

Pyloric Stenosis of Infancy (PS) - Lessons from the Past

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It is a very wise saying-listen to the patient-he is telling you the diagnosis. Clearly infants cannot speak. With PS however the symptoms and signs provide a rich resource of clues which should point to the cause of the pyloric tumour. Over the last hundred years since the condition became recognised, authors have repeatedly confirmed the detailed clinical features. The emergence of pyloromyotomy as a simple and safe cure may have been the reason for their apparent lack of interest in the cause.

In recent years tentative hypotheses about the cause have emerged. Astonishingly none of them has addressed the fundamental obligation to explain the clinical features. Neither the nitric oxide theory; the helicobacter theory; the allegedly abnormal accumulation of growth factors in the sphincter muscle nor the genetic analyses. None of them has done so-not one! Indeed, the monozygotic concordance is only between 0.25 and 0.4 [1]. All these theories have predictably fallen by the wayside.

From this perspective, how do we view the current and innovative theory that PS occurs because of the inheritance of a parietal cell mass at the upper end of normal [2]. When the fasting pH of the stomach is measured there is little difference between PS and normal matched babies. By contrast when the basal acid secretion is measured there is a huge increase in acid secretion [3]. Acid entering the duodenum is the most potent way of causing the pyloric sphincter to contract [4,5] and it is accepted that repeated sphincter contraction causes work hypertrophy of the sphincter. Repeated contraction is the reason why growth factors appear in the sphincter- not the other way round.

What of the need for this theory to explain the clinical features?

Inherited constitutional hyperacidity explains the familial occurrence. It explains the male predominance (males secrete more acid than females) [6]. It explains the presentation at 4 weeks since peak acidities in normal development occur then-probably due to a maturing negative feedback between gastrin and antral acidity [7,8]. It explains the natural resolution if the neonate survives beyond around 8 weeks-the acid being now under negative feedback control and the canal now widened with age. So far in my own researches I have not encountered a single feature of PS which is not explained by the Hyperacidity theory.

In 1903 Dr. Freund, an early medical observer of the extraordinary condition of PS declared that an excess of acid was the cause [9]. This theory was quietly forgotten over the years .and the obvious acidity of the gastric contents was wrongly attributed to collection of acid behind a closed pylorus. There is indeed nothing new under the sun. We do as Newton says simply stand on the shoulders of those who have gone before. So, let it be with the cause of pyloric stenosis of infancy.

A more comprehensive account of the hyperacidity theory may be found in the pages of The consequence and cause of pyloric stenosis of infancy. Dr. Fred. Vanderbom M.A.(Hons) and I.M.Rogers FRCS.

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