Imaging the preterm infant's brain

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Chapter 2

Using cerebral ultrasound effectively in the newborn infant

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Abstract

Cranial ultrasound is the most available and easily repeatable technique for imaging the neonatal brain. Its quality and diagnostic accuracy depends on various factors; the suitability of the ultrasound machine for neonatal cranial work, the use of optimal settings and probes, appropriate scanning protocols, the use of a variety of acoustic windows and, not least, the scanning experience of the examiner. Knowledge of normal anatomy and the echogenicities of different tissues in normal and pathological situations as well as familiarity with the physiological and pathological processes likely to be encountered is vital. This paper assesses the value and appropriate use, safety and diagnostic accuracy of modern, high-quality ultrasound in evaluating the brain of the preterm and term-born infant. Issues of concern regarding teaching, supervision and experience of the examiner are also addressed.

Introduction

Cranial ultrasound (cUS) is the most readily available and easily repeatable technique for imaging the neonatal brain. In contrast to other neuro-imaging tools such as magnetic resonance imaging (MRI) and computed tomography (CT), it can be done bedside with little disturbance to the infant. Neonatal cUS has been used for over 25 years and early studies on intraventricular (IVH) and parenchymal haemorrhage (HPI), post-haemorrhagic ventricular dilatation and cystic periventricular leukomalacia (PVL) have helped greatly our understanding of risk factors for neurodevelopmental abnormalities. Advances in technology have improved the quality of cUS imaging such that it can be a reliable tool for following brain development and showing the most frequently occurring forms of cerebral injury in the preterm and term-born infant brain. The range of cUS diagnoses has increased with the recognition of more subtle patterns of injury and the appreciation of features suggestive of developmental, metabolic and infectious disorders.

In recent years concerns have been raised that cUS is not able to detect more subtle abnormality in the preterm infant (1-4) and that it is not reliable for detecting lesions in the term infant with hypoxic-ischaemic encephalopathy (HIE) and focal infarction. The arrival of MRI with its multislice coverage of the whole brain and large range of sequences has led a very negative press as far as cUS is concerned. This negativity is not well supported by comparative studies where both techniques receive the same time and expertise regarding image acquisition and analysis (5-6).

The quality of cUS imaging and its diagnostic accuracy, as with any other imaging technique, depends on many factors. These include not only the suitability of the equipment for neonatal cranial work and the use of appropriate settings and probes, but also scanning at appropriate times depending on the pathology being sought, the use of different acoustic windows and not least the experience and expertise of the examiner.

This review assesses the value of appropriate timing of modern, high-quality cUS in evaluating both the preterm and term-born infant brain and highlights the current areas of emphasis in this important field of neonatal medicine.

Scanning protocols

There are some technical guidelines on scanning quality and image acquisition (7) but there is no universal agreement about optimal timing for neonatal cUS. Scanning protocols vary considerably between different neonatal units. This variability reflects the different purposes for scanning which include diagnosis, assessing aetiology and predicting outcome, and the population of infants examined, imaging skills and interest of the examiner, the availability of an ultrasound scanner on the neonatal unit and experienced staff, cost-effectiveness and potential hazards.

Preterm infants

Preterm infants have a high risk of hypoxic, haemorrhagic and inflammatory cerebral lesions, mainly IVH, HPI, cystic and non-cystic PVL and more diffuse white matter (WM) injury. Cerebellar abnormality is probably more common than realized (8-11). These findings are of clinical importance, making the use of appropriately timed screening protocols necessary.

Perlman et al. (12) recommended that preterm infants of birth weight < 1000 grams should be scanned between days 3 and 5, 10 and 14, around day 28 and pre-discharge; that those between 1000 and 1250 grams are scanned between days 3 and 5, day 28 and pre-discharge and those between 1250 and 1500 grams between days 3 and 5 and pre-discharge. This recommendation was based on data obtained using this protocol in about 250 preterm neonates. 65% of IVH was detected in the first week after birth, the remaining in the second or third week. Cystic PVL occurred in larger, more mature infants and was not always preceded by increased periventricular echogenicity (PVE). In two out of nine infants it was only detected on the pre-discharge scan. Ventriculomegaly was detected on the initial scan in 50% but only after day 28 in 33%. Significant lesions were detected on the pre-discharge examination in nine infants for the first time. In this study 13% of neonates died in the first 24 hours and were not scanned.

The American Academy of Neurology in 2001 reviewed neuro-imaging strategies for evaluating preterm and encephalopathic term-born infants (13) and recommended practice parameters for imaging of the newborn infant brain. They suggested that in preterm infants < 30 weeks' gestational age (GA), routine cUS should be performed

once between 7 and 14 days of age and optimally repeated between 36 and 40 weeks' postmenstrual age. They argued that with this strategy lesions that influence clinical care (e.g. IVH) and provide information about long-term outcome (e.g. PVL and ventriculomegaly) would be detected.

Both these scanning protocols give rise to concern. Neither recommends scanning on admission, which is essential for detecting lesions of antenatal onset (14). Their recommendations were based on their existing protocol and thus they do not know whether a more extensive protocol would have detected more abnormalities. The protocol by Ment et al. (13) is limited to preterm infants < 30 weeks' gestation with no recommendations for older preterm infants.

Pierrat et al. (15) used frequent cUS examinations and compared the evolution of localized and extensive cystic PVL with neurodevelopmental outcome. Localized cysts developed after the first month from birth in more than half of the infants and were often no longer visible around term equivalent age (TEA). De Vries et al. (16) in a tertiary unit setting, used high-resolution, sequential cUS in preterm infants for predicting cerebral palsy (CP). Infants were divided into those < 32 weeks' GA and those between 33 and 36 weeks' GA, and scanned weekly until discharge and once more at TEA. cUS detected abnormalities in the majority (94%) of children in whom CP was evident by 2 years. The risk for CP was the same for both groups of infants. Infants of 33 to 36 weeks' GA are a population of increasing neurodevelopmental concern that frequently are not scanned at all in the UK. In 29% of infants < 32 weeks' GA cystic PVL, the most predictive marker for CP, was only detected after 28 days. This data gives evidence that cUS can detect most lesions leading to CP if scanning is performed frequently until discharge and again around TEA and supports the need for scanning all gestation preterm infants admitted to a neonatal unit.

Evidence of suboptimal brain growth or atrophy is frequently present in preterm infants, particularly around TEA. Horsch et al. (17) related signs of brain atrophy (enlarged extracerebral spaces (ECS), widened interhemispheric fissure (IHF), reduced complexity of gyral folding) on sequential cUS of preterm infants with neurodevelopmental outcome at 3 years of age. Infants without major lesions but with signs of atrophy at discharge had significantly poorer neurocognitive outcomes. A recent paper by Anderson et al. (18) measuring the length of the corpus callosum and cerebellar vermis

has shown early changes that inform the timing of the effects of preterm birth on brain development – information that would be difficult to obtain by other imaging means in such a large group.

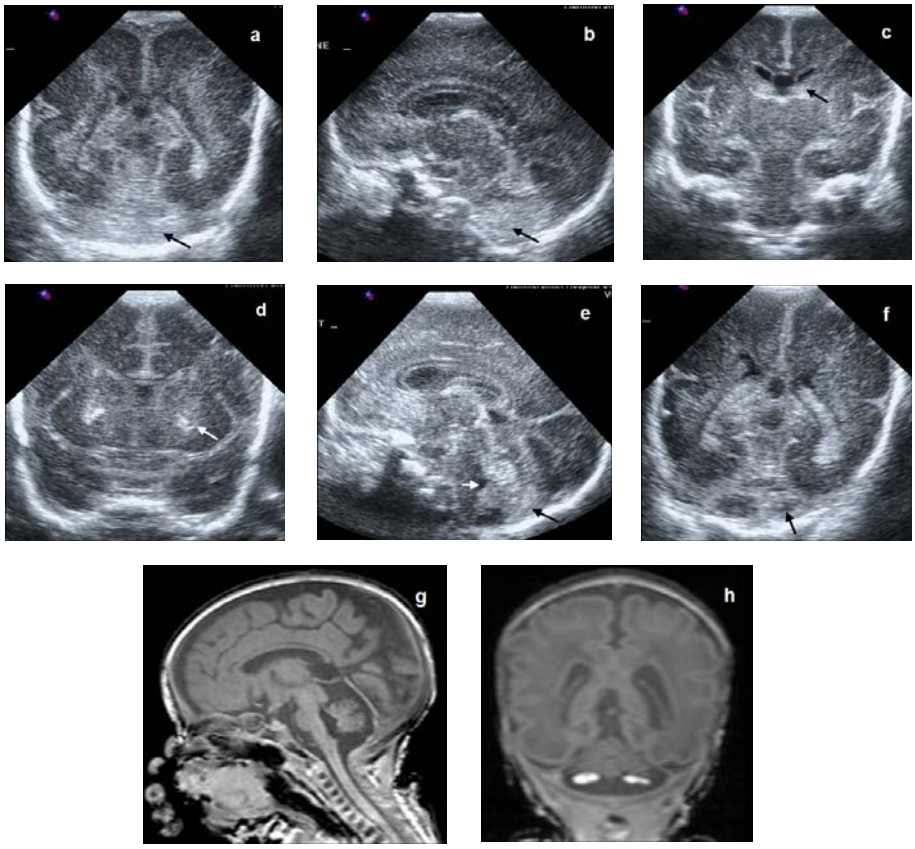
Based on these studies and the population admitted to our tertiary neonatal unit, we developed a scanning protocol for preterm infants (Table 1). All infants admitted to the unit have a cUS scan done by the paediatric specialist registrars (middle grade staff) and reviewed the next day on the ward round. The purpose of this admission scan is to ascertain that neurodevelopmental anatomy looks normal, there are no features to suggest congenital infection, metabolic disease or long-standing brain injury and also to identify recently evolving problems such as haemorrhage or WM echogenicity. Equally important is to note that the scan appears normal at this time.

Table 1. cUS scanning protocol for preterm infants used at the neonatal unit of the Hammersmith and Queen Charlotte's Hospital, London – a tertiary referral centre

	Gestational age at birth (weeks)			
	23-26	27-29	29-32	32-35
Postnatal age at which cUS should be performed	1, 2 and 3 days	Day 1	Day 1	Day 1
	1 week	1 week	1 week	1 week
	2 weeks	2 weeks		
	Weekly to 31 weeks	Weekly to 31 weeks	3 weeks	3 weeks
	Alternating weeks to 36 weeks	At 36 weeks		
	Term	Term	Term	Term

On the early cUS scans special attention should be paid to the presence of IVH and periventricular flaring, while the later cUS scans identify evolving parenchymal lesions (cystic PVL or HPI), the development and progression of post-haemorrhagic ventricular dilatation and signs of atrophy (enlarged lateral or 3rd ventricles especially without evidence of IVH, irregular ventricular margins, a thinned rim of WM around the lateral ventricles, a thin corpus callosum and widened IHF and ECS and changes in the cerebellum) (Figure 1). When assessing the significance of enlarged ventricles and ECS it is necessary to measure the head circumference (HC) and review the pattern of head growth. A small group of infants develop an enlarged ECS associated with increased HC centiles and these children are of less concern.

Figure 1. Series of ultrasound images from a 24-week intrauterine growth restricted infant. Parts a and b: Day 2 showing abnormal echogenicity and loss of definition of the cerebellum suggestive of haemorrhage (arrows); parts c and d: 4- to 6-week scans showing the development of echogenicity in the caudo-thalamic notch (c, arrow) and lenticulostriate vasculopathy (d, arrow); parts e and f: 8-week scans showing a widened 4th ventricle (short arrow) and persisting abnormal echogenicity behind the cerebellum (e, long arrow) and areas of low echogenicity within small cerebellar hemispheres (f, arrow).
MRI scans (parts g and h) at term equivalent age showing an obvious 4th ventricle, small vermis and cerebellar hemispheres with haemorrhage and also an increased interhemispheric fissure and widened extracerebral space (also seen on cUS, images not shown). All the abnormality that is seen on the MRI was seen on cUS. Additionally the cUS showed the development of echogenicity in the caudo-thalamic notch and the lenticulostriate vasculopathy not seen on the term MRI.



In addition to detecting lesions, assessing their site and extent is important for accurate prediction of outcome. In preterm infants, unilateral HPI located at the trigone is associated with a less favourable outcome than lesions in the fronto-parietal region (19). Multiple PVL cysts in the (parieto-)occipital but not the frontal regions are highly predictive of adverse motor outcome (20). Even when cUS is performed optimally, unexpected cases of CP occur. This may in part be due to cerebellar lesions (21) which can be difficult to detect or to subtle injury that has occurred antenatally; awareness of the importance of assessing the posterior fossa structures and scanning through acoustic windows other than the anterior fontanel may be needed (see below).

Stroke occurs in preterm infants (22-23), often affecting the central grey matter, and may only be detected by careful sequential scanning. It is more common in those with congenital heart disease and twins (particularly with twin-to-twin transfusion syndrome), a high risk group that should all have a postnatal cUS scan. It is also important to scan sequentially preterms who become sick or have unexpected postnatal events and also to scan prior to surgery in case of complications (Table 2). Unusual patterns of WM echogenicity and the appearance of cysts may reflect infections in the central nervous system. These include fungal infections, viruses (24-25) and a range of bacterial infections (26). These data apply both to preterm and term-born infants.

Table 2. Clinical indications for additional scans to a standard protocol (CSF, cerebrospinal fluid)

A sudden deterioration in clinical state
A sudden increase in the need for ventilatory support
Necrotizing enterocolitis
Repeated episodes of apneas and/or bradycardias
A sharp fall in haemoglobin level
Onset of seizures or change in neurological status
Increasing ventricular dilatation
Abnormal head growth
Before and after lumbar puncture and for drainage of CSF
Before and after surgery

Term-born infants

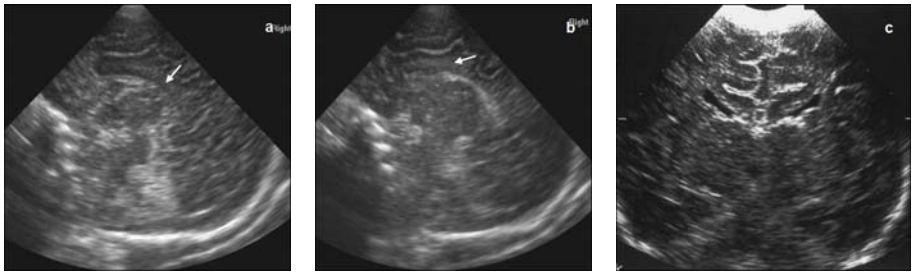
In term-born infants, HIE and stroke are still the major causes of, respectively, diffuse and focal brain injury and neurological morbidity.

Hypoxic-ischaemic encephalopathy

cUS is often considered not very helpful in detecting lesions occurring with HIE or predicting outcome, as the initial swelling makes focal lesions difficult to detect and precisely locate. The American Academy of Neurology (13) recommended that in encephalopathic term infants a CT should be performed to detect haemorrhagic lesions and if findings are inconclusive, MRI should be performed between days 2 and 8 to assess the location and extent of injury. They give no data supporting the diagnostic value of cUS in HIE.

cUS in term-born infants with suspected HIE has several roles. Scanning as part of the admission procedure identifies congenital structural cerebral abnormalities, detects evidence for long-standing or more recently established injury initiated prior to the onset of labour, and detects abnormalities characteristic of non-HIE causes of encephalopathy, e.g. a hypoplastic corpus callosum suggesting a diagnosis of non-ketotic hyperglycinaemia (Figure 2) and germinolytic cysts suggesting mitochondrial or peroxisomal disorders or congenital infection. This information is clearly important for early diagnosis and management. Additionally, with cUS, the evolution of intrapartum injury can be followed and apart from clinical implications this information is very important in medicolegal issues.

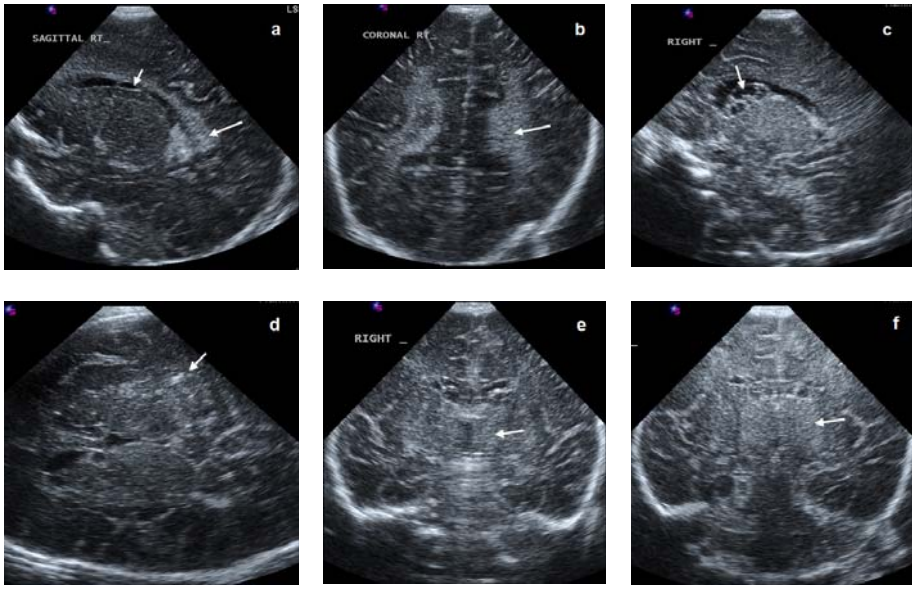
Figure 2. Hypoplastic corpus callosum (a, arrow) and mildly echogenic white matter (b, arrow) in an encephalopathic term infant with non-ketotic hyperglycinaemia. Typical widely spaced ventricles from another patient with non-ketotic hyperglycinaemia (c).



Our protocol for scanning infants with suspected HIE is that a scan should be performed on admission, at 24 hours after birth, days 3 to 4, days 7 and 14, and once again in clinic at 4 to 6 weeks.

The initial cUS, mainly performed to exclude other causes of encephalopathy, by identifying any abnormal anatomy, evidence for established damage (e.g. presence of calcium, marked echogenicity and parenchymal echolucency), lenticulostriate vasculopathy, germinal matrix cysts, diffuse WM echogenicity (Figure 3) or signs of recent haemorrhage. In our experience, haemorrhage is well seen on cUS and the need for CT is restricted to infants with a traumatic assisted delivery, where a midline shift is seen on cUS and neurological intervention is considered. Data from Eken et al. (27) show that the majority of cUS scans in the first 6 hours are normal in HIE but if they are already abnormal the outcome is poor.

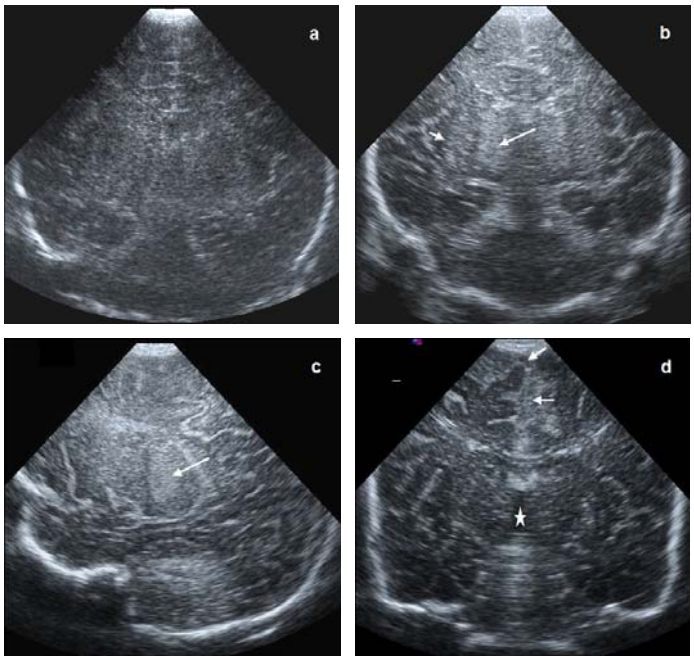
Figure 3. Term infant with evidence of acute asphyxia, retroplacental clot and severe encephalopathy. Initial cUS scans showed small germinolytic cysts (a, short arrow) and an unusual pattern of periventricular white matter echogenicity (a and b, long arrows). Scans at 10 days show enlarging cysts (c, arrow), and specks of calcification (shown on the axial view taken via the mastoid window) (d, arrow). Evolving acute lesions typical of hypoxic-ischaemia particularly to the basal ganglia are seen in parts e and f (arrows). There was a history of an acute gastro-intestinal illness associated with eating uncooked foods in France at 33 weeks' gestational age. No evidence of a viral or metabolic disorder was confirmed; the clinical course is that of a dystonic spastic quadriplegia consistent with the basal ganglia lesions seen in parts e and f.



Acute changes on cUS suggestive of HIE may include diffuse brain swelling on days 1 to 2 (Figure 4, part a), though for infants with only a relatively short hypoxic insult swelling may not be seen. There follows a loss of normal tissue differentiation and increasing echogenicity of basal ganglia (BG) and thalami and WM over the next 3 to 4 days. The cortex often appears very echolucent compared to the adjacent hyperechogenic WM and sulci (Figure 4, part c) in the first days but later may become hyperechogenic if severely damaged (Figure 4, part b). BG and thalamic echogenicity typical of an acute hypoxic-ischaemic insult is usually bilateral, increases daily and has a characteristic appearance (Figure 4, parts b and c). Generalized bilateral WM echogenicity is suggestive of a more long-standing subacute insult; both central grey matter and WM are involved in more severe acute or mixed insults when additionally the cortex appears abnormal. Milder changes can be subtle and are often not seen before 7 days after the insult. With severe widespread injury, the WM becomes increasingly echogenic and then breaks down into cysts. In a recent study by our own group reviewing sequential early cUS of 139 term-born infants with mild-severe HIE (unpublished data), we found that the presence of abnormal echogenicity in the BG and the visualization of the internal capsule, seen as an echolucent line running through the BG region (Figure 4, parts b and c), were highly predictive of an abnormal motor outcome.

Late cUS findings after focal BG lesions include persisting echogenicity in the deep grey matter and dilatation of the 3rd ventricle, signs significant for motor outcome (Figure 4, part d). Widening of the IHF suggest poor hemispheric growth and is associated with poor cognitive outcome. These signs present on later scans are of concern despite apparent clinical improvement in the first few weeks after birth.

Figure 4. Part a (coronal cUS scan): Acute swelling with loss of grey-white matter differentiation. Parts b and c (coronal and parasagittal cUS scans, respectively): Evolving abnormality in the lentiform nucleus (short arrow) and the thalamus (long arrow) with a line of low signal indicating the site of the internal capsule. Part d (coronal cUS scan): Widening of the 3rd ventricle (asterisk), and enlargement of the interhemispheric fissure and the extracerebral space (arrows) at 6 weeks.

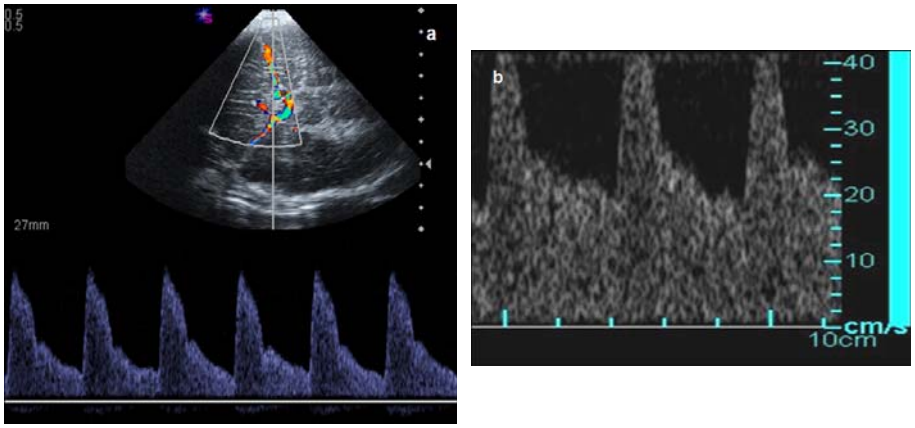


Doppler ultrasound gives useful information in HIE (28). Measurements can be made at the time of the cUS imaging; it is not critical which intracerebral vessel is used – usually the anterior cerebral artery via the anterior fontanel or the middle cerebral artery via the temporal window (Figure 5, part a). Two parameters can be assessed, the total flow velocity (CBFV) and the pulsatility index (PI) (confusingly referred to as the resistance index (RI) in obstetric papers). Normal values for the PI are between 0.65 and 0.85.

$$PI = \frac{\text{peak systolic velocity – the end-diastolic velocity}}{\text{peak systolic velocity}}$$

Abnormal values < 0.55 occur in severe HIE usually between days 2 and 4 after birth (Table 3) (Figure 5, part b). The outcome of infants with abnormal values in the first 6 hours after birth is very poor and suggests a prolonged insult either intrapartum or 1 to 2 days prior to delivery (27). The absolute flow velocities are more difficult to assess as they require the angle of insonation on the vessel to be near to zero and the values vary with the vessel measured on. However, high values carry a poor prognosis and are associated with a low PI.

Figure 5. Part a: View through the temporal window enabling visualization of the middle cerebral artery; below are normal Doppler spectra with a pulsatility index of 0.8. Part b: Abnormal Doppler spectra in hypoxic-ischaemic encephalopathy with a pulsatility index of 0.5.



Doppler measurements are useful in assessing venous flow especially in the straight sinus in suspected venous thrombosis (often associated with IVH or thalamic haemorrhage) (29) and to document abnormal velocity patterns in suspected vascular malformations.

We find cUS, both imaging and Doppler, of great use in excluding non-HIE diagnoses and in following the timing and evolution of acute injury. cUS together with electrophysiological data and clinical progress often allow early prognosis and appropriate clinical management in a situation where it is not possible to obtain a MRI scan. However, there is no doubt that a MRI in the first 1 to 3 weeks is superior for defining lesions and giving more precise long-term prognosis in HIE but the value of

cUS should not be underestimated in the first days after birth and for monitoring at 4 to 6 weeks when a normal cUS scan, clinical examination and head growth are good prognostic indicators.

Table 3. The predictive value of the pulsatility index and cerebral blood flow velocities for adverse neurological outcome in hypoxic-ischaemic encephalopathy in term-born infants (CBFV, cerebral blood flow velocities; PI, pulsatility index; PPV, positive predictive value)

Measurements made 2-4 days after birth - Levene et al. (28)		
	Abnormal CBFV	Low PI < 0.55
Sensitivity	57%	60%
Specificity	88%	63%
PPV	94%	83%

Early (< 6 hours) measurements - Eken et al. (27)		
	Sensitivity	Specificity
Ultrasound	42.1%	60%
Doppler	23.5%	100%

Infant with seizures

Term infants with early neonatal seizures who had fairly normal Apgar scores and were not acidotic at birth are most likely to have had a neonatal stroke; other more common diagnoses are parasagittal infarction and focal haemorrhage. Neonatal stroke has an estimated prevalence of at least 1 / 4000 live births. It is an important cause of hemiplegic CP and other neurological disabilities. cUS is often said to have a poor sensitivity for detecting focal infarction. We found that early cUS (days 1-3) showed abnormalities suggestive of infarction in 68% of cases and in 87% when cUS was performed after day 4 (5). Infants whose late cUS scans remained normal had relatively small, mostly posterior infarcts; none of these infants developed a hemiplegia. Thus, neonatal cUS does allow the detection of abnormality suggestive of focal ischaemic lesions, particularly those likely to lead to hemiplegia.

We perform cUS scans on infants presenting with seizures on admission and then at 2 days, 5 to 7 days and 2 weeks after the onset of seizures. Special attention should be paid to presence of subtle asymmetrical parenchymal echogenicity, a wedge-shaped echogenic area suggestive of middle cerebral artery (MCA) infarction (Figure 6), a

rounded very echogenic lesion suggestive of haemorrhage (Figure 7) and any abnormal structural development suggestive of a developmental abnormality. We suggest however that any infant with seizures a neonatal MRI examination is performed; precise detail of lesion size, location and prognosis are better defined on MRI (30-31) allowing more precise prognosis.

Figure 6. Full-term infant with neonatal seizures. Large wedge-shaped area of echogenicity on the right including the basal ganglia (arrows) is shown (a, b and c, parasagittal cUS scans), typical of a middle cerebral artery territory stroke. MRI confirms the diagnosis; T_1-weighted (d), diffusion-weighted (e) and follow-up (f) scans.

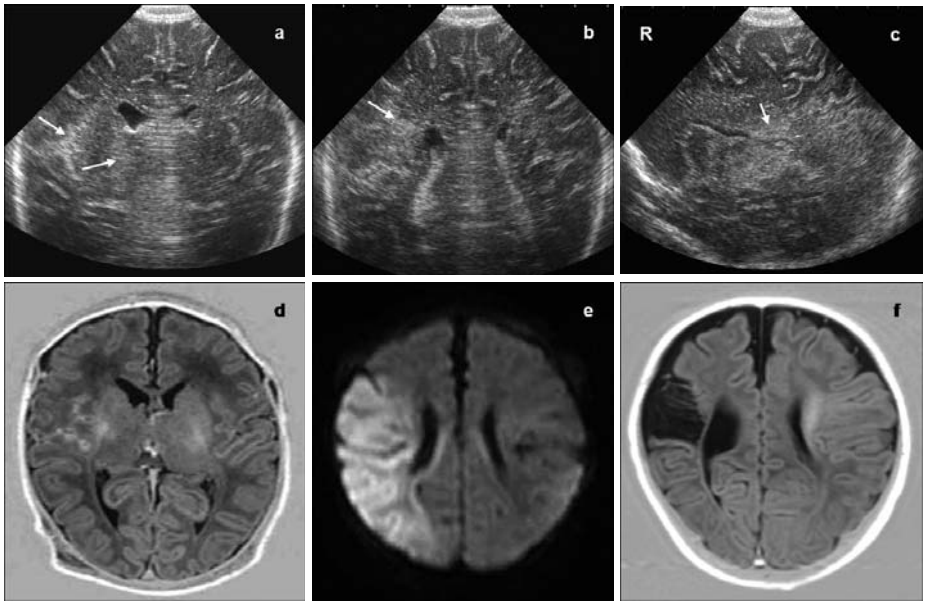
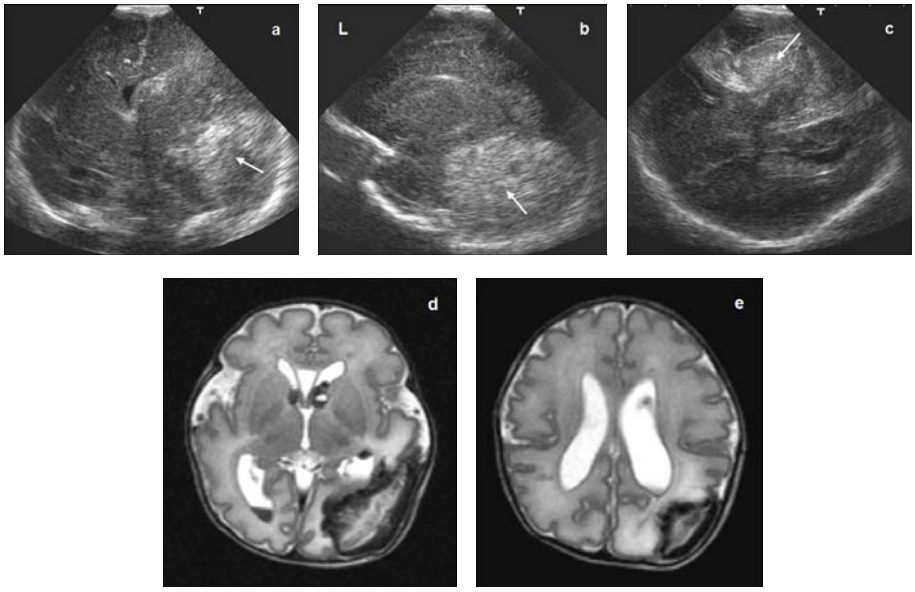


Figure 7. cUS in coronal (a), parasagittal (b) and mastoid view (c) from a 31-week infant with low platelets showing a large recent focal haematoma (long arrows) with a midline shift (a, short arrow). 20 cm^3 of blood was removed by subdural drainage. MRI (d and e) following tap.



The dysmorphic or floppy infant and those with metabolic disorders

Dysmorphic features and the floppy infant syndrome can be indicators of developmental disorders and congenital infection, genetic/chromosomal syndromes, congenital or acquired disorders of the central nervous system and neuromuscular and metabolic disorders (32). The diagnostic work up is often complex and recognition of characteristic cUS findings may aid the diagnostic process. When evaluating the cUS scans attention should be paid to signs of abnormal anatomy, calcification, germinolytic cysts and established injury; ventriculomegaly is common in myotonic dystrophy. As outlined in the section on HIE, term symptomatic infants with germinolytic cysts, lenticulostriate vasculopathy, unusual gyral patterns, abnormal appearances to the cerebellum, diffuse mild increase in WM echogenicity, hypoplasia of the corpus callosum should be investigated for metabolic disorders.

Acoustic windows

The standard acoustic window used for imaging the neonatal brain is the anterior fontanel. However, the cerebellum, brainstem and posterior subcortical WM may be poorly visualized using this approach. The detection of cerebellar abnormality via the anterior fontanel is complicated by the echogenic appearance of the tentorium and cerebellar vermis. The cerebellum is increasingly recognized as an important structure not only for motor control but also for cognitive and behavioural development. Abnormalities due to haemorrhage and infarction and poor growth (8-10) have gone under-recognized. Scanning through the posterior fontanel (junction of the lambdoid and sagittal sutures) and mastoid fontanels (junction of the posterior parietal, temporal and occipital bones) can help to detect lesions and structural malformations in these areas (Figure 7) (19,21,33-34). Imaging through the temporal window allows good views of the mesencephalon and brainstem.

Safety

Although sequential cUS is clearly very important, its potential hazards and burden for the often sick and unstable newborn infant should be kept in mind. These include extra handling, applying pressure and cold gel to the fontanel, the risk of dislodging tubes or lines or introducing infection from equipment that is not kept clean or from the operator. Most of these issues are relatively easy to prevent when appropriate safety and hygienic precautions are taken. All ultrasound equipment for use on a neonatal unit must be kept regularly cleaned, the probes wiped with a damp cloth. We also use hard surface wipes that contain some alcohol but these are not suitable for all probes and the manufacturers should be consulted.

There are also concerns about potential hazards of the ultrasound waves. To date no adverse effects have been shown to result from the sequential use of ultrasound in newborn infants or fetuses, although a slightly increased risk of delayed speech (35), left handedness (36) and intrauterine growth restriction (37) after multiple fetal examinations has been reported. However, ultrasound waves, especially when used

for colour Doppler imaging, may induce temperature elevations in human tissue over time that have the potential to cause damage. It is important to keep the duration of exposure as short as possible. The British Medical Ultrasound Society (BMUS) has published guidelines for diagnostic ultrasound (www.bmus.org). The power used should be kept within the guidelines with the default setting as low as possible compatible with obtaining diagnostic images.

Teaching and experience and accuracy of cUS

Many studies have cast doubt on the accuracy of cUS for detecting cerebral abnormalities predictive of unfavourable outcome. Some have suggested that only in 40-50% of preterm infants with CP (38-39), lesions were detectable on cUS though this contention is not supported by the study of de Vries et al. (14). Few studies have assessed the skills of the examiner and interpreter. In a study comparing cUS and MRI (4), no major lesions were missed on cUS (1-3) though subtle WM change on cUS and MRI were not consistent; the clinical significance of both observations is still not fully understood.

In a survey in the Greater London area in 2000, 56% of those interpreting neonatal cUS were middle grade neonatal registrars, only 44% of whom had any formal training in cUS (40). Assessing the abilities of the doctors in this survey, including consultants, to interpret important cerebral abnormalities on high-resolution cUS images, the mean accurate identification of abnormality was only 59%. In a study in the West Midlands evaluating the training of paediatric specialist registrars in cUS, 26% had never carried out supervised scans; 51% lacked confidence in performance and 57% in interpreting scans (41). Harris et al. (42) recently compared the cUS findings of three reviewers with previously reported rates of WM damage by six neonatal units in New Zealand. They showed that there was only moderate agreement between the reviewers' reports and those of the neonatal units and between the reviewers; the reviewers reported 3 to 6 times more WM damage.

These studies highlight the need for more widespread formal training in scanning techniques and that teaching of cerebral anatomy, the appearance of normal structures,

an understanding of normal growth as well as knowledge of the appearances and evolution of different pathologies are essential for those responsible for acquiring and interpreting cUS scans. These skills need to be assessed and competencies maintained. Increased collaboration between neonatologists, radiographers and radiologists with an interest in neonatal scanning and prognosis is needed in order to improve and rationalise the use of this valuable technique.

Conclusions

Current evidence is that cUS imaging using modern machines, probes, a variety of acoustic windows and sequential scanning at optimal times gives high-quality images that are diagnostically accurate. Issues of teaching, supervision and experience for both paediatric and radiological staff need to be addressed and collaboration between paediatric and radiology departments is needed to improve protocols, image quality and interpretation. Whilst MRI does have advantages over cUS and clear clinical indications, especially in the term-born infant, cUS facilitates early bedside diagnosis and monitoring of pathology in a way that is relatively easy and not disturbing and safe for the newborn infant. Awareness of the timing of injury and its manifestation on cUS are vital and routine scanning must be of high-quality with maximal coverage of the whole brain, and appropriate to the population of infants under review.

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