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Review

The neuropathology of infant subdural haemorrhage

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ABSTRACT

Subdural haemorrhage (SDH) in the infant has a different pattern from that seen in the older child and adult. It is usually a widespread, bilateral, thin film, unlike the thick, space-occupying and often unilateral clot seen in older children and adults after trauma. Whether both arise by the same mechanism is unknown, but it seems unlikely. Most SDH is said to be due to trauma but in infants there are other, atraumatic causes. Birth is also important; recent MRI studies show an incidence of almost 50% in asymptomatic neonates.

Traumatic SDH is said to result from rupture of bridging veins but new insights into the anatomy of infant dura suggest a dural origin for thin film subdural bleeding in young babies. Acute SDH usually rapidly resolves, but sometimes develops into a chronic fluid collection. Healing of SDH is by formation of a granulating membrane which may confer vulnerability to rebleeding, either spontaneously or after an otherwise innocuous event.

SDH has a particular significance as one of the features of the triad (together with retinal haemorrhage and encephalopathy) associated with non-accidental injury. As the possibility of non-accidental injury is often first raised by a radiologic report of subdural bleeding, it becomes critically important in the interpretation of the scan appearances to understand the unique physiology and anatomy of the infant dura.

Contents

1. Incidence

Clinically apparent subdural haemorrhage (SDH) occurs in 12 per 100,000 infants under 2 years and 24 per 100,000 infants under 1 year of age [1]. Most SDH is identified in infants of 0–4 months of age [2].

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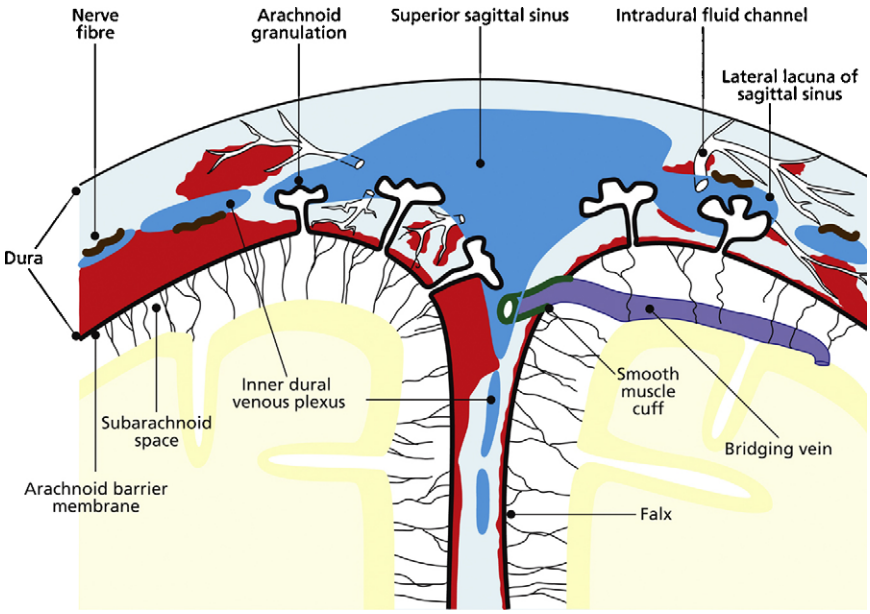

Fig. 1. Diagrammatic representation of a coronal section through the superior sagittal sinus showing the relationships of the sinus, the fluid channels, the arachnoid granulations and the most frequent sites of intradural bleeding.

2. Causes of SDH

SDH is extremely common after birth, recent imaging studies have shown an incidence of SDH of almost 50% in asymptomatic newborns [3]. Trauma was previously considered to be the most common cause of SDH and there are many rarer non-traumatic causes, including coagulation disorders, meningitis, post-neurosurgery, sinus or cortical vein thrombosis, vascular malformations, tumours and metabolic diseases [4,5]. SDH is more common in babies with cranio-cerebral disproportion of any cause, e.g., benign enlargement of the extracerebral spaces, external hydrocephalus and chronic SDH [6,7].

3. Origin of subdural bleeding

It has long been assumed that SDH originates from traumatic rupture of the bridging veins, despite lack of convincing observational data that bridging vein rupture occurs [8,9]. Anatomical and clinical observations indicate that vessels intrinsic to the dura may be a source of bleeding. Bleeding may also occur from a healing subdural membrane and, rarely, SDH may be the result of vascular rupture from other intracranial compartments, for example the subarachnoid space after aneurysmal rupture [10].

In order to consider these alternative hypotheses it is helpful to review the detailed anatomy of the bridging veins and the dura and its blood supply. Fig. 1 is a diagrammatic representation of the dura indicating its detailed structure and frequent sites of intradural bleeding.

4. Bridging veins

Bridging veins are formed by the coalescence of superficial cortical draining veins which run over the brain surface within the subarachnoid space. If ruptured, subarachnoid bleeding will result. As the supratentorial draining veins approach the midline, they unite to form between 9 and 11 large bridging veins which penetrate the arachnoid membrane and pass through the contiguous deep dural layers before entering the sagittal sinus. Recent studies in adults have shown that as the veins pass through the dura they have a muscle coat which acts as a sphincter regulating the flow of blood from the bridging veins, maintaining intravenous pressure when intracranial pressure rises [11,12]. Smooth muscle can be demonstrated in the walls of dural veins close to the superior sagittal sinus, even in fetuses (Fig. 2). The bridging veins are of large calibre, measuring about 3–4 mm in diameter in the adult [13] and carry large volumes of blood from the brain to the dural sinuses. Rupture of such large vessels would

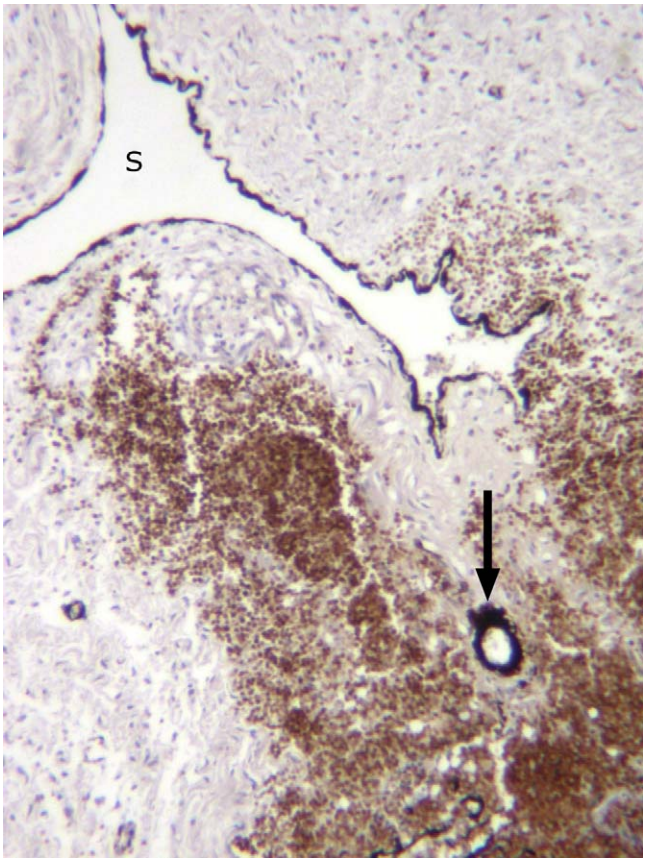

Fig. 2. Infant parasagittal dura stained for smooth muscle actin. Note vessel (arrow) with thick smooth muscle coat close to the superior sagittal sinus (S) (original magnification 4×).

be unlikely to produce the thin film of haemorrhage characteristic of the young infant. However, it is possible that incomplete rupture, or oozing from congested bridging veins, as suggested by Cushing [8] may cause small volume subdural bleed.

5. Dural anatomy

Most of us regard the dura as a tough, fibrous membrane with no other function than the physical protection and support of the brain. However, it is becoming clear, from re-visiting the old literature and from new anatomical studies, that the dura has unique anatomical features in the young infant which may reflect its specific functions and be relevant to the pattern of bleeding in this age group.

All meningeal layers develop from a common primitive mesenchyme; condensation of the outer layer forms the fibrous periosteal and meningeal layers of the dura. The sub- (or inner) dural border cell layer is contiguous with the underlying arachnoid barrier layer, forming a morphological and functional entity. This is unlike development of the lung, for example, where the lung bud grows into a mesenchymal sac giving rise to a potential space between the visceral and parietal pleura. There is no equivalent "subdural space" but there is a subdural compartment consisting of 10–15 layers of loosely arranged flake-like cells with fluid filled spaces between them and few intercellular junctions [14]. The looseness of the cell adhesions in this layer accounts for the ease with which the surgeon or pathologist can lift the dura from the arachnoid membrane. However, careful microsopic examination of the arachnoid frequently reveals flake-like cells from the dural border layer remaining on its dural-facing surface (Fig. 3).

Blood collecting in the dural border cell layer disrupts the loosely adherent cells and forms a "subdural" collection bound superficially by the fibrous dura and deeply by the limiting arachnoid membrane. The widespread distribution of SDH on imaging studies is secondary to the ability of blood to easily dissect through the subdural compartment. Thus, SDH is not haemorrhage into a pre-existing subdural "space", but haemorrhage into the dura, disrupting a specialized compartment of cells. It would be anatomically more correct to replace the term subdural space with subdural compartment.

The dura has an extensive and densely innervated venous plexus between its periosteal and meningeal layers (Fig. 4). This plexus is one of the distinctive features differentiating infant and adult dura, being most extensive in fetal life. In the neonate the venous plexus is so extensive that it forms sinuses in the tentorium, posterior falx and the dura of the floor of the posterior

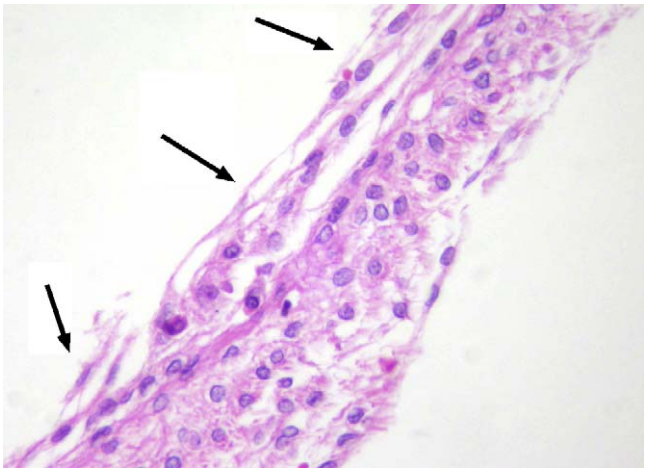

Fig. 3. Infant arachnoid membrane showing flaky cells attached to the dural surface (arrows) (original magnification 40×).

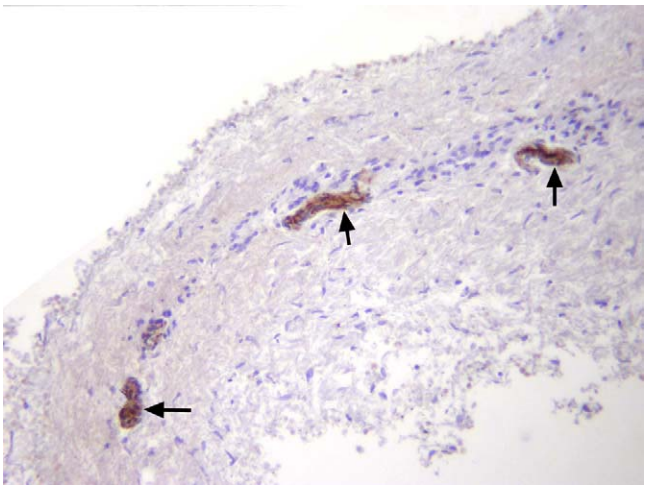

Fig. 4. Section of tentorium in infant showing vascular plexus between the two leaflets of the dura. Arrows indicate frequent nerve bundles (anti-neurofilament with haematoxylin counterstain) (original magnification 10×).

cranial fossa, subsequently dwindling to be represented only by the familiar major sinuses of the adult dura [15,16]. While the function of these venous sinuses is unknown, they may be of significance in subdural bleeding in early infant life. Infant SDH is predominantly found over the dural folds bearing these venous sinuses [3,17]. Friede saw falcine haemorrhage so commonly that he assumed it to be the result of asphyxia rather than trauma [18]. Indeed the radiological finding of a thin, linear, high signal between the hemispheres or over the tentorium has been shown to correlate with intradural bleeding and congestion rather than subdural bleeding (Fig. 5) [19]. Falcine high signal is seen in infants with brain swelling unrelated to trauma [20], and in a series of infants with traumatic brain injury falcine high signal was more common in infants with brain swelling than in those without [21]. In hypoxic-ischaemic injury (HII) bleeding into the dura may be sufficiently extensive to rupture on to the sub- (or inner) dural surface (Fig. 6). This may be associated with macroscopic and radiologically evident collections [22].

There is an extensive network of fluid containing channels in the dura, most dense in the parasagittal regions [24,25,13]. They communicate with the superior sagittal sinus via the lateral lacunae in intimate relation to the arachnoid granulations, above and separate from the sites of entry of the bridging veins. Rounded channels without an endothelial lining, which are prominent in sections of the dura, are thought to represent these channels (Figs. 1 and 7).

A second distinctive feature of the infant dura is immaturity of the arachnoid villi, which are poorly developed in the first 7 months of life. The dura may play a more prominent role in CSF absorption in this period [26,27]. Cells in the deep dural layers abutting the border layer contain many mitochondria and cytoplasmic vesicles, and blood vessels in this layer have thin, fenestrated walls and intracellular vesicles, consistent with fluid uptake [28]. Dye injected into the subarachnoid space appears in dural channels and later in the superior sagittal sinus, indicating a pathway for CSF absorption [27].

These observations lead to the conclusion that the dura is far more than a fibrous supporting membrane. The presence of valveless channels entering the sinuses and the extensive vascularity of the dural folds may well predispose to congestion and bleeding if intracrerebral and intravascular pressure relationships are disturbed in the first months of postnatal life. Pressure changes, congestion, and intradural bleeding may be responsible for the common finding of small volumes of SDH after birth and

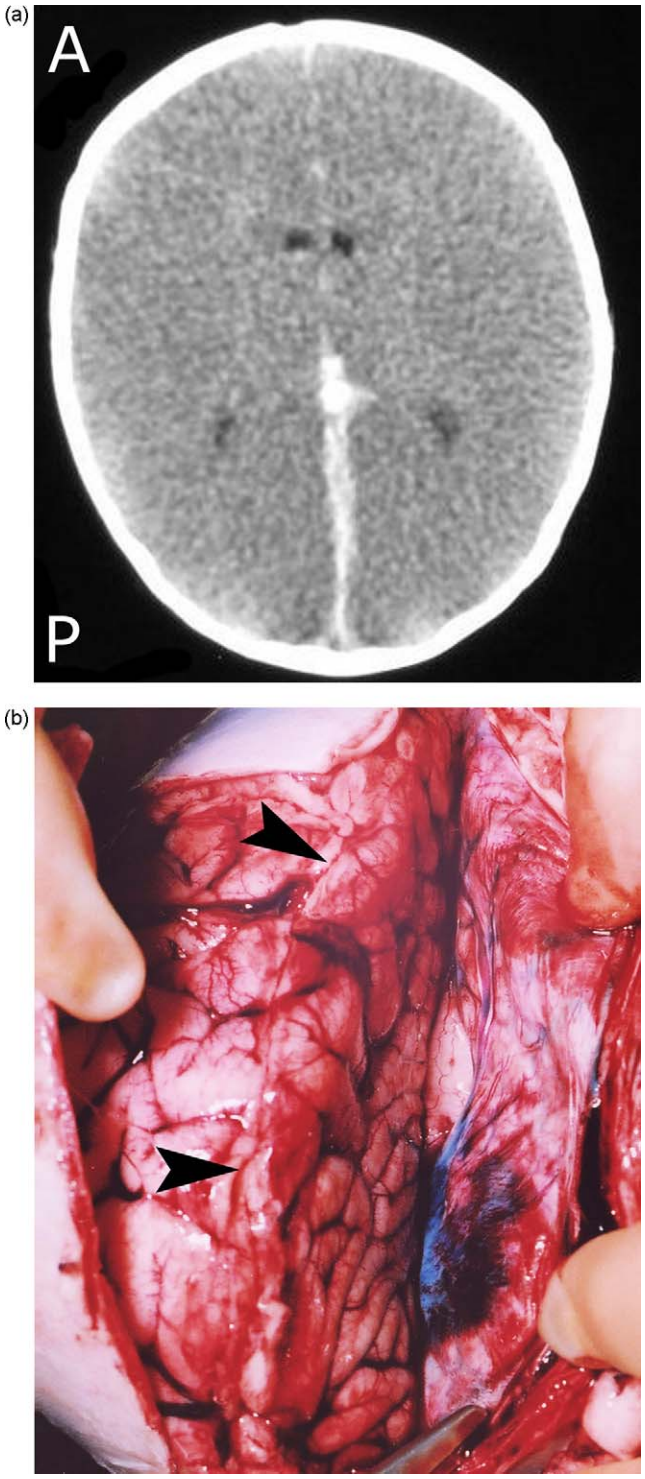

Fig. 5. 4-Month-old infant, sudden unexpected death. (a) CT scan of brain showing midline linear high signal in the position of the falx. (b) Autopsy picture of the same case. The falx is seen in the midline. Arrows indicate the upper parafalcine border of the left cerebral hemisphere. There is a well-defined area of bleeding within the posterior falx but no SDH was seen in relation to any part of the falx or over the hemispheres.

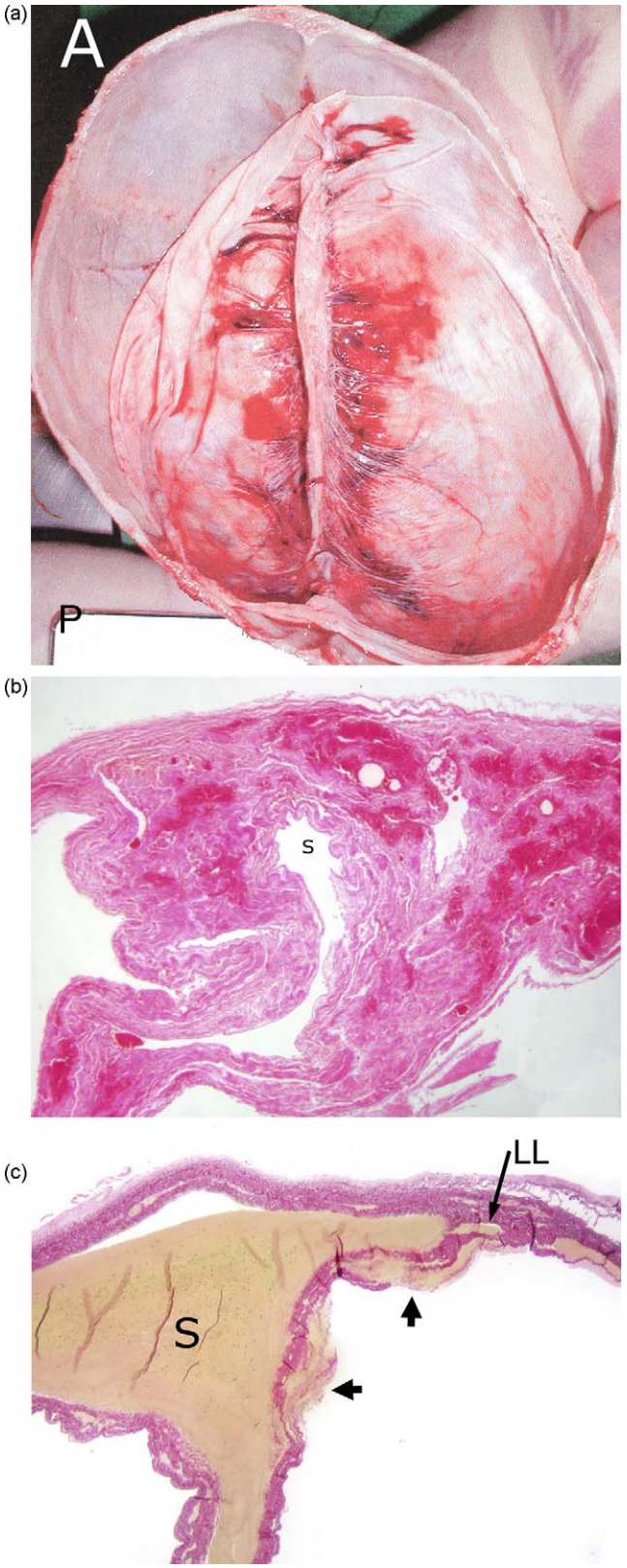

Fig. 6. (a) Subdural haemorrhage in 3-year old with cerebral palsy who died following a prolonged convulsion. Inner aspect of cranial vault showing congestion and bleeding from parasagittal dura. (b) Sagittal sinus (S) and bleeding into adjacent dura. 3-Day infant severe birth asphyxia (original magnification 2×). (c) 2-Month infant. Section of sagittal sinus (S) with lateral lacunae (LL) and adjacent dura. There is fresh blood splitting the layers of the dura and leaking on to the subdural surface (short arrows). (HVG stain; blood is tan, fibrous tissue pink) (original magnification 1×). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

potentially in the first months of postnatal life while the dural anatomy remains immature.

6. Birth related SDH

A series of imaging studies in the last few years have shown an incidence of SDH in between 9 and 46% of asymptomatic neonates

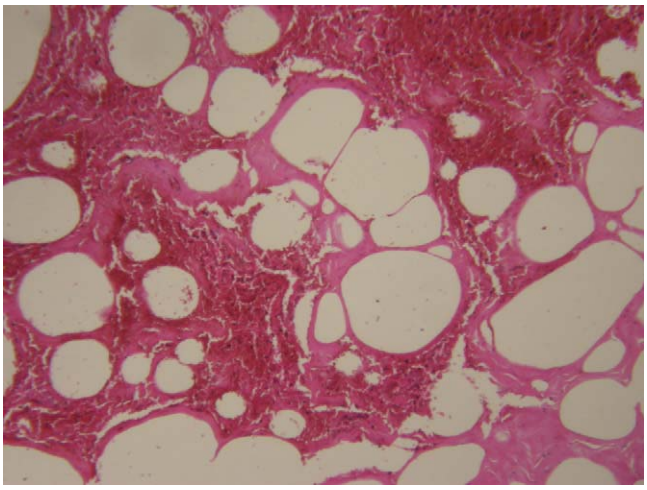

Fig. 7. 2-Year old died after prolonged seizure. The parasagittal dura contains many rounded spaces most without an endothelial lining. These are thought to be intradural fluid channels. There is bleeding into the intervening tissue (original magnification 4×).

after normal, instrumental and caesarian delivery [3,17,29]. The majority are found in the posterior fossa or over the posterior part of the brain; isolated supratentorial haemorrhages are rare [29]. This is highly suggestive of an origin in the vascular dural folds of the posterior fossa and confirms Volpe's belief that neonatal SDH is frequently non-traumatic and arises from the tributaries of the dural sinuses within the dural folds [30]. Craig described

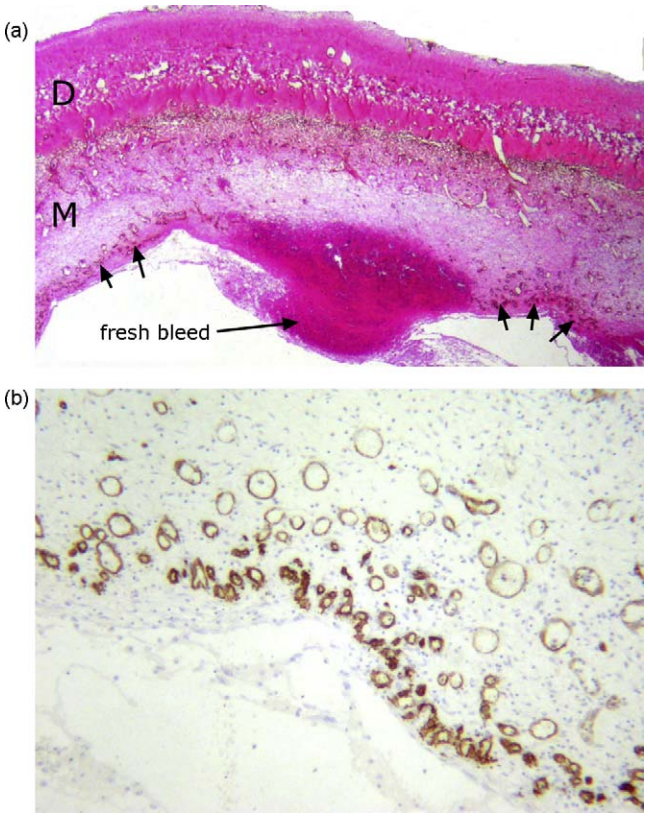

Fig. 8. Section of dura from 7-month-old infant who died in care 4 months after severe non-accidental head injury. (a) There is a thin uniform subdural membrane (M) about the same thickness as the dura (D). A small fresh bleed is seen in the innermost layers of the subdural membrane. Arrows indicate a band of capillaries within the subdural membrane (original magnification 2×). (b) CD 34 stain shows the thick band of macrocapillaries on the free edge of the subdural membrane, the source of the fresh haemorrhage (original magnification 4×).

intrafalcine and intratentorial bleeding in 15% of neonates with SDH and tearing of the bridging veins in only 5%, all of whom had overriding sutures [31].

Two MRI studies have followed a total of 27 babies with birth related subdural haemorrhages with repeat scans at 1–3 months of age. One baby developed further subdural bleeding [3,29]. Due to the very small numbers in these studies, meaningful interpretation is difficult and we have no good data on the consequences of birth related SDH. However, it is likely that most heal without any significant morbidity.

7. What causes bleeding from the infant dura?

A combination of raised intravascular pressure with hypoxic endothelial damage has been implicated as the cause of oozing from meningeal vessels leading to thin film SDH [32]. In the unfused infant skull elasticity of the sutures results in a non-rigid cranial vault. While the lack of rigidity offers the advantage of cranial molding during birth, and may protect against sudden rises of intracranial pressure, the vessel walls are less supported and transmural pressure differentials may become very high [33]. The intradural vessels, thin-walled and adapted for fluid resorption, may be the most likely to leak in these circumstances, resulting in bleeding into the dura.

SDH is seen in hypoxic infants in daily pathological practice [23,34,35] and Larroche noted an association between SDH and ischaemic brain injury in term infants [36]. While two studies have indicated that hypoxia alone is not associated with SDH [37,38], the Byard study [38] included an unselected group of babies up to 3 years in age, and used inadequate histological criteria to confirm HII. Neither study took into account duration of hypoxia, or a history of resuscitation or terminal ventilation. Most babies presenting with thin film SDH have been vigorously resuscitated following a prolonged period of apnoea. These babies are often profoundly hypoxic, acidotic and have deranged clotting. The possible contribution of vascular leakage secondary to reperfusion injury in infant SDH must be considered.

8. Distribution of SDH

In the first few days after bleeding SDH sediments under the influence of gravity and undergoes secondary redistribution to dependent parts of the intracranial compartment [3,39]. Blood spreads widely between all subdural compartments and tracks down around the spinal cord [14]. In babies with intracranial SDH, blood is regularly seen at autopsy in the spinal subdural space, particularly over the dorsal cord and in the most dependent parts of the dural sac. This has been confirmed in MRI studies [40]. Blood is also frequently seen as the nerve roots penetrate the dura, an area of intense vascularity related to local CSF absorption [28,41].

9. Tissue responses and evolution of SDH

Blood clot in the subdural compartment induces a series of tissue responses. In the first days after bleeding macrophages enter the clot and lysis begins. Red cells lose their shape. Haemoglobin is converted to haemosiderin which can be identified with Perl's stain from about 2 to 4 days.

After some 3 or 4 days the infiltrating macrophages ingest red cells and breakdown products are visible within their cytoplasm. These cells may persist for months and even years after injury. Fibroblasts and capillaries grow into the clot and form large thin-walled vascular channels (macro-capillaries) which are often particularly numerous at the deep border of the membrane. By about 10 days the membrane becomes visible with the naked eye. It may be extensive and thin, often no thicker than the dura (Fig. 8). In time the numbers of capillaries are reduced and the membrane

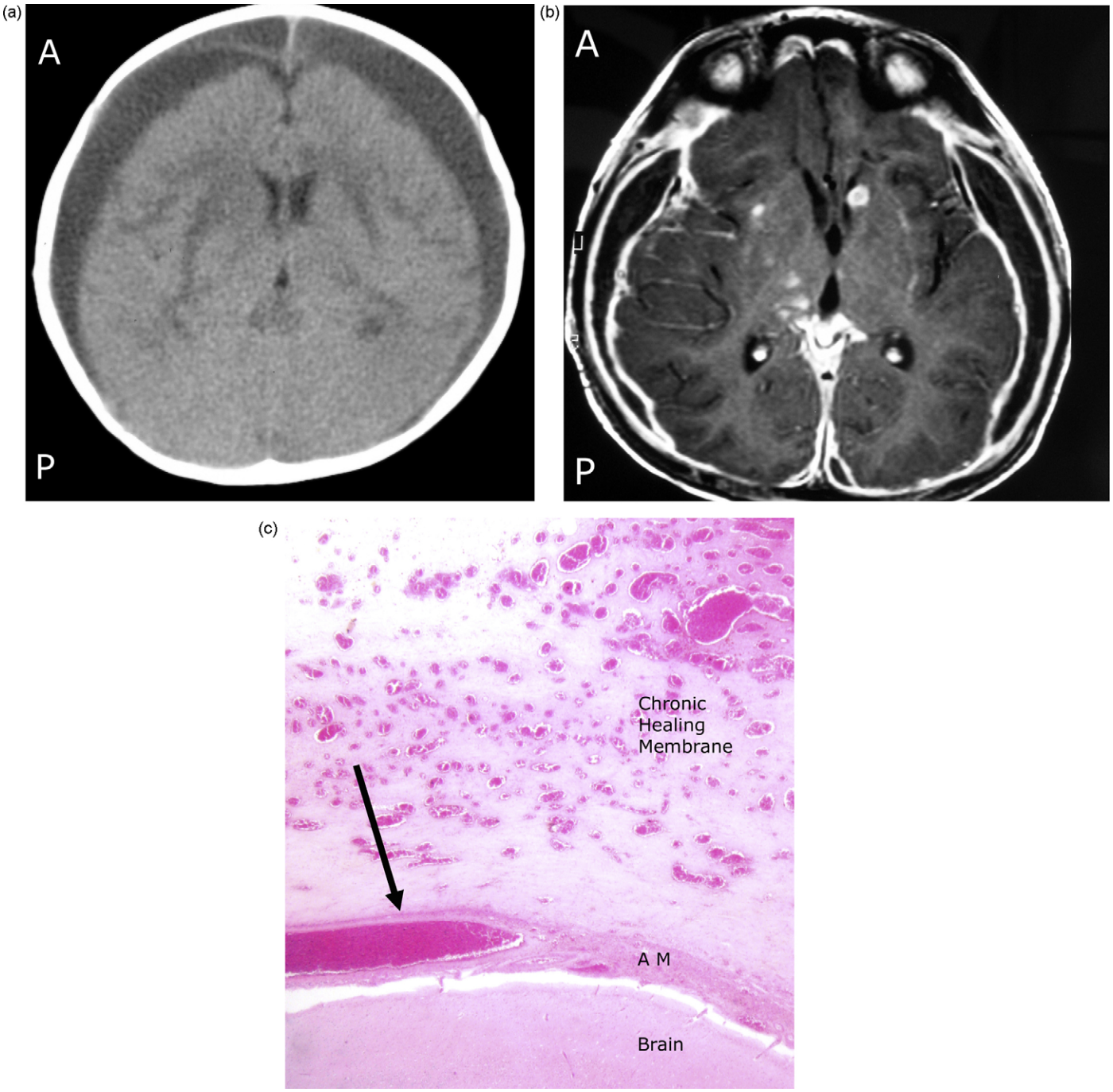

Fig. 9. Infant aged 1 year with chronic SDH following NAHI and meningitis 5 months before death. (a) CT scan shows chronic SDH. (b) MRI scan following contrast enhancement shows a bright line outlining the chronic SDH. This is the signal from the capillaries in the chronic membrane. (c) Histology showing the arachnoid membrane adherent to the chronic healing subdural membrane. A focal fresh bleed is seen (arrow) (original magnification 2×).

becomes predominantly fibrous, with calcification or even areas of ossification.

Healing subdural membranes may be much more common than generally recognised. They are found frequently in neurosurgical series and Rogers found them in 25% of babies dying unexpectedly [42,43]. Such membranes are not seen on routine brain scans but enhance very brightly with contrast [44] (Fig. 9).

As noted, birth related subdural is most commonly found in the posterior fossa [3,17] and tends to redistribute to dependent areas. If the dura from the base of the posterior fossa is not sampled at autopsy then chronic membrane formation and evidence of previous bleeding may be missed.

Although never precise, pathological responses in the first few weeks give some indication of when a bleed has occurred. Timings have mainly been derived from studies on neonates [45] or extrapolated from adult values [46] and so must be interpreted with care and in the light of other features of any individual case. The dura retains its blood supply when a terminally ill infant is nursed on a ventilator, in contrast to the brain where brain swelling and impairment of cerebral blood flow modifies tissue reactive processes (respirator brain). Dural changes may be more helpful than brain reactions in timing injury.

One of the most striking microscopic features of the subdural membrane is the presence of wide, thin-walled capillaries which

are prone to bleed after minor trauma or even spontaneously [47,5,48].

10. Chronic subdural haematoma

Chronic SDH may be a widespread fluid collection, a localised encapsulated "hygroma" or a thin resolving membrane.

Only rarely in infants is the original blood clot encapsulated by a fibrous membrane forming a "subdural hygroma" of similar shape and location to the original haematoma; this is the usual pattern in adults.

More commonly infant chronic SDH is a widespread fluid accumulation. This may be a delayed process, Hwang and Kim [49] described three infants with acute SDH which resolved radiologically, only to reappear as a chronic collection between 68 and 111 days after initial trauma.

Development of chronic subdural fluid collections is far from well understood. Suggested mechanisms include osmotic accumulation of fluid and repeated haemorrhage into the healing granulating membrane or continued exudation or inflammation of the dural border cell layers [50,51]. Leakage of CSF through the subarachnoid membrane has been considered to play a significant role in chronic subdural fluid collections [21,52]. Vinchon et al. [21] repeatedly observed initial bleeding into the subarachnoid space preceding subdural fluid collection in infant SDH and suggested that the infant has particular CSF hydrodynamics related to immature mechanisms of CSF absorption.

11. Neuroradiological identification of SDH

The clinical diagnosis of infant SDH is commonly made by brain imaging. Accurate radiological interpretation depends on a good understanding of the underlying pathological processes.

Radiological demonstration of subdural compartment haemorrhage in the posterior fossa, over the convexities, and in the supratentorial interhemispheric region was once thought to be characteristic of bridging vein rupture and inflicted injury [53–55]. However, it is impossible to identify by radiology whether the blood is confined to the dura (within the folds of the falx) or collecting in the subdural compartment (See Fig. 5). Therefore, interhemispheric and tentorial high density on CT may represent intradural bleeding and venous congestion, not necessarily subdural bleeding. These findings are signs of an age-related response to trauma rather than an indication of the specific cause of that trauma [21,53,56,57].

12. Subdural haemorrhage as part of the triad of injuries in non-accidental head injury (NAHI)

SDH is perhaps the most significant component of the triad (SDH, retinal haemorrhage and encephalopathy) associated with NAHI. Given this significance in non-accidental injury, it is surprising that so few questions have been asked about the histopathological characteristics, origin and natural history of infant SDH.

Nearly 40 years ago Guthkelch proposed that shaking a baby could cause subdural haemorrhage by tearing bridging veins [58]. Biomechanical studies using animals and models have indicated that the forces required to cause SDH are far greater than those attainable by adult volunteers shaking a dummy [59]. Impact generates far more force than shaking alone and appears to be required to produce SDH [54,60]. The forces required to cause bridging vein rupture would exceed the strength of the infant neck; indeed, infants restrained in car seats and subjected to rapid deceleration and neck hyperflexion (whiplash) in road traffic accidents have cervical fractures and nerve root avulsion rather than SDH [61,62]. Any infant shaken sufficiently violently to produce SDH would be expected also to have injury to the bones and soft tissues of the neck and spinal cord [63–65].

13. Conclusion

The very specific anatomy and physiology of the infant dura should cause us to consider alternative mechanisms to bridging vein rupture as the source of SDH in this age group. There is a real need for detailed observational studies of the natural history of infant SDH. Radiological diagnosis can only be validated by well controlled and age matched studies and by pathological correlation.

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References

[1] C. Hobbs, A.M. Childs, J. Wynne, J. Livingston, A. Seal, Subdural haematoma and effusion in infancy: an epidemiological study, Arch. Dis. Child 90 (9) (2005) 952–955.
[2] A.D. Parent, Pediatric chronic subdural haematoma: a retrospectiive comparative analysis, Pediatr. Neurosurg. 18 (1992) 266–271.
[3] V.J. Rooks, J.P. Eaton, L. Ruess, G.W. Petermann, J. Keck-Wherley, R.C. Pedersen, Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants, AJNR Am. J. Neuroradiol. (2008).
[4] A.M. Kemp, Investigating subdural haemorrhage in infants, Arch. Dis. Child 86 (2) (2002) 98–102.
[5] K.P. Hymel, C. Jenny, R.W. Block, Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies, Child Maltreat. 7 (4) (2002) 329–348.
[6] T. Pittman, Significance of a subdural hematoma in a child with external hydrocephalus, Pediatr. Neurosurg. 39 (2) (2003) 57–59.
[7] P.D. McNeely, J.D. Atkinson, G. Saigal, A.M. O'Gorman, J.P. Farmer, Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse, AJNR Am. J. Neuroradiol. 27 (8) (2006) 1725–1728.
[8] H. Cushing, J.T. Goodrich, Reprint of concerning surgical intervention for the intracranial hemorrhages of the newborn by Harvey Cushing M.D., 1905, Childs Nerv. Syst. 16 (8) (2000) 484–492.
[9] T. Jaspan, Current controversies in the interpretation of nonaccidental head injury, Pediatr. Radiol. 38 (Suppl. 3) (2008) S378–S387 (abstract).
[10] N.J. McLellan, R. Prasad, J. Punt, Spontaneous subhyaloid and retinal haemorrhages in an infant, Arch. Dis. Child 61 (11) (1986) 1130–1132.
[11] Z. Si, L. Luan, D. Kong, G. Zhao, H. Wang, K. Zhang, et al., MRI-based investigation on outflow segment of cerebral venous system under increased ICP condition, Eur. J. Med. Res. 13 (3) (2008) 121–126.
[12] J.R. Vignes, A. Dagain, J. Guerin, D. Liguoro, A hypothesis of cerebral venous system regulation based on a study of the junction between the cortical bridging veins and the superior sagittal sinus. Laboratory investigation, J. Neurosurg. 107 (6) (2007) 1205–1210.
[13] H. Han, W. Tao, M. Zhang, The dural entrance of cerebral bridging veins into the superior sagittal sinus: an anatomical comparison between cadavers and digital subtraction angiography, Neuroradiology 49 (2) (2007) 169–175.
[14] J.R. Orlin, K.K. Osen, T. Hovig, Subdural compartment in pig: a morphologic study with blood and horseradish peroxidase infused subdurally, Anat. Rec. 230 (1) (1991) 22–37.
[15] J. Browder, H.A. Kaplan, A.J. Krieger, Venous lakes in the suboccipital dura mater and falx cerebelli of infants: surgical significance, Surg. Neurol. 4 (1) (1975) 53–55.
[16] R.S. Tubbs, R.G. Louis, L. Acakpo-Satchivi, E.G. Salter, Anatomy of the falcine venous plexus, J. Neurosurg. 107 (2007) 155–157.
[17] C.B. Looney, J.K. Smith, L.H. Merck, H.M. Wolfe, N.C. Chescheir, R.M. Hamer, et al., Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors, Radiology 242 (2) (2007) 535–541.
[18] R.L. Friede, Hemorrhages in asphyxiated premature infants, in: R. Friede (Ed.), Developmental Neuropathology, Springer-Verlag, Gottingen, 1989, pp. 44–58.
[19] K. Kibayashi, H. Shojo, T. Sumida, Dural hemorrhage of the tentorium on postmortem cranial computed tomographic scans in children, Forensic Sci. Int. 154 (2-3) (2005) 206–209.
[20] W. Squier, Natural history of subdural haemorrhage in infants: imaging and pathological correlations, Dev. Med. Child Neurol. 47 (6) (2005) (abstract).
[21] M. Vinchon, O. Noizet, S. Defoort-Dhellemmes, G. Soto-Ares, P. Dhellemmes, Infantile subdural hematomas due to traffic accidents, Pediatr. Neurosurg. 37 (5) (2002) 245–253.

[22] M. Cohen, E Whitby, Hypoxia, intradural haemorrhage and subdural bleeding in the paediatric and perinatal postmortem: are they related? A study combining the use of autopsy and post-mortem magnetic resonance, in: BAFM Winter Meeting, Bath, UK, 2007 (abstract).
[23] M.C. Cohen, I. Scheimberg, Evidence of occurrence of intradural and subdural hemorrhage in the perinatal and neonatal period in the context of hypoxic ischemic encephalopathy. An observational study from two referral institutions in the United Kingdom, Pediatr. Dev. Pathol. (2008) 1.
[24] J. Balo, The dural venous sinuses, Anat. Rec. 106 (3) (1950) 319–324.
[25] R.J. Fox, Anatomic details of intradural channels in the parasagittal dura: a possible pathway for flow of cerebrospinal fluid, Neurosurgery 39 (1) (1996) 84–91.
[26] S. Oi, C. Di Rocco, Proposal of "evolution theory in cerebrospinal fluid dynamics" and minor pathway hydrocephalus in developing immature brain, Childs Nerv. Syst. 22 (7) (2006) 662–669.
[27] C. Papaiconomou, A. Zakharov, N. Azizi, J. Djenic, M. Johnston, Reassessment of the pathways responsible for cerebrospinal fluid absorption in the neonate, Childs Nerv. Syst. 20 (1) (2004) 29–36.
[28] W. Zenker, S. Bankoul, J.S. Braun, Morphological indications for considerable diffuse reabsorption of cerebrospinal fluid in spinal meninges particularly in the areas of meningeal funnels. An electronmicroscopical study including tracing experiments in rats, Anat. Embryol. (Berl.) 189 (3) (1994) 243–258.
[29] E.H. Whitby, P.D. Griffiths, S. Rutter, M.F. Smith, A. Sprigg, P. Ohadike, et al., Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors, Lancet 363 (9412) (2004) 846–851.
[30] J.J. Volpe, Intracranial haemorrhage, in: W.B. Saunders (Ed.), Neurology of the Newborn, 1995, 377–378.
[31] W. Craig, Intra cranial haemorrhage in the newborn—a study of diagnosis and differential diagnosis based on pathological and clinical findings in 126 cases, Arch. Dis. Child 13 (1938) 89–124.
[32] J.F. Geddes, R.C. Tasker, A.K. Hackshaw, C.D. Nickols, G.G. Adams, H.L. Whitwell, et al., Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in shaken baby syndrome? Neuropathol. Appl. Neurobiol. 29 (1) (2003) 14–22.
[33] F. Cowan, M. Thoresen, Changes in superior sagittal sinus blood velocities due to postural alterations and pressure on the head of the newborn infant, Pediatrics 75 (6) (1985) 1038–1047.
[34] M. Cohen, P. Cox, L. Kiho, E. Pollina, I. Scheimberg, Letter to the editor. Lack of evidence for a causal relationship between hypoxic-ischemic encephalopathy and subdural hemorrhage in fetal life, infancy, and early childhood, Pediatr. Dev. Pathol. 10 (6) (2007) 500–501.
[35] M. Cohen, E. Whitby, The use of magnetic resonance as an ancillary technique in the paediatric coronial post mortem examination, in: 53rd Annual Meeting of the Paediatric Pathology Society, Istanbul, Turkey, 6–9–2007, 2007, September 6–8 (abstract).
[36] J.-C. Larroche, Lesions of haemorrhagic type, mainly venous, in: Developmental Pathology of the Neonate, Excerpta Medica, Amsterdam, 1977, pp. 355–398.
[37] M. Hurley, J.N. Smith, H.G. Vyas, T. Jaspan, Does cardiorespiratory arrest in young children cause subdural haematomas? Br. Soc. Neuroradiologists (2007) (abstract).
[38] R.W. Byard, P. Blumbergs, G. Rutty, J. Sperhake, J. Banner, H.F. Krous, Lack of evidence for a causal relationship between hypoxic-ischemic encephalopathy and subdural hemorrhage in fetal life, infancy, and early childhood, Pediatr. Dev. Pathol. 10 (5) (2007) 348–350.
[39] M. Vinchon, N. Noule, P.J. Tchofo, G. Soto-Ares, C. Fourier, P. Dhellemmes, Imaging of head injuries in infants: temporal correlates and forensic implications for the diagnosis of child abuse, J. Neurosurg. 101 (Suppl. 1) (2004) 44–52.
[40] P. Koumellis, N.S. McConachie, T. Jaspan, Spinal subdural hematomas in children with non accidental head injury, Arch. Dis. Child (2008).
[41] M. Edsbagge, M. Tisell, L. Jacobsson, C. Wikkelso, Spinal, CSF absorption in healthy individuals, Am. J. Physiol. Regul. Integr. Comp. Physiol. 287 (6) (2004) R1450–R1455.
[42] C.B. Rogers, H.H. Itabashi, U. Tomiyasu, E.T. Heuser, Subdural neomembranes and sudden infant death syndrome, J. Forensic Sci. 43 (2) (1998) 375–376.
[43] A. Ikeda, O. Sato, R. Tsugane, N. Shibuya, I. Yamamoto, M. Shimoda, Infantile acute subdural hematoma, Childs Nerv. Syst. 3 (1) (1987) 19–22.
[44] D.A. Bruce, R.A. Zimmerman, Shaken impact syndrome, Pediatr. Ann. 18 (8) (1989) 492, 482-9.
[45] M. Squier, Acquired diseases of the central nervous system, in: J. Keeling (Ed.), Fetal and Neonatal Pathology, Springer-Verlag, London, 2001, pp. 571–594.
[46] J.E. Leestma, Impact Injuries to the Brain and Head. Forensic Neuropathology, Raven Press, New York, 1988, pp. 184–253.
[47] R. Uscinski, Shaken baby syndrome: fundamental questions, Br. J. Neurosurg. 16 (3) (2002) 217–219.
[48] J. Piatt, A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages, Neurosurg. Focus 7 (4) (1999).
[49] S.K. Hwang, S.L. Kim, Infantile head injury, with special reference to the development of chronic subdural hematoma, Childs Nerv. Syst. 16 (9) (2000) 590–594.
[50] T.M. Markwalder, Chronic subdural hematomas: a review, J. Neurosurg. 54 (5) (1981) 637–645.
[51] J.F. Feng, J.Y. Jiang, Y.H. Bao, Y.M. Liang, Y.H. Pan, Traumatic subdural effusion evolves into chronic subdural hematoma: two stages of the same inflammatory reaction? Med. Hypotheses 70 (6) (2008) 1147–1149.
[52] G. Stroobandt, P. Evrard, C. Laterre, Pathogenesis of persistent subdural effusions in infants (author's transl), Neurochirurgie 24 (1) (1978) 47–51.
[53] L. Ewing-Cobbs, M. Prasad, L. Kramer, P.T. Louis, J. Baumgartner, J.M. Fletcher, et al., Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury, Childs Nerv. Syst. 16 (1) (2000) 25–33.
[54] A.C. Duhaime, T.A. Gennarelli, L.E. Thibault, D.A. Bruce, S.S. Margulies, R. Wiser, The shaken baby syndrome. A clinical, pathological, and biomechanical study, J. Neurosurg. 66 (3) (1987) 409–415.
[55] R.A. Zimmerman, L.T. Bilaniuk, D. Bruce, L. Schut, B. Uzzell, H.I. Goldberg, Computed tomography of craniocerebral injury in the abused child, Radiology 130 (3) (1979) 687–690.
[56] P. Steinbok, Comment, Childs Nerv. Syst. 16 (2) (2000) 110.
[57] G.A. Tung, M. Kumar, R.C. Richardson, C. Jenny, W.D. Brown, Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography, Pediatrics 118 (2) (2006) 626–633.
[58] A.N. Guthkelch, Infantile subdural haematoma and its relationship to whiplash injuries, Br. Med. J. 2 (759) (1971) 430–431.
[59] A.K. Ommaya, W. Goldsmith, L. Thibault, Biomechanics and neuropathology of adult and paediatric head injury, Br. J. Neurosurg. 16 (3) (2002) 220–242.
[60] M.T. Prange, B. Coats, A.C. Duhaime, S.S. Margulies, Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants, J. Neurosurg. 99 (1) (2003) 143–150.
[61] S.C. Winter, G. Quaghebeur, P.G. Richards, Unusual cervical spine injury in a 1 year old, Injury 34 (4) (2003) 316–319.
[62] R.A. Johnston, Paediatric spinal injuries, Injury 35 (2) (2004) 105–106.
[63] J.F. Geddes, A.K. Hackshaw, G.H. Vowles, C.D. Nickols, H.L. Whitwell, Neuropathology of inflicted head injury in children. I. Patterns of brain damage, Brain 124 (Pt 7) (2001) 1290–1298.
[64] P. Shannon, L. Becker, Mechanisms of brain injury in infantile child abuse, Lancet 358 (9283) (2001) 686–687.
[65] M.N. Hadley, V.K. Sonntag, H.L. Rekate, A. Murphy, The infant whiplash-shake injury syndrome: a clinical and pathological study, Neurosurgery 24 (4) (1989) 536–540.