, where the refusal of food and vomiting were apparent from the date of birth, and yet medical treatment was sufficient to allow satisfactory progress to be made. The influence of the size of the feeds and the distension of the stomach is also seen in the duodenal ileus of later infancy and early childhood as has been pointed out elsewhere. In them refusal of food is often the chief symptom until the child reaches an age when it can be forced to take more than it wants, and then vomiting of the obstructive type develops. In case 3 it is probable that the small size of the breast feeds from the primiparous mother accounts for the vomiting being comparatively mild for the first three weeks of the child's life. It is when the factor of gastric distension comes into operation that vomiting becomes of the obstructive type, large, regular, projectile, and showing mucus.

The earliest symptoms therefore consist either of refusal of food or, where normal feeds are taken, of vomiting. After a feed the infant, although not apparently in actual pain, wriggles about as if in discomfort until relieved by vomiting. The stomach is seen much enlarged and standing out under the abdominal wall, but waves of gastric peristalsis are not easily nor frequently to be detected. Their rarity is due to the fact that during times of severe obstruction the stomach is in a state of 'paralytic' distension, just as in some cases of hypertrophic pyloric stenosis peristalsis may be in abeyance until the stomach has been washed out for a day or two.

The presence of mucus in the vomit is of importance. In a study of the gastric contents in wasted infants¹⁷ it was pointed out many years ago that the presence of an excess of mucin was characteristic of hypertrophic pyloric stenosis, and it may now be put forward that in any case of chronic vomiting in an infant the persistence of mucus in the vomit indicates obstruction high in the alimentary tract. It is due to the associated chronic gastritis. The presence of bile in the vomit is not, contrary to what might be expected, a cardinal feature in duodenal ileus of the type here described. It has been recorded in the rare cases of gross megaduodenum associated with duodenal ileus, but in the group now under discussion it is little in evidence, and it is important to realize that this is so. In case 1 no bile was seen in the vomit while the infant was under observation; in case 2 traces of bile were seen in the first two gastric

wash-outs; and in case 3 the vomit was on one occasion seen to be slightly tinged with bile. In hypertrophic pyloric stenosis bile is practically never seen in the vomit or wash-out.

Constipation is not so marked a feature in duodenal ileus as in pyloric stenosis. If the bowels are disordered it is usually by diarrhoea, probably secondary to the gastritis. In case 3, when first coming under observation, there was evident infective enteritis.

Radiography.—Examination by means of an opaque meal before treatment has been instituted usually reveals a much distended stomach, ovoid in shape, with its pyloric end covering the duodenal cap. It also shows that the meal passes very slowly into the small intestine. Such a picture, although characteristic of duodenal ileus to those familiar with it, cannot be taken to prove more than that there is obstruction to the evacuation of the stomach and that the obstruction is incomplete.

If, however, the opaque meal be given directly after gastric lavage more positive evidence may be obtained. The stomach, although much enlarged, may be more normal in outline and the duodenal cap may be seen, especially in oblique views. Oblique and lateral views are nearly always necessary to expose the duodenal cap, because in postero-anterior views it is usually covered by the enlarged stomach. It is essential to see the cap at some period of the examination in order to demonstrate a normal pyloric canal with no increase in the pyloro-duodenal gap, thereby excluding even a minor degree of congenital pyloric stenosis. Lastly, although this cannot be guaranteed at any single examination, evidence of stasis in some part of the duodenum below the cap may be caught.

The use of an opaque meal, therefore, is of great value in diagnosis. It proves the presence of an obstruction to the evacuation of the stomach, and shows that the obstruction is incomplete and not situated at the pylorus. With careful technique and some perseverance it will also show evidence of duodenal stasis. On the other hand the actual emptying time of the stomach is more conveniently estimated by passing a tube into the stomach four hours after a meal than by radiographic examination. In case 3 barium was vomited as long as ten hours after the meal, and it is impracticable to submit a small infant to so many exposures of x-rays as might be necessary to determine the exact emptying time of the stomach.

In order to get the best and most immediate results by radiography two points should be kept in mind: first, the opaque meal should be given directly after the stomach has been thoroughly washed out; and secondly, the meal given should be considerably larger than the normal to allow for the enlargement of the stomach.

Diagnosis.—Where serious vomiting dates from birth (as in cases 1 and 2) the presence of obstruction is obvious, and the chief difficulty is to determine whether it is complete or incomplete. In a new-born baby who is keeping down only small quantities of food it may be

extraordinarily difficult to be sure if any milk is passing into the intestine. An opaque meal will, however, settle this matter at once.

Where, as in case 3, the initial vomiting is less urgent, the chief difficulty is to establish the presence of organic obstruction. Here a positive diagnosis may be reached by a consideration of the distension of the stomach, the discomfort after feeds, the occasional occurrence of visible gastric peristalsis, the forceful nature of the vomiting, and the presence of mucus in the vomit. These signs indicate the presence of obstruction to the evacuation of the contents of the stomach, and in the absence of positive signs of hypertrophic pyloric stenosis tend to incriminate the duodenum. The absence of obvious bile in the vomit must not be taken to exclude duodenal obstruction of an incomplete type. Radiographic examination by an opaque meal, especially if undertaken directly after gastric lavage, will confirm the presence of high obstruction and will exclude pyloric stenosis. It may also give positive evidence locating the obstruction to the duodenum.

In these young infants there is, of course, no great amount of time available for investigation, and it may therefore be urged that, when a diagnosis of incomplete non-pyloric obstruction has been reached, the correct line to take is to order gastric lavage every twelve hours for one or two days before considering opening the abdomen.

Course.—The progress of these cases must depend to some extent on the degree of unalterable compression of the duodenum with which they are born; but from the examples recorded here there appears to be a good chance that, if by lavage and careful dieting the tone of the stomach is maintained and severe distension prevented, compensatory hypertrophy of the gastric wall will enable the obstruction to be overcome. Where the congenital compression is too severe to allow of progress by such means, it may be supposed that gross megaduodenum may develop. Even when progress is being well maintained and symptoms are in complete abeyance a certain amount of obstruction from the congenital compression still remains. This is well seen in the skiagrams of case 2 (figs. 5 and 6) taken at five months of age, where the enlargement of the stomach, the hypertrophy of its walls and the hyperperistalsis indicate the persistence of some degree of obstruction in the absence of any symptoms. The persistence of visible gastric peristalsis is of the same significance.

Study of congenital duodenal ileus in later infancy and early childhood shows the same thing. Although vomiting may have occurred in the earliest weeks of life, giving rise to a suspicion of pyloric stenosis, in most cases the chief early symptom is refusal of food, and by this means vomiting is kept in abeyance and the evidence of any form of obstruction is masked. At about the age of eighteen months, when the child is getting a mixed solid and fluid diet and can be forced to take more than it wants, bouts of obstructive vomiting become common. In the intervals between the attacks both clinical and radiographic examination will show clearly the persistence of some degree of obstruction.

At somewhere between the ages of seven and ten years the symptoms appear to die down spontaneously, owing presumably in some way to the growth of the child. Their disappearance at these ages is much hastened by the administration of a dry diet with drinks two to three hours after meals.

It is possible that not all cases entirely lose their symptoms in adolescence, and it may be suspected that some of the examples of severe dilatation of the stomach seen at this age owe their origin to chronic duodenal ileus. One patient under our observation at the age of seventeen years for extremely severe gastric dilatation, developed acute duodenal ileus after laparotomy fifteen years later.

It may be of interest to mention that during childhood chronic duodenal ileus does not seem to predispose towards such a post-operative risk. Acute duodenal ileus seems almost unknown in children, and several of our cases in children have been through operations for removal of the tonsils or for appendicitis without any untoward symptoms.

Treatment.—The chief aim in treatment is to prevent gastric accumulation and distension, and for this gastric lavage is most effective. A weak alkaline solution may be used, for in spite of the criticisms by biochemists of the use of alkalis for this purpose the method seems harmless and has the advantage of removing the mucus in the stomach more effectively than normal saline solution. The lavage should at first be given twice daily and this should be kept up until the wash-outs remain free of mucus. Then the interval between the treatments may be lengthened to 24 hours, and later to 36 or 48 hours. Any return of copious or projected vomits, or any reappearance of mucus in the stomach, indicates the need for more frequent lavage for the time being. In any case it will probably be twelve weeks or more before lavage can be entirely discontinued.

The second indication for treatment is to give an easily digested feed at properly spaced intervals. If breast milk is not available, dilute evaporated milk, or a half-cream dried milk may be given. It is easy to estimate the emptying time of the stomach by passing a tube at four hours after a feed to find the amount of residue, remembering that with lavage the stomach soon empties more rapidly as the vomiting lessens. Probably it is best to try to get straight on to four-hourly feeds, but where the infant's condition is very frail half the amounts may be given every two hours.

It should be clearly understood that in the class of case described here the enlargement of the duodenum is not sufficient to make the performance of duodeno-jejunostomy a practicable operation. We have had no experience of the other type of case showing gross megaduodenum. Probably here operation is a necessity, and in rather older children duodeno-jejunostomy has been successfully performed. It is generally admitted that a gastro-jejunostomy is not a successful procedure for chronic duodenal ileus and our experience in two cases without megaduodenum in small children beyond the age of infancy confirms this,

Summary and conclusions.

1. Three cases of chronic duodenal ileus from arterio-mesenteric compression in new-born infants are described and the evidence on which the diagnosis is based is submitted.

2. The chief clinical features of the syndrome consist of the enlarged stomach (gastromegaly) causing protrusion of the upper abdomen and showing occasional visible peristalsis; and vomiting of the obstructive type, the vomits being large, projected and containing mucus. Alternatively, refusal of food may keep the vomiting in abeyance for the time being.

3. Radiographic examination will demonstrate the presence of obstruction in mild cases, and in the severe ones will distinguish between complete and incomplete obstruction. It easily excludes any form of pyloric obstruction and, especially in oblique views, may demonstrate duodenal stasis. The opaque meal should be given directly after gastric lavage and should be considerably larger than the normal for the age to allow for the enlargement of the stomach.

4. Medical treatment, chiefly by means of gastric lavage, is capable of restoring the tone of the distended stomach, so that satisfactory progress is possible in spite of the persistence of some degree of duodenal obstruction.

5. It is urged that where symptoms suggesting duodenal stenosis or atresia are found to arise from an incomplete duodenal obstruction, the possibility of duodenal ileus should be considered and a brief trial made of medical measures before submitting the infant to the extreme risk of an abdominal operation.

6. In the exceptional cases, described by other authors, in which gross megaduodenum is present, operation is probably essential; but in the type described here there is no possibility of the performance of duodeno-jejunosomy.

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POSTSCRIPT.

Case 3. —At the age of six months (weight, $12\frac{1}{2}$ lb.) this baby developed difficulty in breathing with signs of bronchopneumonia and died in thirty-six hours. Death was found at autopsy to be due to diphtheria, membrane extending from the larynx into the lungs. The unfortunate fatality appeared unconnected with the condition of the digestive tract.

Autopsy revealed the stomach considerably enlarged, with hypertrophied wall: evidence of chronic gastritis was clear. The pyloric wall partook in the hypertrophy of the rest of the stomach. The pyloric canal was dilated and its rugae flatter than normal. The first part of the duodenum

was enlarged and appeared proportionate to the size of the stomach. Then followed an inch or two where the duodenum was smaller, if not reduced to its normal size. From thence onwards to the duodeno-jejunal flexure the duodenum was much enlarged, at least to twice its normal size, with some hypertrophy of its wall. Emerging from behind the root of the mesentery it resumed its normal size: this change was not absolutely abrupt, but was spread over about two inches of the gut. Although the site of the obstruction thus corresponded to the position of the mesenteric root, it could not be determined that that structure was actually compressing the gut; but on the other hand there were no bands or adhesions to account for the obstruction. Apart from the conditions mentioned the rest of the examination showed nothing of interest.

The autopsy, therefore, fully proved the presence of duodenal obstruction at about the level of the root of the mesentery, and we were satisfied that our diagnosis of arterio-mesenteric compression was confirmed. That the gut, on emerging from behind the root of the mesentery, resumed its normal size gradually rather than abruptly, is probably to be explained by the fact that the infant had been without urgent obstructive symptoms for many weeks before death. That, apart from the obvious effects of obstruction, it could not be made out that the mesenteric root was actually pressing on the gut is in line with what is found in adult cases post mortem. In them it is well recognized that the degree of compression cannot be judged by the anatomical disposition of the parts at autopsy, and that the severity of the obstruction is to be computed by the symptoms during life. In the present case the obstruction was at one time so severe as to cause the infant to vomit barium no less than ten hours after ingestion.

In the light of the autopsy our interpretation of the case perhaps needs modification in one minor matter. It is possible that the influence of the congenital anatomical factor was less, and that of the accessory factor of distension was greater, than we realized; and perhaps it was this, rather than underfeeding, which was responsible for the mildness of the obstructive symptoms in the first three weeks of life.

REFERENCES.

1. Miller, R., & Gage, H. C., *Arch. Dis. Childh.*, Lond., 1930, V, 83.
2. Miller, R., & Gage, H. C., *Ibid.*, 1932, VII, 65.
3. Wilkie, D. P. D., *Am. J. Med. Sci.*, Philad., 1927, CLXXIII, 643.
4. Rowlands, R. P., *Lancet*, Lond., 1933, i, 1107.
5. Cautley, E., *Brit. J. Child. Dis.*, Lond., 1919, XVI, 65.
6. Cameron, H. C., *Brit. Med. J.*, Lond., 1925, i, 765.
7. Bernheim-Karrer, O., *Corr.-blatt. f. Schweiz. Aerzte.*, Basel, 1904, XXXIV, 257.
8. Frank, E. S., *Ztschr. f. Kinderh.*, Berlin, 1913, IX, 99.
9. Downes, W. A., *Ann. Surg.*, Philad., 1917, LXVI, 436.
10. DuBose, F. G., *Surg. Gynec. Obstet.*, Chic., 1919, XXIX, 278.
11. Jewesbury, R., *Proc. R. Soc. Med.*, Lond., 1922-3, XVI, (*Sec. Dis. Child.*), 10.
12. Henske, J. A., & Best, R. R., *Am. J. Dis. Child.*, Chic., 1928, XXXVI, 1224.
13. Camera, U., *Arch. d. méd. des enf.*, Paris, 1930, XXXII, 583.
14. Jewesbury, R., & Page, M., *Proc. R. Soc. Med.*, Lond., 1922-3, XVI, (*Sec. Dis. Child.*), 50.
15. Higgins, T. T., & Paterson, D., *Arch. Dis. Childh.*, Lond., 1926, I, 285.
16. Braid, F., *Proc. R. Soc. Med.*, Lond., 1933, XXVI, ii, 1540.
17. Miller, R., & Willcox, W. H., *Lancet*, Lond., 1907, ii, 1670.