3 ($R=0.597$, $p<0.001$, $n=38$; $R=0.462$, $p=0.009$, $n=31$). TNF-alpha and IL-10 both correlated with free TF in cord samples ($R=0.536$, $p=0.012$, $n=31$, and $R=0.610$, $p=0.003$, $n=22$). No correlations were found between cytokines and F1+2.

Conclusions: Both pro- and anti-inflammatory cytokines correlated with high TF concentration. A difference seems to exist between in utero (cord) and postnatal TF regulation: in cord plasma TNF-alpha as well as IL-10 showed strong associations with TF but lost this role to IL-8 postnatally. Free TF failed to contribute to the rate of thrombin generation. This shift of TF from coagulation to inflammation may have effects on pathogenesis of lung injury, as well as on extrapulmonary morbidity in preterm infants.

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ROUTINE COAGULATION SCREENING IN NICU- TO DO OR NOT TO DO? REVIEW OF 2 U.K TERTIARY NEONATAL UNITS

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Introduction: Routine coagulation screening of neonates admitted for intensive care is common. There is little evidence of efficacy of fresh frozen plasma (FFP) in the treatment of isolated abnormal coagulation values.

Objective: To compare transfusion rates of FFP between 2 tertiary neonatal units with similar admission rates one using routine (NICU1) and other targeted coagulopathy screening (NICU2). Secondary objective was to assess the proportions of cases treated in each unit with FFP whose coagulation parameters reverted to the normal range.

Methods: Retrospective review of neonates receiving FFP from Jan 2003- March 2008 in two UK tertiary-level neonatal units

Results: 164 neonates received 268 FFP transfusions. 131/164 (80%) infants in NiCU1 compared with 33/164 (20%) in NICU2 received FFP. Predominant reason for transfusion was abnormal laboratory measures of coagulation (77% in NICU1 and 47% in NICU 2). Active bleeding

only accounted for 15% of transfusions. Pre and post transfusion coagulation studies (within 24 hours) were measured in 72%. In only 15% did administration of FFP corrected laboratory parameters to the normal range for gestational age, in more than half of instances this occurred with diagnosis of isolated abnormal coagulation values. There was no significant difference in the proportion of babies with corrected values post FFP between two units.

Conclusion: Routine coagulopathy screening may increase the number of FFP transfusions. FFP administration did not correct laboratory parameters in majority of transfusions. The risks of transfusions should be balanced against limited clinical evidence for its administration in isolated coagulopathy.

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HIGH COPEPTIN (ARGININE-VASOPRESSIN) CONCENTRATIONS IN UMBILICAL CORD BLOOD AFTER VAGINAL DELIVERY AND BIRTH ACIDOSIS

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Background: The pituitary-secreted nonapeptide Arginine-Vasopressin (AVP) is unstable and therefore unsuited for diagnostic use but its secretion can be gauged by measuring copeptin, the C-terminal portion of the AVP precursor (CT-proAVP). We determined copeptin in umbilical cord blood and at 3 days of age in order to establish normal values and perinatal factors affecting copeptin concentrations.

Methods: Paired arterial/venous umbilical cord samples were obtained from 117 infants and umbilical venous-only samples from additional 46 infants. In 102 infants, blood was also obtained at 3 days of life. Copeptin levels were determined using the CT-proAVP-Luminescence-immunoassay.

Results: Arterial umbilical cord copeptin concentrations were consistently higher than matched venous ones ($p < 0.001$) but copeptin concentrations were closely related ($R_s = 0.825$, $p < 0.001$). Exceedingly high copeptin concentrations were observed after vaginal birth in umbilical cord venous (median [5-95% range]: 793 [6-4836] pmol/L) and arterial plasma [1610 [85-5000] pmol/L). In addition, copeptin concentrations in umbilical venous and arterial blood were negatively related with birth acidosis (pH, $R_s = -0.578$ and -0.639 , both $p < 0.001$). Postnatal body weight was associated with increased copeptin concentrations at day 3 ($R_s = 0.438$, $p < 0.001$) and was inversely related with copeptin concentrations at birth in umbilical venous and artery plasma ($R_s = -0.289$ and -0.309 , both $p = 0.001$).

Conclusion: Vaginal birth is associated with a huge release of AVP/copeptin. Umbilical cord copeptin concentrations exceed values published so far, including those in critically ill adult patients with myocardial infarction, shock, or brain injury.

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THE LEUKOENCEPHALOPATHIES OF CHILDHOOD: FINDINGS FROM A NATIONAL PROSPECTIVE STUDY

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Background and aims: To report the distribution of leukoencephalopathies in children identified by this prospective population-based study.

Methods: 2802 children with suspected progressive intellectual and neurological deterioration (PIND) were notified via the British Paediatric Surveillance Unit (BPSU) between 1997 and 2009. An expert group reviewed clinical data and confirmed diagnoses where possible. An independent expert reviewed the available scans of children with unclassified leukoencephalopathies.

Results: 1132 of the PIND children had an underlying diagnosis and of these 295 had one of the leukoencephalopathies. The distribution of diagnosed leukoencephalopathies was as follows: metachromatic leukodystrophy 62, X-linked adrenoleukodystrophy 60, Krabbe disease 39, Canavan disease 16, Alexander disease 13,

leukoencephalopathy with vanishing white matter 12, Aicardi-Goutieres syndrome 12, Pelizaeus-Merzbacher disease 11, megalencephalic leukoencephalopathy with subcortical cysts 8, multiple sulphatase deficiency 6, multiple sclerosis 3, peroxisomal bifunctional D-protein deficiency 2, peroxisomal biogenesis defect 1.

There were 50 children in the unclassified leukoencephalopathy group. In 32 the scans were reviewed and 19 were placed in a hypomyelination sub-group, the other 13 remaining in the unclassified leukoencephalopathy group with the 18 whose scans could not be reviewed.

Conclusions: Leukoencephalopathies comprised 26% of PIND children with a known diagnosis. This population based study gives the relative frequency of the different leukoencephalopathies, providing an aid to diagnosis in children with abnormal white matter on brain imaging. After rigorous investigation 16% (50/295) remained in an unclassified group, but this situation is evolving.

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DISTRIBUTION OF MICROGLIA (MG) IN THE IMMATURE BRAIN WITH ISOLATED GERMINAL MATRIX/INTRAVENTRICULAR HAEMORRHAGE (GMH/IVH): A COMBINED MRI AND HISTOLOGICAL APPROACH

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Background: GMH/IVH remains a prevalent form of injury to immature brain. In the absence of overt complications, haemorrhage may result in mild ventriculomegaly and poor developmental outcome. The imaging appearances are suggestive of periventricular injury with subsequent tissue atrophy. Although essential for normal brain development, presence of resident MG may exacerbate the injury. We have shown increased activated MG in normally appearing PVWM compared with less susceptible