Results: Arterial umbilical cord copeptin concentrations were consistently higher that matched venous ones ($p< 0.001$) but copeptin concentrations were closely related ($Rs= 0.825$, $p < 0.001$). 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, $Rs = -0.578$ and -0.639, both $p < 0.001$). Postnatal body weight was associated with increased copeptin concentrations at day 3 ($Rs = 0.438$, $p < 0.001$) and was inversely related with copeptin concentrations at birth in umbilical venous and artery plasma ($Rs = -0.289$ 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 periventicular 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