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EPIGASTRIC IMPEDANCE: A ROBUST AND SENSITIVE MEASURE OF GASTRIC EMPTYING OF LIQUIDS IN HEALTH AND DISEASE **HL SMITH, GW HOLLINS, IW BOOTH** Institute of Child Health, University of Birmingham & Birmingham Maternity Hospital

Gastric emptying (GE) may be measured by recording changes in epigastric impedance (EPI), as the stomach fills with, and subsequently empties, a liquid of low electrical conductivity. We have evaluated this method, and used it to record physiological and pathophysiological changes in GE. In 45 healthy children (27 M, 18F; mean age 8.9 years, range 7 mo-17y) EPI was recorded fasting and following ingestion of 400 ml/m² orange squash. Ingestion of the test meal in aliquots revealed a highly significant positive correlation between impedance increase (Δ EPI) and volumes consumed ($r > 0.95$; $p < 0.001$). Log transformation of Δ EPI showed significant negative correlations with height, weight, body surface area, triceps skinfold thickness and age. Mean time to half-emptying ($t_{1/2}$) was 14.8 min (SD 6.8); however, $t_{1/2}$ showed no correlation with anthropometric indices or with age.

In 12 children the test was repeated with fat added to the test meal. The addition of only 1.25% fat (as triglyceride emulsion) delayed GE by 57% (mean $t_{1/2} = 10.4$ min with orange squash; mean $t_{1/2} = 15.6$ min for squash with fat; $p < 0.01$). The test was also useful clinically, and we have shown rapid GE in 2 infants with dumping ($t_{1/2} = 4$ and 5 mins); GE returned to normal with thickened feeds.

This method is therefore non-invasive and non-isotopic and well tolerated, and is sensitive enough for both physiological and pathophysiological applications in childhood.

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VITAMIN E AND PREVENTION OF LIVER DAMAGE IN HETEROZYGOTES WITH ALPHA-1-ANTITRYPSIN DEFICIENCY **K. Pittschieler** Paediatric Gastroenterology, Regional Hospital, Bolzano 1

From our previous experience on more than 900 MZ and MS heterozygous alpha-1-antitrypsin deficient infants we know that liver enzymes can be, especially in their first six months, pathologically elevated. The pathogenesis of the liver damage in homo- and heterozygous patients is still not well known and superoxide radicals may be involved in this process. It is established that vitamin E has anti oxidative activity and is together with the superoxide dismutase the main scavenger for toxic waste products of the oxygen. 156 MZ and 146 MS phenotypes were divided in a double blind way in two groups, one with and one without a daily oral intake of 50 mg vitamin E for six months: at the age of two and four months liver enzymes as AST, ALT, GGT, ALP and bile acids were determined. The following table shows the distribution of abnormal values in both groups at 2 and 4 months

2 ^m Vit E	151	31	21	4 ^m Vit E	151	12	8
No	151	49	32	151	21	14	
tot.	n	%		tot.	n	%	

In conclusion: Vitamin E seems to be able to reduce in this age group the incidence of a more or less severe liver involvement in heterozygous infants supporting the previously described pathogenetic hypothesis.

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ANOMALOUS HIGH PANCREATICO-BILIARY UNION **-THE MAIN CAUSE OF CHRONIC PANCREATITIS IN CHILDREN**

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The assessment of pancreatitis by existing techniques is often insufficient for precise diagnosis of its aetiology. 17 children with history of recurrent abdominal pain, vomiting and increase of serum and urinary amylase and serum lipase level were diagnosed to have chronic pancreatitis. Beside metabolic studies and ultrasound investigation endoscopic retrograde cholangiopancreatography (ERCP) was successfully performed in 15 children.

ERCP was made under diazepam sedation with Olympus JFB3 duodenoscope within 30 minutes. For cannulation we used standard adult cannulas. ERCP was incomplete in 2 children; pancreatograms were obtained only Anomalous high pancreatico-biliary union (P-B type) with long common channel exceeding 15 mm, pancreas divisum, choledochal cyst with anomalous high biliary-pancreatic union (p-B type) and long common channel, sclerosing papillitis were diagnosed. ERCP was normal in 5 children.

This study shows that migration failure of the pancreatico-biliary union with long common channel is the most common single cause of chronic pancreatitis in children. This anomaly cannot be diagnosed by any other means than by ERCP.

ERCP is the method of choice in the investigation of children with chronic pancreatitis.

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INCREASED SUPERIOR MESENTERIC VASCULAR RESISTANCE IN INFANTS AT RISK OF NECROTISING ENTEROCOLITIS (NEC) **Coombs RC, Morgan MEI, Durbin GM, Booth IW, McNeish AS** Neonatal Unit, Birmingham Maternity Hospital, & Institute of Child Health, University of Birmingham

To determine whether initial mesenteric hypoperfusion predisposes to NEC, we assessed superior mesenteric artery (SMA) peak systolic velocities (PSV) relative to those in the coeliac axis (CA) using duplex pulsed Doppler ultrasound, in a group of infants with perinatal complications which placed them at increased risk of NEC. The ratio of PSV in these adjacent vessels (CA/SMA) was used to determine relative downstream resistance in the SMA. We studied two control groups: i) 14 term neonates (mean wt 3.3 kg); ii) 18 preterm infants with no history of perinatal asphyxia (mean wt 1.6 kg; 27-35 wk). The study group comprised 27 infants at increased risk of developing NEC who had sustained perinatal asphyxia; or were growth retarded and required intensive care; or had received an exchange transfusion (mean wt 1.5 kg; 26-39 wk).

MEAN PSV	TERM		PRE-TERM		AT RISK	
(cm/sec)	SMA	CA/SMA	SMA	CA/SMA	SMA	CA/SMA
Day 1	49 b	1.40	52	1.16 a	37 b	2.61 a
Day 4	71	0.88 c	63	1.3	50	1.54 c

(All p values between data pairs (a-c) < 0.05)

On Day 1, the CA/SMA ratio in the AT RISK group was significantly higher than the PRETERM group ($p < 0.03$); this reflected lower SMA velocities in the AT RISK group compared with controls ($p < 0.02$ of TERM). In the AT RISK group, abnormalities in SMA velocities and in the CA/SMA ratio were still significantly abnormal compared with TERM controls on Day 4 ($p < 0.02$). These data provide a rational explanation for the link between NEC and its known risk factors.

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AVOIDANCE OF INDOMETHACIN INDUCED REDUCTION IN MESENTERIC BLOOD FLOW VELOCITY USING SLOW INFUSIONS **Coombs RC, Morgan MEI, Durbin GM, Booth IW, McNeish AS** Neonatal Unit Birmingham Maternity Hospital and Institute of Child Health, University of Birmingham

Indomethacin used in the management of patent ductus arteriosus predisposes to necrotising enterocolitis. We have previously shown that the first dose of indomethacin given as an iv bolus (< 20 sec) causes a rapid and profound fall in gut blood flow velocities: mean peak systolic velocity (PSV) in superior mesenteric artery (SMA) decreased from 74 to 38 cm/sec; median time to max fall 7.4 min. We have therefore prospectively studied the changes in PSV in the coeliac axis (CA) and SMA following slow infusion of indomethacin (IND) using Doppler ultrasound.

10 subjects were studied each receiving their first dose of indomethacin iv (0.2 mg/kg) infused slowly over 30 minutes. Although in 8 cases there was a fall in PSV in the SMA, and in 5 cases in CA, in neither vessel was this reduction significant. The median time to max fall in the SMA was 37 min (15-55), and for the CA 37 min (9-46). In comparison to rapidly infused IND the percentage reduction in PSV was significantly less with the slow infusion ($p < 0.03$) and the time to maximal fall longer ($p < 0.0009$).

MEAN ($\pm$ SD)	PSV PRIOR to IND	PSV POST IND
CA	65 $\pm$ 19 cm/sec	66 $\pm$ 33 cm/sec NS
SMA	71 $\pm$ 21 cm/sec	56 $\pm$ 32 cm/sec NS

We conclude that the profound fall in mesenteric blood flow velocity seen following a rapid iv bolus of IND (suggesting an ischaemic insult) can be avoided by its slow infusion, which may reduce the prevalence of necrotising enterocolitis post-indomethacin.

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IS CHOLEDOCHAL CYST A CONGENITAL ANOMALY, AN ACQUIRED DILATATION OR BOTH? **Y. Yamashiro, T. Miyano, S. Oguchi, T. Shimizu, M. Satoh** Dept of Pediatrics and Pediatric Surgery, Juntendo University School of Medicine, Tokyo, Japan.

In recent years, anomalous confluence of the common bile duct (CB) and the pancreatic (P) duct, that is, anomalous choledochopancreatic ductal junction (ACPDJ) has aroused interest as a possible cause of choledochal cyst (common bile duct dilatation: CBD). Our experimental study advocated this theory. ACPDJ forming long common channel is believed to allow reflux of the P juice into the CB and the resultant irritation of the CB by reflux P juice, which is mixed with bile resulting in activation of P enzymes, gives rise to its dilatation. Thus, it is conceivable that CBD is an acquired dilatation as a complication of ACPDJ. To clarify whether ACPDJ is the sole cause of CBD, we conducted a study to analyse P enzyme levels in bile aspirated from the dilated CB at laparotomy because existence of activated P enzymes in the CB is highly suggestive of acting as curative factor. The subjects consisted of 24 cases, aged 4 days to 15 years. P enzymes analysed were amylase, trypsin, elastase, lipase and phospholipase A. Activation of trypsin was found in 15 cases (62.5%) in whom other enzymes were also activated. But no activation of these enzymes was detected in the remaining 9 cases (37.4%) irrespective of ACPDJ, of whom 7 (29.6%) were less than 2 months of age and presented with jaundice from early infant days. These findings suggest that ACPDJ is not the sole cause of CBD and "congenital" CBD also possesses some proportions of the disorder.

1) Y. Yamashiro, et al. J Pediatr Gastroenterol Nutr 1984;3:721-727.