PYLORIC HYPERTROPHY IN NEW-BORN INFANTS

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During the last ten or fifteen years, in which hypertrophic pyloric stenosis and pylorospasm in infants have been the objects of absorbing interest, the prevailing tendency has been to draw well-defined clinical pictures of these two affections, and to distinguish them sharply from normal conditions, as well as from each other. With regard to the latter distinction, the chief object has been to define the operative indication; we have now come to understand that this end is not always attainable, and that difficult borderline cases form a large proportion of the total. As a matter of fact, the absence of any sharp dividing line was more than suspected ten years ago by Ibrahim,¹ and has recently been reaffirmed by Holt.² Careful study of the best contributions to the subject conveys the impression that this attitude of uncertainty pervades most of the literature; only the surgeons, possessed of a more limited view, seem to maintain rather positive opinions, where the pediatricists are vexed with doubts.

With the aid of certain postmortem material that has recently been at my disposal, I shall endeavor to show that even the first proposition, namely, the diagnosis between pyloric hypertrophy and presumably normal conditions, is beset with difficulties, and that, as a consequence, the distinction between spasm and true organic obstruction is likely to remain, in many cases, a matter of great uncertainty unless new diagnostic methods are brought to our aid.

The following new-born infants were examined:

CASE 1 (No. 15887).—Baby S., premature, died in the third week, of atelectasis. The pylorus forms an obviously thickened mass, its walls being about 4 mm. in diameter, but the lumen appears to be fully normal. The pylorus as a whole projects into the duodenal canal very much in the manner of a cervix uteri, and as described in cases of true pyloric hypertrophy.

CASE 2 (No. 15894).—Baby S., died on the fifth day, of hemophilia of unknown origin. The same condition of the pylorus is found, the lumen of the organ measures 6 to 7 mm. in diameter.

CASE 3 (No. 16149).—Baby S., died on the fifth day of multiple hemorrhages. The pylorus presents the same thickening of the walls, and projection into the duodenum, with a lumen of 8 to 9 mm.

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1. Ibrahim: *Die angeborene Pylorusstenose im Säuglingsalter*, 1905.

2. Holt: *Medical Versus Surgical Treatment of Pyloric Stenosis in Infancy*, Jour. Am. Med. Assn., June 17, 1914, p. 2014.

CASE 4 (No. 16148).—Baby C., died in the third week, of congenital atresia of the bile ducts. Pylorus exactly as noted in Case 3.

By way of comparison, I give two autopsies of older infants.

CASE 5 (No. 15818).—Frances K., died at 3 months of meningitis. The pylorus is little, if at all, thicker than the duodenum, and does not project into it; the pyloric wall is of about normal thickness.

CASE 6 (No. 16364).—Frank E. died in the fourth month, of an exceptionally severe purulent meningitis. There is in this case a slight thickening of the pyloric wall, but less marked than in any one of the four new-born infants.

To these I may add a previously³ reported control case:

CASE 7.—Died in the eighth week, of gastro-enteritis. Pyloric wall is not at all thickened, measuring 2.5 mm. in diameter; the viscus does not project into the duodenum.

SUMMARY

In summarizing the above cases, I wish, in the first place, to call attention to the substantial uniformity of conditions in the first four cases, which were consecutive, not selected, and in which the pyloric findings were accidental, and not sought for. The conditions in any one of these four infants might have led to pyloric spasm, which, according to Ibrahim,¹ is a probable causative factor in the development of true organic obstruction.

In the middle period of infancy, we have plainly a different state of affairs in the normal subject. There is usually no thickening, or it is, at any rate, very slight, as in Case 6. As far as can be determined from so small a material, a retrogressive change normally takes place between the third week and the third month, in the course of which the very perceptible thickening of the pylorus gives way to a condition in which the borders of that organ are macroscopically almost undistinguishable.

The first group of cases presented no symptoms of spasm or obstruction. The latter was plainly not present, and the former does not occur, even in predisposed cases, until the third week of life. The stated findings do not, therefore, conflict with our clinical observations. The question of a postmortem contracture, long since thrashed out, is here refuted by the conditions noted in the second group; also in a few of these cases by the performance of the autopsy after rigor mortis had disappeared. As in obstructive hypertrophy, so in these cases, the total bulk of the viscus was greatly increased; in postmortem spasm the increased thickness would be compensated by an equivalent shortening of the organ.

The thickness of the pyloric wall in the normal infant of two months is 2 to 2.5 mm., in the new-born it is greater, 3 to 4 mm., in established hypertrophic stenosis fully 5 mm.; evidently the conditions in the new-

3. Wachenheim, F. L.: *Am. Jour. Med. Sc.*, April, 1905.

born are intermediate, and it depends on circumstances whether the normal retrogressive change takes place, or the thickening increases to such a degree as to cause obstruction. Possibly the development of the latter condition is due to hyperacidity and consequent pylorospasm, leading to hypertrophy, as is believed by some authors, but this sequence is so far purely conjectural. At any rate, the average new-born infant bears within itself the latent tendency to develop pyloric hypertrophy, and it depends on factors hitherto uncontrollable whether the stated condition develops or the pyloric wall thins out in the normal course.

Owing to the small number of cases in my series it would be presumptuous to state that the pyloric thickening in the new-born is universal; its occurrence in four consecutive cases seems to show that it is present at least in the majority, but further research along these lines is imperative, and may lead to valuable results if my observation is confirmed by other investigators. Further research may also lead to an explanation why some infants never get beyond the clinical picture of pylorospasm, whereas a considerable number go on to true organic obstruction. The working out of prophylactic measures may likewise result from further studies; in this respect the present dietetic treatment of pylorospasm possibly points the way.

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