painful stage, where in addition to callosities on the toes and under the anterior arch, there exist subluxations and arthritis of the metatarsal phalangeal joints. It is advisable in these cases to attempt to procure relief by palliative measures, such as by special boots and supports, because operative interference is usually disappointing.

From the foregoing it will be realized that it is impossible to have perfect action of the foot unless the toes have free play and are under adequate muscular control.

Even in this short article, which was intended primarily to draw attention to the function of the toes, it was found necessary to refer to the structure and function of the arches of the feet as well. It is irrational to lay stress upon the importance of one part of an organ to the exclusion of another, yet for years, in regard to foot matters, the public, both lay and medical, has adopted this pernicious habit and has concentrated its thought almost entirely on the arches of the feet alone.

It is the business of the medical practitioner to instruct the public in matters of hygiene, and how to obtain the maximum efficiency out of their bodies. In this connection, small and insignificant though they may appear to be, the toes, nevertheless, are deserving of more respect than is usually meted out to them.

N.B.—In order to simplify the diagrams as much as possible, the flexor brevis digitorum muscle has not been shown, and the toes have been made to consist of two phalanges instead of three. But this, of course, does not alter the validity of the arguments.

NOTES ON SO-CALLED CONGENITAL HYPERTROPHIC PYLORIC STENOSIS IN INFANTS.

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THE subject of congenital hypertrophic pyloric stenosis is always considered in one lecture at least at each of our three Post-Graduate Courses which are held every year at the Infants Hospital. Further, it is often dealt with independently by the physician, the surgeon, the pathologist, and the radiologist. This is done because we have very abundant material on which to demonstrate, and because from our respective points of view we all take a great interest in the subject.

For my part, my views on this subject are not exactly orthodox, and they never have been ever since the time I made it the subject of the thesis for my M.D. degree at Oxford some thirty years ago, and was compelled to alter the title to please the examiners and fit in with prevailing opinion. The name usually given to this condition is in my opinion conceived in misunderstanding and born in violation of the very obvious underlying pathogenesis. The term *congenital* is misleading, because the hypertrophy is practically always acquired through overaction of the muscles of the antrum, not of the sphincter proper, and usually *after* birth. The term *stenosis* is wrong, because it implies that the obstruction is due to some organic narrowing of the sphincter, whereas it is actually functional and due to want of relaxation of muscles which are normally in a condition of tonus. The orifice of the sphincter is not

narrowed or stenosed, it is locked, barred and bolted. If the obstruction were organic it could never be cured, as is so often the case, by simple medical expedients.

When I suggest that so-called pyloric stenosis is never congenital, I do not mean that there are no differences in the actual size of the muscles which form the sphincter at the time of birth, nor of the antrum; such differences must necessarily exist. Moreover, I do not suggest for one moment that this sphincter always acts functionally in a uniform manner. It is obvious that neuro-muscular mechanisms of this kind must offer every degree of sensitiveness to stimulation at the time of birth. It would be contrary to everything that we know of physiology or pathology to imagine it could be otherwise, and in my opinion it is largely on the degree of sensitiveness at birth that the fate of the pyloric reflex is sealed. The pyloric reflex is a very valuable one and protects the delicate intestinal tract against excess in quantity as well as against indigestibility of the food. This sphincter, however, does not relax if the stomach contents are unsuitable for admission to the duodenum. The reflex is frequently, perhaps invariably, brought into play before birth—indeed—as the late Dr. John Thomson suggested many years ago, early vomiting in newborn infants may represent the acquisition of a very sensitive pyloric reflex acquired from ante-natal experiences and due to the swallowing and vomiting of abnormally large quantities of amniotic fluid.

Some little time ago a baby was born at Queen Charlotte's Hospital, which immediately after birth vomited about 9 oz. of amniotic fluid. It was subsequently admitted into the Infants Hospital, where it died when it was about two weeks old. At the autopsy we found the stomach enormously enlarged, with great hypertrophy of the pylorus, antrum, and sphincter. Such a condition must be extremely rare, but I take it that it only represents in exaggerated degree what usually occurs, and that in this case the enormous dilatation of the stomach and hypertrophy of the antrum were the results of swallowing abnormal quantities of amniotic fluid, followed by vomiting before birth. Male infants, with their more highly elaborated and impressionable nervous systems, are naturally more likely to develop sensitive pyloric reflexes than female infants, and this explains the greater frequency of cases of so-called pyloric stenosis in the former.

The inevitable consequence of overaction of any muscle is hypertrophy, and the earlier the date at which the overaction comes into play, the greater will be the hypertrophy. Hypertrophy of the antrum is, however, comparatively rare in newborn infants, although on palpation so-called pyloric tumours can often be felt through the abdominal wall. This is due much more to the fact that the antrum is in spasm, than that there is any appreciable enlargement of the antrum itself. Some of the severest cases of vomiting of the projectile type, with all the usual concomitants of so-called pyloric stenosis, prove on operation or autopsy to have very little enlargement of the sphincter muscles, or indeed of the antrum, whereas, post mortem, some of the largest tumours that I have seen at autopsy have been in infants who have had little or no vomiting during life.

The pyloric sphincter does not obstruct the outflow of the stomach contents owing to organic stenosis, it stops it because the opening is actually occluded or shut, and refuses to relax. The normal resting condition of all sphincters is one of tonus, in other words, they are closed. When relaxation occurs, the opening represents the active phase. It is therefore a contradiction in terms to speak of any

sphincter being functionally "stenosed," it is under normal conditions completely shut. It is either closed or it is open, it is never in any half-way position, although it is true that it can be in a condition of exaggerated tonus or spasm. The whole question of the innervation of the sphincter mechanisms is very obscure, but it is agreed on all sides that when the sphincter is at rest it is closed, and when it is in its active phase it is open, but the exact manner in which the nervous system or the pneumogastric nerve controls the action of opening is still in doubt. In the case of the pylorus, it appears that the peristaltic wave, controlled and initiated by the vagus, passes along the pyloric end of the stomach and finally terminates in relaxation of the sphincter proper. It commences in the muscles of the antrum and ends in an active phase of relaxation of the pyloric sphincter. The pyloric sphincter itself is a comparatively small structure, and only constitutes an insignificant part of what is commonly known as the pyloric tumour in cases of so-called hypertrophic pyloric stenosis. The pyloric reflex proper is therefore one of *relaxation* and not of closure, and cases in which the contents of the stomach are retained owing to want of opening of the sphincter are due to the establishment of what one may call a perverted pyloric reflex—that is to say, the pylorus acquires the habit of remaining in a condition of tonus and refusing to relax in the ordinary way as part of the peristaltic wave that starts in the antrum. This habit represents the exaggeration of a natural protective reflex, and is due in almost every case to improper feeding soon after birth, and by improper feeding I mean errors as regards quantity as well as regards quality in the food. This occurs as often in breast-fed as in bottle-fed infants, and is due, of course, to excess in quantity.

At Queen Charlotte's Hospital, since I substituted, about three years ago, **three hourly instead of four-hourly feeds, the checking of the intake of breast milk by means of the "test" feed, and the substitution of peptonized for ordinary cow's milk, the number of cases of vomiting have been reduced almost to zero.** Indeed, I have heard of only one doubtful case of hypertrophic pyloric stenosis which required operation, although previously they had been common enough. On the other hand, there have been quite a number of cases of *projectile vomiting* due to *sepsis* and other infections, cases which were ordinarily diagnosed as ones of so-called hypertrophic pyloric stenosis, but which prove on more careful examination to be due to some infective condition, the vomiting passing off with the subsidence of the infection. I practically never meet with cases of vomiting in young babies due to primary gastric trouble if adequate care is taken during the first ten days of life to prevent excess in quantity of food, or any inappropriateness as regards quality in those artificially fed—that is to say, if the pyloric reflex is educated and developed on the right lines. Although it is not always easy to do so, I find that in the vast majority of cases which are diagnosed as hypertrophic pyloric stenosis, the symptoms can be overcome by *ordinary medical means*, namely, by the exhibition of sedative drugs, such as bromide, opium, chloral, or belladonna, combined with re-education of the sphincter. The only cases that require operation are those in which the sphincter so obstinately refuses to relax that even normal glucose saline cannot be retained in the stomach. On an average I find that about one case in ten of those which are sent up to the Infants Hospital, supposed to be suffering from so-called hypertrophic pyloric stenosis, requires operation, the rest of them respond readily enough to sedatives and re-educational expedients. It is quite

easy to settle whether operation is necessary or not. If an infant can *retain glucose saline* in small *quantities without vomiting*, operation is not necessary, for by very easy stages the re-education of the pyloric reflex can usually be effected. Personally, I attach very little importance to the size of the tumour as a guide to whether operation is necessary or not. I attach even less importance to projectile vomiting, presence of visible peristalsis, or obstinate constipation. These are merely the natural consequences of persistent closure of the pylorus. I believe more in the evidence of X-rays after an opaque meal, but it is perfectly possible, and indeed it often happens, that all the symptoms supposed to be pathognomonic of hypertrophic pyloric stenosis are present in infants who have been perfectly well up to a point, and then suddenly succumb to some sudden infection, such as influenza. I have had several instances of this kind in which all the symptoms quickly subsided as the infection passed off. In other cases the projectile vomiting may occur simultaneously with an equally rapid emptying of the stomach through the pylorus. I have had several instances of this kind also in which, according to most of the clinical evidences the case was one of pyloric stenosis, and yet the X-ray has revealed that the pylorus was not only open but allowed an unduly rapid emptying into the duodenum, this being due to violent and inco-ordinated peristalsis.

Persistent vomiting, especially of the projectile type, is nearly always accompanied by loss of acid to the system. Vomiting that occurs immediately after the intake of food—usually of the non-projectile type—is not nearly so likely to cause loss of acid and lead to a blood alkalosis, for the reason that the stomach has not in such cases had time to secrete much acid. In those cases in which the pylorus does not relax, food is retained in the stomach, perhaps for many hours, becoming gradually more and more acid, so that when projectile vomiting ultimately takes place a large amount of acid is lost to the body. The usual treatment adopted in such cases is to wash out the stomach with bicarbonate of soda. This is obviously an irrational proceeding. *The treatment is to restore acid to the system* and this can best be done by subcutaneous injections of fluid containing sodium chloride, or better still, by intravenous transfusions. We feel at the Infants Hospital that we have made a great advance by the substitution of large intravenous injections of this kind for the usual plan of giving small subcutaneous injections which are usually absorbed slowly. In all cases of so-called pyloric stenosis one of the chief dangers to combat is that of dehydration, and here again we find that intravenous transfusions are of far greater value than mere subcutaneous injections.

To sum up the gist of these few notes on hypertrophic pyloric stenosis—I would repeat:

Firstly, that this condition is always due to want of relaxation of the sphincter, and not to organic stenosis.

Secondly, that the treatment is one of re-education.

Thirdly, that a condition of alkalosis usually accompanies this condition, and is best treated by intravenous transfusions of a saline containing ordinary sodium chloride.

Fourthly, that the restoration of fluid to the circulation is an important means of preventing the collapse which often accompanies both operative and non-operative methods of treatment.