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Magnetic resonance imaging of the newborn brain

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KEYWORDS
chronic pelvic pain, conscious pain mapping, pelvic congestion, nerve entrapment, LUNA, trigger points, adhesions

Summary Magnetic resonance (MR) is an ideal tool for imaging the neonatal brain, although adaptations may need to be made to both hardware and sequences. It is useful for assessing many diseases in the immature brain including hypoxic–ischaemic encephalopathy, infarction and infection, examples of which are given in this review. A thorough knowledge of the normal MR imaging (MRI) appearances at different gestations as well as the variation with different sequences is necessary for correct scan interpretation. The imaging appearance of lesions in the newborn brain depends on the timing and nature of the insult and the pulse sequence used. Techniques such as diffusion-weighted imaging provide important early information in ischaemic lesions. Quantitative MRI techniques, such as three-dimensional volumetric MRI, can be used to provide objective assessment of brain development in both health and disease.

PRACTICE POINTS

- MR imaging is useful for assessing many diseases in the neonatal and preterm brain including hypoxic–ischaemic encephalopathy, infarction and infection
- The imaging appearance of lesions in the newborn brain depends on the timing of the insult and the pulse sequence used
- A thorough knowledge of the normal appearances at different gestations as well as the variation with different sequences is necessary
- Quantitative MR imaging techniques, such as three-dimensional volumetric MRI, can be used to provide objective assessment of development and disease progression

INTRODUCTION

Magnetic resonance (MR) imaging is non-invasive and non-ionizing and provides excellent soft tissue differentiation, making it the modality of choice for investigating numerous diseases of the brain. Its use in adults is established, and an increasing number of centres are now using it to investigate the newborn brain.

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MR PULSE SEQUENCES

The term neonatal brain contains approximately 92–95% water and this decreases over the first 2 years of life to adult values of 80–85%. The high water content of the neonatal brain is associated with a marked increase in T1 (longitudinal) and T2 (transverse) relaxation times in comparison to adults. As a result the pulse sequences need to be adjusted to allow for the different MR properties of the immature brain.

We routinely use a T1 weighted conventional spin echo sequence (CSE) (TR 860/TE 20 ms) (TR—repetition time, TE—echo time), a T2 weighted CSE (TR 2700/TE 120 ms) or a T2 weighted fast spin echo (FSE) sequence (TR 3500/TE_{eff} 208 ms) and an inversion recovery (IR) sequence (TE 3800/TI 950/TE 30 ms).

In addition to standard T1 and T2 weighted imaging, which are used for qualitative image assessment, quantitative MR techniques are available. Three-dimensional (3D) volumetric MR imaging allows measurement of whole brain volume, as well as different structures such as the ventricles and cerebellum. This technique can provide data to identify subtle deviations from normal[1–3] and change in brain size over time.[4] MR imaging can also be used to assess physiology, with techniques such as diffusion (DWI) and perfusion weighted imaging (PWI). DWI is able to demonstrate areas of acute infarction before they are visualized with conventional MR imaging. As in

the adult patient, intravenous contrast enhancement should always be used in cases of suspected infection. Using T1 weighted imaging, contrast enhances areas where there is a breakdown in the blood–brain barrier, such as some parenchymal lesions and infected meninges.

TIMING THE MR IMAGING EXAMINATION

The ideal time to image depends on the information required but is often constrained by the resources available for imaging sick neonates. Conventional scans performed within the first 24 h may appear normal even when there has been severe perinatal injury to the brain. Early imaging will help to differentiate antenatal from perinatal lesions. Perinatally acquired abnormalities 'mature' and become easier to identify by the end of the first week. For information on the exact pattern of injury a scan between 1 and 2 weeks of age is usually ideal. After 2 weeks there may be signs of cystic breakdown and atrophy, which may make the initial pattern of injury more difficult to define.

INFANT PREPARATION FOR SCANNING

Neonates can often be scanned without sedation during natural sleep. Infants over the age of 3 months almost always require sedation and we usually use chloral hydrate orally or via suppository. It is rarely necessary to use general anaesthesia to image a child under 2 years old. All neonates and infants undergoing MR scanning are monitored with ECG and pulse oximetry and a paediatrician is in attendance throughout. Infants who require assisted ventilation can be imaged in an MR scanner providing MR compatible ventilation and monitoring equipment is available. As with all MR examinations, a thorough metal check is essential before imaging, and particular neonatal metallic hazards need to be considered, such as Searle arterial lines, electronic name tags and portable oxygen cylinders.

MR IMAGING OF THE NORMAL NEONATAL BRAIN

In the neonatal brain, unmyelinated white matter (WM) has a low signal intensity (SI) on T1 weighted images and high SI on T2 weighted images. Cerebrospinal fluid (CSF) is hypointense on T1 weighted imaging and hyperintense on T2 weighted imaging (Fig. 1).

Myelination begins at around 20 weeks gestational age (GA) and continues up to around 2 years of age. As myelination proceeds, the water content of WM decreases, causing a reduction in SI on T2 weighted imaging. There is a corresponding increase in glycolipids, cholesterol and proteins, which causes an increase in SI on T1

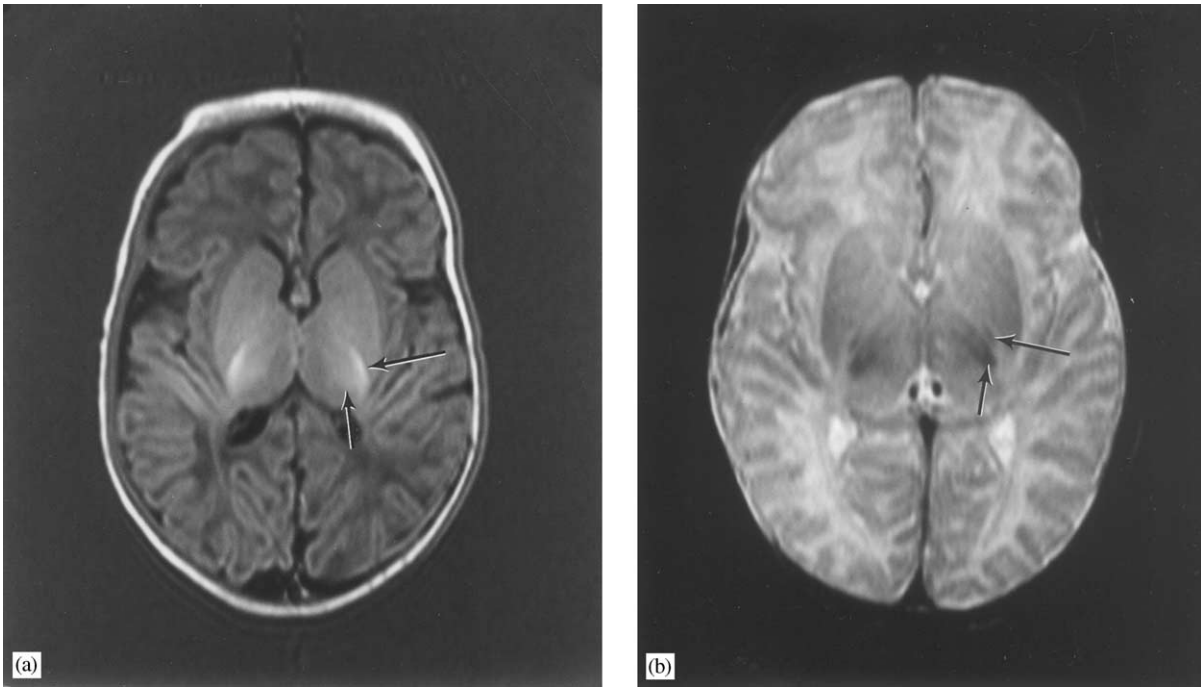

Figure 1 Normal appearances of the brain at term. Inversion recovery sequence (IR 3800/30/950 ms). (a) There is high signal in the posterior limb of the internal capsule (long arrow) and in the region of the ventrolateral nuclei of the thalami (short arrow). (b) T2 weighted imaging (SE 2700/120 ms). The myelin in the internal capsule has a low signal intensity (long arrow). There is also low signal intensity in the region of the ventrolateral nuclei of the thalamus (short arrow).

weighted imaging.[5] Changes in the SI of WM due to myelination are demonstrated at different ages on T1 and T2 weighted imaging. T1 weighted imaging is better at demonstrating myelination in the first 6–8 months after birth and T2 weighting is better between 6 and 18 months.[6]

MR IMAGING OF THE NORMAL PRETERM BRAIN

The cerebral cortex is demonstrated as high SI on T1 weighted imaging and low SI on T2 weighted imaging. The very premature brain has little sulcation and gyration at 24 weeks GA but this rapidly evolves. On T2 weighted imaging prior to 30 weeks GA, bands of low SI are visible within the cerebral WM, around the lateral ventricles,[7] representing glia migrating from the germinal matrix to the developing cerebral cortex[8] (Fig. 2). Areas of extremely high SI on T2 weighted FSE images are visualized around the anterior and posterior horns of the lateral ventricles between 24 and 36 weeks GA[7] (Fig. 2). Histologically, these extremely high SI areas are comprised of dense fibre bundles, which are less organized than elsewhere in the white matter of the centrum semiovale, and have a relatively low cellular density.[9]

The germinal matrix (GM) is visible up to around 32 weeks GA as a prominent structure at the margins of the lateral ventricles (Fig. 2). After this age, small residual areas of germinal matrix are visualized at the anterolateral angles of the lateral ventricles, adjacent to the head of the caudate nucleus and in the roof of the temporal horns. GM is demonstrated as high SI on T1 weighted imaging and low SI on T2 weighted imaging (Fig. 2).

Myelin has been demonstrated in numerous central structures in the very preterm brain such as the brainstem, cerebellum and thalami.[10] Myelin is not seen in the white matter of the cerebral hemispheres until 35 weeks GA. Quantitative MR imaging studies have demonstrated a steady increase in brain surface area and cortical folding[1] and a reduction in T2 values in the cerebral WM with increasing GA.[11]

MR IMAGING ASSESSMENT OF BRAIN INJURY IN TERM INFANTS

Acute asphyxia

The term infant with a history suggestive of acute asphyxia is at risk of developing hypoxic–ischaemic encephalopathy (HIE). The term 'neonatal encephalopathy' is now considered more appropriate as it reflects the problems in proving that a fetus has suffered a hypoxic–ischaemic event.

Basal ganglia and thalamic lesions

Term infants that develop HIE following a well-defined acute hypoxic–ischaemic insult (e.g. placental abruption,

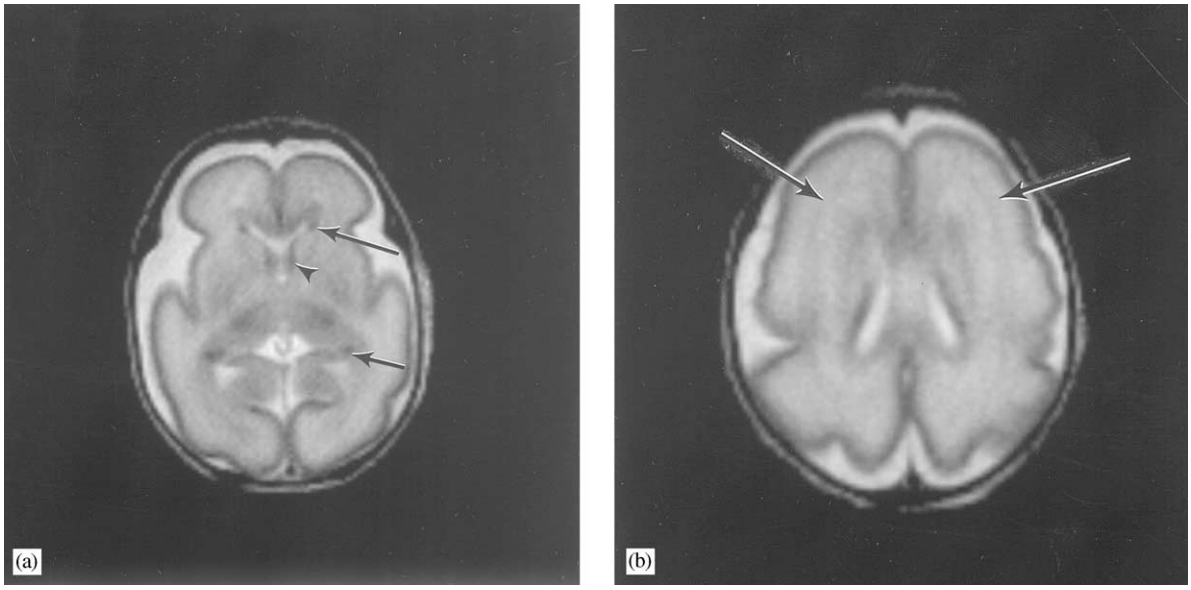

Figure 2 Preterm infant at 26 weeks gestation T2 weighted fast spin echo sequence (FSE 3500/208$_{eff}$ ms). (a) Low ventricular level. There is a relatively smooth cortex. Low signal intensity areas of germinal matrix are seen in the anterior horns (long arrow), over the caudate heads (arrowhead) and in the roof of the temporal horns (short arrow). There are high signal intensity areas in the anterior periventricular white matter. (b) High ventricular level. There are low signal intensity bands (arrows) within the white matter corresponding to cells migrating out from the germinal zones to the developing cortex. There is high SI in the periventricular white matter.

uterine rupture) typically sustain bilateral lesions within the basal ganglia and thalami (BGT).[12]

Occasionally, severe BGT lesions are seen with less obvious precipitating events. This may reflect failure to recognize the severity of asphyxia or an individual susceptibility to damage because of previous hypoxic–ischaemic events or an underlying metabolic or thrombotic disorder. BGT lesions that occur many weeks before term delivery have been described following attempted maternal suicide.[13]

The BGT are susceptible to injury because they are metabolically very active in the immature brain.[14] The changes seen on MR imaging are thought to be secondary to ischaemia although the SI abnormalities are such that they were originally described as haemorrhagic. The short TI and T2 lesions seen as the lesions evolve are probably due to capillary proliferation and later to mineralization.[9]

In term infants with HIE the main sites of abnormality are the posterior and lateral lentiform nucleus and the ventrolateral nuclei of thalami (Fig. 3). More extensive lesions may involve all of the basal ganglia and medial areas of thalami (Fig. 4) and there may be spread downwards to involve the midbrain and the dorsal aspects of the pons and medulla (Fig. 5). Infants with such extensive lesions usually have stage-III HIE and die within days or weeks of birth.

In the term infant, the most clinically useful association with BGT lesions is the SI within the posterior limb of the internal capsule (PLIC). In the normal brain at term, there should be evidence of myelin in the posterior 1/3–1/2 of the PLIC (Fig. 1). Myelination is usually first evident at around 37 weeks gestation and slightly earlier in infants born preterm. Absence of the normal SI in infants older than 37 weeks GA is often seen with severe BGT lesions (Fig. 4) and it is able to predict the neurodevelopmental outcome in term infants with HIE.[15] Its advantage for predicting outcome is that it may appear before abnormal SI within other areas of the brain. However, it may still take up to 48 h to become abnormal in some infants, and the appearances of the abnormal PLIC can vary and may sometimes be difficult to interpret.

Additional abnormalities associated with BGT lesions include early brain swelling, with diminished extracerebral spaces and slit like ventricles. Abnormal, mainly short TI, appearances in the cortex so-called 'cortical highlighting' almost always accompany significant BGT lesions at term. The predominant sites are the central fissure, the interhemispheric fissure and the insula. The depths of the cortical sulci are preferentially affected (Fig. 6). The highlighting is consistent with capillary proliferation occurring as a result of infarction in the deepest layers of cortex. An abnormal SI may take several days to evolve and be seen most clearly during the second week. By this

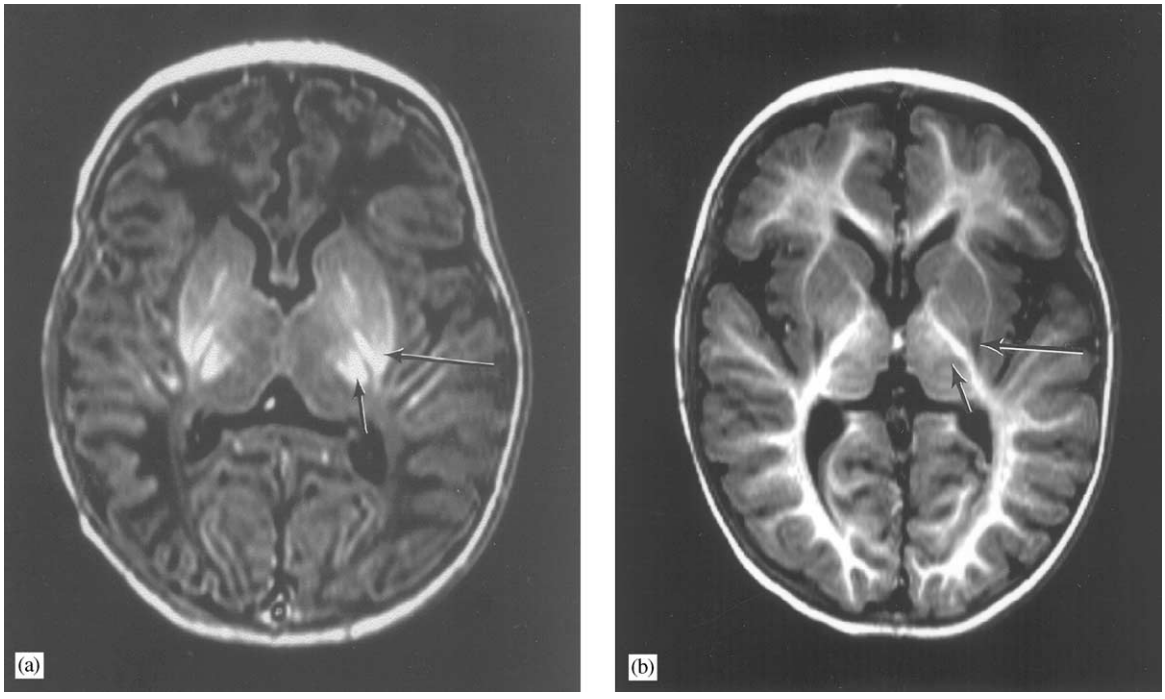

Figure 3 Bilateral basal ganglia (BGT) lesions. (a) Infant with stage-II HIE imaged at 5 d. Inversion recovery sequence (IR 3800/30/950 ms). There are several areas of very high signal intensity within the lentiform nuclei and thalami (arrows). There is some high signal from myelin within the posterior limb of the internal capsule but there is an additional parallel low signal intensity. (b) At 1 year of age, there are characteristic focal low signal intensity cysts in the posterior part of the putamen (long arrow) and in the thalamus (short arrow). This infant developed athetoid quadriplegia.

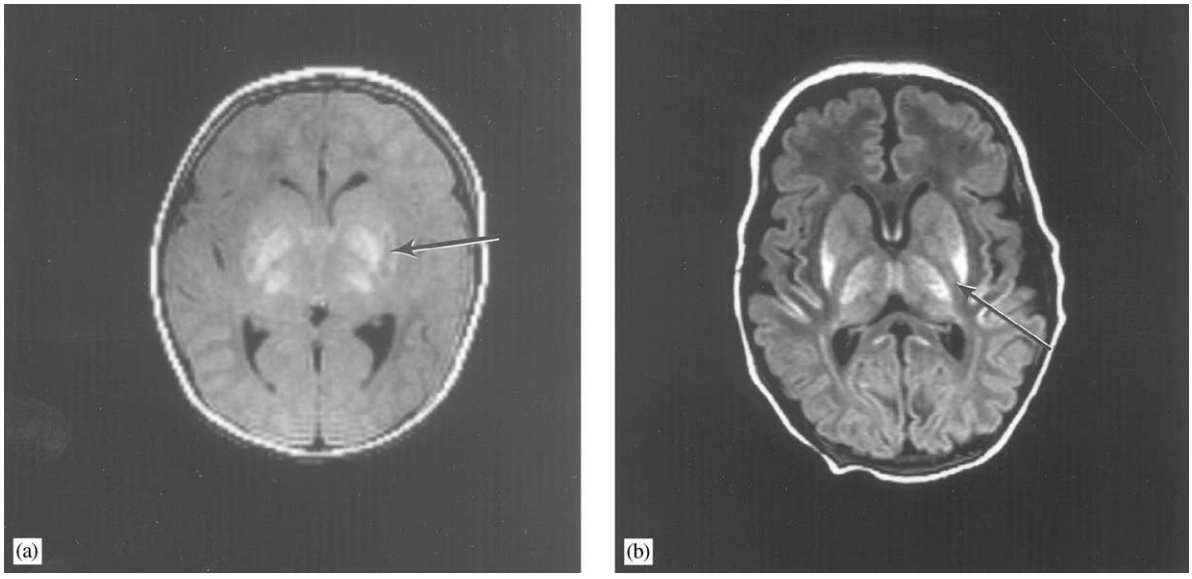

Figure 4 Severe BGT abnormalities. (a) Infant with stage-II HIE imaged at 7 d. There are widespread abnormal high SI throughout the lentiform nuclei and thalami. There are additional low signal intensity cystic areas laterally. (b) Infant with stage-III HIE at 12 d of age. There is abnormal low signal intensity within the posterior limb of the internal capsule (arrow). There are abnormal high signal intensities either side of the PLIC.

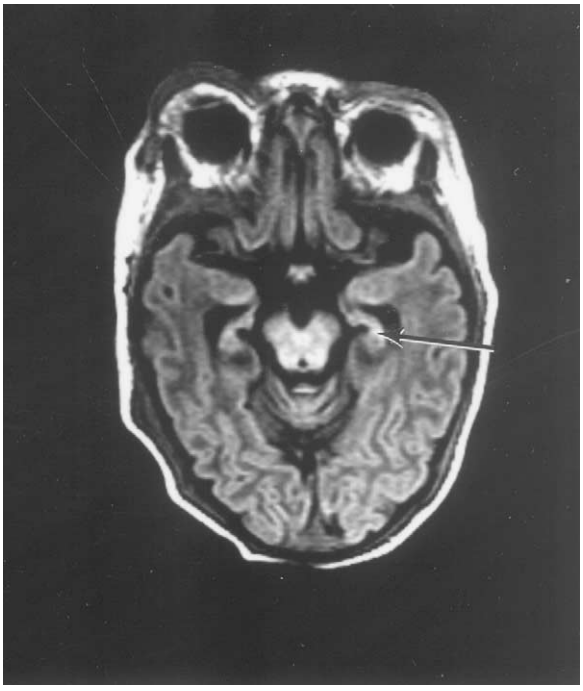

Figure 5 Brain stem and hippocampal abnormalities. Inversion recovery sequence (IR 3800/300/950 ms). There are areas of abnormal high signal intensity within the mesencephalon. There is abnormal high signal intensity in the hippocampal region (arrow). There is already some dilatation of the temporal horn of the lateral ventricle consistent with atrophy. This infant also had diffuse changes throughout the basal ganglia and thalami on imaging and at postmortem.

time the adjacent subcortical white matter often develops abnormal SI consistent with infarction (Fig. 6).

Severe BGT lesions are also associated with abnormalities in the medial temporal lobe. These are not immediately obvious but by the end of the second week, there are often definite short TI areas within the hippocampal region and dilatation of the temporal horn as a result of adjacent tissue atrophy (Fig. 5). In some infants with severe BGT, there are additional widespread abnormalities in the white matter with appearance of infarction giving rise to multicystic leucomalacia (Fig. 7). In infants with HIE who develop both BGT and early white matter infarction, there may be compounding factors that prime the white matter. These factors may include chronic or repetitive ischaemic insults, infection or an inherent susceptibility to ischaemia.

The clinical outcome of the infant is dependent on the severity of the BGT lesions. Severe BGT lesions are associated with a spastic or mixed dystonic/spastic quadriplegia, secondary microcephaly and marked intellectual impairment. There are usually persistent feeding difficulties, which often requires a gastrostomy to avoid long-term nasogastric tube feeding. These children make little developmental progress and may have seizures, which are difficult to control. Early death is common, usually as a result of respiratory complications. In infants with a combination of BGT and multicystic leucoencephalopathy (Fig. 7), the neurodevelopmental outcome is determined mainly by the severity of the BGT, particularly in terms of motor impairment. Less severe BGT lesions (Fig. 3) are associated with the development of an

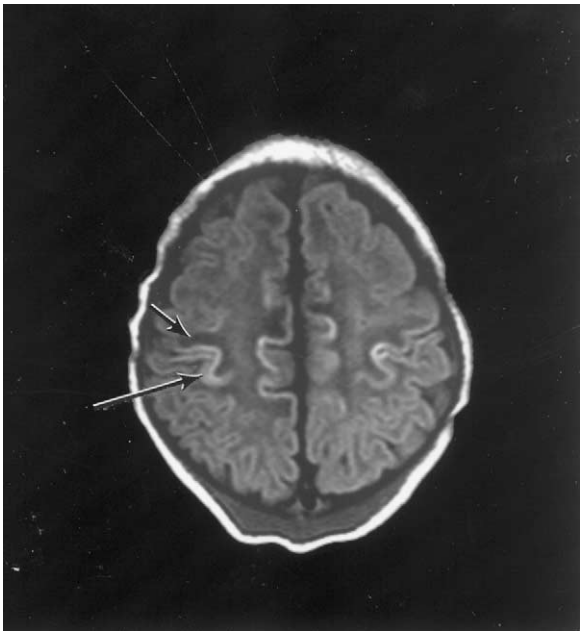

Figure 6 Abnormal highlighting of the cortex. Term infant stage-II HIE aged 12 d. Inversion recovery sequence (IR 3800/30/950 ms). There is cortical highlighting around the central fissure (long arrow), which is maximal at the depths of sulci. There is additional low signal intensity in the adjacent subcortical white matter (short arrow).

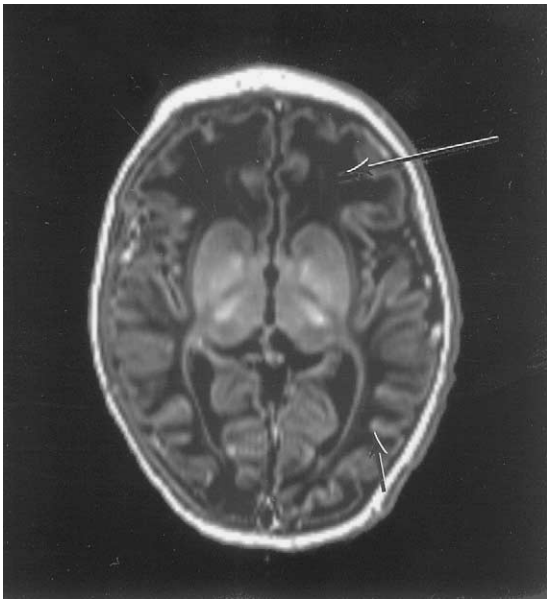

Figure 7 Severe basal ganglia and white matter abnormalities. Inversion recovery sequence (IR 3800/30/950 ms). Infant with stage-II HIE at 8 d of age. Low signal intensity is present throughout the white matter (long arrow) with areas of cortical highlighting at the depths of sulci (short arrow). There is abnormal high signal intensity in the basal ganglia and thalami as well as abnormal low signal intensity within the PLIC.

athetoid quadriplegia usually with good preservation of intellect and normal head growth. Mild BGT lesions may be associated with late onset tremor, and mild but often transient abnormalities of tone.

EARLY SEIZURES WITH MINIMAL DEPRESSION OF APGAR SCORES

Infants who present with seizures but in whom Apgar scores were near normal show a variety of pathologies.[16] These include haemorrhagic or infarctive lesions, congenital or acquired infection, congenital abnormalities and metabolic disorders.

Despite the good condition at birth, these infants may still have sustained a perinatal injury to the brain although the lesions are more likely to involve the white matter and cortex. These abnormalities include areas of infarction that may be focal, multifocal, widespread or parasagittal in distribution.

Evolution of perinatally acquired infarction

Acute infarction is best detected with DWI, then with T2 weighted images and lastly with T1 weighted images (Fig. 8). Areas of infarction show restricted diffusion of water molecules, which gives an abnormal high SI on DWI. This is often detectable within hours of the insult and is certainly present at the time the infant comes to imaging. The abnormal high SI on DWI gradually decreases over the first 10 d while abnormal SI becomes more obvious with conventional imaging. T2 weighted images show abnormal high SI and a loss of the normal low SI of cortical markings, presumably due to oedema and/or infarction of the cortex (Fig. 8). T1 weighted images show loss of grey/white matter differentiation initially (Fig. 9). This loss of differentiation may return during the second week and become exaggerated reflecting abnormal low SI within the WM and abnormal high SI within the cortex (Fig. 7). Breakdown and atrophy of the infarcted tissue may become obvious after 2 weeks and generally lasts until about 6 weeks. This pattern of abnormal SI using the different sequences is similar whatever the extent of infarction.

Parasagittal infarction in the term infant

Parasagittal infarction involves the deep WM at border zones of major artery territories (Fig. 8). Some of these infants may present with 'full blown' HIE although the clinical history is often atypical and there may be an abnormal antenatal history.

Parasagittal lesions may occur in the presence of severe hypoglycaemia.[17,18] Infants with parasagittal

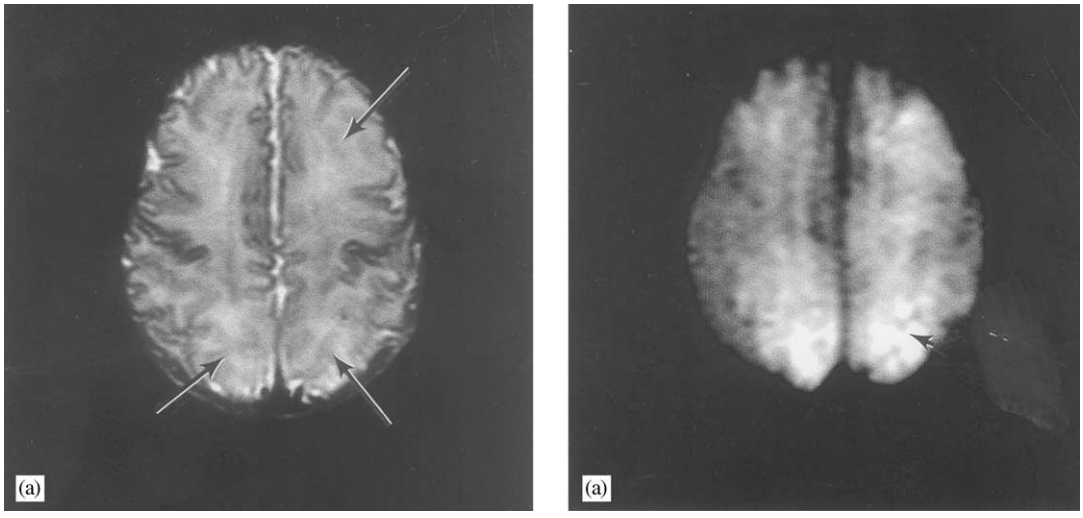

Figure 8 Parasagittal infarction. (a) T2 weighted fast spin echo sequence in an infant aged 3 d. There is loss of grey/white matter differentiation in the anterior and posterior parietal lobes. (b) This is more obvious on diffusion weighted image where the abnormal high signal intensity is caused by restricted motion of water molecules within the acute infarcts. There is also normal high signal intensity from myelin in the PLIC and normal signal intensity within the basal ganglia and thalami.

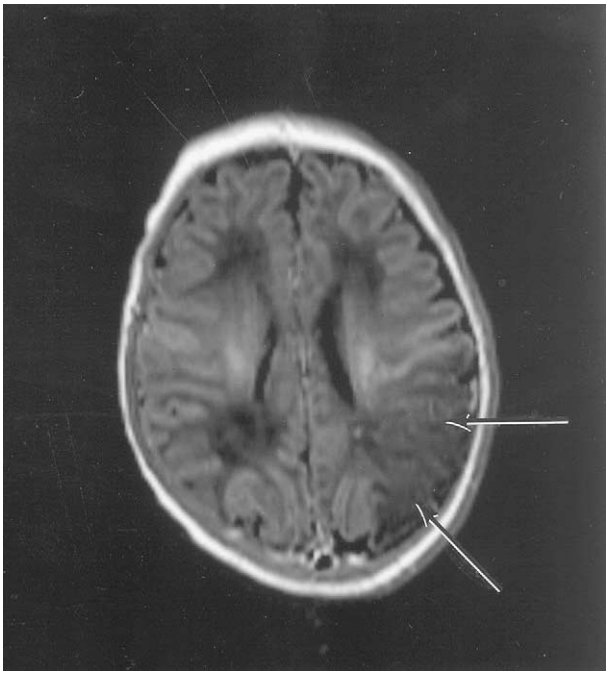

Figure 9 Middle cerebral artery infarction . Term born infant presenting with neonatal convulsions. Inversion recovery sequence (IR 3800/30/850 ms) aged 5 d. This shows loss of grey/white matter differentiation consistent with a left-sided middle cerebral artery infarction involving the cortex and white matter of the posterior parietal and temporal lobe (arrows). The basal ganglia, thalami and the PLIC were normal at a lower level in this infant.

infarction usually develop a secondary microcephaly although the neurodevelopmental outcome is often surprisingly good, particularly for motor function. This is because there is usually minimal or no involvement of the basal ganglia and thalami. However, underlying pathologies such as severe persistent hypoglycaemia are associated with major cognitive problems.

Large areas of haemorrhagic white matter infarction may also be seen in infants with or without some signs of HIE. Once again the basal ganglia and thalami may be spared. These infants may show more profound metabolic abnormalities such as prolonged conjugated hyperbilirubinaemia and recurrent hypoglycaemia. These infants often develop marked cognitive with some motor impairment such as a mild diplegia. A search for an underlying metabolic disorder should be carried out but this may not be successful.

Multifocal areas of infarction that do not appear to be in a parasagittal distribution may be secondary to infection, e.g. herpes, varicella, listeria (Fig. I0). Contrast enhancement should always be used if acquired infection is suspected. Prompt detection and treatment of early lesions may modify or prevent brain damage. White matter lesions may also be seen in congenitally acquired infections such as cytomegalovirus (CMV) (Fig. II) or rubella. In CMV the white matter may have a persistent abnormal SI for years but not atrophy. The presence of subependymal cysts that protrude into the ventricle are common in congenital infections and should always provoke appropriate investigations.

Focal infarction in the term infant

Perinatally acquired focal infarction, or 'neonatal stroke', is usually left sided and involves the territory supplied by

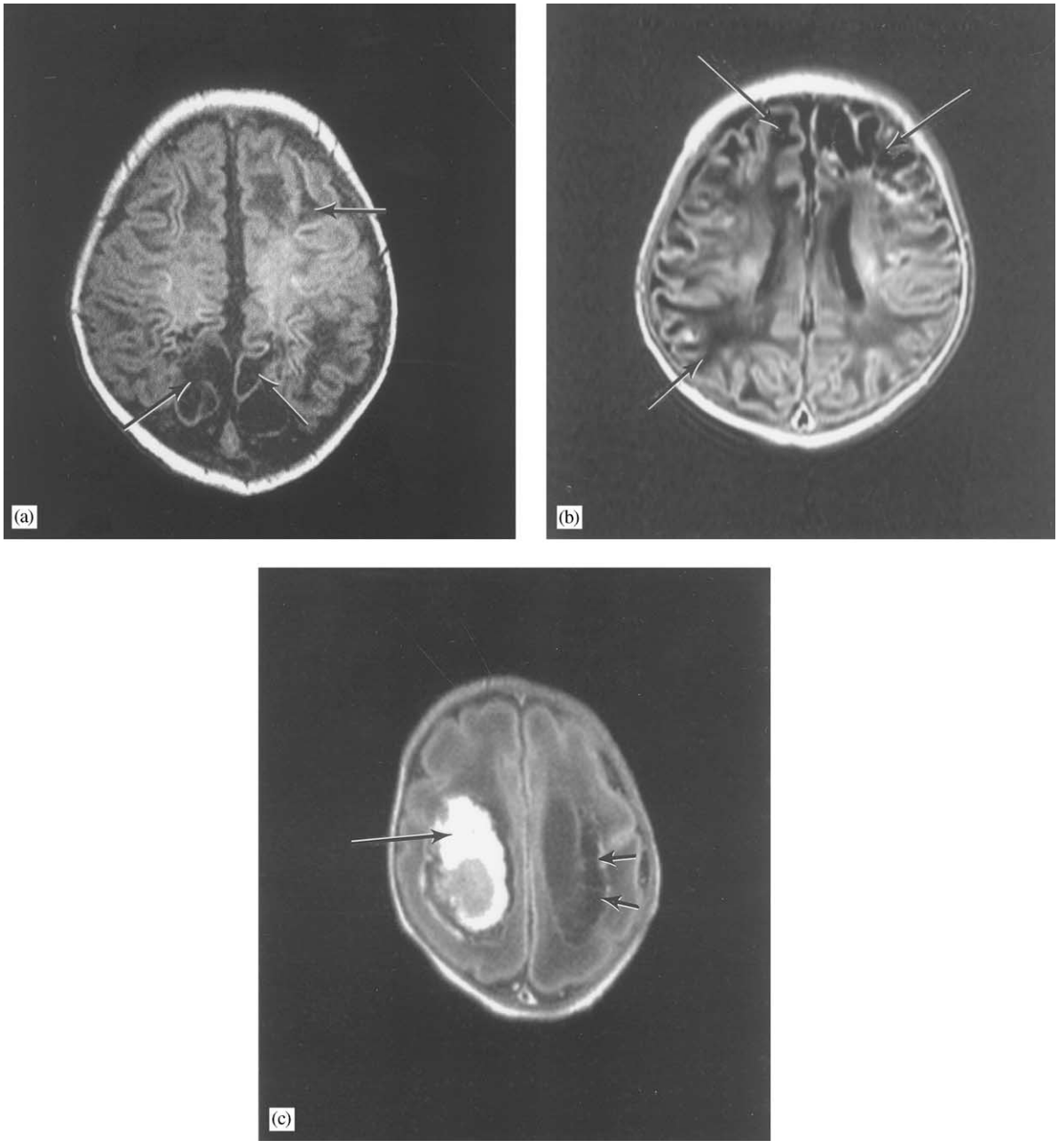

Figure 10 (a) Term born infant with a history of maternal varicella infection at 15 weeks gestation. Inversion recovery sequence (IR 3800/30/950 ms). There are areas of infarction within a mature looking brain (arrow) consistent with infarction within the late third trimester. (b) Term born infant with perinatally acquired herpes infection. Inversion recovery sequence (IR 3800/30/950 ms) demonstrating multiple areas of white matter and cortical infarction (arrows). These were not confined to the temporal regions. (c) Preterm infant at 32 weeks gestation with a history of maternal listeria just prior to delivery. T1 weighted spin echo sequence (SE 860/20 ms). There are bilateral abnormalities. The lesions on the right are mainly haemorrhagic (long arrow). There is extensive periventricular cyst formation on the left (short arrows).

the middle cerebral artery (Fig. 9). Neonatal stroke is associated with some specific antenatal factors, e.g. primigravida, history of abdominal pain, fetal distress, instrumental delivery, but may be asymptomatic. The clinical picture is not usually one of 'full blown' HIE and the infants usually go to the postnatal ward following delivery. It is unusual for focal arterial infarctions in the term infant to be haemorrhagic although there may be haemorrhage in other sites of the brain and a well-recognized combination is the presence of focal infarction adjacent to an area of subdural haemorrhage. A unilateral lesion within the thalamus or basal ganglia may be

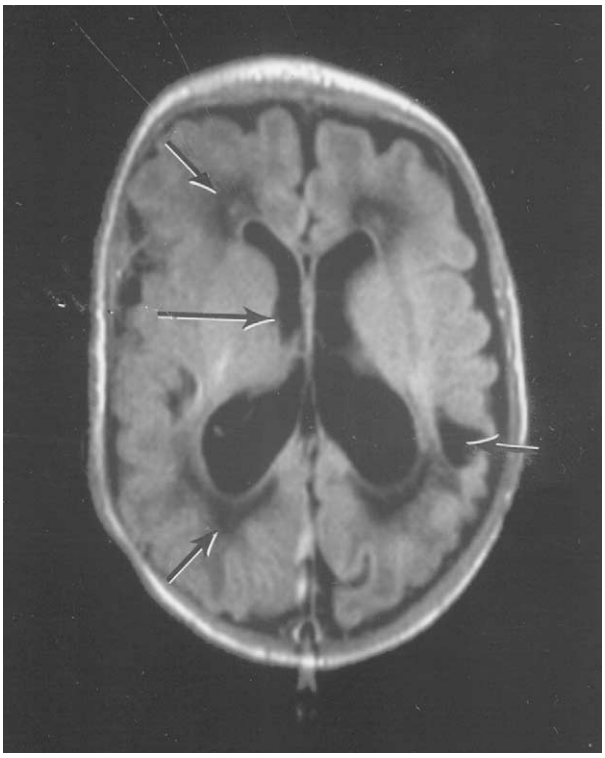

Figure 11 Cytomegalovirus infection. (a) Preterm infant with early postnatally acquired cytomegalovirus infection. Imaged at term equivalent age. Inversion recovery sequence. There is a widened caudothalamic notch (long arrow) consistent with previous subependymal cysts. Subependymal cysts which are clearly visible on ultrasound are easily missed on MR imaging. There is additional abnormal low signal intensity within the white matter (short arrows).

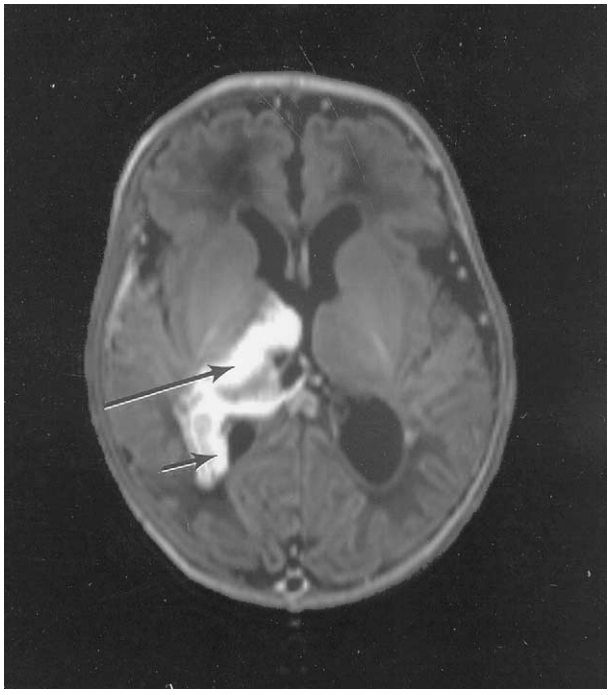

Figure 12 Thalamic haemorrhage. Term infant who presented with convulsions. (a) Inversion recovery sequence (IR 3800/30/850 ms). There is unilateral high signal intensity in the thalamus consistent with primary haemorrhage or haemorrhagic infarction (long arrow). (b) There is additional haemorrhage within the right lateral ventricle (short arrow).

infarctive, particularly in the more preterm infant.[19] Occasionally, they may be found as an additional antenatal lesion in infants with neonatal stroke. They may then highlight an inherent predisposition to infarction either because of a thrombotic disorder or because of a maternal embolic source.

Outcome following neonatal stroke depends on the sites involved, so that those infants that have abnormalities within the hemispheric white matter/cortex, the BGT and the PLIC have a high incidence of later hemiplegia. The development of hemiplegia in children with perinatal stroke has also been associated with the presence of factor V Leiden heterogenicity.[20]

Haemorrhagic lesions

Infants with seizures and normal Apgar scores may have sustained a haemorrhagic lesion within or external to the brain.[16] The SI of haemorrhage on MRI varies depending on the site of the haemorrhage, the age of the haemorrhage and the pulse sequence used.[21] Multiple small haemorrhages may be seen in the parenchyma. These may be associated with a period of hypoglycaemia. A more unusual but well-recognized lesion is that of haemorrhage within the thalamus (Fig. 12). The aetiology of thalamic haemorrhage is obscure. Imaging may identify associated intraventricular haemorrhage (IVH) (Fig. 12) and can help with dating the lesion although haemorrhage within the thalamus and the ventricle may evolve differently. The IVH may be severe enough to cause ventricular dilatation that requires intervention. The haemorrhage gradually resolves to leave an atrophied thalamus. These infants may develop a hemiplegia and cognitive problems.

Infants with seizures and a history of a traumatic delivery, shoulder dystocia or an expanding head may have sustained an extracerebral haemorrhage. Subdural haemorrhage is associated with difficult deliveries and is relatively common in the asphyxiated infant (Fig. 13). It occasionally occurs secondary to a congenital clotting disorder. Subdural haemorrhage may also occur without an obvious precipitating event. In the infant who has already been discharged from hospital, non-accidental injury needs to be considered. Subarachnoid haemorrhage may be asymptomatic and is probably quite frequent following even normal deliveries. It follows the contours of the brain and, if small, may be difficult to identify. If more extensive, it may be difficult to distinguish from subdural haemorrhage.

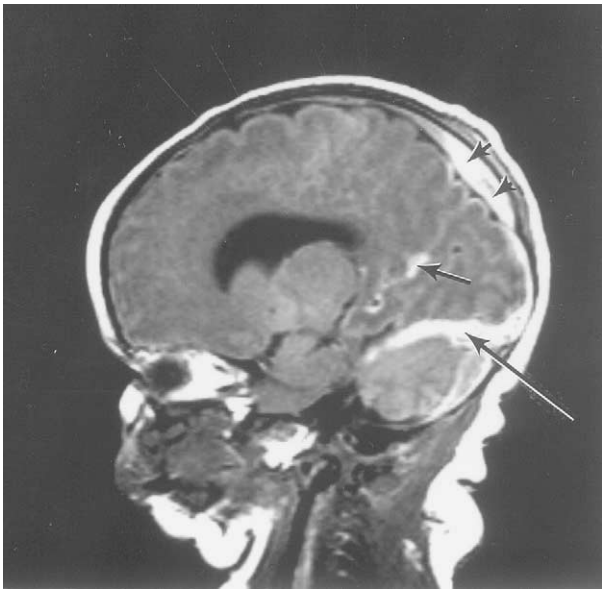

Figure 13 Subdural haemorrhage in an infant with stage-II HIE, delivered by vacuum extraction following a failed attempt at forceps. T1 weighted spin echo sequence (SE 860/20 ms). There is extensive abnormal high signal intensity consistent with haemorrhage in the subdural space over the parietal lobes (arrowheads), in the posterior fossa and around the tentorium (long arrow). There is a small amount of intraventricular haemorrhage in the posterior horns (short arrow).

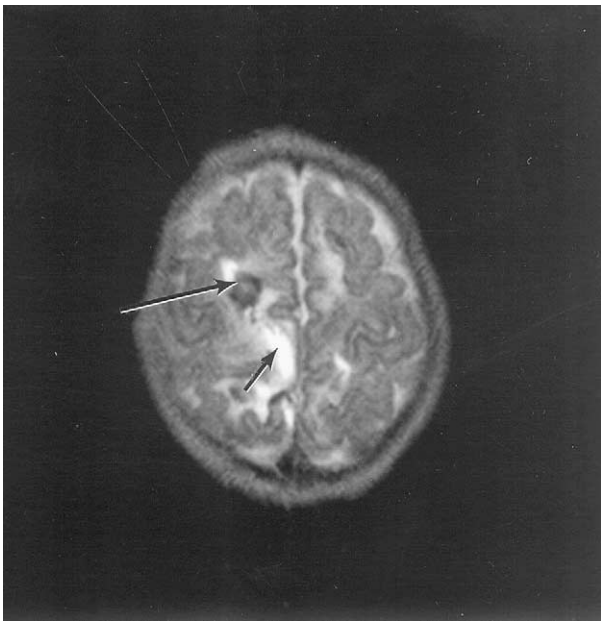

Figure 14 Sagittal sinus thrombosis. T2 weighted (FSE 3000/208_{eff} ms) image in an infant with stage-II HIE and a high haematocrit. There are multiple areas of haemorrhagic infarction in the cortex and subcortical white matter (arrows). Imaging of the sagittal sinus was consistent with thrombosis.

Sinus thrombosis occurs due to trauma, sepsis, cardiac failure, dehydration or an increased haematocrit. Sinus thrombosis may be difficult to confirm. Normally, the sinuses are depicted as low signal on T1 weighted MRI, due to flowing blood. This is dependent on the position of blood flow in each slice. In sagittal sinus thrombosis, the SI is not pathognomonic. In thrombosis, the sinus is typically high SI on T1 weighted images and low signal on T2 weighted images on all slices and in all planes. T1 weighted sagittal images from a 3D volume set with an unselected radiofrequency pulse which is largely flow independent may be useful. Contrast enhancement may help to confirm a patent sinus or demonstrate a filling defect. Sinus thrombosis is associated with specific patterns of parenchymal injury, including haemorrhagic lesions in the cortex and white matter (Fig. 14). Straight sinus thrombosis is associated with lesions within the basal ganglia and thalami.

Metabolic disorders

Neonates with early seizures but apparently normal Apgar scores may have a neurometabolic disorder, such as non-ketotic hyperglycinaemia or Zellweger's syndrome. Imaging may be normal, show delayed myelination or be associated congenital malformations such as agenesis of the corpus callosum and cortical migration defects. Bilateral BGT lesions may also be seen in certain metabolic disorders that present in the neonatal period, e.g. Leigh's disease. These infants may be differentiated from those with HIE by the evolution of their clinical signs but there is often overlap, and so all infants with HIE or those with basal ganglia lesions should have a full metabolic screen and a congenital infection screen. In neonates with kernicterus abnormal signal intensity within the globus pallidus and subthalamic nuclei may be relatively subtle for the first few months (Fig. 15).

MRI ASSESSMENT OF BRAIN INJURY IN PRETERM INFANTS

The developing brain is highly susceptible to injury including periventricular leucomalacia (PVL), intraventricular haemorrhage/germinal layer haemorrhage (IVH/GLH) and parenchymal haemorrhagic infarction. The majority of preterm infants demonstrate some abnormality on brain MR imaging in the early neonatal period.[8] Additionally, quantitative MRI has revealed delayed myelination in adolescents who were born preterm[22] and reduced cortical folding[1] in the preterm brain at term equivalent age compared with term born infants.[21]

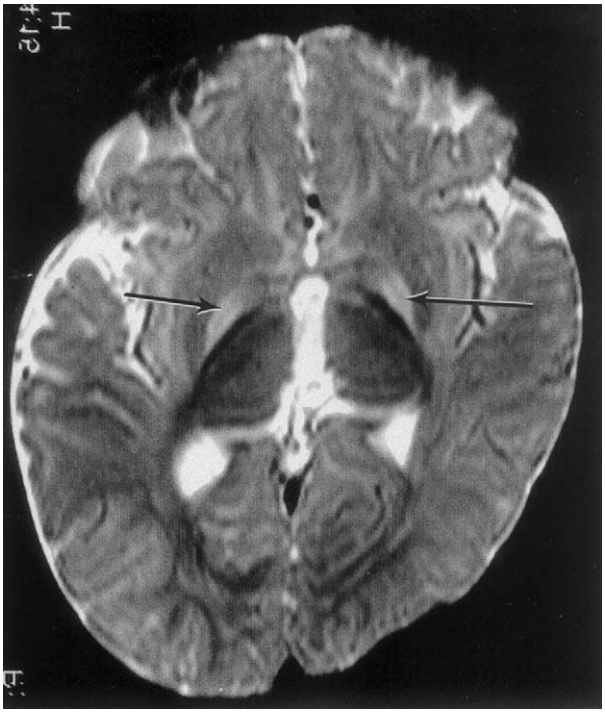

Figure 15 Kernicterus. Term born infant with hyperbilirubinaemia. Neonatal imaging was equivocal with some increased signal intensity within the globus pallidum on T1 weighted imaging, but at 11 months of age there are obvious abnormal areas of increased signal intensity on the T2 weighted images (arrows).

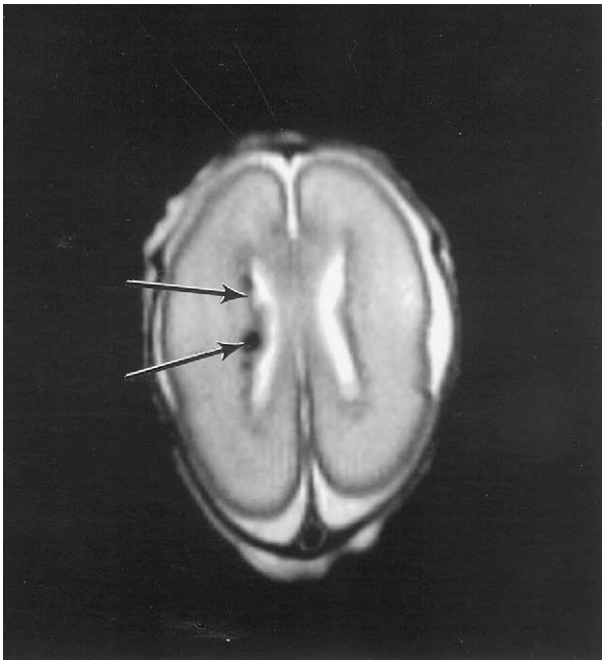

Figure 16 Germinal matrix haemorrhage. Preterm infant born at 26 weeks gestation T2 weighted fast spin echo sequence (FSE 3000/208$_{eff}$ ms). The germinal matrix haemorrhages are seen as low signal intensity (arrows).

Intraventricular/germinal layer haemorrhage

GLH is demonstrated as low signal on T2 weighted imaging and high SI on T1 weighted imaging. It can be differentiated from the normal germinal layer by its irregular appearance and its slightly more hypointense SI on T2 weighted imaging (Fig. 16).

Periventricular haemorrhagic infarction

PHI is a consequence of large IVH obstructing the terminal veins, and results in interruption of projection and association fibres as well as oligodendroglial damage, which disrupts myelination. It is demonstrated as a fan-shaped lesion of low SI on T2 weighted FSE imaging (Fig. 17). In surviving infants, a porencephalic cyst usually develops at the site of the lesion. The appearance of myelin in the PLIC at term equivalent age has been shown to predict the development of hemiplegia in these infants.[23]

Periventricular leukomalacia

PVL is a form of white matter damage that has two components: focal cyst formation and diffuse white matter

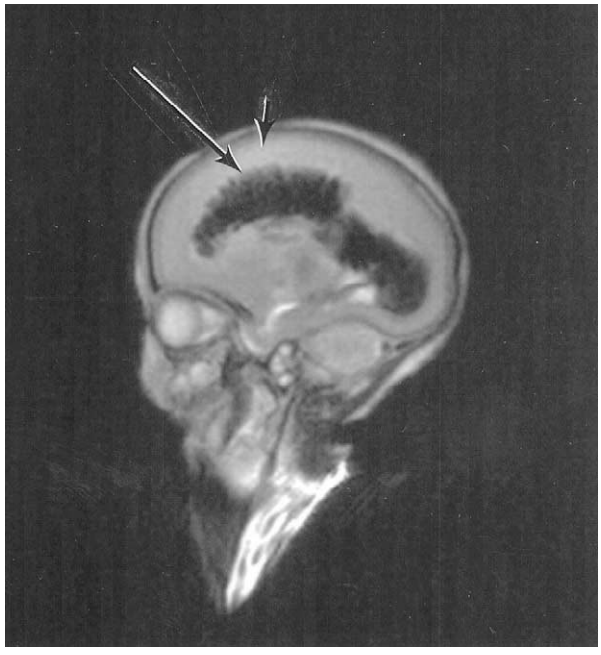

Figure 17 Periventricular haemorrhagic infarction. Preterm infant born at 27 weeks gestation and imaged at 3 d. T2 weighted fast spin echo sequence (FSE 3500/208$_{eff}$ ms). In the sagittal plane. There is extensive low signal intensity consistent with intraventricular haemorrhage. There is additional low signal intensity in a fan-shaped distribution in the frontal white matter (long arrow). There is adjacent abnormal increased signal intensity extending out to the cortex (short arrow). This is consistent with oedematous or ischaemic tissue. It showed signs of infarction at histology.

injury. Traditionally, PVL was thought to be due to ischaemia but recent studies also implicate infection.[24,25] Acutely, PVL is demonstrated as hypointense periventricular regions on T1 weighted imaging which are hyperintense on T2 weighted imaging. Additionally, areas of short T1, presumably a haemorrhagic component, have been identified in the acute/subacute stage.[26] On DWI, PVL is demonstrated as high SI in the acute stage. These areas may become cystic (Fig. 18) and lead to dilatation of the lateral ventricles, particularly in the region of the occipital horn (Fig. 18). PVL is associated with delayed or deficient myelination. The late imaging findings in PVL may resemble those found in term born infants with perinatal acquired haemorrhagic lesions.[21]

Three-dimensional MRI has demonstrated that preterm infants with PVL have a reduced cortical grey matter volume at term compared with both preterm infants with no evidence of PVL and normal term control infants.[3] These findings suggest that PVL has an impact on cerebral cortical development, which may explain the cognitive deficits associated with this condition.

Diffuse excessive high signal intensity (DEHSI)

Previous studies have also shown that the preterm infant at term appears to have areas of excessive long T2 (DEHSI) within the cerebral white matter.[8] These changes are most marked in the periventricular white matter and decrease with age (Fig. 19). DEHSI can be quantified and is associated with an increase in T1 and T2 values. It is unclear what this represents in pathological terms but it may represent a form of diffuse white matter disease in the preterm infant.

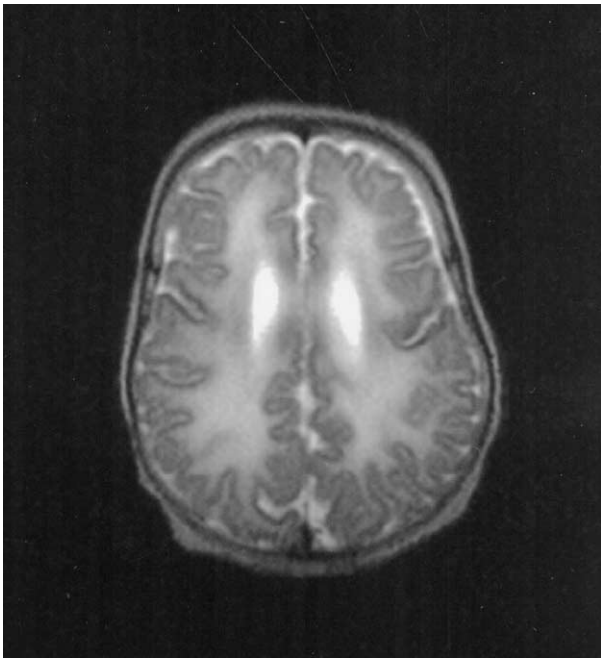

Figure 19 Diffuse excessive high signal intensity (DEHSI). Preterm infant born at 26 weeks gestation and imaged at term equivalent age. T2 weighted fast spin echo sequence (FSE 3500/208_{eff} ms). There is excessive high signal intensity throughout the periventricular white matter.

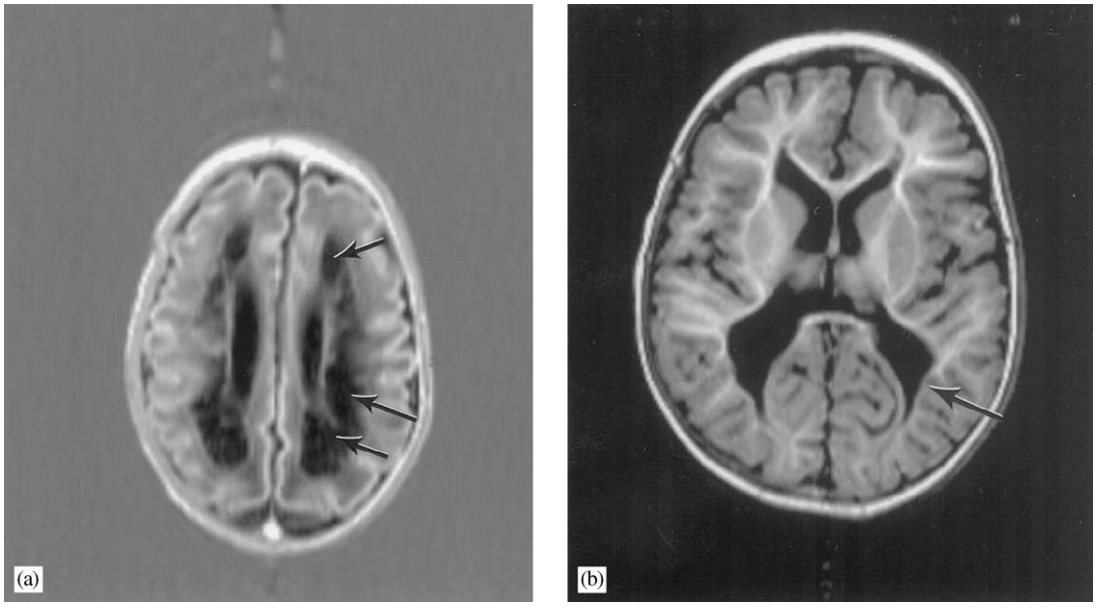

Figure 18 Periventricular leucomalacia. Infant born at 32 weeks gestation with evidence of cyst formation on cranial ultrasound a few days later. (a) Inversion recovery sequence (IR 3800/30/950 ms). There is extensive cyst formation around both ventricles (arrows). (b) Inversion recovery sequence (IR 3600/30/700 ms) at 15 months of age. There is irregular ventricular dilatation, most marked posteriorly (arrow) and a paucity of myelination.

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