

## Conservative Management of Infantile Pyloric Stenosis by Nasoduodenal Feeding

Y. Yamashiro, H. Mayama, K. Yamamoto, M. Sato, and G. Nawate

Department of Paediatrics, Juntendo University, School of Medicine, and Department of Paediatrics, Hino City General Hospital, Tokyo, Japan

**Abstract.** Fifty cases of infantile pyloric stenosis were treated conservatively by transpyloric nasoduodenal tube feeding.

The mean age on admission was 38.2 days. Transpyloric intubation was carried out in all patients and 45 (90%) with infantile pyloric stenosis were cured by nasoduodenal feeding. Among 5 (10%) requiring surgical intervention, abandonment of nasoduodenal feeding was the cause in only 3, and the parents chose an operation in the remaining 2 a few days after nasoduodenal feeding had been started. In non-surgically cured cases (45), mean body weights on admission and at discharge were 3,750 g and 5,177 g respectively; the duration of nasoduodenal feeding was 8 to 37 days (mean 17.2 days), mean weight gain during nasoduodenal feeding was 42.7 g/day and mean hospital stay was 39.7 days (38.0 days in 43 cases without any associated disorder).

This experience suggests that nasoduodenal feeding in this report is a more effective treatment for infantile pyloric stenosis than any traditional medical treatment and it could be the preferred management in the small group of patients for whom an operation could carry a high risk, or whose parents refuse operation.

**Key words:** Infantile pyloric stenosis – Nasoduodenal tube feeding

### Introduction

Since Ramstedt's operation for infantile pyloric stenosis (IPS) was developed most patients with IPS have been treated surgically and there is no doubt that the results of this form of treatment are excellent. Paedia-

tricians sometimes find, however, that some patients with IPS can be cured by medical treatment. Indeed, there are some reports which indicate remarkable success when patients with IPS were treated medically [1, 2, 7], especially in less severe cases [6, 11], but the problems of the lower success rate of medical as compared with surgical treatment, with prolonged undernutrition and other factors, have still remained.

The present authors thought that if there were a more efficient and reliable treatment for IPS than traditional conservative methods it would bring benefit to the patients who carry a high operative risk, or whose parents requested non-surgical treatment.

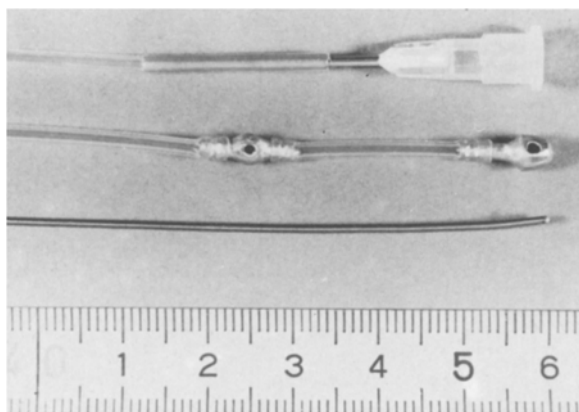
We have employed a modification of nasoduodenal (transpyloric) feeding for the non-surgical treatment of IPS. Our experience using this method in 50 patients is detailed in this paper.

### Patients and Methods

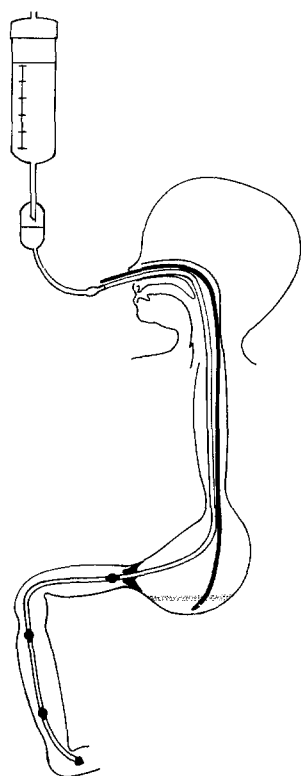
#### Patients

To establish whether this method could be effective, not only for mild cases but also for moderately severe and severe cases, nasoduodenal feeding (NDF) was randomly allocated for all patients with IPS. However, in order to quantitate the degree of pyloric obstruction before treatment was instituted, all patients in this study were diagnosed not only by clinical signs and symptoms but also by roentgenographical investigations. A patient whose pyloric canal did not open for more than 30 min after ingestion of gastrografen, and in whom only a long "string sign" was demonstrable, was classified as having a "severe" degree of obstruction; a pyloric canal opening between 16 to 29 min after gastrografen was classified as "moderate" and a pyloric canal opening within 15 min was classified as "mild." Of the total of 50 cases, 10 were classified as "severe," 31 as "moderate" and 9 as "mild." Forty-five patients (90%) were male. The mean age on admission was 38.2 days. Immediately after admission, every patient received intravenous fluid therapy for 1 to 2 days for correction of dehydration and biochemical disturbances. Informed consent was obtained for all the patients.

*Offprint requests to:* Y. Yamashiro, Department of Paediatrics, Juntendo University, School of Medicine, 2-1-1 Hongo, Bunkyo-ku, Tokyo, Japan



**Fig. 1.** Nasoduodenal silicone tube with gold plugs. Top: proximal end of tube, 1.0 mm internal diameter into which an 18 gauge needle is inserted as a connector. Centre: distal end of tube connected to a hollow gold plug, 4.5 mm maximum external diameter. The tube also has 3 to 4 additional oval shaped hollow "connecting" plugs (one is shown). Bottom: a cardiac guidewire



**Fig. 2.** Diagrammatic representation of nasoduodenal tube feeding, showing also the nasogastric tube

#### *Duodenal Tube*

Figure 1 shows the duodenal tube with a gold plug which was remodelled from that described by Rhea et al. [10]. Silicon rubber tubing (2.0 mm external diameter and 1.0 mm internal diameter) was used. The tube was connected to the distal end of the smooth, bullet-shaped head of the hollow plug which was made of 16–18 carat gold. The tube also had 3 or 4 additional oval shaped hollow "connecting" plugs which acted as weights

to prevent displacement of the tube from the duodenum into the stomach. Each plug was tightly fixed by surgical thread.

#### *Methods*

Gastric contents were aspirated and 5–10 ml of gastrografen injected by syringe through a nasogastric tube (No. 5 French) which was then left in position and strapped to the patient's cheek. Next, a 50 to 60 cm length of duodenal tube was prepared by threading a lubricated, stainless steel cardiac guidewire through the tube until it was just visible in the plug but not protruding. The patient was placed in the supine position and the duodenal tube with guidewire was passed gently through the nose, posterior pharynx, and well into the oesophagus. At this point, the patient was placed prone and, under fluoroscopy, the tube advanced until it reached the greater curvature so that the end plug moved towards the pylorus. When gastric peristalsis occurred, the guidewire was used to insert the end plug and tube into the pyloric canal. After the end plug had passed successfully beyond the pyloric obstruction, the tube was advanced so that the connecting plugs passed this point as well. The position of the tube was considered optimal if its tip lay in the duodenum, somewhere between the second portion and the ligament of Treitz. The guidewire was then slowly withdrawn without pulling the tube tip back and the tube was attached to the patient's face, using adhesive tape. An 18 gauge needle was cut to provide a square edge and then inserted into the proximal tube as a connector.

#### *Feeding*

A reservoir containing the milk feed was connected to the nasoduodenal feeding tube (Fig. 2). The desired formula or fluid was delivered at room temperature to the infant every 3 h at a rate which provided the desired amount of fluid and calories. A formula milk (Wakodoh, Japan), 74 Cal/100 ml of full strength (329 mOsm/l), or a casein-hydrolysed milk MA1 (Morinaga, Japan), 70 Cal/100 ml of full strength (340 mOsm/l) was used to provide 120 to 150 Cal/kg/day. Gastric juice was aspirated through the nasogastric tube with a syringe, and was infused slowly into the duodenum, immediately after each nasoduodenal feeding. While the feeding tube was in place a dummy was given to the patients to maintain their sucking abilities, and the nursing staff and parents stimulated them by holding or handling them as often as possible.

#### *Discontinuation of Feeding*

Two factors were adopted as criteria for a change from nasoduodenal to nasogastric feeding: these were (a) the attainment of a certain body weight and (b) the volume of fluid, as measured by gastric aspiration, which could pass from the stomach into the duodenum. With regard to body weight, 3 hourly nasogastric feeding was attempted at 5,000 g. This procedure enabled measurement by gastric aspiration of the volume of fluid passing from the stomach to the duodenum. 70 ml was taken as the critical volume at which a decision was made to change from nasoduodenal to nasogastric feeding. Volumes of feed were increased gradually until 100 ml volumes could be tolerated via the nasogastric tube without vomiting. Bottle feeds were then instituted. Small volumes (50 ml) were given by bottle initially and the remainder by nasogastric tube; proportions were adjusted gradually until the whole feed could be taken by bottle. After this, volumes of bottle feed were increased if necessary. In a few cases, it was not possible to

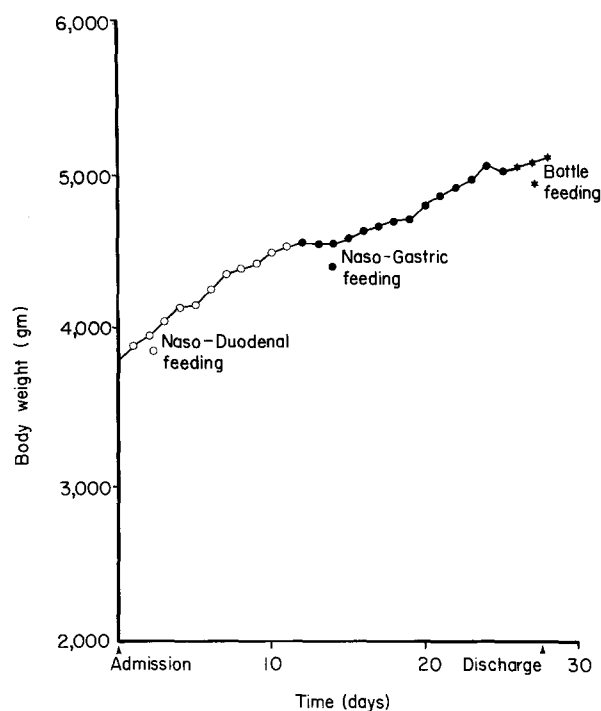


Fig. 3. Clinical course of case 1

establish nasogastric feeding even though a body weight of 5,000 g had been attained. In these cases, reintubation with a nasoduodenal tube was performed and alimentation continued until the baby weighed approximately 5,500 g. At this weight all patients could tolerate nasogastric feeds and subsequent bottle feeds.

### Typical Case Histories

#### Case 1

A 41 day old boy with projectile vomiting was admitted to hospital. He was born normally; birth weight 2,700 g. No neonatal problems were present until he started vomiting twice to three times a day at the age of 7 days, and vomiting becoming more frequent and projectile after the age of 35 days. He was diagnosed as having IPS by his doctor and referred to hospital where the diagnosis was confirmed radiographically. On admission he was in a fairly good nutritional state but moderately dehydrated. The pyloric tumour was palpable. Body weight was 3,640 g. Laboratory tests showed: serum sodium 131 mmol/l; potassium 3.2 mmol/l; chloride 89 mmol/l; pH 7.5; base excess +8 mmol/l.

On the day of admission, nasoduodenal feeding was started giving 100 ml of half strength formula milk every 3 h. On the following day the dehydration and the imbalance of electrolytes had recovered. The milk volumes and strength were built up gradually and finally reached 140 ml per feed of full strength milk. During the period of the duodenal alimentation he gained weight steadily as shown by a gain of 595 g in 11 days (45 g/day) (Fig. 3). Feeds were switched to nasogastric when the nasoduodenal tube spontaneously was displayed into the stomach on the 12th day. Good weight gain continued with nasogastric feeding without vomiting, and body weight reached 5,000 g on the 21st day of admission. He could then tolerate oral bottle feeding and was discharged on the 26th day. Subsequent progress was uneventful.

#### Case 2

An 18 day old girl was born normally and weighed 3,200 g. She began to vomit a few times a day in the second week after birth and gradually this became more frequent and projectile. As dyspnoea also appeared in the third week after birth and rapidly progressed, she was referred to hospital. Physical examination on admission showed an ill baby with poor nutrition, a dry skin and a dusky hue. A pansystolic murmur was present. Body weight was 2,700 g only. Pulse rate was 140/min and respiratory rate 60/min. Laboratory tests showed: serum sodium 138 mmol/l; potassium 5.7 mmol/l; chloride 96 mmol/l; pH 7.4; base excess +1 mmol/l. She was diagnosed as having IPS associated with ventricular septal defect from the evidence of the pansystolic murmur, radiographic findings and ECG.

After digitalization, rehydration with fluid therapy and small frequent milk feeds by mouth, nasoduodenal feeding was begun on the 8th day after admission. Tachypnoea, rib retraction and tachycardia persisted for about six weeks after admission. Although duodenal feeding of 100–110 Cal/kg/day was carried out for 16 days, she gained only 100 g (6 g/day) in this period. This was presumed to be due to exhaustion due to heart failure. Subsequently, feeding was switched to nasogastric and coincidentally the symptoms of heart failure gradually improved. When the body weight reached 4,000 g two months after admission, oral bottle feeding was started and no vomiting occurred.

A further two months' hospital stay was necessary for cardiac care and she was discharged 115 days after admission with a body weight of 5,388 g.

### Results

Data from individual babies are given in Tables 1 and 2.

Transpyloric intubation was achieved in all 50 patients and the total time for successful tube placement varied from 2 to 20 min.

With regard to X-ray exposure, the average time taken for duodenal tube placement was 3.5 min and the amount of radiation given was 8.7 Rads. This X-ray exposure is comparable to that given during a barium meal examination.

Forty-five of the 50 (90%) IPS patients were cured by nasoduodenal feeding (Table 1).

Five patients (10%) (Table 2) required pyloromyotomy. Of these five, surgical intervention was necessary in three cases only on account of the abandonment of nasoduodenal feeding. In these three babies, the first few intubations were successful and duodenal infusions could be performed, but the tubes were repeatedly and spontaneously displaced from the duodenum into the stomach so that infusions could not be maintained. In the cases of the remaining two babies the parents requested surgery.

Comparative data for body weight on admission, weight gain during intubation, duration of nasoduodenal feeding, and length of stay in hospital for the conservatively treated babies (45) and the babies treated by pyloromyotomy (5) are shown in Table 3.

**Table 1.** Infants with pyloric stenosis who responded satisfactorily to naso-duodenal feeding

Case No.	Age on admission (days)	Body weight (g)			Pyloric obstruction on X-ray study	Duration of NDF (days)	Mean weight gain by NDF (g/day)	Stay in hospital (days)	Remarks
		at birth	at admission	at discharge					
1.	41	2700	3840	5145	++	12	54	26	VSD
2.	18*	3200	2700	5388	++	16	6	115	
3.	45	3230	3245	5465	+++	11	45	68	
4.	38	2740	3000	5069	+++	22	39	70	
5.	40	2865	2600	4772	+	37	52	40	
6.	65	3800	4915	5920	++	14	40	31	
7.	30	3303	3814	5495	+	12	38	52	
8.	35	3220	3765	5050	+	10	44	56	
9.	30*	3520	3581	4445	+	8	40	17	
10.	36*	2500	2670	4973	+++	29	52	68	
11.	46	2350	2984	5090	++	13	46	28	Down syndrome with VSD
12.	49	3400	4420	5600	++	15	48	35	
13.	27	2820	3340	4505	+	10	49	27	
14.	57	3550	4881	5954	++	18	35	33	
15.	46	3000	3873	5542	++	12	34	40	
16.	42	2700	3704	4990	++	20	45	35	
17.	12	2965	3165	4796	+++	8	48	41	
18.	61	3350	4778	6010	++	34	35	41	
19.	33	3420	4346	5542	++	28	38	38	
20.	54	3400	4417	6103	++	8	57	37	
21.	36	3680	4551	6014	+++	25	42	42	
22.	47	2890	4070	5167	++	13	38	31	
23.	40	2222	2698	4600	+++	36	39	44	
24.	32	3060	3563	4980	++	18	41	36	
25.	21	2800	3630	4960	+	13	52	23	
26.	46	2790	4332	5532	++	30	37	40	
27.	35	3280	3279	4599	+++	14	32	46	
28.	27	3210	3840	5090	++	16	43	27	
29.	66	3780	3843	5330	++	17	36	53	
30.	38	3190	3144	5281	+++	13	46	50	
31.	39	3620	4099	5161	++	15	42	39	
32.	26	3230	3600	4890	++	16	38	38	
33.	61	2820	4050	5120	++	15	41	26	
34.	64	3800	4915	5920	+	12	47	31	
35.	34	3250	4020	5210	++	13	43	29	
36.	41	3300	4380	5070	++	17	44	35	
37.	30*	3212	3460	5040	++	37	51	49	
38.	32	3862	4529	5090	++	12	57	17	
39.	15	2600	2470	3800	++	15	31	36	
40.	53	2800	4500	5308	+	10	46	21	
41.	28	3007	3800	5010	+	10	44	25	
42.	25	3200	3500	4930	++	14	43	26	
43.	37	3000	3640	5250	+++	34	46	53	
44.	28	3150	3203	4950	++	13	48	46	
45.	33	3350	3600	4810	++	12	48	28	
Mean	38.6	3130	3750	5177		17.2	42.7	39.7	

NDF = naso-duodenal feeding, VSD = ventricular septal defect,

**Table 2.** Infants with pyloric stenosis who required pyloromyotomy after brief nasoduodenal feeding

Case No.	Age on admission (days)	Body weight (g)		Pyloric obstruction on X-ray study	Duration of NDF (days)	Mean weight gain by NDF (g/day)	Remarks
		at birth	at admission				
1.	41*	3160	3530	+++	7	35	
2.	40	3630	3800	++	11	43	
3.	18	3420	2994	++	23	25	
4.	63	3255	4391	++	2	50	Parents requested operative treatment
5.	12	3347	3303	++	5	18	
Mean	34.8	3362	3603		5	34.2	

NDF = nasoduodenal feeding, \* = female

+ = mild, ++ = moderate, +++ = severe degree of pyloric obstruction on X-ray study

**Table 3.** Comparative data for body weight on admission, weight gain during nasoduodenal feeding (NDF) and mean weight gain during nasoduodenal feeding for the conservatively treated babies and the babies treated by pyloromyotomy

	Conservatively treated cases (45)	Surgically treated cases (5)
Mean body weight on admission (g)	3,750	3,603
Mean body weight at discharge (g)	5,177	—
Mean duration of NDF (days)	17.2	5 (before operation)
Mean weight gain during NDF (days)	42.7	34.2 (before operation)
Mean stay in hospital (days)	39.7 (38.0) <sup>a</sup>	6 (before operation)

<sup>a</sup> Cases without any associated disorder

Mean body weight for all cases on admission was 3,775 g (3,750 g on conservatively treated cases, 3,603 g in the five who came to operation). At discharge the mean body weight for the 45 babies treated by intubation was 5,177 g.

The duration of nasoduodenal feeding was 8–37 days in conservatively treated cases (mean 17.2 days) and 2–23 days before operation (mean 5 days) in the five surgically treated cases. Mean stay in hospital for conservatively treated cases was 39.7 days (38.0 days in cases without any associated disorder). Mean weight gain during the duodenal alimentation for total cases was 41.8 g/day (conservatively treated cases 42.7 g/day, operated cases 34.2 g/day).

Diarrhoea was often observed in the first 10 patients who were given standard formula milk by duodenal infusion, although no reducing substance or steatorrhea

were found in the stools. However in the 40 patients treated later, in whom casein hydrolysed milk was introduced, diarrhoea was much less frequent and no metabolic disturbances were encountered. None of the 50 patients had serious complications which were attributable to the nasoduodenal tube. In particular, no baby developed duodenal perforation, necrotizing enterocolitis or intussusception, as have been reported in low birth weight infants. The babies in this present study were followed by the authors for one year after the end of nasoduodenal feeding. Growth and development were within normal limits for age and no long-term complications were observed.

## Discussion

As experience was gained with nasoduodenal feeding, it became apparent that it usually produced a good result and that 90% of patients with IPS were curable by conservative management. The principal problem of treatment was frequent duodenal tube displacement from the duodenum into the stomach, necessitating earlier attempts at nasogastric feeding or oral feeding than had been shown by previous experience to be appropriate. The success or failure of duodenal feeding in IPS was not correlated with the degree of pyloric obstruction, as determined by roentgenographic examination, but no failure occurred in “mild” cases. In other words, in patients with IPS whose pyloric canal opens within 15 min under fluorescopy the success rate of conservative management can be expected to be very high.

Some precautions should be observed when nasoduodenal feeding is used in IPS patients. To avoid the “dumping syndrome”, osmotic diarrhoea, and perhaps necrotizing enterocolitis, hypertonic fluid and rapid infusion are not advisable. Thus, in all patients

duodenal feeding was started with a half strength formula milk at an infusion rate of 1 ml/min and gradually increased to full strength at the same rate of infusion. With regard to the possible reason for the occurrence of diarrhoea in the babies given milk feeds, as opposed to the relatively normal stools in those given casein hydrolysate, it may be that bypassing the stomach and displacing its secretions from their normal environment interfered with the breakdown of whole milk and that casein hydrolysate is more suitable. This appeared to provide adequate nutrition, calories and fluid, and its osmolality was similar to that of whole milk. In theory, fully adapted milk preparations should be better than casein hydrolysate in view of the lesser acidity and greater cystine content of whole milk; however the authors did not recognize any problems with casein hydrolysate.

With regard to absence of acute complications during nasoduodenal feeding, this may have been associated with the facts that our cases were older than newborn infants (mean age 38.2 days on admission) and that the intubation tubes were made of silicone rubber.

Traditional medical management of IPS consists of supportive therapy, frequent feeding of small amounts of diluted milk with gastric lavage, and anticholinergic drugs, e.g., atropine methylnitrate and methylscopolamine nitrate, given intramuscularly or orally [3, 5, 8]. The main problems of traditional medical treatment compared with surgical treatment are: the lower success rate (less than 30 to 70%) [3, 5, 8], although if mild cases are selected higher success rates can be expected [6, 11], the higher mortality rate; the slowness of improvement, and the severe and prolonged undernutrition which may occur [12]. These unfavourable points of traditional medical treatment have not been fully solved by nasoduodenal feeding, especially with regard to the slowness of improvement. It is noteworthy however that the rate of weight gain in the 45 babies treated by duodenal intubation was similar to their expected rate of gain under normal conditions at home.

The observation that it was nearly always possible to change from nasoduodenal to nasogastric feeding when the baby weighed around 5,000 g may offer a clue about the reasons for improvement in IPS. This may be due to a simple anatomical enlargement of the pyloric canal with growth, or alternatively, to bypassing the stomach and so decreasing the effect of hypergastrinaemia which has been noted and thought to play an important role in the development of IPS [4].

It has been observed that the hypergastrinaemic response to intragastric milk infusion in patients with IPS who had been treated by nasoduodenal feeding was much smaller than in similar babies before treatment [13]. The duodenal infusion itself, inducing secretion of some gut hormones, i.e., secretin, cholecystokinin and

gastric inhibitory peptide from the duodenal mucosa may also contribute to further reduction of the elevated blood gastrin level [9].

## Conclusion

Our experience suggests that nasoduodenal feeding as described in this report is a more effective treatment for IPS than traditional non-surgical methods. However, it is not indicated for routine management but may be the preferred management in exceptional cases. These include a small group of children for whom an operation could carry a high risk irrespective of the severity of pyloric stenosis, or whose parents requested conservative management.

We are grateful to Dr. K. Makino and Mr. S. Aoki for their constructive comments and technical contributions. We thank the medical and nursing staff of the Infant Care Unit for their participation and co-operation, and Professor H. Kato for his support.

## References

1. Bøggild-Madsen NB (1975) Medicinsk behandling af kongenit pylorusstenose. En oversigt behandlingen på et central-sygehus. *Ugeskr Læg* 137:201-203
2. Coroner BD (1955) Hypertrophic pyloric stenosis in infancy treated with methyl scopolamine nitrate. *Arch Dis Child* 30:377-386
3. Day LR (1969) Medical management of pyloric stenosis. *JAMA* 207:948-950
4. Dodge JA, Karim AA (1976) Induction of pyloric hypertrophy by pentagastrin. *Gut* 17:280-284
5. Eschenbacher HL, Göbel H (1972) Zur Behandlung der spastisch-hypertrophischen Pylorusstenose mit Methylscopolaminnitrat. *Klin Päd* 184:367-371
6. Jacoby NM (1962) Pyloric stenosis: Selective medical treatment. A survey of 16 years experience. *Lancet* 1:119-121
7. Malmberg N (1949) Hypertrophic pyloric stenosis — A survey of 136 successive cases with special reference to treatment with scopol. *Acta Paediatr* 38:472-483
8. Mellin GW, Santulli T, Altman HS (1965) Congenital pyloric stenosis: A controlled evaluation of medical treatment utilizing methyl-scopolamine. *J Pediatr* 66:649-657
9. Rayford PL, Miller TA, Thompson JC (1976) Secretin, Cholecystokinin and newer gastrointestinal hormones. *N Engl J Med* 294:1093-1101
10. Rhea JW, Kilby JO (1970) A nasojejunal tube for infant feeding. *Pediatrics* 46:36-40
11. Schäfer KH, Bingel G (1975) Konservative und chirurgische Therapie der spastischen, hypertrophischen Pylorusstenose (s.h.P.). Eine katamnestiche Studie. *Msschr Kinderheilk* 123:503-508
12. Shandling B (1979) Congenital hypertrophic pyloric stenosis. In: Vaughan VC III, McKay RC Jr, Behrman RE (eds) *Textbook of Pediatrics*. W.B. Saunders Company, Philadelphia, pp 1050-1053
13. Yamashiro Y, Mayama H, Yamamoto K, Nawate G, Kato H (1977) Serum gastrin levels and G cell in the antral mucosa in congenital hypertrophic pyloric stenosis. Abstracts of papers XV International Congress of Pediatrics, New Delhi, India, p 51

Received April 9, 1980