Comment on 'value of cranial ultrasound at initiation of therapeutic hypothermia for neonatal encephalopathy'

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CORRESPONDENCE

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We read with interest the paper by Sanislow et al. [1]. on the role of cranial ultrasound (cUS) prior to starting therapeutic hypothermia (TH) for infants with hypoxic-ischaemic encephalopathy (HIE). They suggest that cUS is mainly used to detect major intracranial hemorrhage (ICH), a potential reason not to start TH. They compare day 1 cUSs to day 4 post-TH MRIs and argue that pre-TH cUS is unnecessary as no major ICH was detected. In our longstanding experience of admission cUS we have seldom encountered an infant with severe ICH meeting criteria for TH; such infants usually present later with a different history [2].

The main reason for performing cUS prior to TH is to look for evidence of antenatally acquired lesions, HIE mimics, congenital infections and malformations. Table 1 lists some cases we have come across over many years. Such findings are uncommon and unsurprisingly not reported among the 108 infants studied here [1]. The abnormalities in Table 1, although suggestive of non-HIE diagnoses, need confirmation and generally TH will not be withheld. Meanwhile investigations can be instituted early and parental counselling can include concerns about another possible diagnosis from the outset rather than later on.

Changes in echogenicity on cUS following acute hypoxic-ischaemia take time to develop, as do changes on MRI. A normal cUS soon after birth is strong supporting evidence that injury seen later is of immediate perinatal onset. White matter echogenicity takes at least 12–24 h to develop and basal ganglia/thalamic echogenicity even longer depending on insult severity. Echogenicity seen on admission, would suggest an insult of recent origin but not immediately antepartum. However a common finding on cUS in term infants especially after vaginal delivery is slit-like ventricles [3]. Thus Sanislow and colleagues´ observation of slit-like ventricles in almost half their infants should not be interpreted as cerebral oedema unless accompanied by other indicators of swelling, such as loss of tissue definition and small extracerebral spaces. Comparing this finding on a day 1 cUS with day 4 MRIs is therefore unhelpful.

In contrast to preterm infants, intraventricular haemorrhage (IVH) in full-term infants, whilst uncommon, usually develops from the choroid plexus and when small, can be difficult to detect on cUS. The example in Fig. 2A could be easily missed, but the small temporal periventricular haemorrhagic infarct (Fig. 2B) should have been diagnosed, though not on the coronal view shown (Fig. 2F). It may be that it developed after day 1 and was not present when the cUS was done.

Whilst fully agreeing that MRI is the neuro-imaging gold standard for infants treated with TH we strongly recommend an admission cUS for assessing anatomy, the absence of evolving acute injury or evidence for antenatal injury and for detecting indicators of underlying problems rather than only for detecting severe haemorrhage. A second cUS at the end of TH will allow recognition of abnormalities developing following a peripartum insult and allow a valid comparison of cUS and MRI findings. We consider the two neuro-imaging techniques complimentary for the encephalopathic full-term infant.

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Table 1. Observations made by the authors on admission cUS in newborn term infants presenting with encephalopathy.

Ultrasound observation	Possible interpretation
Enlarged ventricles, sometimes with widening of the interhemispheric fissure and extracerebral space	Established antenatal insult
Porencephaly	Antenatal parenchymal haemorrhage suggestive of a coagulopathy or mutation of the *COL4A1* gene
Germinolytic cysts, lenticulostriate vasculopathy and enlarged ventricles and a shallow Sylvian fissure	Peroxisomal disorder or cytomegalovirus
Diffuse echogenicity and/or anterior white matter cysts with early-onset seizures	Molybdenum cofactor deficiency or sulphite oxidase deficiency
Small vermis and pons	Pontocerebellar hypoplasia
Hypoplastic corpus callosum with and without an abnormal cerebellum	Non-ketotic hyperglycinaemia

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AUTHOR CONTRIBUTIONS

LSdeV wrote the first draft of the comment and finalized it following reviews by the other authors. FC, SS and FG reviewed and approved the comment.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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