The Turkish Journal of Pediatrics 28:115 - 121, 1986

BASAL AND POSTPRANDIAL GASTRIN LEVELS IN THE EARLY NEONATAL PERIOD*

*Ahmet Aydın MD***, Makbule Haktan MD***, Erol Çakır PhD*****

Key words: gastric acid secretion, gastrin receptor, newborn, postprandial serum gastrin level

It has been shown by several authors that gastric acid secretion during the first five and six hours of life is very low. It has also been shown that it reaches a maximum level in the following 24 hours, and gradually becomes achlorhydric by the end of the first month[1-6]. Many authors have reported that the level of gastrin, the chief inducer of gastric acid secretion, is higher in the neonate than in the adult[7-19]. Animal studies have proved that gastrin probably plays a role in the structural and functional development of the gastrointestinal system[20,21].

Although the physiological stimulus of protein-containing foods is the most important factor in the secretion of gastrin in adults, the results of experiments on the activity of G-cells in the neonate in response to such stimulus are controversial[10,11,15,17,19].

Our goal was to study whether gastrin plays a role in gastric acid secretion in the early neonatal period by determining basal serum gastrin levels and to see the response of the G-cells.

Material and Methods

The samples for this study were drawn from babies born to healthy mothers in the Obstetrical Clinic of Cerrahpaşa Medical Faculty of Istanbul University between April and July 1982. All subjects were born spontaneously via the vaginal route, had an Apgar score of 9 or 10, and a gestational age of 39.0 ± 0.9 weeks. Our study group included 90 babies, 46 girls and 44 boys. Their mean birthweight was 3335 ± 564 grams and their mean birth height was 50.3 ± 1.0 cm. Babies were matched in six equal groups. Venous blood samples were obtained only once from each baby. They were taken from the umbilical vein during delivery and from the antecubital vein in the postnatal period.

---

* From the Department of Pediatrics, Istanbul University Cerrahpaşa Faculty of Medicine, Istanbul.

** Pediatrician, Eskişehir State Hospital.

*** Associate Professor of Pediatrics, Istanbul University Cerrahpaşa Faculty of Medicine.

**** Doctor of Chemistry, Department of Pediatrics, Istanbul University Cerrahpaşa Faculty of Medicine.

Groups A, B, C and D were each composed of 15 babies aged 0 hours, 3-7 hours, 2 days and 7 days respectively. Basal serum gastrin levels in these groups were determined. Postprandial serum gastrin levels were measured in two other groups of 15 babies each, one group aged 3-7 hours, the other 7 days. The blood samples were taken 30 minutes after a meal in the latter groups. All babies were fed 10-20 ml of formula (SMA®) at four-hour intervals, sufficient for one-week-old infants. Blood samples approximately two ml each were drawn and centrifuged within an hour, at room temperature. Hemolyzed samples were excluded from the study. All serum samples were kept at −29 °C and were treated the same day.

We used a Gastrin Radioimmunoassay Kit (Biomedica) for determining serum gastrin levels. Each sample was measured twice and the results were expressed in picograms/ml. We used Student's *t* test in analyzing our data.

Results

Basal serum gastrin levels were found to be approximately similar in 0-hour, 3-7-hour, 2-day- and 7-day-old babies; however, they were quite high when compared to adult basal serum gastrin levels (Figure 1, Tablo I).

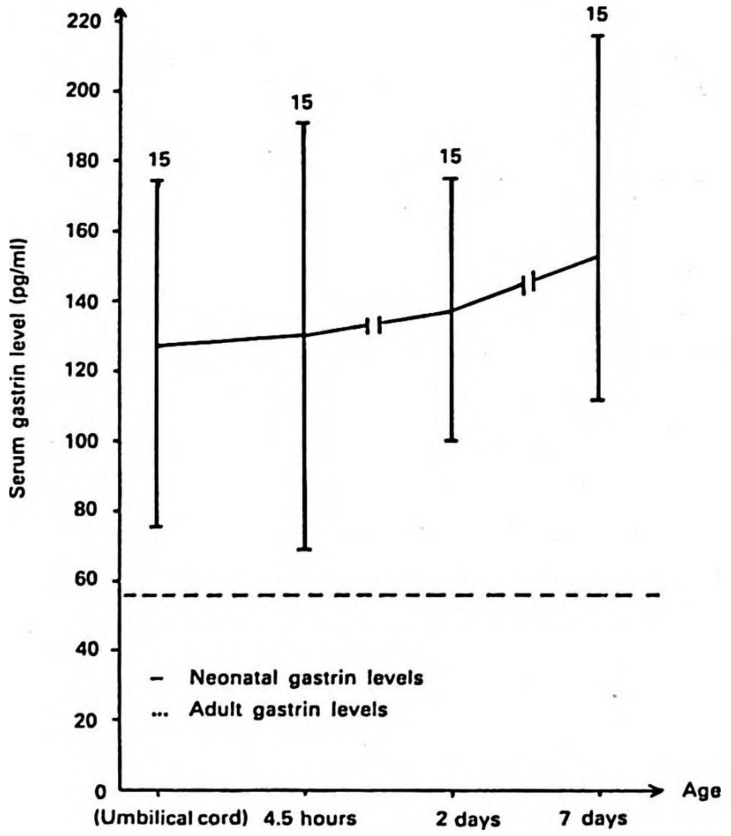

Figure 1

Basal serum gastrin levels in the early neonatal period.

TABLE I

Comparison of Fasting Levels of Serum Gastrin in Early Neonates and Adults

Group	Age	No. of cases	Gastrin level	Student's *t* test
A	0 hour	15	127.7 ± 49.3	A – B $p > 0.05$ A – C $p > 0.05$ A – D $p > 0.05$ A – E $p < 0.001$
B	3 - 7 hours	15	129.0 ± 56.9	B – A $p > 0.05$ B – C $p > 0.05$ B – D $p > 0.05$ B – E $p < 0.001$
C	2 days	15	138.8 ± 36.2	C – A $p > 0.05$ C – B $p > 0.05$ C – D $p > 0.05$ C – E $p < 0.001$
D	7 days	15	167.6 ± 55.1	D – A $p > 0.05$ D – B $p > 0.05$ D – C $p > 0.05$ D – E $p < 0.001$
E	Adult	25	56.6 ± 5.6	E – A $p < 0.001$ E – B $p < 0.001$ E – C $p < 0.001$ E – D $p < 0.001$

We observed no significant difference between the basal serum gastrin levels and the postprandial serum gastrin levels in the 3-7-hour- and 7-day-old neonates (Table II).

Discussion

Even though our knowledge of the role of gastrin on the secretion of hydrochloric acid and its trophic effects on the gastrointestinal tract has increased recently, we still know little about its role in the neonate. In 1974, it was reported by Rodgers et al[7] that the gastrin level determined from the umbilical vein of neonates was higher than in adults. This finding is confirmed in the literature[8-9] and by our study (Figure 1).

TABLE II

Comparison of Basal and Postprandial Serum Gastrin Levels of 3-7-hour- and 7-day-old Infants

	3-7-hour-old infants (n = 15)	7-day-old infants (n = 15)
Gastrin level before feeding (pg/ml)	129.0 ± 56.9	167.6 ± 55.1
Gastrin level 30 min after feeding (pg/ml)	136.1 ± 34.2	161.6 ± 52.7
Student's *t* test	$p > 0.05$	$p > 0.05$

Early experiments had suggested that high neonatal serum gastrin levels would depend on the maternal serum gastrin[7]. Although the levels of umbilical venous and arterial gastrin are similar, the high levels of umbilical gastrin when compared to maternal levels during delivery[7,10], and the low concentrations in placentas, as well as the fact that there is a transplacental blockage to gastrin[5] when injected in pregnant mice[11] and dogs[19] supports the hypothesis that the maternal gastrin level has no effect on the high serum gastrin levels in the fetus. Besides, although gastrin has a short half-life, it remains at a high level throughout the neonatal period[18-19]. Gastrin also has been found in the G-cells of the antral mucosa beginning from the nineteenth week of fetal life[22]. These findings strongly suggest that gastrin is actively secreted by newborns.

Previously reported studies into the cause of neonatal hypergastrinemia have been unable to resolve this question. Although increased vagal activity and increased discharge of catecholamines during delivery, together with renal insufficiency of the neonate, have been suggested as probable causes of the high neonatal levels of gastrin, still they are far from fully clarifying this finding[5,16].

There remains a logical explanation, which is the idiopathic hyperactivity of G-cells. According to this theory, G-cells discharge all of their gastrin content into the circulation under the effect of a high pH of the gastric juices[5,16].

In spite of the sufficient parietal cell mass and the hypergastrinemia, low gastric acid secretion is most probably due to the lack of gastrin receptors in the neonate[23]. Takeuchi et al[21] showed the absence of gastrin receptors in newborn rats, as well as their appearance in the third week of life, and thereafter the increase in hydrochloric acid secretion and antral gastrin concentrations, achieve-

ment of normal serum gastrin levels and the beginning of the trophic effects of gastrin on the gastrointestinal tract.

Results of experiments on the activity of G-cells in the presence of the physiological stimulus of protein-containing foods are controversial[10,11,15,17,19]. We found no significant difference between basal and postprandial serum gastrin levels measured in 3-7-hour-old and 7-day-old babies (Table II). In 1975 Sann et al[10] reported an increase in postprandial serum gastrin levels in three infants whose exact ages were not identified. Von Berges et al[11] in 1976 showed a significant increase in postprandial serum gastrin levels in their study on ten full-term healthy babies whose mean age was 4.5 hours. It should be noted, however, that if one of their cases, the one in which the postprandial gastrin level increased fourfold, is excluded from the series, the results become insignificant.

Aynsley-Green et al[19] in 1979, have shown in 18 asphyctic full-term babies, half of whom were fed with carbohydrates and half with proteins, an increase in serum gastrin after meals. As it has been clearly established that carbohydrates have no effect on the serum concentration of gastrin[24], the primary illness of these babies may have been responsible for the increase mentioned in their study. Factors such as the small number of cases involved in the studies, lack of attention to the age of the patients, the inclusion of ill babies, and faulty interpretation of statistical data cast doubt on the results of the above mentioned experiments, which contrast with our findings.

Rogers et al[15] in 1978 found no postprandial hypergastrinemia in 26 full-term healthy babies either in the first hours of life or on the second day. In 1980 Lucas et al[17] reported no increase in postprandial serum gastrin levels during the first six days in healthy premature babies. The results of these two studies are in accord with ours.

In our opinion, the reason for the inability of G-cells to respond to protein stimulus in the early neonatal period is the low antral concentrations of gastrin, due to the continuous and uncontrolled G-cell hypersecretion. We should mention that later in life, as gastrin receptors develop, the gastric acid secretion increases and with it the inhibitory effect of gastric acid on the G-cells. This would make G-cells capable of responding to protein-containing foods. Since increase in postprandiaal gastrin occurs in the third month of life[25], the appearance of gastrin receptors most probably coincides with this increase.

Summary

We investigated the basal and postprandial serum levels of gastrin in 60 full-term healthy infants in the early neonatal period. Basal values in cord blood and in the peripheral veins of these babies before the first feeding (3-7 hours), and on the second and seventh days were similar to each other and higher than the basal

values of adults. Despite the sufficient parietal cell mass and high serum gastrin levels, the hyposecretion of gastric acid in newborns suggests that the parietal cells of newborn infants lack gastrin receptors. Pre- and postprandial serum gastrin values on the first and seventh day did not demonstrate a significant difference. These results show that serum gastrin levels are maximal under basal conditions in the early neonatal period and also suggest that antral gastrin concentrations are extremely low as a result of maximal G-cell hypersecretions.

REFERENCES

1. Hess AF. The gastric acid secretion of infants at birth. *Am J Dis Child* 6:264, 1913.
2. Miller RA. Observations on gastric acidity during first month of life. *Arch Dis Child* 16:22, 1941.
3. Ebers DW, Smith DI, Gibbs G. Gastric acidity on the first day of life. *Pediatrics* 18:800, 1956.
4. Ahn CJ. Kim YJ. Acidity and volume of gastric contents in the first week of life. *J Korean Med Assoc* 6:72, 1963.
5. Avery GB, Randolph JG, Weaver T. Gastric acidity in the first day of life. *Pediatrics* 37:1004, 1966.
6. Euler AR, Byrne WJ, Cousins LM, et al. Increased serum gastrin concentrations and gastric acid hyposecretion in the immediate newborn period. *Gastroenterology* 72:1271, 1977.
7. Rogers IM, Davidson DC, Lawrence J, et al. Neonatal secretion of gastrin and glucagon. *Arch Dis Child* 49:796, 1974.
8. Delle Fave GF, Assuma M, Sopranzi N, et al. Neonatal hypergastrinemia. *Gastroenterol* 7:36, 1975.
9. Torsoli A, Delle Fave GF, Melchiorri P, et al. Proceedings: neonatal hypergastrinaemia in man. *Gut* 14:834, 1975.
10. San L, Chayvialle JAP, Bremond A, Lambert R. Serum gastrin level in early childhood. *Arch Dis Child* 50:782, 1975.
11. von Berger L. Henrichs I, Raptis S, et al. Gastrin concentration in plasma of the neonate at birth and after the first feeding. *Pediatrics* 58:264, 1976.
12. Euler AR, Byrne WJ, Cousins LM, et al. Increased serum gastrin concentrations and gastric acid hyposecretion in the immediate newborn period. *Gastroenterology* 72:1271, 1977.
13. Aynsley-Green A, Bloom SR, Williamson DH, Turner RC. Endocrine and metabolic response in the human newborn to first feed of breast milk. *Arch Dis Child* 52:291, 1977.
14. Janik JS, Akbar AM, Burrington JD, et al. Serum gastrin levels in infants and children. *Pediatrics* 60:60, 1977.
15. Rodgers BM, Dix PM, Talbert JL, MacGuigan JE. Fasting and postprandial serum gastrin in normal human neonates. *J Pediatr Surg* 13:13, 1978.
16. Euler AR, Ament ME, Walsh JH. Human newborn hypergastrinemia: an investigation of prenatal and perinatal factors and their effects on gastrin. *Pediatr Res* 12:652, 1978.
17. Lucas A, Bloom SR, Aynsley-Green A. Development of gut hormone responses to feeding in neonates. *Arch Dis Child* 55:678, 1980.
18. Lucas A, Adrian TE, Christofides N, et al. Plasma motilin, gastrin, and enteroglucagon and feeding in the human newborn. *Arch Dis Child* 55:673, 1980.
19. Aynsley-Green A, Lucas A, Bloom SR. The effects of feeds of differing composition on entero insular hormone secretion in the first hours of life in human neonates. *Acta Pediatr Scand* 68:265, 1979.

20. Johnson LR, Lichtenberger LM, Copeland, EM et al. Action of Gastrin on gastrointestinal structure and function. *Gastroenterology* 68:1184, 1975.

21. Takeuchi K, Peitsch F, Johnson LR. Mucosal gastrin receptor V. Development in newborn rats. *Am J Physiol* 240:6163, 1981.

22. Larsson IM, Davidson DC, Lawrence J, et al. Fluorescence histochemistry of the gastrin cell in fetal and adult man. *Gastroenterol* 68:1152, 1975.

23. Polacek MA, Ellison EH. Gastric acid secretion and parietal cell mass in the stomach of a newborn infant. *Am J Surg* 111:777, 1966.

24. Walsh JH, Grossman MI. Gastrin (first of two parts). *New Engl J Med* 292:1324, 1975.

25. Collina A, Lazzari R, Golinucci G, et al. Le curve gastrinemiche dopo pasto in varie eta pediatriche. *Minerva Pediatr* 31:625, 1979.