HYPERTROPHIC PYLORIC STENOSIS WITHOUT SYMPTOMS

BY

LEONARD FINDLAY, M.D., F.R.C.P.

B. D., a male aged four weeks, the second child of healthy parents, was admitted to the Princess Elizabeth of York Hospital for Children on May 8, 1937, because of 'peeling of the skin' of two weeks' duration and vomiting after feeds for one week. The child was reported to have appeared healthy at birth and to have had a weight of 6 lb. 1 oz. He was breast-fed and seemed to make good progress until the age of twelve days, when the mother noticed 'peeling of the skin' round the mouth, which thereafter gradually extended over the face and head and on to the trunk and limbs.

At the age of three weeks he ceased to put on weight and in consequence the question of insufficient breast milk was raised, but weighing before and after a feed showed an increase on the average of 3 oz. Nevertheless, extra-feeding was started in the form of a dried milk but this the child promptly and invariably vomited, not forcibly according to the mother's story but simply being 'brought up with wind.' The bowels continued to move regularly twice or thrice daily.

On admission to hospital the child had a weight of 6 lb. There was generalized desquamation of the skin except round the mouth, where it had started and where the skin had a pink colour, was quite smooth and delicate-looking as is seen in the floor of a recently ruptured blister. The skin of the face and body generally had a rather curious appearance. It was whitish and taut and drawn into wrinkles and suggested that the body had been covered with a thin layer of collodion. Wherever the skin had desquamated the exposed dermis was red and delicate-looking but it did not seem that any further peeling would take place, and this in fact was the course of events.

The mouth was normal except for slight enlargement of the tonsils and congestion of the pharynx. The chest was normal. On examination of the abdomen a pyloric tumour was easily palpable and gastric peristalsis visible. A barium meal showed great interference with motility of the stomach; no passage of barium from the stomach was noted within three-quarters of an hour and complete emptying had not occurred by the end of seven hours. The urinary chlorides were 0.24 gm. per cent. Wassermann reaction in mother and child was negative.

In view of the physical signs a diagnosis of hypertrophic pyloric stenosis was made in an infant the subject of dermatitis exfoliativa. The question of ichthyosis vulgaris was considered because the mother reported that in some of her relatives a dry scaly skin had been a marked feature. Two of her cousins, indeed, were said to suffer from the condition at present, although it was worse during their childhood and was responsible for their being sent home from school because of a 'dirty skin.'

For the first three days of residence in hospital the child was given breast-milk (3 oz. every four hours) when available, and if not the feed consisted of an equal quantity of peptonized milk. The skin was anointed with olive oil. During this time there had been no vomiting and the bowels had moved regularly. On the fourth day, however, but probably because

of a rather prolonged physical examination for the purpose of demonstrating the pyloric tumour and gastric peristalsis, vomiting did occur. This was more of the nature of regurgitation and was definitely not expulsive. Nevertheless, because of this development in the presence of a hypertrophied pylorus, eumydrin (1 in 10,000 solution, 2.5 c.c. half-an-hour before each feed) was instituted, but as there was no recurrence it was stopped after four days. However, within a further forty-eight hours the regurgitation of the feeds started again, taking place several times daily, but the bowels continued to move regularly and the weight steadily to rise. Within twenty-four hours of the onset of this latter spell of regurgitation the urinary chlorides, which had previously ranged between .24 and .26 gm. per cent., fell to .06 per cent., the blood chlorides were reduced to .404 gm. per cent. and the blood CO₂ registered 73 vol. per cent. The rejection of some of the feeds continued for five days; for the first three it was described by the nurse in charge as merely regurgitant in nature but on each of the fourth and fifth days the child had one expulsive vomit. At the end of this period chlorides were absent from the urine. The great bulk of the food, however, must have been retained as the motions, one or two daily, were of a normal character. Moreover, the child did not look ill and there was no evidence of depletion although he did lose weight slightly. In consequence of the return of the vomiting the eumydrin was again instituted (2.5 c.c. of a 1 in 10,000 solution before each feed), but from then until the child's dismissal from hospital, three weeks later, there was no recurrence of vomiting or regurgitation and he steadily increased in weight. During the residence the condition of the skin gradually became normal with no return of the desquamation. Details regarding the temperature, weight, feeding, medicinal treatment, vomiting, bowel action and urinary chlorides are shown in the accompanying chart (fig. 1).

Comment

It may be objected that the designation 'pyloric stenosis without symptoms' given to the above case is unwarranted, but the symptoms were so slight (real expulsive vomiting only occurred on two occasions), the bowels moved daily, and the child never appeared ill, that the symptoms could hardly have been less prominent unless they had been absent

altogether. It is almost unknown for the vomiting in pyloric stenosis to intermit for more than twenty-four hours after it has once appeared, whereas in this patient this was the case for a period of more than seventy-two hours. Yet that hypertrophic pyloric stenosis was present there cannot be the slightest doubt. During the whole period (four weeks) that the child was under observation in hospital a pyloric tumour was palpable and peristaltic waves in an apparently dilated stomach were invariably observed after a feed. Furthermore, the result of the barium meal presented the characteristic features met with in this malady, viz., much diminished gastric motility, the opaque substance not entering the duodenum until between three-quarters and one hour after ingestion and not having entirely left the stomach within seven hours. It may be stated that the child returned to hospital for a second barium meal five weeks after dismissal, i.e., at the age of thirteen weeks, and that exactly the same result was obtained.

The behaviour of the chloride metabolism in this case seems to be of particular interest and importance. On admission to hospital the urinary chlorides were normal in amount ($\cdot 24$ gm. per cent.) and they continued at this level during the first week of residence, registering $\cdot 24$ gm. per cent. on the eighth day, but on the eleventh day, i.e., the day following the onset of food regurgitation, they had fallen to $\cdot 06$ gm. per cent., and on the fifteenth day, i.e., the day following the two explosive vomits, chlorides were absent from the urine. On the succeeding day chloride excretion began again but continued low throughout the remainder of the residence in spite of the fact that no more vomiting occurred, the child was receiving an ample diet, and was steadily increasing in weight.

A fall in, or even the complete absence of, urinary chlorides is one of the most characteristic features of hypertrophic pyloric stenosis. Although generally attributed to the vomiting, it has never been possible to correlate the degree of the fall with the severity of the vomiting. This fall in chlorides is, on the other hand, one of the rarest occurrences in vomiting, and even severe and prolonged vomiting, from any other cause than pyloric stenosis. These facts have always cast doubt on the view that the whole explanation is to be found in the loss of HCl in the gastric juice. While it is admitted that there was some vomiting in this case, and hence a possible cause of chlorine depletion, it is difficult to believe that the deprivation resulting from only two explosive vomits during the course of forty-eight hours, was sufficient to account for the complete absence of chlorine from the urine.

I have seen one other case of hypertrophic pyloric stenosis (confirmed at autopsy) in which there was no history of vomiting and in which vomiting never once occurred during a residence of four weeks in hospital. Nevertheless, this particular child developed a most severe degree of alkalosis (indeed it was the presence of this state that led to a careful examination of the abdomen and the detection of a pyloric tumour and visible gastric peristalsis) as shown by slow shallow breathing and a blood CO_2 content of 102 vol. per cent. so that in all probability there was also a fall in the

blood and urinary chlorides though unfortunately neither of these estimations was carried out.

The findings in these two undoubted examples of hypertrophic pyloric stenosis surely suggest that it is some factor other than vomiting which is responsible for the fall in the urinary chlorides.

It will be recalled that Haden and Orr¹ in the first instance suggested as an explanation for the absence of chloride excretion in pyloric stenosis that the chlorine was fixed in the tissues by some toxin, and that later Drake and Tisdall² showed that the injection of histamin led to a low blood chloride content which could not be attributed to vomiting. At first the low chloride content of the tissues in pyloric stenosis demonstrated by Morris and Graham³, and the high retention of chlorine when administered intravenously⁴, seemed to negative this explanation, but the finding of Morris and Morris⁵ that the chlorine is present in organic and inorganic combination, and that the former exists in a volatile and a non-volatile form, suggested that the original hypothesis of Haden and Orr might be after all correct.

It may be that not only is the chlorine deflected to the tissues but that in addition a greater proportion is present in the volatile form and therefore not readily estimated. Was it the development of pyloric spasm in the present case, as indicated by the regurgitation, that precipitated the change in chlorine metabolism? In support of such a hypothesis the usually more rapid rise in the urinary chlorides to the normal level after the cessation of vomiting in those cases submitted to operation than in those treated medically may be instanced. It is assumed that by means of the operation all spasm must be immediately relieved.

In conclusion, attention is drawn to the variation of the symptomatology in different examples of hypertrophic pyloric stenosis, and it is suggested that it is this factor which accounts for the varying experience of different workers regarding the efficacy of eumydrin. These physicians who have had under their care a larger proportion of mild cases will have good results to record, whereas those who have had a high proportion of severe cases will have had the reverse experience. In the present case it is felt that any variety of medical treatment would have succeeded, so mild, if not indeed non-existent, were the symptoms. However, such a successful issue has not been a usual experience. Ever since Svensgaard⁶ reported her success with this line of treatment eumydrin has been given a trial in all examples of this condition coming under observation, but as yet it is only in the minority that it has proved efficacious and spared the necessity of operative interference. Nevertheless, as eumydrin often acts in a most unexpected fashion, it should always be given a trial.

REFERENCES.

1. Haden, R. L., & Orr, T. G., *J. Exper. Med.*, New York, 1923, XXXVII, 377.
2. Drake, T. G. H., & Tisdall, F. F., *J. Biol. Chem.*, Baltimore, 1926, LXVII, 91.
3. Morris, N., & Graham, S., *Arch. Dis. Childh.*, London, 1931, VI, 27.
4. *Idem*, *ibid.*, 1929, IV, 335.
5. Morris, N., & Morris, S., *Biochem. J.*, London, 1930, XXIV, 1716.
6. Svensgaard, E., *Arch. Dis. Childh.*, London, 1935, X, 443.