CONGENITAL HYPERTROPHIC PYLORIC STENOSIS.

By LEONARD FINDLAY, M.D., D.Sc.

(Visiting Physician, Princess Elizabeth of York Hospital for Children.)

Introduction.

In view of an analysis⁽¹⁾ of a series of cases of hypertrophic pyloric stenosis observed in the Edinburgh School and reported in the Brit. Med. Jour. only last year, and of the subsequent comments which appeared in the correspondence columns of that same journal, the record of a typical example of the condition with some remarks on the diagnosis and treatment would not seem to be out of place. In the above-mentioned paper by Drs. Wallace and Wevill it is implied that the diagnosis is somewhat difficult, and in many cases uncertain, and not a few of the writers who contributed letters of comment seemed to be of a like opinion.

Would that there were many diseases which could be so definitely diagnosed by ordinary clinical or bed-side methods as can hypertrophic pyloric stenosis! If this were the case the practice of medicine would be a much easier and less harassing occupation than it is. In our considered opinion failure to diagnose hypertrophic pyloric stenosis is entirely due to inattention to certain important points and to not spending sufficient time and care over the examination. If due attention is paid to these matters there should, we are convinced, never be any doubt regarding the presence or otherwise of this not uncommon congenital anomaly.

Case Record.

The case which we wish to describe was that of a male infant, the first-born of healthy parents. The pregnancy was unaccompanied by any trouble; it proceeded to full term and the child was delivered spontaneously and apparently healthy, with a weight of $7\frac{1}{2}$ lbs. Breast feeding every four hours was adopted but vomiting occurred from the first day: this did not happen at first after every feed but it steadily got worse and ultimately became projectile in nature, so that at the age of two weeks the child was put on the bottle, each feed consisting of Nestle's milk 1 drachm to 8 ounces of water. In spite of this change in the diet the vomiting continued to increase in severity and the bowels, which up till that time had been moving regularly, became constipated.

The child came under observation at the age of three weeks, weighing 6 lbs. 11 ozs., of fair colour (hæmoglobin 76% and red blood cells 4,740,000 per c.mm.) quite vigorous and with no appearance of dehydration. The mouth, ears, chest and genitalia were normal. Inspection of the abdomen revealed the outline of a full stomach (the child had had a feed shortly before) and an occasional abortive peristaltic wave was observed, but the typical recurring waves of peristalsis could not be elicited. The abdomen was lax and careful palpation disclosed the presence of a small pyloric tumour in the mid-line and high up in the epigastrium. During the examination vomiting, typically expulsive in nature, occurred and on palpating once more the pyloric tumour could no longer be detected.

Examination of the urine disclosed a complete absence of chlorides, but the breathing seemed normal in depth and rate and the blood CO_2 amounted to 65 vols. % (normal 55 to 75%), so that there was no evidence of alkalosis.

Although a definite diagnosis of pyloric stenosis was made and the child was not acutely ill, and would have stood an operation quite well, this was not done, but it was decided to temporize and by proper feeding, gastric lavage and subcutaneous saline transfusions to get the child into perhaps even a better state for operation. Subsequent events, however, did not altogether justify the delay, for, although the weight did not decline, no doubt due to the subcutaneous salines, the vomiting became more frequent and severe, the whole feeds being almost immediately rejected. Gastric peristalsis became more evident and at times was very marked. Still, however, the tumour could only be felt when the stomach was full. After a vomit the pyloric tumour could not be palpated, but if another feed were given it again came within the reach of the fingers. One concluded that this intermittent appearance of the tumour was due to its high position in the abdomen and it occurred to one that perhaps by holding the child up by supporting it under the armpits and shaking gently the pylorus might be induced to descend, as indeed it did, for immediately after this manoeuvre the tumour could be comparatively easily rolled under the palpating fingers.

Four days after coming under observation the child was operated upon by our colleague, Mr. H. S. Wright. Local anaesthesia after a preliminary dose of 1/75th grain of morphia was employed and Ramstedt's technique adopted. The child stood the operation well. Peptonised milk in dram doses every hour, and in gradually increasing amounts, was the method of feeding practised and the child rapidly improved. No vomiting occurred after the operation, the bowels moved regularly and by the end of two weeks the child had a weight of 8 lbs.

Commentary.

A.—Diagnosis.

Sex and position in family. As is usual, the patient suffering from hypertrophic pyloric stenosis was a male and the first-born. Female children, however, are so frequently affected that sex is of little moment in the diagnosis. The late Dr. John Thomson always told that the first seven examples which he encountered were girls. Of a consecutive series of 100 cases personally observed, 83 were boys and 17 were girls. Too much stress also need not be laid on the position of the child in the family. Whatever the disease, the patient is most likely to be the first-born, for the simple reason that there are most first children. If there is a family at all there must be a first child. Nevertheless, the proportion of first-born children is higher in the case of hypertrophic pyloric stenosis than in most other diseases.

Vomiting. Vomiting of course is the paramount symptom, and it may be laid down as a golden rule that in the new born or very young infant, and especially if being breast-fed, vomiting should always raise the suspicion of pyloric stenosis. Vomiting, however, is not necessarily present. We ourselves have seen a typical example, verified by post-mortem examination, in which the child only vomited once and the bowels moved regularly every day. The patient came under observation with the slow shallow breathing characteristic of alkalosis. This prompted a careful examination of the abdomen, because pyloric stenosis is the almost invariable cause of this state during infancy, when a pyloric tumour and visible gastric peristalsis were revealed. The child died at the age of four months from an intercurrent pneumonia after having been under observation in hospital for one month.

This absence of vomiting is to our mind conclusive proof that the obstruction in this disease is not entirely organic in nature, at least from the presence of a

pyloric tumour, but that it is chiefly due to spasm. By no other conception than one of spasm can be explained the sudden and complete cessation of all vomiting for as long as 24 or 48 hours, which is not so very uncommon.

It will have been noticed that in our case the vomiting commenced immediately after birth, which is unusual. As a rule, the vomiting does not set in till some time later—most frequently about the third week, although it may be delayed till the eighth week. Because of this fact it seems difficult for many to believe that the pyloric hypertrophy, the fundamental lesion, is congenital in origin. But that the condition is congenital there is no doubt. The hypertrophy of the pylorus has been observed in the foetus. The hypertrophy of the pylorus almost certainly precedes the vomiting, at least in our experience a tumour can always be felt whenever the symptom of vomiting appears, and as just noted, may be present when there is no vomiting at all. This surely is further proof, if such were required, that the cause of the obstruction and vomiting is spasm superimposed on the hypertrophy.

Vomiting, however, is almost invariably present and is usually, although not necessarily, of a peculiarly characteristic nature. It tends to occur immediately or soon after a feed. The vomitus is ejected with great force as if shot out of a gun; hence the terms explosive or expulsive vomiting. The amount vomited may equal a whole feed, but if each feed is not vomited it may have a bulk of more than one feed. In the early stages the type of vomiting may be anything but characteristic; it may be infrequent and slight in degree, but with time it as a rule assumes its severe and typical form.

Pyloric Tumour and Visible Peristalsis. However characteristic the vomiting may be and suspicious the combination of vomiting and constipation in the young infant, the ultimate diagnosis rests on the palpation of a hypertrophied pylorus and the presence of a dilated stomach with visible peristalsis. But to appreciate these two signs the examination must be carried out in a regular manner and with the utmost patience.

It is a fortunate circumstance that in the majority of instances the abdomen is quite lax and permits of easy palpation. This is no reason, however, that every precaution should not be taken. The patient must be placed in a comfortable position so that all wriggling and movements from general restlessness are eliminated. As a rule it is advisable to have the child lying in the lap of the mother or nurse, and preferably in front of the fire, so that there may be no risk of chilling with consequent restlessness. The physician should be comfortably seated on the left of the patient and he should use the left hand for palpation. The hand, warmed, is placed on the abdominal wall and gradually, with each succeeding respiration, explores a deeper level. When once the pyloric tumour is felt there is no mistaking it. The sensation is very characteristic, a small hard rounded mass about the size of the terminal phalynx of the little finger, slightly moveable and able to be rolled under the fingers. There may be appreciated slight variations in consistence but the tumour never disappears, as some writers state. A further remarkable feature is that when once it has been detected it can always at subsequent examinations be felt with ease.

The tumour is usually felt in the region of the gall bladder, but it may be situated more towards, or in, the middle line. The position of the pylorus is governed to a great extent by the degree of distension of the stomach and when this is extreme the tumour may be found in the right iliac fossa. On occasion the pylorus is situated high up under the liver and is difficult to feel properly,

and indeed at times it may be beyond the reach of the fingers. In these circumstances the device practised in the case of our own patient, viz., holding him up by the armpits and gently shaking, may result in bringing the pylorus to a level at which it can be felt. It may be for this reason, too, that occasionally the tumour is felt best when the stomach is full as was so in our case. In other instances the pylorus is obscured by the stomach, perhaps because the latter is full, and hence, before deciding that there is no pyloric hypertrophy, the examination must be carried out sometime after a feed, or immediately after vomiting, but the requisite conditions can always be obtained by passing a stomach tube and siphoning off the stomach contents.

The presence of a pyloric tumour is pathognomonic of pyloric hypertrophy and fortunately it can be detected in practically every example of the condition; in our experience in every example. During the past 10-15 years we can honestly state that we have not observed a case of hypertrophic pyloric stenosis in which a tumour was not palpable. That we were not missing cases is surely proven by the fact that during the same period, we have not met with the example which was disclosed as an accidental finding either at operation or on the post-mortem table. It is because of this experience that we remarked in the introductory paragraph that there are few diseases which can be so definitely recognized in such a large proportion of instances. But the sign must be looked for; its search requires time and patience and it is because of a failure to observe these desiderata that we explain why Drs. Wallace and Wevill only palpated a tumour in 24.1 per cent. of their cases.

The other characteristic sign is *visible gastric peristalsis*, although it must be remembered that this has not the same pathognomonic significance as it may be present in other conditions, e.g., duodenal stenosis and atresia. Indeed, it is the combination of vomiting and constipation, in a new-born or very young infant, with visible gastric peristalsis *but no palpable tumour* which points to duodenal stenosis or atresia and not to hypertrophic pyloric stenosis. And, just as its presence is not pathognomonic of hypertrophic pyloric stenosis, its absence is not proof of the absence of the condition, for in our experience it has not been so constantly present as a palpable tumour.

Visible gastric peristalsis, like the pyloric tumour, often requires time and patience for its demonstration. One cannot expect that it will always be present. Peristalsis will only occur if there is something in the stomach which is to be passed on into the duodenum. Thus, if the viscus is empty, it may be in complete abeyance. Moreover, if there has just occurred violent vomiting, there may be for a time paresis of the stomach musculature. Hence, if one wishes to elicit this important diagnostic feature, the conditions favourable for its development must be present. These are first and foremost food in the stomach. So, if the child has recently vomited, or if he has not been fed for some time, one should give a feed (a variety of test-meal), and with all the conditions favourable for the child lying comfortably, observe the abdomen and every now and again apply gentle stimulation by tapping. If the stomach is dilated and hypertrophied, waves of peristalsis will be seen to pass in a regular sequence from left to right. It appears as a ball emerging from under the left costal margin, slowly moving across to or beyond the middle line where it disappears but only to be followed by another in its wake. When once seen the picture can never be forgotten. Indefinite or abortive waves have no significance but only the definite and regular passage of a ball-like tumour. In any infant, and particularly if emaciated, the stomach after a meal stands out and abortive attempts at peristalsis are visible.

In these cases vermicular movements in the transverse colon passing from right to left and peristalsis in coils of the small intestine are often seen but, curiously, these extraneous peristaltic waves are seldom observed in the true case of hypertrophic pyloric stenosis.

Urinary Chlorides. From the record of the above case it will have been observed that examination of the urine revealed a complete absence of chlorides. This is very strong evidence of duodenal obstruction but not necessarily proof that the obstruction is due to hypertrophy of the pylorus. Duodenal atresia also gives rise to an absence of chlorides in the urine, and duodenal atresia to a very low urinary chlorine content, but, as already emphasized, in neither of these conditions is a tumour palpable and in both of them an X-ray examination gives a characteristic picture. And, as a rule, the patient suffering from either of these latter conditions is only a few days old, whereas in the case of pyloric stenosis the patient has an age of three or more weeks. It can also be stated that just as an absence of urinary chlorides is strong evidence of pyloric stenosis abundant chlorides in the urine makes it unlikely that this is present.

X-ray examination after a barium meal was not carried out in our case because, in our opinion, it is of little help in the diagnosis. By this means all that can be revealed is a dilated stomach and delay in emptying, but both of these are so variable that in a certain proportion of cases the findings are within normal limits. Maizels⁽²⁾ records from the Infants Hospital that X-ray examination was negative in three cases and inconclusive in eleven other cases, in all of which operation subsequently revealed hypertrophy of the pylorus. If, however, one is suspicious of duodenal obstruction and, because of the absence of a pyloric tumour, hypertrophic pyloric stenosis can be eliminated, a barium meal may demonstrate that the cause of the mischief is atresia or stenosis of the duodenum. In these conditions the characteristic X-ray appearance is ballooning of the first part of the duodenum with or without the passage of the contents into the small intestine, depending on whether the obstruction is partial (stenosis) or complete (atresia).

B.—Treatment.

By way of treatment both medicine and surgery play a part. Although it has been abundantly shown that the condition can be successfully treated by medical means alone, most authorities at the present time would give surgery first place. Medicine, however, always plays a part in the therapy, even when operation is decided upon, because of its importance in the pre- and post-operative treatment. Whether or not it will be employed entirely depends on the period of the illness at which the diagnosis is made. On the average the disease or at least the active manifestations of the disease, has a maximum course of three months. As we have already remarked, the symptoms are due to spasm superimposed on the hypertrophy of the muscle and this tends to abate during the fourth month of life. Seldom or never does one see a case acutely ill after five months of age. By this time the patient has either died or recovered. Hence, if the child first comes under observation when the course of the disease is well advanced, say when he is over three months of age, it may be wise to consider temporizing by diet and gastric lavage as we know that the condition will soon of its own accord get well. If such a course is decided upon the diet, next to breast milk, which is borne best is peptonised milk, given in amounts and at the intervals normal for the age. If the child vomits soon after a feed a repeat feed, which is usually retained, should be given. The vomiting can usually be con-

trolled by lavage of the stomach which should be carried out shortly before a feed once or twice daily. Warmed sterile water is best for this purpose. Normal saline by the mouth may also be given but this must be used with caution, as in this disease there is a tendency to salt retention with the development of œdema which, by causing a great increase in weight, gives a false feeling of security. If the child is much dehydrated saline transfusions may be of assistance. One gauges the efficacy of these various measures by the weight of the child and the amount of faecal output, which latter is a measure of the degree of obstruction.

If, however, the case is seen early in the course of the disease surgical measures should be entertained and carried out as soon as the child is in a fit condition. One knows that in a child of three or four weeks, and the same applies with more force in a still younger child, with medical measures there will be many anxious weeks ahead and that the condition may become desperate at any time from the development of some intercurrent infection. One might delay operation for a day or so if the child were much depleted and by saline transfusion attempt to get him into a more suitable state for operation. Blood transfusion, unless there is definite anæmia, so fashionable at the present moment, has never seemed to us to have any advantage over subcutaneous transfusion with normal saline reinforced by glucose solution intravenously.

Perhaps the two most important features of the operation itself are, (1) the time which its performance occupies, and (2) the method of anæsthesia employed. All authorities are agreed that the least that is necessary to relieve the obstruction is best, and that for this purpose Ramstedt's technique, which simply consists in incising the muscular coat of the pylorus and the pyloric portion of the stomach and the arrest of any bleeding, is the ideal. An expert operator can complete this operation from start to finish within five to ten minutes.

So far as anæsthesia is concerned, chloroform or ether, or a mixture of these, should not be employed. The most satisfactory general anæsthetic is gas and oxygen, although perhaps better results are obtained by the use of local anæsthesia. The child in these latter circumstances is given 1/75 grain morphia hypodermically beforehand, and the area of the abdominal incision is surrounded by a rampart of skin infiltrated with novocain or percaine.

Equally important with the operation is the post-operative treatment, which aims at the child's intestine being gradually accustomed to food. Owing to the obstruction little food has entered the intestine and a full meal would be liable to set up enteritis, which is one of the most serious complications. It is not necessary to abstain from feeding for any length of time but this can be commenced soon after the operation so long as the amount is small and only gradually increased. Breast milk of course is best but if this is not available peptonized milk will be found the best substitute. One hour after the completion of the operation a beginning is made with one dram doses every hour, increasing the amount by one dram every second hour until a ½-ounce feed has been reached when the interval between the feeds is increased to 1½ hours. Then the feeds are increased to 1-oz. every 2 hours, to 1½-oz. every 2½ hours, then by two dram increases every alternate feed to 2-oz. every 3 hours, and finally to 3 or 3½-oz. every 3 hours. In this way the child receives 4-oz. during the first 12 hours, 5-oz. during the next 12 hours and 16 during the following 24 hours. During the first day or two the intake of fluid is very limited but this can be compensated for by the administration of saline per rectum or subcutaneously.

(1) Wallace, H. L. and Wevill, L.B., *Brit. Med. Jour.*, 1934, i, 1153.

(2) Maizels, M., *Arch. Dis. Child.*, 1931, vi, 293.