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Feeding and communication impairments in infants with central grey matter lesions following perinatal hypoxic–ischaemic injury

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ABSTRACT

Background: Basal ganglia and thalamic (BGT) injury is common after acute perinatal hypoxia–ischaemia. Cerebral palsy is the most obvious consequence of BGT injury affecting 70–75% of survivors and is predictable from neonatal magnetic resonance imaging (MRI). However there is no equivalent predictive data for other specific outcomes. Feeding and communication impairments are also common in children following hypoxic–ischaemic encephalopathy (HIE) and BGT injury.

Aims: To describe, in infants with HIE and BGT injury, the prevalence of feeding and communication impairments; and to evaluate the accuracy of early MRI for predicting these outcomes.

Methods: 175 term infants with HIE and BGT injury were studied. Brain lesions were classified by site and severity from the MRI scans. Motor, feeding and communication impairments were documented at 2 years.

Results: Feeding and communication impairments occurred in 65% and 82% of 126 survivors respectively and related strongly to the severity of motor impairment. Forty-one children had a gastrostomy or long-term nasogastric tube. Injury severity in all brain regions was significantly associated with feeding and communication impairment on univariate analysis. On logistic regression analysis BGT (OR 10.9) and mesencephalic lesions (OR 3.7) were independently associated with feeding impairment; BGT (OR 10.5) and pontine lesions (OR 3.8) were associated with gastrostomy; the severity of BGT lesions (OR 20.1) was related to the severity of communication impairment.

Conclusions: Feeding and communication impairment are very common in children with BGT and brainstem injury of neonatal origin and can be well predicted from early MRI scans.

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1. Introduction

Feeding and communication are vital functions for newborns and young children. In infants with neonatal hypoxic–ischaemic encephalopathy (HIE), injury to the basal ganglia and thalami (BGT) is common and the hallmark of acute perinatal injury.[1] While cerebral palsy (CP) is the most obvious consequence affecting 70–75% of survivors,[2] the ability to suck, swallow and develop functional verbal communication may also be seriously affected. Oromotor dysfunction may lead to feeding and speech problems. Other factors such as poor facial expression, physical difficulties limiting gestures, learning disability and language delays may also impede normal communication in these children.

Whilst the prevalence and severity of feeding and communication impairment in children with CP have been studied,[3–7] CP has multiple aetiologies often not related to perinatal HIE. Thus these data are difficult to apply when discussing prognosis for a newborn with HIE. In addition to seeking information about possible CP and mobility, parents often ask about other abilities and particularly whether their baby will be able to feed and speak. Feeding and communication impairments have not been described in children with HIE and BGT lesions.

Magnetic resonance imaging (MRI) provides the most accurate early information about the site and type of brain injury and the prediction of individual outcomes in infants with HIE. The pattern and severity of lesions in the BGT and the posterior limb of the internal capsule (PLIC) are predictive of CP,[2,8,9] whereas the degree of brainstem injury is predictive of death.[2] The value of neonatal MRI for the prediction of other specific outcomes following HIE has not yet been explored.

The aims of this study were: (1) to describe the prevalence of functional feeding and communication impairment in infants with HIE and BGT injury and (2) to identify patterns of injury on neonatal MRI that are predictive of these outcomes.

2. Methods

Ethical permission for scanning the infants in this study was obtained from the Hammersmith Hospital research ethics committee and consent was obtained individually from the parents.

2.1. Participants

Infants with neonatal encephalopathy investigated with MRI brain scans at our tertiary unit in Hammersmith/Queen Charlotte's Hospitals (London, UK) were included in our prospectively maintained database from 1993 to 2007. From this database of 555 infants we retrospectively selected those who met all the following criteria: (1) ≥35 weeks gestation; (2) signs of foetal compromise (abnormal foetal rate tracing, meconium stained liquor, or a sentinel event during labour); (3) poor condition at birth (5-min Apgar score <5, cord blood pH <7.1, or need for major resuscitation); (4) neonatal encephalopathy (difficulty with initiating and maintaining respiration, altered consciousness, abnormal tone and reflexes, with/without seizures)[10] (5) BGT injury seen on MRI within 6 postnatal weeks; (6) assessment of outcome at a minimal age of 12 months. Exclusion criteria were: identifiable metabolic disorders, severe congenital malformation/infections, genetic abnormalities or hypothermia treatment. For included infants, medical records were reviewed to extract demographic data, family history, antenatal and perinatal data which had been recorded neonatally on a detailed proforma.

2.2. MRI imaging

Infants were imaged with conventional T1-weighted (T1W) spin echo (SE), inversion recovery, and T2-weighted (T2W) SE sequences in a 1.0, 1.5, or 3 T MR scanner, depending on the system being used in our unit at the time of presentation. Images were assessed for normal anatomical development, evidence of prolonged/subacute problems or long-standing established injury, and for unusual patterns of injury. Abnormal signal intensities (SI) within the BGT, PLIC, white matter (WM), cortex, brainstem and cerebellum were documented and classified as follows (Figs. 1 and 2)[2]:

BGT:
- mild, focal abnormal SI;
- moderate, multifocal abnormal SI;
- severe, widespread abnormal SI

Posterior limb of the internal capsule:
- normal SI from myelin;
- equivocal, reduced or asymmetrical SI
- abnormal, reversed or abnormal SI bilaterally on T1W and/or T2W sequences.

White matter:
- normal/mild, exaggerated long T1/T2 in the periventricular WM only;
- moderate, long T1/T2 extending out to the subcortical WM and/or focal punctate lesions and/or focal area of infarction;
- severe, widespread abnormalities including overt infarction, haemorrhage and loss of grey/white matter differentiation.

Cortex: loss of grey/WM differentiation (usually seen in the first week) and/or cortical highlighting (usually seen after the first week).
- normal,
- mild (1–2 sites),
- moderate (3 sites),
- severe (>3 sites)

The sites documented were the central sulcus, interhemispheric fissure, insula, occipital and medial temporal lobes.

Brainstem:
- normal,
- mild–moderate (loss of anatomic details, excessive differentiation between anterior and posterior pons and/or mild asymmetries),
- severe (marked atrophy/asymmetries, abnormal SI/myelination).

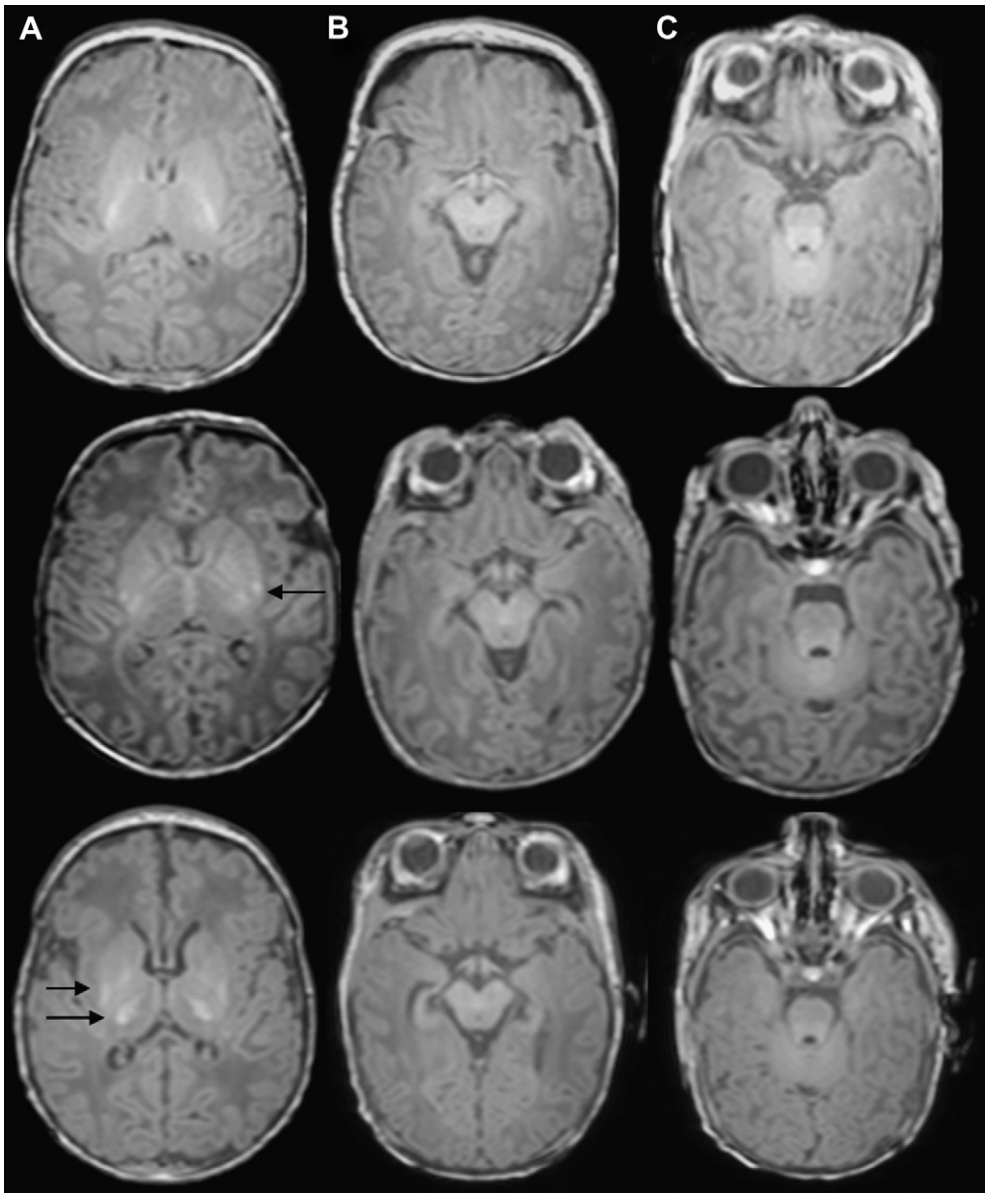

Fig. 1 – *Top row*: Example of T1-weighted images at the level of the basal ganglia and thalami (A), the mesencephalon (B) and the pons (C) of a normal term infant scanned on day 3. *Middle row*: T1-weighted images of an infant scanned on day 5 illustrating mild basal ganglia and thalamic (BGT) injury (A) (arrow) with a normal looking mesencephalon (B) and pons (C). He did not have cerebral palsy and started walking at 16 months. He did not have feeding impairment, but he had moderate speech problems and needed assistive communication methods. *Bottom row*: T1-weighted images of an infant scanned on day 18 illustrating moderate basal ganglia and thalamic (BGT) injury (A) (arrows) with a normal looking mesencephalon (B) and pons (C). This child developed dystonic cerebral palsy, GMFCS level IV. He had some problems with solids but not with liquids and did not need a gastrostomy. However he had severe speech problems and was only able to communicate using assistive communication systems.

Abnormalities were assessed separately in the mesencephalon and the pons.

2.3. *Outcome data*

Infants were assessed using a standardised neurological examination,[11] head circumference measurement and Griffiths Mental Development Scales.[12] We also documented by direct questioning and observation problems relating to feeding, communication, vision, hearing, behaviour and later seizures. The Surveillance of Cerebral Palsy in Europe definition and classification for CP were applied.[13] Gross Motor Function Classification System (GMFCS)[14] was used to grade motor functional impairment.

Feeding and communication impairment were defined and classified as follows:

Feeding impairment included poor suck or need for spoon-feeding of liquids, coughing, spluttering or choking during

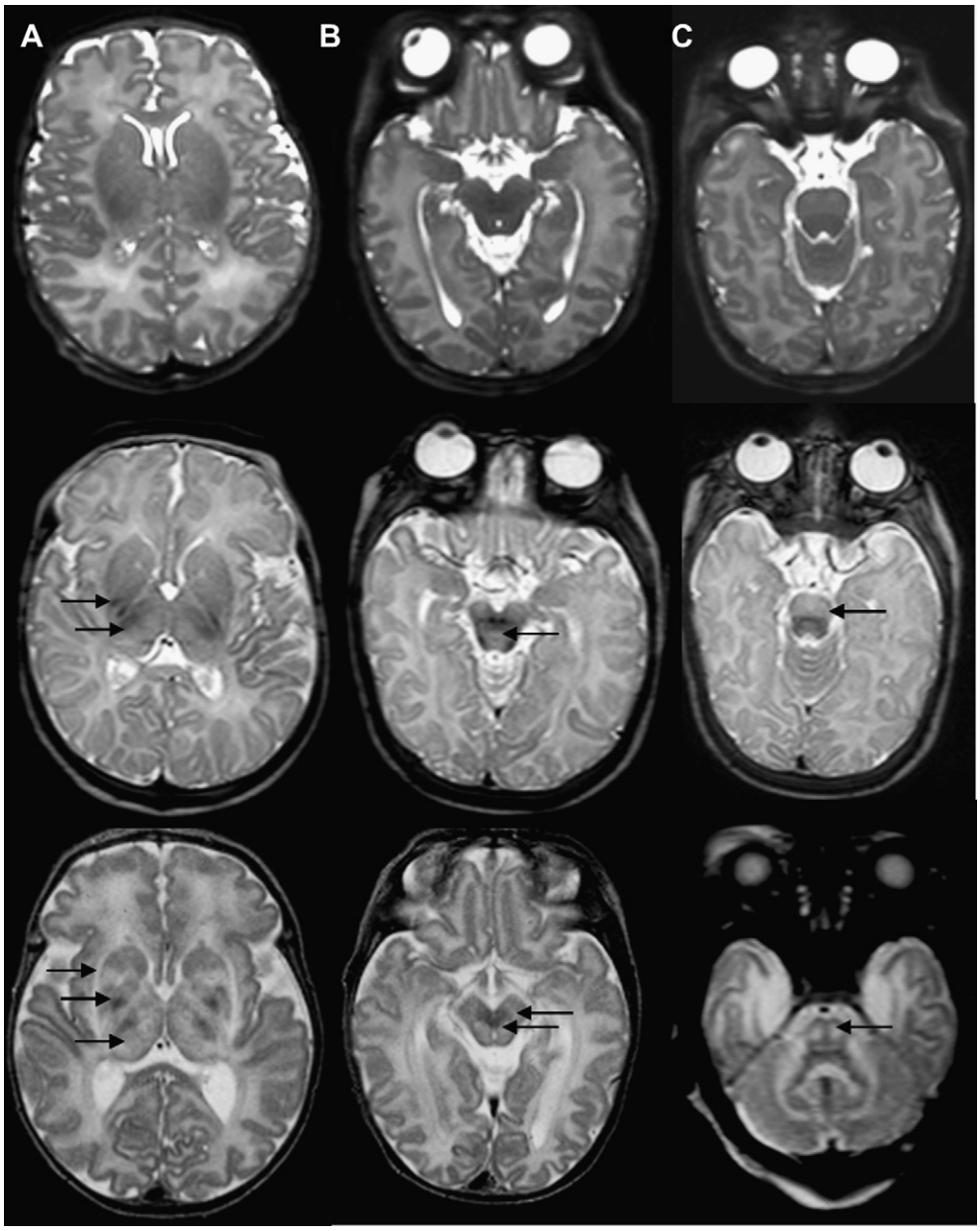

Fig. 2 – *Top row*: Example T2-weighted images at the level of the basal ganglia and thalami (A), mesencephalon (B) and pons (C) of a normal infant scanned on day 1. *Middle row*: T2-weighted images of an infant scanned on day 6 illustrating moderate injury to the BGT (A) (arrows), and moderate abnormal signal intensity at the level of the mesencephalon (B), and the pons (C) (arrows). She had spastic quadriplegia, GMFCS level III. She had feeding impairment and could only eat pureed food and liquids, but she did not need a gastrostomy. She had moderate speech impairment and needed assistive communication methods. *Bottom row*: T2-weighted images of an infant scanned on day 15 illustrating severe, widespread injury to the BGT (A) (arrows), and severe abnormal signal intensity at the level of the mesencephalon (B), and the pons (C) (arrows). He developed spastic quadriplegia, GMFCS level V. He had severe feeding impairment since the neonatal period and a gastrostomy was inserted when he was 7 months old. He was unable to communicate by no discernible means.

feeds, feeds that took a long time by parental assessment and difficulty with swallowing solids at all or lumpy foods[3,15,16]:

1. None,
2. Mild–moderate. Problems swallowing solids or liquids, can feed orally but require mashed or pureed foods,
3. Severe. Requiring assisted feeding (nasogastric or gastrostomy tube).

Communication impairment included any expressive speech or language difficulty, and was classified as[7]:

1. None. Age-appropriate expressive speech and language,
2. Mild–moderate. Able to use speech, but may need formal assistive communication methods,
3. Severe. Non-verbal communication (assistive methods alone or unable to communicate by any ways).

2.4. Statistical analyses

The data were analysed using SPSS version 11.5 (SPSS, IL, USA). The relation between the presence and severity of WM, cortex, BGT, PLIC, cerebellum and brainstem injury on MRI with feeding and communication impairment was assessed by univariate analysis using the Fisher's exact test. A logistic regression analysis was then performed to identify brain structures with an independent association with feeding and communication impairment. We assessed the relationship between motor function and feeding and communication problems. Prenatal and perinatal factors were also included in the univariate analysis and if they were found to be significantly associated with outcome they were entered into the logistic regression analysis. Differences with p-level <0.05 were considered statistically significant.

3. Results

Of the 555 infants in our neonatal encephalopathy database (1993–2007), 175 fulfilled all the entry criteria. Patient demographics are summarised in Table 1. Of the infants excluded were 64 with metabolic diagnoses, congenital malformations/infections and/or genetic abnormalities; 20 <35 weeks gestation, 41 treated with hypothermia, 59 scanned after six weeks, 10 lost to follow up and 186 with HIE but without BGT lesions.

Table 1 – Demographic data of the study population.

Factor	$n = 175$
Antepartum data	
Family history of seizures, *n* (%)	10 (6)
Maternal age, mean ± SD	30 ± 5.5
Primiparity, *n* (%)	85 (48)
Maternal blood pressure abnormalities, *n* (%)	21 (12)
Maternal thyroid disease, *n* (%)	5 (3)
Intrapartum data	
Sentinel event, *n* (%)	47 (27)
Abnormal cardiotocography, *n* (%)	118 (67)
Emergency C-section, *n* (%)	83 (47)
Apgar score at 1 min, median (range)	1 (0–6)
Apgar score at 5 min, median (range)	4 (0–9)
Cord or 1st blood sample pH, mean ± SD	6.87 ± 0.2
Major resuscitation, *n* (%)	143 (82)
Infant characteristics	
Male, *n* (%)	96 (54)
Twins, *n* (%)	5 (3)
Gestational age, median (range)	40 (35–42)
Birthweight, median (range) kg	3.3 (1.9–4.6)
Corrected birthweight, mean ± SD[a]	−0.28 ± 0.05
Birthweight <10th centile, *n* (%)	21 (12)
Head circumference, median (range), cm	35 (31.5–38)
Corrected head circumference, mean ± SD[a]	0.13 ± 0.20
Head circumference <10th centile, *n* (%)	21 (12)

a Corrected for GA and gender and expressed as SD from the mean for the British population.

3.1. Motor outcome

The median age at follow up was 24 (range: 12–48) months; 82% of surviving children were ≥18 months and 61% ≥ 24 months at the time of assessment. 28% (49) had died from neurological problems, 23 neonatally, 17 during year one and 9 later.

Of the 126 surviving infants, 89 (71%) had CP (spastic 54%, spastic–dystonic 35%, athetoid 11%). Only two children had a hemiplegia and none had an isolated diplegia. The severity of motor impairment was mild (GMFCS level I) in 9%, moderate (levels II/III) in 14% and severe (levels IV/V) in 77%.

3.2. Feeding impairment

All the children who died after the neonatal period and 65% of survivors ($n = 82$) had feeding impairment. Infants who died neonatally were too severely affected to suck feed.

Information about the type and severity of the feeding impairment was available for 77 of the 82 affected children. Problems were mild–moderate in 41 (50%) and severe in 41; 36 children had a gastrostomy (median age 11 months, range 3–48 months) and 5 had long-term (minimum 6 months) nasogastric tube feeding.

In most infants, feeding impairments were present from the neonatal period or started in the first 6 months, but in 10 children (8% of survivors) who appeared initially to feed well, difficulties were present at the time of follow up; two of them required a gastrostomy. All had also significant motor impairment (levels III–V). Six of the 82 infants (5% of survivors) with early feeding difficulties improved during the first year, and they could feed with no or minimal difficulties at follow up. These 16 children had a wide range of feeding problems and different injury severities on the MR scans; and no clear injury pattern was identified either in those that improved or those that deteriorated with regard to feeding, but overall children with more severe brain injury had more severe feeding difficulties in the longer term and vice versa.

Functional feeding impairment was strongly related to motor function (Table 2). All but 3 children with feeding impairment also had CP. Of the 3 without CP, two had transient problems during the first year that spontaneously improved and the third had persistent problems only with solids. All 41 children who needed a gastrostomy or long-term nasogastric tube had CP; generally with severe functional impairment (GMFCS levels IV/V in 39 and level I in 2). One child with only mild motor impairment had a gastrostomy inserted at 7 months because of early feeding refusal and very poor growth, but later the feeding problems resolved and the gastrostomy was removed. This child started walking at 19 months and did not have speech problems. The other child with mild motor impairment but requiring gastrostomy had dystonia, and started walking at 20 months. She had a gastrostomy at 21 months because she was unable to cope with liquids by mouth.

3.2.1. MRI and feeding impairment (Table 3, Figs. 1 and 2)

The presence and severity of injury to all brain structures were significantly associated with the presence of feeding impairment. However in the logistic regression model only the severity of BGT (OR 10.9; 95% CI 4.2–43.6; $p < 0.001$) and mesencephalic (OR 3.7; 95% CI 2.1–8.2; $p < 0.001$) injury were independently associated with feeding impairment. When

Table 2 – Relation between level of motor impairment (GMFCS score) and any feeding or communication impairments.

	No CP N = 37	CP n = 89					p
		GMFCS I n = 8	GMFCS II n = 6	GMFCS III n = 6	GMFCS IV n = 23	GMFCS V n = 45	
Feeding impairment	3 (8)	2 (25)	3 (50)	5 (83)	18 (78)	43 (96)	<0.001
Gastrostomy/long-term nasogastric tube	0	2 (25)	0	1 (17)	5 (22)	29 (64)	<0.001
Communication impairment	10 (27)	3 (37)	6 (100)	6 (100)	18/22 (82)[a]	41/41 (100)[b]	<0.001
Severe communication impairment	1 (3)	0	0	1 (17)	14 (64)	38 (93)	<0.001

CP: cerebral palsy; GMFCS: Gross Motor Function Classification System.
a Missing information in 1 child.
b Missing information in 4 children.

infants had severe BGT and mesencephalic injury the probability of having feeding impairment was 84% (area under the curve 0.82, 95% CI 0.73–0.91; $p < 0.001$).

The presence and severity of injury in all brain structures except the WM were significantly associated with the need for gastrostomy insertion or prolonged nasogastric tube feeding. However in the logistic regression model only the severity of BGT injury (OR 10.5; 95% CI 2.9–56.7; $p < 0.001$) and pontine involvement (OR 3.8; 95% CI 1.8–10.2; $p < 0.001$) were independently associated with gastrostomy insertion. Severe BGT and pontine injury were associated with a 91% probability of needing a gastrostomy/long-term nasogastric tube (area under the curve 0.87, 95% CI 0.81–0.94; $p < 0.001$).

There was a small group of 22 infants with apparently minor cerebellar variants or abnormality, all also with severe BGT and brainstem injury, who had a disproportionately adverse outcome: 14 died and all the 8 survivors had severe feeding and communication impairments (7 needed a gastrostomy).

3.3. *Communication impairment*

Communication impairment occurred in 82% of survivors. Of these survivors 43 children (34%) had mild–moderate impairment and 61 (48%) had severe impairment and were completely non-verbal. Communication impairment was strongly related to motor outcome: it was almost universal in children with CP GMFCS levels II–V but was present even in children without CP (Table 2).

3.3.1. *MRI and communication impairment (Table 4, Figs. 1 and 2)*

The presence and severity of injury in all brain structures were significantly associated with the presence and severity of communication impairment, but only the severity of BGT injury (OR 20.1; 95% CI 9.3–72.9; area under the curve 0.87, 95% CI: 0.81–0.94; $p < 0.001$) was independently associated with the presence and severity of communication impairment.

3.4. *Relation between feeding and communication impairments*

Functional feeding and communication impairments were closely related. Communication problems were more common: of the 82 children with feeding impairment, only one did not have communication impairment and 92% had severe communication impairment; 69% of children with communication impairment had also feeding problems ($p < 0.001$). There were 27 children with no feeding and no communication impairments; they had significantly less severe injury in all brain structures than the rest of the cohort, but the high prevalence of both impairments in the cohort did not allow us to find a good prediction model from neonatal MR scans for the absence of these problems.

4. Discussion

Functional feeding and communication impairments, as reported by parents and observed in a clinical setting, are highly

Table 3 – Relation between severity of basal ganglia–thalamic and brainstem injury and feeding impairment.

		Severity of BGT injury				Severity of mesencephalic injury				Severity of pontine injury			
		Mild n = 28	Moderate n = 35	Severe n = 70	p	Normal n = 59	Moderate n = 24	Severe n = 50	p	Normal n = 63	Moderate n = 45	Severe n = 22	p
Feeding impairment	Any (n = 82)	3 (11)	16 (46)	63 (90)	<0.001	18 (30)	19 (79)	45 (90)	<0.001	22 (35)	38 (84)	20 (91)	<0.001
	Severe[a] (n = 41)	0	3 (9)	38 (54)	<0.001	6 (10)	6 (25)	29 (58)	<0.001	6 (9)	17 (38)	18 (82)	<0.001

BGT: basal ganglia–thalami.
a Severe feeding impairment: gastrostomy (n = 36) or long-term (minimum 6 months nasogastric tube feeding, n = 5).

Table 4 – Relation between severity of basal ganglia–thalamic injury and communication impairment.

		Severity of basal ganglia–thalamic injury			
		Mild $n = 28$	Moderate $n = 35$	Severe $n = 70$	p
Communication impairment	Any ($n = 104$)	15 (54)	32 (91)	57 (81)	0.0009
	Severe ($n = 61$)	2 (7)	3 (9)	50 (71)	<0.001

prevalent in children with neonatal HIE and BGT injury, the most common pattern of injury seen after perinatal hypoxia–ischaemia. Although feeding impairment was rare in children without CP, importantly communication impairment was present at 2 years even in children without motor difficulties.

We found that the severity of BGT and brainstem lesions seen on neonatal MRI could predict with considerable accuracy the presence and severity of feeding and communication impairments at 2-year follow up. BGT and brainstem injury were significantly related to the presence and severity of feeding impairment but only BGT injury was independently associated with communication impairment. Brainstem injury is a typical site of injury in term and preterm infants with severe HIE[18,19] and is the best predictor of death in infants with HIE.[2,17] These data show that assessment of the brainstem on neonatal MRI may provide important prognostic information about the severity of feeding impairments in survivors of neonatal HIE.

Oromotor dysfunction is caused by motor neuronal damage producing bulbar and/or pseudobulbar palsy. The corticonuclear tract connecting the motor cortex with the brainstem motor nuclei runs mainly through the genu of the internal capsule, although a few fibres are located in the PLIC.[20] The dorsal brainstem is a watershed zone with a high metabolic demand in the perinatal period and thus particularly vulnerable to ischaemic insults. It contains very important structures including the cranial nerve IV–XII nuclei, the solitary tract nucleus, the *nucleus ambiguus* and the reticular formation, that are involved in oromotor function.[21,22] In children investigated for oromotor dysfunction and with a history of hypoxic–ischaemic events brainstem lesions were reported on MRI after 5 months of age.[23] In addition to the central role of the corticonuclear tract and brainstem nuclei in feeding difficulties, the basal ganglia are also essential as they modulate the final motor response via the thalamus.[24] Dysphagia and dysarthria are classical signs of the commonest adult diseases related to basal ganglia. Thus, our finding that lesions in the BGT and brainstem predict later functional feeding impairment has a plausible pathophysiologic basis.

In neonatal HIE, obtaining good quality MR images and correctly interpreting the findings are of paramount importance in order to facilitate accurate prognosis.[25] This is especially applicable to brainstem lesions, which may be difficult to assess.[23,26] It is crucial that good imaging of the BGT, PLICs and brainstem is obtained in this clinical context.

Functional feeding problems are a major problem for infants and their families and contribute to malnutrition in children with neurological conditions.[27] Malnutrition has been associated with poorer health status and growth and limitations in social participation in children with CP.[28] Early adequate nutrition might have advantageous effects on neurological function.[29] Feeding impairments also have consequences on family life: parents frequently report mealtimes as being stressful and prolonged,[4] and concerns about safe and reliable drug administration, risk of aspiration and general health status are common.[30] There were 10 children who apparently fed well and then deteriorated; in part this may have been due to parental determination that feeding was not difficult. All 10 infants had moderate or severe BGT lesions and 7 had brainstem abnormalities, thus their feeding problems could have been anticipated and earlier review with this as the focus would likely have been helpful.

About one third of our survivors required gastrostomy insertion to maintain their nutritional status and growth because of their severe feeding impairment. Despite its good results in improving nutritional status and health, and it being a safe procedure with few complications,[30,31] there is often initial parental resistance to gastrostomy insertion, as it requires surgery, they may think of it as unnatural and emphasizing their child's disability[32]; and being unable to feed their child orally is perceived as a sign of parental failure; deciding to allow gastrostomy insertion is difficult and stressful for parents.[33] However gastrostomy usually improves the quality of the child's life and that of the family.[30,31] Early identification of infants likely to need a gastrostomy could be very helpful in planning the most appropriate follow up, in exploring parental feelings about feeding and in the prevention of malnutrition and other complications.

Communication impairment was even more prevalent than feeding impairment. It occurred in over 80% of survivors half of whom were completely non-verbal. These figures are higher than those reported previously in populations of children with CP.[5,6,34] This may be because children with GMFCS levels I–III, who usually represent the majority of children with CP, were a minority in our cohort with BGT lesions, and communication problems are related to the level of motor impairment.[34,35] Although we suspect speech difficulties, specifically dysarthria, were the main cause of communication impairment in our population, we cannot exclude other underlying conditions. Because communication impairment has consequences for all aspects of a child's development,[3,5,7] children at risk of speech and language problems should be identified early in order to optimise their communication. Indeed, our data suggest that such problems could be anticipated, with proactive advice about communication and follow up provided.

Our large and well characterized cohort of infants with HIE and BGT injury offers for the first time useful data for clinicians involved in the care of these infants. A limitation of our

study is that independent feeding and speech assessments were not systematically performed. Further studies are needed to explore the underlying causes of these conditions, particularly speech and language development and communication in infants with HIE and BGT injury. Another limitation is that the scales we used to classify feeding and communication impairments, although adapted from previously used systems, are not standardized. In contrast to the widely used classification system of motor impairment (GMFCS), there are as yet no such equivalents for the functional classification of feeding or communication impairments; a classification for communication difficulties is being developed but so far has assessed few children of this young age. Their preliminary data suggest a high rate of communication problems in children with CP.[36]

In conclusion, we found that functional feeding and communication impairments are very common in children with BGT injury following HIE; neonatal MRI can be used to predict their likely occurrence and severity. We suggest that these data help in anticipating problems hopefully reducing the risk of failure to thrive, infant and parental exhaustion from attempts at oral/nasogastric feeding and food refusal from distressing oromotor experiences. These findings highlight the need to be proactive in looking for communication impairment and also in supporting parents and children in communicating from an early stage when it may be very hard for the child with severe injury to respond at all even with facial expression or gestures.

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