

GASTRIC EMPTYING (GE) IN INFANTS AND CHILDREN WITH GASTRO-OESOPHAGEAL REFLUX (GOR): A 5-YEAR RETROSPECTIVE STUDY

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GASTRIC EMPTYING TIME OF A LIQUID OR SEMI-SOLID MEAL WAS EVALUATED IN 477 INFANTS AND CHILDREN REFERRED FOR SUSPICION OF GOR WHO UNDERWENT GASTRO-OESOPHAGEAL SCINTISCANNING, PROLONGED PH-METRY, MANOMETRIC EVALUATION OF THE LOWER OESOPHAGEAL SPHINCTER (LOS) PRESSURE AND FIBERENDOSCOPY. NO DIFFERENCE IN GE WAS OBSERVED IN CHILDREN LESS THAN 3y REGARDLESS THE PRESENCE OR ABSENCE OF GOR, THE PRESSURE OF THE LOS OR THE PRESENCE OF OESOPHAGITIS. IN CHILDREN OLDER THAN 6y, HOWEVER, GE WAS SIGNIFICANTLY DELAYED IN THOSE PRESENTING A GOR (COMPARED TO THOSE WITHOUT REFLUX). IN CHILDREN OLDER THAN 3y A SLOWER GE WAS PRESENT IN THOSE WITH A DECREASED LOS PRESSURE (COMPARED TO THOSE WITH HIGHER LOS PRESSURE) AND IN THOSE WITH OVERT OESOPHAGITIS (COMPARED TO THOSE WITHOUT OESOPHAGITIS). THIS STUDY SUGGESTS THAT GOR IS A MORE SEVERE DISEASE IN CHILDHOOD THAN IN INFANCY WITH A MORE COMPLEX MOTOR DISORDER AFFECTING NOT ONLY THE LOS FUNCTION BUT THE GASTRIC FUNDUS AS WELL.

INFLUENCE OF THE FAT CONCENTRATION OF A FORMULA ON THE GASTRIC ANACIDITY TIME. Yvan Vandenplas, Helmut Loeb. Academic Children's Hospital Free University of Brussels, Belgium.

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Whereas milk- (or formula-)feeding neutralizes the gastric content (pH>4), gastric pH monitoring can be used studying the gastric acidity, and subsequently the gastric emptying, time (Sutphen, AJDC 1986;140:1062-1064).

Using a new technique, extended simultaneous esophageal and gastric pH monitoring, we measured the postcibal time gastric pH was > 4 in 11 asymptomatic infants according to a double-blind cross-over technique. Two isocaloric formulae with a different fat concentration were studied. The composition of both formulae (Fo1/Fo2) is as follows (g/100ml): protein 2.2/2.2; Fat 2.6/3.6; Carbohydrates 8.2/7.6; Lactose 5.3/5.4.

The results of the esophageal pH monitoring were in all infants within normal ranges, but GER occurred more in the postcibal period of infants receiving Fo2. The duration gastric pH was > 4, was significantly (P<0.001; Student's t-test) shorter in the infants receiving Fo1 (52 +/- 10 min) (mean +/- 1 SD) than in the infants receiving Fo2 (79 +/- 11 min). Whereas a delayed gastric emptying time has been reported as one of the causes of GER pathology (Hillemeier, J Pediatr 1981;98:190-193), the administration of a low-fat formula (milk) could possibly decrease the incidence of reflux by shortening the gastric emptying time.

A DOUBLE-BLIND CONTROLLED STUDY OF CIMETIDINE IN REFLUX ESOPHAGITIS (RE). S.Cucchiara, L.Gobio Casali, F. Balli, G.Magazzù. Clinical Pediatrics of Naples, Modena, Messina and Dpt Pediatrics of Mantova, Italy.

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Most infants with gastroesophageal reflux (GER) disease (GERD) are successfully treated only by conservative care. To assess whether pharmacologic therapy is needed in GERD with RE, 32 patients (pts) were randomized to either Cimetidine (C) (17) (30 mg/kg/d) or placebo (P) (15), in addition to intensive postural therapy, in a 12 week double-blind trial. Mean(+SD) age(months) was 21.7+37.6 (C) and 29.03+39.7 (P). GER was diagnosed by prolonged pH test, RE by endoscopy and histology and graded by a scoring system. Based on clinical, pHmetric and histological data, 12 Cpts (70.6%) and 3 Ppts (20%) healed ($X^2:5.6, p<0.01$), 4 Cpts (23.5%) and 3 Ppts (20%) improved (N.S.), 4 Cpts (23.5%) and 9 Ppts (60%) worsened ($X^2:4.7, p<0.01$). The RE score (mean+SD) significantly decreased only in Cpts (pre:6.3+2.4, post:1.6+2.4, $p<0.01$), not in Ppts (pre:6.2+2.7, post:5.4+3.8, N.S.). Histological improvement was seen in all (100%) of Cpts with moderate RE vs 42.9% of Ppts ($X^2:6.8, p<0.01$) and in 87.5% of Cpts with severe RE vs 37.5% of Ppts ($X^2:4.9, p<0.01$). In conclusion, although GERD in infancy has a naturally self limited course with conservative care, extensive pharmacologic therapy is needed in presence of RE.

PHOSPHOLIPIDS AND TRYPSIN GASTRIC OUTFLOW : A MARKER OF DUODENO-GASTRIC REFLUX (DGR) IN CHILDREN.

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Potential toxicity of bilio-pancreatic secretions on oesophageal and gastric mucosa is wellknown in adults. Nevertheless DGR had never been assessed in children. After a 6 hours fast, we measured the following parameters by their one hour gastric outflow, collected with a naso-gastric tube : trypsin (TR), marker of pancreatic reflux, by radio-immunologic assay ; phospholipids (PLP), i.e. lecithin and lysolecithins, markers of biliary reflux, by colorimetry ; sialic acid (SA), marker of gastric mucus erosion, by Aminoff method. The measurements were done in 38 controls, divided into 3 groups : 0-2 months (n=8), 2-12 months (n=20), 1-4 years (n=10)

		TR(mcg/kg/h)	PLP(mcmol/kg/h)	SA(mcg/kg/h)
0 - 2 m	m + 2 SD	2,9	0,42	370
2 - 12m	m + 2 SD	4,6	0,25	295
1 - 4 y	m + 2 SD	0,41	0,08	100

As in adults, DGR was defined by a value above mean plus 2 SD for one or the 2 bilio-pancreatic markers. In a preliminary study DGR could be demonstrated in 3 children, with an antral gastritis. The levels of the 3 parameters were then much higher than the definite standards. This non invasive method seems to be a very usefull approach for the diagnosis, mechanisms and treatment of gastric and oesophageal pathologies.

A NEW METHOD FOR DETECTING ANTI LIVER CELL MEMBRANE ANTIBODY USING A HUMAN HEPATOMA CELL LINE.

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A simple technique for detection of serum autoantibodies that bind to liver membrane antigens on the surface of liver cells has been developed using Alexander cells [PLC/PRF/5]. Alexander cells were incubated with test serum (1:160), washed then adherent antibodies were detected by incubating with ¹²⁵I-Protein A. Specific binding to cell surface was calculated as the ratio between the counts with patient's serum divided by 2SD above the mean count obtained with serum from controls. The ratio was significantly higher in those with active autoimmune chronic active hepatitis (aCAH) [median 4.5, range 1.1-23.1] when compared to inactive cases [1.5, 0.3-4.1; $p<0.01$], to alpha-1 antitrypsin deficiency [1.2, 0.3-2.3; $p<0.01$], and to Wilson's disease [1.7, 0.3-2.9; $p<0.01$]. The ratio in primary sclerosing cholangitis was similar to active aCAH [4.2, 0.8-6.7] which would support recent evidence implicating autoimmune mechanisms in PSC. In 11 aCAH cases tested for anti-LSP [liver specific lipoprotein] antibody a positive correlation was found between Alexander cell binding assay & anti-LSP titres ($r = 0.56, p<0.02$). In 4 aCAH tested @ diagnosis [4.8, 3-23.1] the ratio fell after effective immunosuppressive therapy [0.9, 0.6-4.5]. Alexander cell binding assay provides a simple, rapid & sensitive technique to detect specific antibody to liver cell membrane which may help in the management of autoimmune liver disease.

DIFFERING EXPRESSION OF ANTIBODIES TO DOUBLE STRANDED DNA IN AUTOIMMUNE CHRONIC ACTIVE HEPATITIS (aCAH) & PRIMARY SCLEROSING CHOLANGITIS (PSC)

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Non organ-specific autoantibodies, in particular antinuclear antibody (ANA) are present in both aCAH and PSC of childhood, suggesting an imbalance in immune-regulation akin to that observed in systemic lupus erythematosus (SLE). The aim of this study was to investigate whether an increase in antibody to double-stranded DNA (dsDNA), typical of SLE, is present in these two conditions. Binding to dsDNA was determined by RIA in 8 ANA positive children with PSC [4 male, median age 7yrs, range 3-14yrs] and in 20 patients with aCAH [5 male, median age 12.5yrs, range 8-21yrs], 6 of whom are ANA positive, the remainder being positive for antibodies to smooth muscle (SMA), liver/kidney microsomes (LKM) and/or gastric parietal cells (GPC). Anti-dsDNA antibodies in the range diagnostic for SLE (>25 U/ml) were found in 5 children with aCAH [2 ANA positive, 2 LKM positive, 1 SMA positive] but in none of the PSC group. The mean dsDNA binding in children with aCAH was 15.0 IU/ml ± SD 10.63 compared to 7.78 U/ml ± SD 1.7 in those with PSC ($p < 0.05$). Anti-dsDNA antibodies of more than 25 U/ml in aCAH occurred in the absence of ANA. In the PSC group where ANA is commoner & generally of higher titre, anti-dsDNA antibodies did not correlate with ANA titre. This is a further demonstration of different immune dysfunction in these two chronic liver disorders.