REVIEW

Outcome following subdural haemorrhages in infancy

Sandeep Jayawant, Jeremy Parr

Arch Dis Child 2007;**92**:343–347. doi: 10.1136/adc.2005.084988

Subdural haemorrhages (SDH) are associated with significant neurodisability in affected individuals. The incidence of SDH in infants is between 12 and 25 cases per 100 000 children and most detected SDH are due to physical abuse. In the infant brain, SDH are caused by tearing of the bridging veins in the subdural space and may result in significant brain injury. The challenge of assessing outcome in infants with SDH is evaluating whether SDH or other accompanying brain insults are instrumental in the neurodevelopmental outcome.

Subdural haemorrhages (SDH) are associated with significant neurodisability in affected individuals. The incidence of SDH in infants is 20–25 cases per 100 000 children under the age of 1 year[1–3] and 12 cases per 100 000 children under the age of 2 years.[3] Most detected SDH are due to physical abuse.[1 3 4] In the infant brain, SDH are caused by tearing of the bridging veins in the subdural space as a result of rotational and deceleration forces, or by other pathological processes. While the causal mechanisms of SDH include meningitis, coagulopathy, vascular malformations and rare metabolic disorders, the outcome in affected children has been mostly studied in those who have suffered traumatic (accidental or non-accidental) brain injuries. As the developing infant brain is more susceptible to shaking or impact injuries due to poor head control and the relatively large size and weight of the infant head compared with the body, accidental or non-accidental (inflicted) trauma may cause significant brain injury. In individuals who have suffered trauma, other brain injuries are associated with the presence of SDH, for example, subarachnoid and intraparenchymal haemorrhages, diffuse axonal injury and injury to the brainstem and spinal cord. As a result of all mechanisms of brain injury, secondary hypoxic ischaemic injury and resulting cerebral oedema may occur due to decreased cerebral perfusion secondary to raised intracranial pressure, apnoea or seizures. Thus, outcomes may relate more to the associated brain injury than to the SDH per se. The challenge of assessing outcome in infants with SDH is evaluating whether SDH or other accompanying brain insults are instrumental in the neurodevelopmental outcome.

See end of article for authors' affiliations

Correspondence to: Dr Sandeep Jayawant, John Radcliffe Hospital, Oxford OX3 9DY, UK; Sandeep.Jayawant@orh.nhs.uk

Accepted 12 December 2006

OUTCOME STUDIES: METHODOLOGICAL CONSIDERATIONS

Retrospective studies performed during the 1970s gave initial insight into the outcomes in children with accidental or non-accidental traumatic SDH (for a historical review, see Minns and Brown[5]). However, more recently, several prospective studies have significantly increased our knowledge of outcome following injury.[6–10] SDH are recognised in association with a variety of causal mechanisms, but published data on the outcome in children with non-traumatic SDH is sparse. One considerable confounding factor in studies of outcome following both traumatic and non-traumatic SDH is that accompanying brain injuries are likely to be present but differ with varying mechanism of injury. This variation, as well as the variation in injury severity, introduces bias into outcome studies as outcome is related to both the mechanism and the severity of injury. Additionally, specific methodological difficulties remain; stringent diagnostic criteria were not used to categorise injury in some studies, casting doubt on the validity of data, and also the definition of injury varies between studies (ranging from clinical examination findings to radiological features) making the comparison of data complex. Furthermore, the duration of follow-up is extremely variable between studies; a period of at least 2 years after injury might be considered adequate to assess motor and cognitive outcome, but many studies did not standardise the period and reported a mean duration of follow-up, while in other studies the follow-up period was very short. With reference to measuring outcome, the assessment tools used have been diverse. Finally, none of the listed studies reported subgroup analysis of outcome by either the presence of SDH, age or radiological classification of injury.

The absence of a ''gold standard'' battery of outcome measures and the subsequent use of non-standardised and variable outcome measures has led to difficulties in the comparability of outcome data following SDH. While some studies have simply assessed outcome using neurological examination[11] and carer report,[12] other measures used have included the Glasgow Outcome Scale (GOS), Seshia's global outcome score, King's Outcome Scale for Childhood Head Injury (KOSCHI) and the Pediatric Overall Performance Category (POPC)[13] (table 1). Of these outcome measures, the GOS[14] and its modifications have been most widely used in measuring long-term outcomes; however, the GOS may not identify cognitive and fine motor deficits.[15] That the scales listed categorise individuals within a rather narrow scale (for example, the GOS has a 5 point outcome scale and the Seshia score a 6 point scale) results in

Abbreviations: GOS, Glasgow Outcome Scale; KOSCHI, King's Outcome Scale for Childhood Head Injury; POPC, Pediatric Overall Performance Category; SDH, subdural haemorrhages

categorisation rather than dimensionalisation of outcome. In order to achieve standardisation and adequately identify and quantify gross and more subtle difficulties, assessment with outcome scales, developmental scales and neuropsychological tests in combination is desirable.

GROUPING OF OUTCOME DATA FOLLOWING SDH

For this review, in order to critically assess outcome data, studies have been grouped by causal aetiology. Thus the outcomes in infants with SDH due to trauma are divided into those caused by accidental and non-accidental mechanisms; the outcomes of SDH in infants following non-traumatic injury are described separately.

OUTCOME FOLLOWING NON-ACCIDENTAL (INFLICTED) HEAD INJURY

The short-term outcome in infants with inflicted SDH is poor and the associated mortality ranges from 11% to 36%[1 5 8 16]; in surviving children, long-term morbidity is usual and ranges from mild learning difficulties to severe physical and cognitive impairment.[1 9 10 17] Table 1 lists some of the recent short- and long-term retrospective and prospective studies of outcome in infants and children who have sustained non-accidental (inflicted) head injury.

OUTCOME FOLLOWING OTHER CAUSES OF SDH

Accidental head injury

SDH are more likely to be associated with parenchymal haemorrhage in children who have suffered accidental head injury than inflicted injury.[6] Despite this, several recent studies have confirmed that outcomes are much better following accidental head injury than after non-accidental head injury.[6 10 12 22 23] In contrast, a recent large study by Keenan *et al* showed that 61% of children who sustained accidental head injury had SDH (mostly road traffic accidents or significant falls); 57% of these individuals had a good outcome, 12% a poor outcome and 31% died.[9] These data may be confounded by the large number of individuals who suffered head injury following motor vehicle accidents, resulting in more reported deaths but less disability than in other studies.

The difference in reported outcomes between children who have been accidentally injured and those who have suffered inflicted injury is likely due to two main factors. Firstly, in reports to date, accidentally injured children have tended to be older than non-accidentally injured children[22] due to an increased likelihood of older children being injured in road traffic accidents (both as passengers or pedestrians) rather than having a brain injury secondary to shaking (although see Salehi-Had *et al*[24]). Secondly, differences are also likely to be due to the mechanisms of inflicted injury and because children with inflicted injury are likely to have a relatively late presentation to medical care, resulting in delayed treatment during a critical period.

Non-traumatic SDH

Although trauma is the commonest cause of SDH in infancy,[1 3] subdural collections of blood, effusions or empyemas are seen following birth, meningitis, metabolic disorders and in association with haematological disorders.

SDH may occur following instrumental deliveries and also after the normal birth process.[3 25] In contrast to other traumatic causes of SDH, those occurring following childbirth are largely asymptomatic, resolve shortly after birth and do not evolve into

Table 1 Retrospective and prospective studies of outcome following inflicted brain injury

	Number of patients	Follow-up period	Outcome measure	Died	Disabled	Good recovery
Retrospective studies						
Bonnier *et al*, 1995[18]	13	Mean 7 years 2 months	Neurological exam Psychological exam Developmental tests (various including WISC)	1	5 (+1 vegetative state)	7 initially, 1 at 5 years
Duhaime *et al*,1996[19]	84	Mean 9 years	Modified GOS (telephone interview)	22	9/14 (data available on 14 children)	5/14
Haviland and Ross Russell, 1997[12]	15	3 months to 3 years	Mild, moderate or severe disability	2	12	1
Barlow and Minns, 1999[20]	17	Mean 33 months	Seshia outcome scale	2	8	7
Barlow and Minns, 2000[2]	44	3 years	Seshia outcome scale	6	22/42 (data available on 42 children)	14/42
King *et al*, 2003[17]	364	At hospital discharge	Paediatric Cerebral Performance Category scale	69	274 (including 251 requiring ongoing care)	21
Karandikar *et al*, 2004[21]	65	2 years	KOSCHI	16	20/45 (data available on 45 children)	25/45
Keenan *et al*, 2004[22]	80	At discharge	POPC	18	28	34
Keenan *et al*, 2006[9]	41	1 year	POPC Stein-Jessop Functional Status II (Revised) Global Health Index	Only survivors reported	29	12
Prospective studies						
Ewing-Cobbs *et al*, 1998[6]	20	Mean 1.3 months	Modified GOS Standardised developmental tests	Only survivors reported	16	4
Ewing-Cobbs *et al*, 1999[7]	28	1–3 months	Modified GOS Bayley development scales	Only survivors reported	21	7
Barlow *et al*, 2005[8]	25	59 months	GOS Seshia outcome scale Standardised developmental tests	1	16	8

GOS, Glasgow Outcome Scale; KOSCHI, King's Outcome Scale for Childhood Head Injury; POPC, Pediatric Overall Performance Category; WISC, Wechsler Intelligence Scale for Children.

chronic subdural effusions; in such patients, SDH do not need intervention and resolve spontaneously within 4 weeks.[25]

The outcome in individuals with SDH associated with bacterial meningitis is similar to the outcome in individuals with meningitis alone,[26] suggesting that the poor outcome is related to the global brain insult caused by the associated inflammatory process rather than as a result of subdural empyemas alone. Subdural empyemas following bacterial meningitis may cause pressure effects and require neurosurgical intervention; a large retrospective study including both children and adults with SDH showed a mortality of 12.2%, morbidity of 26% and good outcome by Glasgow Outcome Scores in 82% of patients.[27]

Considering metabolic disorders, type 1 glutaric aciduria results in microencephalic macrocephaly and haemorrhages may result from associated stretched bridging veins secondary to cerebral atrophy.[28] In keeping with the various causes of SDH, other mechanisms (in this case metabolic encephalopathy affecting the basal ganglia) affect neuronal integrity and influence outcome. Outcome in type 1 glutaric aciduria is also affected by the degree to which acute striatal necrosis affects all muscle groups.[29] The outcome in patients with and without SDH alone is not reported.

The final group of children who may present with SDH are those with blood clotting disorders; however, no cases series report on outcomes in such individuals. In contrast to other mechanisms of SDH, as haemorrhage may occur spontaneously or following seemingly innocuous trauma, individuals with clotting disorders may represent a useful study group in which to assess outcome following SDH and their immediate sequelae alone, without the complicating factors of other brain related insults.

SPECIFIC NEURODISABILITY OUTCOMES

In addition to the global outcomes reported in table 1, specific neurodisabilities are also associated with the presence of SDH and are described below. Within each of these outcome categories, the severity of disability varies with the causal mechanism.

Neurological outcome

Nearly two thirds of infants who sustain non-accidental head injury will have neurological sequelae.[1] Various motor patterns evolve including hemiplegia, quadriplegia, ataxia, hypotonia and dystonia.[1 8 17 21 30] Other abnormalities include speech and language difficulties, hearing impairment, cranial nerve abnormalities, microcephaly, hydrocephalus and epilepsy.[8 21 31] As previously discussed, specific neurological sequelae are less likely to occur in individuals with accidental head injury than with inflicted head injury.[6] Neurological outcome in children with SDH and bacterial meningitis is very similar to that in children with meningitis alone.[26] Individuals with birth-related SDH are thought to have normal neurological outcome, but longitudinal studies are lacking.

Seizures

Individuals with inflicted SDH may experience acute seizures. However, post-traumatic epilepsy is also seen in children who have sustained subdural haemorrhage[1 2 21 32]; multifocal seizures and infantile spasms have also been reported.[8] In contrast, children are less likely to have seizures following SDH associated with accidental trauma than with inflicted injury.[6] Seizures are no more common in those with meningitis and SDH than in those with meningitis alone.[26]

Vision

Following traumatic brain injury, visual sequelae can result from vitreous and retinal haemorrhages as well as cortical visual dysfunction. Most intraocular haemorrhages resolve within 4 weeks.[33] Visual dysfunction includes visual field defects, visual agnosia, visual acuity loss and ocular movement abnormalities.[8 21] Retinal haemorrhages are more likely to be seen in children with inflicted than accidentally caused SDH; infants with inflicted injury frequently have visual impairment.[34] Accidentally caused retinal haemorrhages are more likely to be unilateral than bilateral and thus may result in less overall morbidity.[6 35] Individuals who suffer SDH and associated brain injuries may develop cortical blindness and optic atrophy due to mechanisms unrelated to the SDH itself.

Speech and language

Following SDH, speech and language abnormalities may occur and are usually associated with a broader cognitive impairment.[6–8] Some infants may develop an autistic spectrum disorder following SDH.[8 30] Other speech problems in children with SDH are a result of dysarthria and bulbar dysfunction secondary to associated brain injuries. Children are more likely to require speech therapy following SDH secondary to inflicted than accidental causes.[9]

COGNITIVE, BEHAVIOURAL AND NEUROPSYCHIATRIC OUTCOMES

Behavioural problems are seen in a large proportion of children who have suffered SDH in infancy; occasionally developmental arrest and autistic regression occur.[8] Rage reactions, self-injurious behaviour, temper tantrums, hyperactivity, attention deficit disorders and stereotypical ritualistic behaviours are all described and sleep disturbances are also seen.[8] Poor concentration and reduced attention result in deteriorating school performance.[8 21] Special educational needs are identified in a large proportion of children with inflicted brain injury. In a study of school age children, five of 18 children attending mainstream school had a statement of special educational needs. In addition, seven children attended special schools.[21]

Neuropsychological testing frequently confirms significant impairment of intellectual function following SDH and traumatic brain injury in children.[6 7 36–38] In a careful and detailed study of cognitive function using Bayley Scales of Infant Development, British Ability Scales and Vineland Adaptive Behavioural Scales, Barlow *et al* have shown memory deficits, attention deficits, poor social skills, low initiative, emotional liability, inappropriate sexual behaviours and poor motor skills[8]; these features are mainly indicative of frontal lobe dysfunction. Since the frontal lobes continue to mature during late childhood and adolescence, related difficulties may only be seen several years after SDH and brain injury, emphasising the need for sufficient long-term follow-up.[21]

SOCIAL AND LEGAL OUTCOMES

Of particular relevance to SDH secondary to inflicted injuries, and beyond the neurodevelopmental outcome, is the outcome for the child's family unit. The social well-being of the child depends on the legal and social outcome in every case. Legal and social outcomes have been largely neglected by researchers who have understandably concentrated on the outcome in the individual involved. Systematic non-biased research in this important area is methodologically difficult due to data protection, patient confidentiality, achieving consent from the child's parents and the emotive nature of physical abuse. Inflicted SDH are more likely to occur in lower socioeconomic class families[3]; demographic data relating to the alleged perpetrators and their victims (social class, employment and area of residence) is available to a limited extent.[1 3 39] Furthermore, limited data are available on outcomes of child protection conferences and criminal prosecutions. Successful prosecutions are achieved in a minority of cases[17] and most

children are returned to their previous homes.[12 17] In summary, longitudinal outcome studies gathering data on the result of social and legal outcomes for infants and families (child protection procedures and criminal prosecution, estrangement within the family, fostering and adoption and further abuse within the family) are badly needed.

RADIOLOGICAL OUTCOME

Most acute SDH show radiological evidence of resolution within 4–6 weeks. Following a traumatic cause of SDH, associated brain parenchymal injuries are often present. Associated injuries are diverse and relate to the mechanism of injury; radiologically identified damage in the form of cerebral oedema, infarction, parenchymal and ventricular haemorrhages, contusions, hypoxic ischaemic injury and diffuse axonal injury, and gliotic scaring in subcortical white matter, cerebellar hemispheres and corpus callosum is seen in association with SDH.[40 41] Follow-up imaging after an appropriate interval may then reveal residual gliosis, cerebral atrophy, hydrocephalus or porencephaly.[31 41] Cranial growth deceleration and intraparenchymal brain abnormalities detected in the first 3 months following injury have been found to be significantly associated with poor short- and long-term neurodevelopmental outcome.[6 41]

PROGNOSTIC AND PREDICTIVE FACTORS FOR OUTCOME FOLLOWING SDH

In studies of outcome following SDH, the premorbid factors may influence results. For example, genetic, social and environmental factors, premorbid developmental disorders and neurodevelopmental progress are all important. Management during the acute phase following injury also affects outcome; however, aggressive management was not associated with better outcome in one study.[19] In traumatic brain injury, Glasgow Coma Scale, cerebral perfusion pressure and mean arterial pressure control may affect outcome.[20] Other factors such as duration of impaired consciousness and number of lesions on imaging are also important.[38] Various other predictive factors for poor prognosis include the presence of early seizures, apnoea, raised intracranial pressure, hypotension, vitreous haemorrhages, skull fractures, intraparenchymal brain abnormalities, brain swelling and diffuse axonal injury.[20 41–44]

Important post-injury factors include the legal and social outcomes of child protection proceedings (whether the child returns to an abusive environment with resultant further physical and/or emotional abuse and neglect) and whether further insults to the brain occur from causes such as uncontrolled epilepsy, hydrocephalus, visual impairment and cognitive impairment.

Finally, we should consider whether outcome in children with SDH can be predicted in other ways. Berger *et al*[45] have recently investigated the measurement of the degree of brain injury through endogenous markers rather than neuroradiological factors or observed clinical symptoms or signs. The combined use of specific biochemical markers of neuronal damage,[45 46] clinical features such as bilateral retinal haemorrhages[35] as well as radiological features such as diffuse axonal injury[40] in order to predict outcome following SDH may lead to new prognostic opportunities in the field. Were an endogenous marker considered sensitive and specific enough to indicate that a neuronal injury had occurred, this could form the basis of a screening test which could be used to identify individuals who required neuroimaging. If chemical or radiological markers were predictors of potential poor outcome,[46] increased early intervention and rehabilitation might be appropriate in order to improve future progress.

Key points and recommendations

- Studies of outcome in infants with SDH have been limited by research methodology.
- Outcome in infants with inflicted SDH is poor compared to those with SDH from other causes.
- Studies of social and legal outcomes are necessary in order to assess the importance of appropriate support measures for the child and family.
- Studies are required to ascertain whether specific clinical, radiological and biochemical features at acute clinical presentation predict long-term outcome.
- Large, prospective multicentre longitudinal studies of infants with SDH are required. Clinical and neuropsychological assessment by multidisciplinary teams using standardised outcome measures are necessary to detect subtle neurodevelopmental and cognitive deficits which otherwise may be overlooked.

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

In summary, there are few published studies that compare and contrast the differences in outcome between SDH of differing aetiology. Furthermore, studies of outcome following accidental or non-accidental trauma do not report subgroup outcomes by either age or the presence of SDH with or without other brain injury. Multicentre prospective longitudinal studies of outcome following SDH (as measured by clinical examination, standardised outcome scores and neuropsychological tests) are required. Such studies should include children of all ages who have suffered SDH of all aetiologies, in order to show whether outcomes differ by causal mechanism and age, and to identify which of the associated markers of injury correlate best with poor outcome.

Authors' affiliations
Sandeep Jayawant, Jeremy Parr, Department of Paediatric Neurology, John Radcliffe Hospital, Oxford, UK

Competing interests: None.

REFERENCES

1 **Jayawant S**, Rawlinson A, Gibbon F, *et al*. Subdural haemorrhages in infants: population based study. *BMJ* 1998;**317**:1558–61.
2 **Barlow KM**, Minns RA. Annual incidence of shaken impact syndrome in young children. *Lancet* 2000;**356**:1571–2.
3 **Hobbs C**, Childs AM, Wynne J, *et al*. Subdural haematoma and effusion in infancy: an epidemiological study. *Arch Dis Child* 2005;**90**(9):952–5.
4 **Caffey J**. Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. *Am J Roentgenol* 1946;**56**:163–7.
5 Minns RA, Brown JK, eds. *Shaking and other non-accidental head injuries in children*. Clinics in developmental medicine, No. 162. London: Mac Keith Press, 2005.
6 **Ewing-Cobbs L**, Kramer L, Prasad M, *et al*. Neuroimaging, physical and developmental findings after inflicted and non-inflicted traumatic brain injury in young children. *Pediatrics* 1998;**102**:300–7.
7 **Ewing-Cobbs L**, Prasad M, Kramer L, *et al*. Inflicted traumatic brain injury: relationship of developmental outcome to severity of injury. *Pediatr Neurosurg* 1999;**31**(5):251–8.
8 **Barlow KM**, Thomson E, Johnson D, *et al*. Late neurological and cognitive sequelae of inflicted traumatic brain injury in infancy. *Pediatrics* 2005;**116**:174–85.
9 **Keenan HT**, Runyan DK, Nocera M. Child outcomes and family characteristics 1 year after severe inflicted or noninflicted traumatic brain injury. *Pediatrics* 2006;**117**(2):317–24.
10 **Keenan HT**, Runyan DK, Nocera M. Longitudinal follow-up of families and young children with traumatic brain injury. *Pediatrics* 2006;**117**(4):1291–7.
11 **Ludwig S**. Shaken baby syndrome: a review of 20 cases. *Ann Emerg Med* 1984;**13**:104–7.

12 **Haviland J**, Ross Russell RI. Outcome after severe non-accidental head injury. *Arch Dis Child* 1997;**77**:504–7.
13 **Seshia SS**, Johnston B, Kasian G. Non-traumatic coma in childhood: clinical variables in prediction of outcome. *Dev Med Child Neurol* 1983;**25**:492–501.
14 **Jennett B**, Bond M. Assessment of outcome after severe brain damage: a practical scale: *Lancet* 1975;**1**(7905):480–4.
15 **Koelfen W**, Freund M, Dinter D, *et al*. Long-term follow up of children with head injuries-classified as ''good recovery'' using the Glasgow Outcome Scale: neurological, neuropsychological and magnetic resonance imaging results. *Eur J Pediatr* 1997;**156**(3):230–5.
16 **Goldstein FC**, Levin HS. Epidemiology of pediatric closed head injury: incidence, clinical characteristics and risk factors. *J Learn Disabil* 1987;**20**:518–25.
17 **King WJ**, MacKay M, Sirnick A; Canadian Shaken Baby Study Group. Shaken baby syndrome in Canada: clinical characteristics and outcomes of hospital cases. *CMAJ* 2003;**168**(2):155–9.
18 **Bonnier C**, Nassogne MC, Evrard P. Outcome and prognosis of whiplash shaken infant syndrome; late consequences after a symptom free interval. *Dev Med Child Neurol* 1995;**37**:943–56.
19 **Duhaime AC**, Christian C, Moss E, *et al*. Long term outcome in infants with the shaking impact syndrome. *Pediatr Neurosurg* 1996;**24**:292–8.
20 **Barlow KM**, Minns RA. The relationship between intracranial pressure and outcome in non-accidental head injury. *Dev Med Child Neurol* 1999;**41**:220–5.
21 **Karandikar S**, Coles L, Jayawant S, *et al*. The neurodevelopmental outcome in infants who have sustained a subdural haemorrhage from non-accidental head injury. *Child Abuse Review* 2004;**13**:178–87.
22 **Keenan HT**, Runyan DK, Marshall SW, *et al*. A population-based comparison of clinical and outcome characteristics of young children with serious inflicted and noninflicted traumatic brain injury. *Pediatrics* 2004;**114**(3):633–9.
23 **Vinchon M**, Defoort-Dhellemmes S, Noule N, *et al*. [Accidental or non-accidental brain injury in infants. Prospective study of 88 cases]. *Presse Med* 2004;**33**(17):1174–9.
24 **Salehi-Had H**, Brandt JD, Rosas AJ, *et al*. Findings in older children with abusive head injury: does shaken-child syndrome exist? *Pediatrics* 2006;**117**(5):e1039–44.
25 **Whitby EH**, Griffiths PD, Rutter S, *et al*. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet* 2004;**363**(9412):846–51.
26 **Snedeker JD**, Kaplan SL, Dodge PR, *et al*. Subdural effusion and its relationship with neurological sequelae of bacterial meningitis in infancy: a prospective study. *Pediatrics* 1990;**86**:163–70.
27 **Nathoo N**, Nadvi SS, van Dellen JR, *et al*. Intracranial subdural empyemas in the era of computed tomography: a review of 699 cases. *Neurosurgery* 1999;**44**(3):529–35.
28 **Morris AA**, Hoffmann GF, Naughten ER, *et al*. Glutaric aciduria and suspected child abuse. *Arch Dis Child* 1999;**80**(5):404–5.
29 **Strauss KA**, Puffenberger EG, Robinson DL, *et al*. Type I glutaric aciduria, part 1: natural history of 77 patients. *Am J Med Genet C Semin Med Genet* 2003;**121**(1):38–52.
30 **Barlow K**, Thompson E, Johnson D, *et al*. The neurological outcome of non-accidental injury. *Pediatr Rehabil* 2004;**7**(3):195–203.
31 **Lo TY**, Phillips M, Minns RA, *et al*. Cerebral atrophy following shaken impact syndrome and other non-accidental head injury. *Pediatr Rehabil* 2003;**6**(1):47–55.
32 **Barlow KM**, Spowart JJ, Minns RA. Early post traumatic seizures in non-accidental head injury: relation to outcome. *Dev Med Child Neurol* 2000;**42**(9):591–4.
33 **Pierre-Kahn V**, Roche O, Dureau P, *et al*. Ophthalmologic findings in suspected child abuse victims with subdural haematomas. *Ophthalmology* 2003;**10**(9):1718–23.
34 **Wilkinson WS**, Han DP, Rappley MD, *et al*. Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. *Arch Ophthalmol* 1989;**107**(10):1472–4.
35 **Bechtel K**, Stoessel K, Leventhal JM, *et al*. Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. *Pediatrics* 2004;**114**(1):165–8.
36 **Ewing-Cobbs L**, Prasad MR, Landry SH, *et al*. Executive functions following traumatic brain injury in young children: a preliminary analysis. *Dev Neuropsychol* 2004;**26**(1):487–512.
37 **Taylor HG**, Yeates KO, Wade SL, *et al*. A prospective study of short- and long-term outcomes after traumatic brain injury in children: behaviour and achievement. *Neuropsychology* 2002;**16**(1):15–27.
38 **Prasad MR**, Kramer LA, Ewing-Cobbs L. Cognitive and neuroimaging findings in physically abused preschoolers. *Arch Dis Child* 2005;**90**(1):82–5.
39 **Sanders T**, Cobley C, Coles L, *et al*. Factors affecting clinical referral of young children with a subdural haemorrhage to child protection agencies. *Child Abuse Review* 2003;**12**:358–73.
40 **Datta S**, Stoodley N, Jayawant S, *et al*. Neuroradiological aspects of subdural haemorrhages. *Arch Dis Child* 2005;**90**:947–51.
41 **Bonnier C**, Nassogne MC, Saint-Martin C, *et al*. Neuroimaging of intraparenchymal lesions predicts outcome in shaken baby syndrome. *Pediatrics* 2003;**112**:808–14.
42 **Kemp AM**, Stoodley N, Cobley C, *et al*. Apnoea and brain swelling in non-accidental head injury. *Arch Dis Child* 2003;**88**:472–6.
43 **Coates BM**, Vavilala MS, Mack CD, *et al*. The influence of definition and location of hypotension on outcome following severe pediatric traumatic brain injury. *Crit Care Med* 2005;**33**(11):2645–50.
44 **Chung CY**, Chen CL, Cheng PT, *et al*. Critical score of Glasgow Coma Scale for pediatric traumatic brain injury. *Pediatr Neurol* 2006;**34**:379–87.
45 **Berger RP**, Adelson PD, Pierce MC, *et al*. Serum neuron-specific enolase, S100B, and myelin basic protein concentrations after inflicted and noninflicted traumatic brain injury in children. *J Neurosurg* 2005;**103**(1 Suppl):61–8.
46 **Berger RP**, Dulani T, Adelson PD, *et al*. Identification of inflicted traumatic brain injury in well-appearing infants using serum and cerebrospinal markers: a possible screening tool. *Pediatrics* 2006;**117**(2):325–32.