

fasting gastrin levels in our patients (125 ng/l) were identical with the control (128 ng/l). The gastrin responses to a Sustagen meal (providing 1.7 g/kg protein) were likewise similar. The finding of a somewhat flattened postprandial gastrin curve in Hambourg's infants after pyloromyotomy is most likely explained by residual antral stasis. As these authors properly point out, radiological evidence of impaired gastric emptying frequently persists in the immediate postoperative period in these patients. We would agree with the conclusions of these authors that there is no evidence to support the hypothesis that patients with pyloric stenosis have an abnormal gastrin mechanism.

References

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Dr Hambourg and co-workers comment:

We agree with the conclusions of Dr Rodgers and Dr Moazam although the results of their recent study are not comparable with ours (Moazam *et al.*, 1978). Regarding the curve showing changes in serum gastrin levels after ingestion of a high protein meal (Sustagen, providing 1.7 g/kg protein) these authors obtained a slope that increased more slowly from baseline values. Moreover the peak gastrin values were noted in control infants at 45 min, and in patients with hypertrophic pyloric stenosis at 30 min preoperatively and 45 min postoperatively. Analysis of the decreasing slope was not described. Our study was performed by administering a casein hydrolysate (Amirige, providing 0.7 g/kg protein) containing free amino-acids. In 11 control infants and 10 patients with pyloric stenosis the mean peak gastrin level was noted within the first 10

minutes after the feeding, showing a maximum value at 10 min and a return to basal gastrin levels after 60 and 90 min. The differences may be explained by differences in the two methods. Certainly high protein preparations are capable of producing a gastrin release. However hydrolysed proteins, especially free amino-acids, were found to be more potent gastrin stimulants (Elwin, 1974), thus increasing the accuracy of the test. Our results with the gastrin secretion test show the importance of recording the serum gastrin levels within the first 10 min after feeding and continuing to record it for at least 60 min. In the study by Moazam *et al.* (1978), 11 infants with pyloric stenosis were evaluated with postprandial serum gastrin levels before pyloromyotomy and 2 to 10 weeks after surgery. Interestingly, a gastrin secretion test was performed on patients with untreated hypertrophy of the pylorus. In the postoperative studies a similar rise in serum gastrin concentration was noted in response to protein ingestion, however no significant differences were observed from preoperative studies.

In our patients, increased fasting gastrin levels were noted after pyloromyotomy and were still present 2 weeks after the operation. The postoperative hypergastrinaemia may be explained by a residual antral stasis on the 7th and 15th days after surgical pyloromyotomy, represented by a postprandial gastrin curve with a 'levelling-off' effect between 30 and 60 min, as Dr Rodgers and Dr Moazam clearly point out. Nevertheless, in these two studies test meals did not produce an exaggerated gastrin response, and the results were of the same order as those obtained by the same trial control stimulation. Therefore we agree that there is no evidence that patients with pyloric stenosis have an abnormal gastrin mechanism.

References

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