Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies

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Does an expanded subarachnoid space predispose to subdural bleeding? What does heterogeneity in the appearance of a subdural collection on CT or MRI imaging indicate? Spontaneous rebleeding? Minor re-injury? Major re-injury? In some specific cases, answers to these questions have important forensic implications. To conclude objectively that an infant's intracranial hemorrhage or rebleeding resulted from inflicted injury or re-injury requires an in-depth understanding of the pathogenesis of posttraumatic subdural and subarachnoid collections. The authors present two cases of indoor, accidental, pediatric, closed-head trauma that resulted in intracranial rebleeding. Both accidental cranial impacts occurred in medical settings and were independently witnessed by medical personnel. In addition, the authors summarize the relevant medical literature regarding pediatric intracranial bleeding and rebleeding.

Abusive head trauma is the leading cause of traumatic death during infancy. Subdural hematoma is a frequent finding in these young victims. There are many nontraumatic etiologies for subdural hemorrhage. On the other hand, in the absence of an adequate alternate explanation for subdural bleeding during infancy or childhood, child abuse must be considered.

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How can we *objectively* interpret the forensic significance of pediatric intracranial hemorrhage or rebleeding? We will explore this question in depth by addressing the following questions in order:

1. What are the potential explanations for enlargement of the subarachnoid space in an infant or young child?
2. Is an infant or young child with an enlarged subarachnoid space predisposed to subdural bleeding?
3. What is the differential diagnosis for subdural hematoma?
4. What is the pathophysiology of traumatic subdural hemorrhage?
5. What does serial cranial imaging reveal in young victims of head trauma?
6. What are the potential explanations for heterogeneity in the appearance of a subdural collection on CT or MR imaging?
7. Under what circumstances do subdural hemorrhages rebleed?
8. What are the expected clinical consequences of traumatic subdural bleeding and rebleeding in an infant or young child?

What Are the Potential Explanations for Enlargement of the Subarachnoid Space in an Infant or Young Child?

In the first 2 years of life, the subarachnoid spaces are relatively larger than in older children or adults (see Table 1) (Alper et al., 1999; Fessell, Frankel, &

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TABLE 1: Differential Diagnosis of an Expanded Subarachnoid Space

Benign congenital enlargement (secondary to immaturity of the arachnoid villi)
Acute subarachnoid hemorrhage
Posttraumatic communicating hydrocephalus (secondary to adhesive arachnoiditis induced by acute subarachnoid hemorrhage)
Expansion of the subarachnoid space secondary to partial obstruction of regional CSF flow by a contiguous or overlying acute subdural hematoma
Cerebral atrophy
Subdural hygroma (misinterpreted as an expanded subarachnoid space)
Chronic subdural hematoma (misinterpreted as an expanded subarachnoid space)

Wolfson, 1997; Kleinman, Zito, Davidson, & Raptopoulos, 1983; Libicher & Troger, 1992; Prassopoulos & Cavouras, 1994). This benign condition is more common in premature infants and in young infants with macrocephaly. Alvarez, Maytal, and Shinnar (1986) found a positive family history of macrocephaly in 88% of cases. Congenital expansion of the subarachnoid spaces during infancy is characterized by reversible enlargement of the subarachnoid spaces (most evident in the frontal regions), prominence of the basilar cisterns and the anterior interhemispheric fissure, and mild ventriculomegaly. Early CT studies incorrectly suggested that these extracerebral collections resided in the subdural space (Mori, Handa, Ito, & Okuno, 1980; Robertson, Chun, Orrison, & Sackett, 1979). Benign enlargement of the subarachnoid spaces probably represents a physiologic and transient form of communicating or external hydrocephalus resulting from immaturity of the arachnoid villi (Wolpert & Barnes, 1992).

Enlargement of the subarachnoid space may result from subarachnoid hemorrhage—a common finding in young victims of inflicted cranial trauma (Cohen, Kaufman, Myers, & Towbin, 1986; Dolinskas, Zimmerman, & Bilaniuk, 1978; Duhaime et al., 1992; Ewing-Cobbs et al., 2000; Reece & Sege, 2000; Wolpert & Barnes, 1992). Blood products in the subarachnoid space may cause an adhesive arachnoiditis, which impedes absorption of cerebrospinal fluid (CSF) by the arachnoid villi (Barkovich, 1995; Fitz & Harwood-Nash, 1978). Through this mechanism, isolated subarachnoid hemorrhage may predispose to posttraumatic, communicating hydrocephalus manifesting as expansion of the subarachnoid space.

More commonly in cases of child abuse, enlargement of the subarachnoid space is seen in association with contiguous or overlying acute subdural hemorrhage (Kapila, Trice, Spies, Siegel, & Gado, 1982; Mori et al., 1980; Orrison, Robertson, & Sackett, 1978; Robertson et al., 1979; Rothenberger & Brandl, 1980). The presence of extensive subdural hemorrhage overlying a large surface of the cerebral convexity may impede the flow of cerebrospinal fluid into the region underlying the hematoma (Elvidge & Jackson, 1949; Mori et al., 1980), thereby expanding the surrounding subarachnoid spaces. In some cases of inflicted pediatric head trauma with subdural hematoma, a definitive expansion of the subarachnoid spaces and/or the ventricles is noted on follow-up cranial imaging a few days after injury (Kleinman & Barnes, 1998).

Finally, an expanded subarachnoid space may also represent cerebral atrophy—a frequent and oftentimes devastating complication of inflicted pediatric head trauma. Using cranial CT imaging alone, it may be difficult or impossible to differentiate between cerebral atrophy, posttraumatic communicating hydrocephalus, and/or a chronic subdural collection. An enlarged or enlarging head circumference suggests communicating hydrocephalus or a more chronic subdural collection. Static or decreasing head circumference suggests cerebral atrophy or subdural hygroma—a space-filling lesion (Barkovich, 1995). In most cases of abusive head trauma, the acute subdural hemorrhage, with associated diffuse brain injury, precedes the development of cerebral atrophy, communicating hydrocephalus with expansion of the subarachnoid space, and/or a chronic subdural collection (Kleinman & Barnes, 1998).

Is an Infant or Young Child With an Enlarged Subarachnoid Space Predisposed to Subdural Bleeding?

The frequent coexistance of an enlarged subarachnoid space with overlying subdural collections has led some authors to suggest that an enlarged CSF space predisposes to the development of acute subdural hemorrhage. Authors have postulated that the amount of trauma needed to produce subdural hemorrhage in these infants is less than that required to produce acute subdural bleeding in infants with "normal" subarachnoid spaces (Aoki, 1994; Aoki & Masuzawa, 1984; Howard, Bell, & Uttley, 1993; Ikeda et al., 1987; Kapila et al., 1982; Papasian & Frim, 2000; Veyrac, Couture, & Baud, 1990). In addition, authors have proposed that susceptibility to subdural hemorrhage is related to racial variations in the size of the subarachnoid space and head circumference (Aoki, 1994; Howard et al., 1993; Rekate, 1985).

Does an expanded subarachnoid space predispose a child to traumatic subdural hemorrhage (e.g., as a

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complication of a minor fall)? The medical literature does not give us a definitive answer to this question. More likely, however, the opposite is true. Acute subdural hemorrhage may facilitate secondary expansion of the subarachnoid space (Kleinman & Barnes, 1998). Consider the following:

1. Benign prominence of the subarachnoid space is common in normal infants.
2. Though limited in number, long-term observations of infants with benign, congenital expansion of their subarachnoid spaces revealed no increased frequency of subdural hematomas over time (Briner & Bodensteiner, 1981; Ment, Duncan, & Geehr, 1981; Robertson et al., 1979).
3. Very likely, some severely head-injured infants with traumatic subdural hemorrhage had pre-existing and benign enlargement of their subarachnoid spaces unrelated to the development of their subdural hematoma (Kleinman & Barnes, 1998).
4. Finally, as described previously, displacement of the subarachnoid compartment by an overlying subdural collection may cause secondary expansion of the surrounding subarachnoid spaces (Elvidge & Jackson, 1949; Mori et al., 1980).

Until a prospective, comparative study concludes that normal infants with prominent subarachnoid spaces are at a statistically greater risk for subdural hemorrhage, this concept should be viewed with caution (Kleinman & Barnes, 1998). On the other hand, our first case report of subdural bleeding secondary to witnessed, minor, closed-head trauma suggests that expansion of the subarachnoid space may indeed predispose to subdural bleeding under certain abnormal clinical circumstances.

A 20-month-old boy suffered a linear skull fracture, an epidural hemorrhage, and transtentorial herniation after he fell down several concrete stairs. Despite neurosurgical intervention, CT imaging one month later revealed right hemispheric cerebral atrophy, ventriculomegaly, and an expanded subarachnoid space (see Figure 1). Three months later, he struck his forehead against a low windowsill during physical therapy. He cried immediately and was not easily consoled. He remained persistently irritable with decreased appetite over the following week. Cranial CT imaging several days later revealed an acute, right occipital, subdural hematoma in the region of his maximal cerebral atrophy (see Figure 2). He recovered gradually thereafter with resolution of his new subdural hematoma without additional neurosurgical intervention.

His first injury did not cause subdural or subarachnoid hemorrhage. Therefore, the later expansion of his subarachnoid space reflects cerebral atrophy—not posttraumatic communicating hydrocephalus or a chronic subdural collection. At the time of his second, minor, closed-head injury during physical therapy, acute subdural bleeding occurred in the region of his pre-existing cerebral atrophy and expanded subarachnoid space. His clinical signs were limited to irritability and loss of appetite. Because the second impact was relatively minor, the child did not suffer severe and acute clinical deterioration suggestive of traumatic axonal injury. Instead, his clinical signs were caused by the limited mass effect of focal subdural bleeding.

Because this child's expanded subarachnoid space was an acquired, posttraumatic condition, this case example demonstrates that an expanded subarachnoid space resulting from cerebral atrophy can pre-

FIGURE 1: This 20-Month-Old Male Suffered a Linear Skull Fracture, an Epidural Hemorrhage, and Transtentorial Herniation After He Fell Down Several Concrete Steps. Despite Neurosurgical Intervention, CT Imaging One Month Later Revealed Right Hemispheric Cerebral Atrophy, Ventriculomegaly, and an Expanded Subarachnoid Space.

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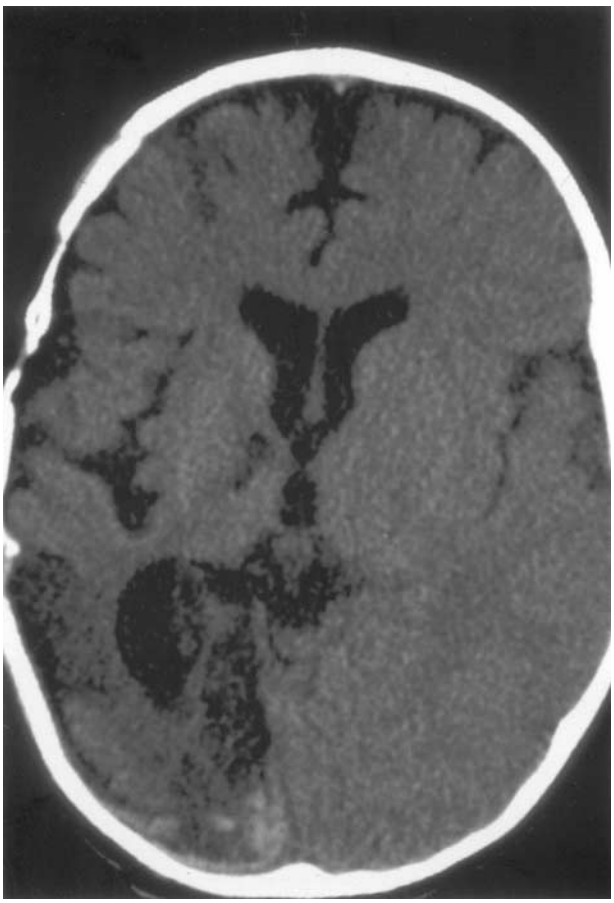

FIGURE 2: Three Months After His Initial Cranial Injury, This 20-Month-Old Male Struck His Forehead Against a Low Windowsill During Physical Therapy. He Cried Immediately and Was Not Easily Consoled. Over the Following Week, He Remained Persistently Irritable With Decreased Appetite. Cranial CT Imaging Several Days After This Witnessed, Minor Fall Revealed an Acute, Right Occipital, Subdural Hematoma in the Region of His Maximal Cerebral Atrophy.

dispose to regional subdural hemorrhage as a consequence of a minor, indoor fall. This witnessed case example should not be extrapolated to predict that benign expansion of the subarachnoid spaces during infancy predisposes to subdural bleeding.

What Is the Differential Diagnosis for Subdural Hemorrhage?

Subdural hemorrhages have been linked etiologically to accidental trauma; inflicted trauma; medical or surgical interventions; prenatal, perinatal, and pregnancy-related conditions; birth trauma; metabolic diseases; congenital malformations; genetic diseases; oncologic diseases; autoimmune disorders; clotting disorders; infectious diseases; the effects of poisons, toxins, or drugs; and other miscellaneous conditions. The vast majority of these entities can be diagnostically excluded (or confirmed) by careful history, physical examination, radiological studies, and/or laboratory testing (see Table 2).

What Is the Pathophysiology of Traumatic Subdural Hemorrhage?

The dura mater is composed of fibroblasts and large amounts of extracellular collagen. The dural border cell layer forms its innermost region. The arachnoid consists of an outer barrier cell layer and the inner arachnoid trabeculae, which bridge the subarachnoid space. An actual or potential subdural space does not exist in humans. In most instances, when a tissue space is created traumatically in this general area of the meninges, it represents a cleaving of the innermost dural cell border layer (Haines, 1991). Traumatic, acute, subdural hemorrhage is a blood clot without a membrane within the cleaved dural border cell layer (also known as an intradural hematoma). Given its common usage, in this article, we use the term *subdural* to refer to these posttraumatic, intradural collections.

Acute, traumatic subdural hemorrhage may result from either a contact or noncontact mechanism of injury. When subdural hemorrhage is limited to the impact site or the region underlying a skull fracture, it likely represents an isolated contact injury (i.e., resulting primarily from skull deformation and not from cranial acceleration). Because linear skull fractures actually begin in a region of cranial outbending and not necessarily at the point of impact and inbending (Arnholz, Hymel, Hay, & Jenny, 1998; Gurdjian, Webster, & Lissner, 1950), a contact subdural hemorrhage can occur at a location other than the impact site as a complication of a linear skull fracture.

Occasionally, intracranial hemorrhage resulting from contact forces alone may cause coma or death if sufficiently large to cause brain shifts, herniation, or brainstem compression (Gennarelli, 1984). If cranial motion is restricted but impact occurs with sufficient force and energy over a very large cranial surface area (e.g., crush injury), large and/or bilateral underlying subdural hematomas can occur as a massive contact injury unrelated to cranial acceleration (Duhaime, Eppley, Margulies, Heher, & Bartlett, 1995; Hymel, Bandak, Partington, & Winston, 1998). On the other hand, a small, focal subdural collection can disappear rapidly on neuroimaging (Duhaime, Christian, Armonda, Hunter, & Hertle, 1996).

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TABLE 2: Differential Diagnosis for Subdural Hemorrhage

	Differentiate by				
Condition	*History*	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	*Comments*
Trauma					
Nonaccidental trauma (Deb et al., 2000; Jayawant et al., 1998)	Yes/No	Yes/No	Yes/No	No	History may be unreliable. Physical exam and radiologic studies often yield information leading to diagnosis. Blunt and penetrating trauma can cause SDH.
Accidental trauma (Wilkins, 1997)	Yes	Yes/No	Yes/No	No	Validate history. When history matches injury, accidental trauma is more likely.
Traumatic aneurysm of the middle meningeal artery (Aoki, Sakai, & Kaneko, 1992)	Yes	No	Yes/No	No	Seven reported cases in adults. Can cause delayed SDH after severe injury.
"Headbanging" to rock music (Mackenzie, 1991)	Yes	No	No	No	Two adults who violently shook their heads to the beat of rock music developed SDH. One also had a traumatic dissection of the carotid artery.
"Breakdancing" (McNeil, Spruill, Langley, Shuping, & Leonard, 1987)	Yes	Yes/No	No	No	Breakdancing can involve repetitive trauma to the head. One case is reported in the literature of SDH caused by breakdancing.
Roller-coaster rides (Kettaneh, Biousse, & Bousser, 2000)	Yes	No	No	No	Three cases of SDH are reported in the French literature after roller-coaster rides.
Medical or surgical interventions					
Spinal tap (Hart, Bone, & Hadley, 1988)	Yes	No	No	No	SDH has been reported as a rare complication of a spinal tap in an adult. SDH symptoms developed after the tap for an unrelated condition.
Spinal anesthesia (Ortiz et al., 1991)	Yes	No	No	No	Intracranial SDH have rarely been reported as complications of spinal anesthesia. The mechanism is debated.
Epidural anesthesia (Diemunsch et al., 1998)	Yes	No	No	No	Few reports are noted.
Lumbar myelography (Suess, Stendel, Baur, Schilling, & Brock, 2000)	Yes	No	No	No	In adults, 16 cases of intracerebral SDH have been reported after lumbar myelograms.
Ventriculoperitoneal or ventriculoarterial shunt (Badiane et al., 1992; Sharma, Mahapatra, Pawar, Sousa, & Athale, 1999)	Yes	Yes	Yes	No	SDH can be a long-term complication of shunts for hydrocephalus.
Craniotomy (Wyllie, Comair, Kotagal, Raja, & Ruggieri, 1996)	Yes	Yes	Yes	No	Disruption of vessels during surgery can cause subdural bleeding.
Prenatal, perinatal, and pregnancy-related conditions					
Intrauterine trauma (Gunn & Becroft, 1984; Stephens, Richardson, & Lewis, 1997)	Yes/No	Yes/No	No	No	Usually occurs with skull fracture. History may not be reliable, particularly if trauma is the result of domestic violence.
Idiopathic intrauterine SDH (Green, Wilson, Romaniuk, May, & Welch, 1999)	Yes	No	Yes	No	In some cases, the cause of intrauterine SDH cannot be identified.
Intrauterine isoimmune thrombocytopenic purpura (Zalneraitis, Young, & Krishnamoorthy, 1979)	Yes	No	No	Yes	Rare condition where maternal antibodies destroy fetal platelets.
Pre-eclampsia (Giannina, Smith, Belfort, & Moise, 1997)	Yes	Yes	No	Yes	SDH is rarely reported in pre-eclamptic, pregnant women.

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TABLE 2 (continued)

Condition	*Differentiate by*				*Comments*
	History	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	
Postnatal cerebral infarction (Steinbok, Haw, Cochrane, & Kestle, 1995)	No	No	Yes	No	Cerebral infarction associated with SDH has been reported shortly after birth. The etiology of this condition is unknown.
Birth trauma					
Forceps delivery (Hankins, Leicht, Van Hook, & Uckan, 1999)	Yes	Yes/No	No	No	Occurs in less that 0.01% of deliveries. Scalp trauma is obvious initially.
Vacuum extraction (Hanigan, Morgan, Stahlberg, & Hiller, 1990)	Yes	Yes/No	No	No	SDH around the tentorium has been reported as a complication.
Other birth trauma (Hayashi, Hashimoto, Fukuda, Ohshima, & Moritaka, 1987)	Yes	No	No	No	Births other than breech births and suction deliveries can cause SDH. It is more likely to occur in infants of primiparous mothers. Symptoms develop shortly after birth.
Metabolic diseases					
Glutaric aciduria type I (Baric et al., 1998)	Yes/No	Yes	Yes	Yes	Can present with SDH. Often have large heads and dystonia. Persistent acidosis occurs. Urine organic acids abnormal. Degeneration of putamen and caudate seen on CT.
Galactosemia (Sychlowy and Pyda 1971)	Yes	No	No	Yes	One case is reported in the Polish literature.
Pyruvate carboxylase deficiency (Ahmad et al., 1999)	No	No	No	Yes	An inborn error of carbohydrate metabolism presenting with lactic acidemia and acidosis.
Congenital malformations					
Intracranial arteriovenous malformations (Oikawa, Aoki, & Sakai, 1993)	No	No	Yes	No	Not common in children under 6 years.
Cerebral aneurysm (O'Leary & Sweeny, 1986)	No	No	Yes	No	Ruptured aneurysm usually causes subarachnoid hemorrhage.
Arachnoid cyst (Romero, Rovira, Ibarra, Piqueras, & Rovira, 1989)	No	No	Yes	No	Rupture of vessels surrounding arachnoid cyst can cause SDH.
Spontaneous rupture of a cerebral artery (Koc, Pasaoglu, Kurtsoy, Oktem, & Kavuncu, 1997; Tokoro, Nakajima, & Yamataki, 1988)	No	No	No	No	Rare condition reported only in adults. Deformed artery is often found at autopsy or at surgery.
Schizencephaly or porencephaly (Osaka, Shirataki, Matsumoto, Yokoyama, & Ogino, 1977)	No	No	Yes	No	Subdural effusions of CSF can be caused by direct connection of the dura to the lateral ventricle through a brain malformation.
Genetic diseases					
Osteogenesis imperfecta (Cole & Lam, 1996)	Yes/No	Yes	Yes	Yes	Familial, but can occur as a spontaneous mutation. Physical characteristics may include poor growth, poor teeth, blue sclera, and joint laxity. Bones usually appear demineralized on X-ray. Testing of collagen production in fibroblasts is positive in 85% of cases.
Sickle-cell anemia (Falter, Sutton, & Robinson, 1973)	Yes	No	No	Yes	The mechanism of SDH in patients with sickle-cell anemia is unknown. The SDH is frequently fatal.
Alagille syndrome (Woolfenden et al., 1999)	Yes	Yes	Yes	Yes	Children with Alagille syndrome can develop vasculitis similar to Moyamoya disease. They are also more likely to develop other types of serious intracranial hemorrhages such as epidural hematomas, presumably secondary to the thinness of their skulls.

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Ehlers-Danlos syndrome (Ortiz Remacha, Candia, & Conde, 2000)	Yes	Yes	No	Yes	SDH can be the presenting manifestation of Ehlers-Danlos syndrome.
Autosomal dominant polycystic kidney disease (Wijdicks, Torres, & Schievink, 2000)	Yes/No	No	Yes	Yes	Chronic SDH was noted in 5 patients. ADPKD is known to be associated with vascular anomalies of the CNS.
Menkes' kinky hair syndrome (Seay et al., 1979)	Yes	Yes	Yes	Yes	SDH is associated with brain atrophy.
Oncologic diseases					
Meningeal carcinomatosis (McKenzie, Rengachary, McGregor, Dixon, & Suskind, 1990)	Yes	Yes/No	Yes	Yes/No	Metastases of neoplasms to the leptomeninges can cause SDH. This has been reported with many types of cancer, including sarcomas, lung cancers, prostate cancers, breast cancers, and others.
Leukemia (Mashiyama et al., 2000)	Yes/No	Yes/No	No	Yes	SDH has been reported as the presenting symptom of both acute monocytic leukemia in a child and chronic lymphocytic leukemia in adults.
Solid CNS tumors (Ozhan, Tali, Isik, Saygili, & Baykaner, 1999; Timothy, Lafuente, Chakrabarty, Saxena, & Marks, 1999)	Yes/No	Yes/No	Yes	Yes/No	Solid tumors can present as SDH or be complicated by SDH, including fibrous histiocytoma, meningiomas, and others.
Primary mucosa-associated lymphoma in the dura (Kambham, Chang, & Matsushima, 1998)	No	No	No	Yes	Diagnosis is made by histology of tumor cells.
Mass lesions in the subdural space (Cinalli et al., 1997)	No	No	Yes	Yes	Two cases of mass lesions in the subdural space (liposarcoma and fibrohistiocytic sarcoma) are reported, where a subdural effusion around the tumor was misdiagnosed as a chronic SDH.
Autoimmune disorders					
Lupus erythematosus (Bovim, Jorstad, & Schrader, 1990)	Yes	Yes	No	Yes	Spontaneous SDH has been described in adults with lupus, not necessarily related to antiplatelet antibodies. Other lupus patients with lupus anticoagulant factors are also reported to have SDH.
Clotting disorders					
Anticoagulant therapies (Massicotte, Marzinotto, Vegh, Adams, & Andrew, 1995; Snyder, Sridharan, & Pagnanelli, 1997)	Yes	Yes/No	No	Yes	Warfarin and other anticoagulants can lead to abnormal bleeding from minor injuries. SDH has been reported in anticoagulated patients after roller-coaster rides (adults).
Ginko biloba ingestions (Rowin & Lewis, 1996)	Yes	Yes/No	No	Yes/No	One case of an adult developing a spontaneous SDH after chronic ingestion. The herb has platelet-inhibiting properties.
Coagulopathy related to cirrhosis of the liver (Furui, Yamada, & Iwata, 1989)	Yes	Yes	No	Yes	Liver disease can impair clotting.
Hemophilia A and B (de Tezanos Pinto, Fernandez, & Perez Bianco, 1992; Lutschg & Vassella, 1981; Ohga et al., 1988)	Yes/No	Yes/No	No	Yes	In the absence of surgical procedures, most hemophilia patients do not develop bleeding problems during the neonatal period. SDH is usually related to minor trauma, rather than spontaneous. SDH is rare in these patients.
Factor V deficiency (Salooja, Martin, Khair, Liesner, & Hann, 2000)	Yes/No	Yes/No	No	Yes	SDH has presented at birth in an infant with severe Factor V deficiency (<1%).
Factor XII deficiency (Nicholls, Chan, Koo, Kwong, & Tsoi, 1993)	Yes/No	Yes/No	No	Yes	Was reported in an infant.
Idiopathic or drug-induced thrombocytopenic purpura (Kolluri, Reddy, Reddy, Naidu, & Kumari, 1986)	Yes/No	No	No	Yes	Platelet deficiency creates coagulopathy.
Hemorrhagic disease of the newborn (vitamin K deficiency) (Behrmann, Chan, & Finer, 1985)	Yes	No	No	Yes	Occurs in infants not receiving vitamin K injections at birth.

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TABLE 2 (continued)

Condition	*Differentiate by*				*Comments*
	History	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	
Diffuse intravascular coagulation (Furui et al., 1983; Hymel et al., 1997; Stein, Young, Taluchi, Greenbaum, & Ross, 1992)	Yes	Yes	No	Yes	DIC from cancer, infection, or trauma can be associated with SDH.
Acquired inhibitors of plasma clotting factors (Ryan, Arkel, Walters, Frimmer, & Desposito, 1986)	Yes/No	No	No	Yes	Thought to be a postviral phenomenon. Can cause a severe coagulopathy.
Hermansky-Pudlak syndrome (Russell-Eggitt, Thompson, Khair, Liesner, & Hann, 2000)	No	Yes	No	Yes	Associated with a bleeding diathesis caused by impaired platelet function. Affected children usually have albinism and nystagmus.
Alpha 1-antitrypsin deficiency (Israels & Gilfix, 1999)	No	No	No	Yes	Presents as "late hemorrhagic disease of the newborn" with vitamin K deficiency.
Infectious diseases					
Hemophilus influenzae meningitis (Ogilvy, Chapman, & McGrail, 1992)	Yes	Yes	Yes	Yes	Meningitis can cause subdural effusions and empyemas that can be confused with old hematomas.
Streptococcus pneumoniae meningitis (Ogilvy et al., 1992)	Yes	Yes	Yes	Yes	Meningitis can cause subdural effusions and empyemas that can be confused with old hematomas.
Other bacterial meningitis (Syrogiannopoulos, Nelson, & McCracken, 1986)	Yes	Yes	Yes	Yes	Subdural effusions are caused less commonly by other organisms such as *Neisseria*, Group B *Streptococcus*, *Escherichia coli, Listeria monocytogenes*, and others.
Kawasaki disease (Aoki, 1988)	Yes	Yes	No	Yes/No	Subdural *effusions* (rather than hematomas) can occur with Kawasaki disease secondary to leptomeningeal vasculitis.
Endocarditis leading to septic emboli of a cranial artery causing aneurismal rupture of the vessel (Bandoh, Sugimura, Hosaka, & Takagi, 1987)	Yes	Yes	No	Yes	Rare, not reported in children.
Chronic otitis media (Gower & McGuirt, 1983)	Yes	Yes	No	No	Subdural effusions and empyemas (not hematomas) have been reported with chronic middle ear disease.
Intracranial extension of sinus infections (Dolan & Chowdhury, 1995)	Yes/No	No	Yes	No	Subdural empyemas can form from intracranial extension of sinusitis. (More common in the pre-antibiotic era.)
Malaria (Omanga, Shako, Ntihinyurwa, Mbuyu, & Beltchika, 1979)	Yes	No	No	Yes	Malaria can cause subdural effusions (not hematomas).
Herpes simplex encephalitis (Saito et al., 1983)	Yes	Yes/No	No	Yes	One case of chronic SDH is reported after HSV encephalitis in the Japanese literature.
Congenital toxoplasmosis	Yes	Yes	Yes/No	Yes	Reported in the Polish literature. Possibly related to cerebral atrophy.
Poisoning, toxins, or drug effects					
Lead poisoning (Sensirivatana, Supachadhiwong, Phancharoen, & Mitrakul, 1983)	Yes	No	Yes	Yes	One case of subdural effusions with high protein content after lead poisoning has been reported. The etiology of the effusions was unknown.
Cocaine (Keller & Chappell, 1997)	Yes	No	No	Yes/No	Cocaine use has been associated with spontaneous SDH.
Tamoxifen (Missori et al., 1998)	Yes	Yes/No	No	No	Chronic SDH has been reported in patients taking tamoxifen. It is unknown if this is a primary effect or an occurrence related to other aspects of the patients' disease.

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Other					
Hemodialysis of patients with kidney disease (Bechar, Lakke, van der Hem, Beks, & Penning, 1972)	Yes	Yes	No	No	Spontaneous SDH in dialysis patients can be related to liver disease or anticoagulant therapy, but sometimes the cause is not apparent.
Open heart surgery (Yokote et al., 1985)	Yes	Yes	No	No	Rarely reported complication; etiology is unclear, but may be related to anticoagulation.
Moyamoya disease (Takeuchi, Ichikawa, Koike, Tanaka, & Arai, 1992)	No	No	Yes	No	Moyamoya is a rare vascular disease of the central nervous system affecting the distal arteries. Moyamoya means "hazy puff of smoke" in Japanese, referring to the angiographic appearance of the abnormal vessels developing at the base of the brain.
Bone marrow transplant (Colosimo et al., 2000)	Yes	Yes	No	Yes	In one series, 2.6% of patients receiving allogeneic or autologous bone marrow transplants developed SDH. The therapeutic maneuver involves many factors leading to central nervous system bleeding, including intrathecal methotrexate therapy, low platelet counts, and coagulopathies.
Hyperostosis frontalis interna (Ishiguro, Nakagaw, Yamamura, & Kurokawa, 1997)	Yes	Yes	Yes	No	A rare condition causing frontal bossing.
Wegener's granulomatosis (Yokote, Terada, Nakai, & Itakura, 1997)	Yes	No	Yes	Yes	Subdural effusions containing blood and pus reported in a few cases.
Hemorrhagic shock and encephalopathy	Yes/No	Yes	No	Yes	A rare condition of unknown etiology, usually fatal, causing severe hyperthermia, coagulaopathy, and shock. Possibly is a postviral condition. Although subdural hematomas have not been reported in the literature resulting from this condition, the author (CJ) has seen one case.
Complication of parenteral nutrition (Rushforth, Green, Levene, & Puntis, 1991)	Yes	No	No	Yes	One case is reported in the literature of a preterm infant developing subdural "fat effusions" as a result of parenteral nutrition through a central venous catheter.
Spontaneous intracranial hypotension (Nakajima, Sakai, Aoki, & Takakura, 1996)	Yes	No	Yes	No	Bilateral chronic subdural hematomas have been linked to cerebral hypotension caused by a CSF leak; sometimes associated with collagen abnormalities; has not been reported in children.

NOTE: Some of the conditions listed cause subdural effusions or empyemas, which can be confused with subdural hemorrhage.

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Traumatic, acute, subdural hemorrhage can also occur as a noncontact injury (i.e., resulting primarily from cranial acceleration and not from skull deformation). Whole head, rotational, cranial acceleration induced by impact or impulsively (i.e., without impact) can rupture parasagittal bridging veins. Noncontact, subdural hematomas in infants and young children tend to be large, interhemispheric, and/or bilateral. Noncontact subdural hematoma is a frequent finding in young victims of inflicted head trauma (Alexander, Schor, & Smith, 1986; Aoki & Masuzawa, 1986; Bruce & Zimmerman, 1989; Cohen et al., 1986; Duhaime et al., 1992; Ewing-Cobbs et al., 1998, 2000; Guthkelch, 1971; Hymel, Rumack, Hay, Strain, & Jenny, 1997; Jayawant et al., 1998; Ludwig & Warman, 1984; Merten & Carpenter, 1990; Merten, Osborne, Radkowski, & Leonidas, 1984; Reece & Sege, 2000; Sato et al., 1989; Tsai, Zee, Apthorp, & Dixon, 1980).

It is widely presumed that chronic subdural hematoma develops directly from acute subdural hematoma. Radiologically, subdurals evolve from high density in the acute phase, to isodensity with brain parenchyma in the subacute phase, and finally to low density similar to cerebrospinal fluid in the chronic phase. For this reason, a subdural collection that was observed to have evolved from an acute subdural hematoma is appropriately referred to as a *chronic subdural hematoma* (Kleinman & Barnes, 1998). However, at least in adults, only a few cases of chronic subdural hematoma evolve directly from acute subdural hematoma (Croce et al., 1994; Dolinskas, Zimmerman, Bilaniuk, & Gennarelli, 1979; Lee et al., 1996; Mathew et al., 1993). Experimental models largely fail to produce an enlarging, chronic, subdural hematoma from an acute solid clot (Goodell & Mealey, 1963; Watanabe, Shimada, & Ishii, 1963). Instead, the vast majority of chronic, posttraumatic, subdural collections evolve from subdural hygroma (Lee, Bae, Doh, Bae, & Yun, 1998).

A posttraumatic, subdural hygroma represents an accumulation of cerebrospinal fluid in the subdural space without a membrane, frequently with modified composition (Lee, 1998; Lee, Bae, Park, & Yun, 1994). An arachnoid tear has been widely proposed as a potential pathogenic mechanism (Borzone et al., 1983; Fobben et al., 1989; Gade et al., 1990; Miller, 1987; Murata, 1993). More likely, subdural hygroma develops by effusion upon resolution of acute subdural hemorrhage (Gutierrez, McLone, & Raimondi, 1979; Wetterling et al., 1988).

Acute subdural hematoma often resolves rapidly. Such rapid resolution likely reflects the high levels of tissue thromboplastin in brain tissue and cerebrospinal fluid (Astrup, 1965). As it resolves, a posttraumatic space may persist within the cleaved intradural membrane, particularly in the presence of decreased intracranial pressure. In the presence of sufficient, persistent, intradural space, subdural hygroma develops by effusion. Persistence of the traumatic, intradural defect may be facilitated by prolonged spinal fluid drainage, excessive therapy with osmotic agents, dehydration, and/or evolving cerebral atrophy. Increased arachnoid and vascular permeability resulting from trauma may also facilitate the effusion (Gutierrez et al., 1979; Wetterling et al., 1988). Thus, subdural hygroma begins not as a mass lesion but as a delayed, space-filling lesion. For this reason, a majority of subdural hygromas are aymptomatic (Lee, 1998; Lee et al., 1994, 1998).

Typically, subdural hygroma appears as an extra-axial collection of similar density to cerebrospinal fluid and must be differentiated radiologically from chronic subdural hematoma and/or cerebral atrophy with a widened subarachnoid space (Kleinman & Barnes, 1998) (see Table 1). The early fate of a posttraumatic subdural hygroma is either resolution or continued expansion. Early expansion is related to continued effusion and brain shrinkage. Resolution of a subdural hygroma is related to fluid absorption and brain re-expansion. When the brain shrinks and/or effusion exceeds absorption, a subdural hygroma will enlarge (Lee, 1998; Lee et al., 1996, 1998).

If the hygroma persists, a neomembrane will form eventually from proliferating dural border cells. Once a neomembrane has formed, neovascularization occurs. Spontaneous microhemorrhages from this neomembrane occur frequently into the expanding subdural effusion. For this reason, the content of subdural hygroma is frequently mixed with blood or is xanthochromic. Microhemorrhages may occur with little or no trauma (Kleinman & Barnes, 1998; Lee et al., 1998). Ultimately, repeated hemorrhages can transform an enlarging subdural hygroma into an expanding chronic subdural hematoma—a collection of liquefied blood within a membrane (Fujisawa et al. 1991, 1995; Ito et al., 1976, 1987; Markwalder, 1989; Nakamura & Tsubokawa, 1989; Yamashima, Yamamoto, & Friede, 1983).

Chronic subdural hematoma fluid does not coagulate. Even with the addition of thrombin, a fibrin clot often does not form. All laboratory measures of coagulation indicate a dysfunction of the coagulation system in chronic subdural hematomas (Kawakami, Chikama, Tamiya, & Shimamura, 1989). A vicious cycle of rebleeding and hyperfibrinolysis is believed to ultimately result in hematoma enlargement (Toyosawa et al., 1997).

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TABLE 3: Differentiation Between Subdural Hygroma and Chronic Subdural Hematoma

Feature	*Subdural Hygroma*	*Chronic Subdural Hematoma*
Appearance on CT/MRI	Similar to CSF	Similar to CSF
Mean interval from injury to diagnosis	1 to 3 weeks	Over 3 weeks
Head circumference	Static or decreasing	Enlarged or increasing
Fluid collection	Clear, xanthochromic, or blood-tinged	Dark brown "crank case oil"
Total protein, albumin and hemoglobin content	Substantially lower than blood	Similar to blood
Neomembrane	Often lacking	Usually present
Mass lesion	Rarely	Potentially

The distinction between subdural hygroma and chronic subdural hematoma may be difficult. Both subdural collections demonstrate density or intensity similar to cerebrospinal fluid on cranial CT or MR imaging. The mean interval from injury to diagnosis is 1 to 3 weeks for subdural hygroma and more than 3 weeks for chronic subdural hematoma (Lee et al., 1998). The fluid collection within a subdural hygroma is clear, xanthochromic, or blood-tinged. Subdural fluid within a chronic subdural hematoma may have a dark brown "crank case oil" appearance (Lee, 1998). The total protein, albumin, and hemoglobin contents in subdural hygroma are substantially lower than in chronic subdural hematoma or in blood (Weir, 1971). Neomembrane is usually present with chronic subdural hematoma, but subdural hygroma often lacks a membrane. In adults, subdural fluid within a chronic subdural hematoma may act as a mass lesion, whereas subdural hygroma rarely acts as a mass lesion and often disappears (Lee, 1998; Lee et al., 1994, 1998; Lee et al., 1996) (see Table 3).

During infancy, the differentiation between subdural hygroma and chronic subdural hematoma may be even more difficult or impossible. In the abused, head-injured infant, large, bilateral, subdural collections may lead to craniocerebral disproportion with an enlarging head, virtual disappearance of brain white matter, and massive subdural collections of CSF density (Duhaime & Sutton, 1992). With such intracranial findings, it is often difficult to determine whether a chronic subdural collection reflects a delayed space-filling lesion, a mass lesion, or both of these sequentially.

An infant's brain is very deformable. The skull is both unfused and elastic. Very likely, these characteristics facilitate the formation of posttraumatic subdural hygroma as a space-filling lesion. Thereafter, the extent and severity of the child's posttraumatic cerebral atrophy will significantly influence his/her propensity for development of an enlarged subarachnoid space and/or a chronic subdural hematoma. As the head-injured infant with cerebral atrophy grows older, the cranial sutures will fuse, the fontanels will close, and both skull elasticity and brain deformity will decrease. Under these circumstances, the child's chronic subdural hematoma may begin to act with mass effect, and subdural rebleeding from any etiology may be less well-tolerated (see Figure 3).

What Does Serial Cranial Imaging Reveal in Young Victims of Head Trauma?

We reviewed the available medical literature regarding the results of serial cranial CT imaging of head-injured children (Dias, Backstrom, Falk, & Li, 1998; Feldman, Brewer, & Shaw, 1995; Giangiacomo, Khan, Levine, & Thompson, 1988; Sinal & Ball, 1987; Stein, Spettell, Young, & Ross, 1993; Tabori et al., 2000). Our findings are summarized in Table 4. These reports were published between 1987 and 2000, representing a potentially wide range of CT imaging technology, technique and/or sensitivity for detection of subtle intracranial hemorrhage. Serial MR cranial imaging in young victims of head trauma has not been described. The specific timing of subsequent CT imaging varied widely across studies, dictated by clinical necessity rather than prospective research design. In addition, cases of potential delayed hemorrhage or rebleeding were not always described or interpreted in light of the associated clinical findings, making pathophysiologic extrapolations difficult. For example, initial intracranial hemorrhage may be self-limited by increased intracranial pressure.

Despite their limitations, these reports lead us to conclude the following:

1. The first appearance of intracranial hemorrhage in young, head-injured children can be delayed.
2. Serial CT cranial imaging frequently reveals progressive intracranial hemorrhage in young children hospitalized with head injury.
3. Some head-injured children with delayed and/or progressive intracranial hemorrhage will require neurosurgical intervention.

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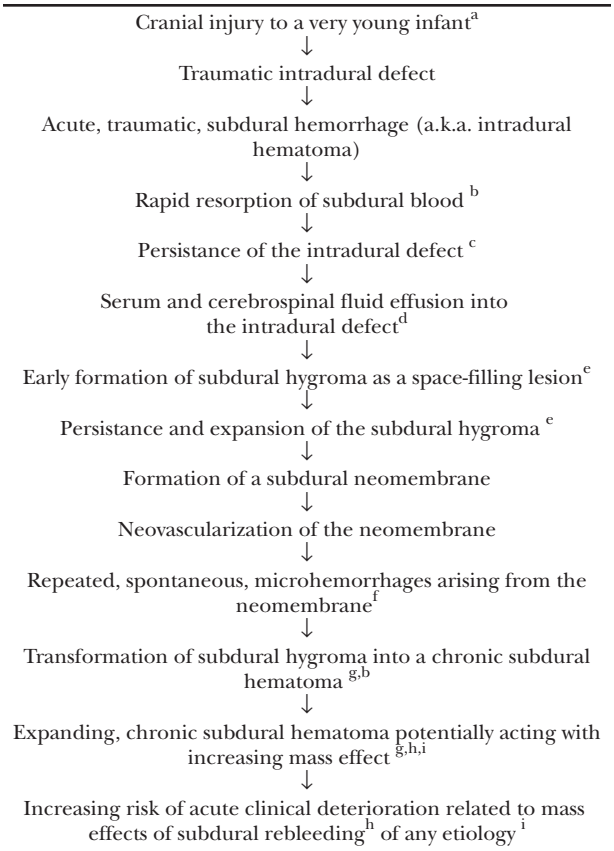

FIGURE 3: Pathophysiological Pathway Explaining Changing Clinical Manifestations of a Posttraumatic Subdural Collection in the Same Infant or Young Child Over Time

a. The acute clinical manifestations at the time of an acute, posttraumatic, subdural hemorrhage likely reflect the primary injury mechanism(s) and severity.
b. Facilitated by high levels of tissue thromboplastin in brain and cerebrospinal fluid.
c. Facilitated by decreasing intracranial pressure.
d. Facilitated by increased arachnoid and vascular permeability resulting from trauma.
e. Facilitated by evolving cerebral atrophy, open fontanels, and a young skull that is both unfused and elastic.
f. Spontaneously or as a consequence of minor closed head trauma.
g. Facilitated by the combination of rebleeding and hyperfibrinolysis.
h. Facilitated by skull maturation leading to decreasing elasticity, closed fontanels, and fused sutures.
i. Including spontaneous subdural rebleeding, rebleeding due to minor trauma, or major re-injury.

4. Spontaneous subdural rebleeding can and does occur in young children hospitalized with a head injury.
5. However, only a single case has been reported in the medical literature of a hospitalized, head-injured infant with spontaneous subdural rebleeding, documented on serial CT imaging and requiring neurosurgical intervention (Dias et al., 1998). Unfortunately, little or no details regarding this case are provided.

What Are the Expected Clinical Consequences of Subdural Bleeding or Rebleeding in an Infant or Young Child?

Acute subdural bleeding or rebleeding in an infant or young child has been linked directly or indirectly to a wide variety of acute and chronic clinical presentations (Alexander, Crabbe, Sato, Smith, & Bennett, 1990; Ewing-Cobbs et al., 1998; Gilles & Nelson, 1998; Jenny, Hymel, Ritzen, Reinert, & Hay, 1999; Johnson, Boal, & Baule, 1995; Willman, Bank, Senac, & Chadwick, 1997; Reece & Sege, 2000). Microhemorrhages originating from the neomembrane of a subdural hygroma may produce little or no clinical manifestations. On the other extreme, cranial acceleration injury mechanisms sufficient to cause subdural bleeding or rebleeding may precipitate traumatic axonal injuries at the craniocervical junction (Geddes et al., 2001; Geddes, Whitwell, & Graham, 2000) or more diffusely (Vowles, Scholtz, & Cameron, 1987). These primary brain injuries may manifest acutely as loss of consciousness, apnea, hypotension, prolonged traumatic coma, and/or death (Johnson et al., 1995; Willman et al., 1997).

Based on complex considerations, we may be able to predict the expected, acute, clinical consequences of subdural bleeding or rebleeding in young victims of head trauma (see Table 5). Adverse, acute clinical deterioration is *less* likely at the time of subdural bleeding or rebleeding if and when the subdural bleeding or rebleeding

- is microscopic and spontaneous (rather than induced by re-injury);
- occurs in a younger child with an elastic skull, unfused sutures, open fontanels, and a deformable brain;
- occurs into a space-filling subdural hygroma (as compared to chronic subdural hematoma acting with mass effect); and/or
- is induced by re-injury that does not precipitate clinically significant, primary brain injury (e.g., craniocervical or diffuse traumatic axonal injury).

On the other hand, adverse, acute clinical deterioration is *more* likely at the time of subdural bleeding or rebleeding if and when the subdural bleeding or rebleeding

- is induced by re-injury (rather than spontaneous and microscopic);
- occurs in an older child with decreasing skull elasticity, closed fontanels, fused sutures, and decreasing brain deformability;
- occurs into a chronic subdural hematoma acting with mass effect (as compared to a space-filling subdural hygroma); or

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TABLE 4: Results of Serial Cranial CT Imaging in Young Victims of Head Trauma

Source	*Study Subjects*	*Changes on Follow-up Imaging Studies*
Stein, Spettell, Young, and Ross (1993)	351 children and adolescents with serious head injury	No spontaneous rebleeding; delayed or progressive intracranial lesions in 145 cases (41%); delayed parenchymal injury correlated with severity of initial injury, initial subarachnoid or intraventricular hemorrhage, and abnormal coagulation on admission.
Giangiacomo, Khan, Levine, and Thompson (1988)	5 whiplash shaken infants	All had abnormal imaging studies. One infant had bifrontal, hypodense subdural effusions. The child's CT at 13 days postadmission showed new, acute subdural hemorrhage.
Sinal and Ball (1987)	12 victims of abusive head trauma	3 developed new subdural hemorrhage seen 4 to 10 days after admission.
Feldman, Brewer, and Shaw (1995)	34 children with abusive head trauma	3 developed new, delayed abnormalities, including enlarging subdural effusion, evolving infarct, and delayed subdural and subarachnoid hemorrhage.
Tabori et al. (2000)	173 children with head trauma, Glascow Coma Score <12	47 (27%) of the repeat CT scans showed new lesions, including 6 new intracranial hemorrhages. 2 required surgical intervention.
Dias, Backstrom, Falk, and Li (1998)	21 infants with abusive head trauma	All initially had acute subdural hematoma. Four evolved to chronic subdural hematomas. One re-bled at 7 days and required surgical intervention.

TABLE 5: Expected Clinical Consequences of Subdural Bleeding or Rebleeding in Young Victims of Head Trauma

At the Time of Acute Subdural Bleeding or Rebleeding, Adverse Clinical Deterioration Is	
Less Likely	*More Likely*
If and When the Subdural Bleeding or Rebleeding	
. . . is microscopic and spontaneous	. . . induced by re-injury
. . . occurs in a younger child with increased skull elasticity, unfused sutures, open fontanels, and a very deformable brain	. . . occurs in an older child with decreasing skull elasticity, fused sutures, closed fontanels, and decreasing brain deformability
. . . occurs into a space-filling subdural hygroma	. . . occurs into a chronic subdural hematoma acting with mass effect
. . . is induced by re-injury that does not precipitate clinically significant primary brain injury	. . . is induced by re-injury that precipitates clinically significant primary brain injury

- is induced by re-injury that precipitates clinically significant, primary brain injury (e.g., craniocervical or diffuse traumatic axonal injury).

What Are the Potential Explanations for Heterogeneity in the Appearance of a Subdural Collection on CT or MR Imaging?

Pediatric victims of inflicted head trauma frequently suffer repetitive intracranial injuries (Alexander et al., 1990; Ewing-Cobbs et al., 2000; Jenny et al., 1999) (see Table 6). Therefore, discovery of intracranial hemorrhages of multiple ages during infancy should raise concern about the possibility of repetitive, inflicted cranial trauma.

In the majority of cases, an acute subdural hemorrhage is of uniform high density on CT imaging. However, exceptions do occur. Dias and colleagues described the appearance of a hypodense subdural collection 20 hours after head injury in an infant whose initial CT scan was considered normal (Dias et al., 1998). A hyperacute subdural hematoma may reveal low attenuation similar to the density of CSF or brain (Barnes & Robson, 2000). Leakage of cerebrospinal fluid into the subdural space may result from a traumatic disruption or tear of the arachnoid membrane (Borzone et al., 1983; Fobben et al., 1989; Gade et al., 1990; Miller, 1987; Murata, 1993). Rarely, this occurs as an isolated traumatic injury with little or no associated hemorrhage (Kleinman & Barnes, 1998). This is called an acute subdural hygroma—a lesion that may be misinterpreted radiologically as an older, chronic subdural hygroma or hematoma.

A hyperacute subdural may also reveal high- and low-density components that are arranged concentri-

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TABLE 6: Potential Explanations for Heterogeneity in the Appearance of a Subdural Collection on CT or MR Imaging

Hyperacute subdural hematoma
Combined acute subdural hematoma and acute subdural hygroma
Heterogeneity within a single, evolving, subdural hemorrhage
Subdural rebleeding induced by acquired coagulopathy (e.g., DIC)
Spontaneous subdural rebleeding within a chronic subdural collection
Subdural rebleeding within a chronic subdural collection induced by minor closed head trauma
Repetitive and severe head trauma inducing multiple subdural hemorrhages

cally or intermixed. These mixed-density, hyperacute collections are frequently misinterpreted as chronic subdural hemorrhage, or chronic subdural hematoma with rehemorrhage (Sargent, Kennedy, & Kaplan, 1996). The low density component of a heterogenous, hyperacute subdural hematoma may represent active hemorrhaging, unclotted hemorrhage (particularly in a patient with low hematocrit or a clotting abnormality), or serum extrusion (low density) associated with early clot retraction (high density) (Kleinman & Barnes, 1998). Finally, an evolving subdural hemorrhage may demonstrate heterogeneity from one region to another over time within a single subdural collection.

Clinicians and radiologists should exercise considerable caution before estimating the age of a subdural collection or concluding with certainty that heterogeneity within a subdural collection represents rehemorrhage or re-injury (Kleinman & Barnes, 1998). Magnetic resonance imaging is the preferable modality for differentiating between aging hematoma and cerebrospinal fluid and for estimating the age of subdural bleeding (Alexander et al., 1986; Ball, 1989; Fobben et al., 1989; Gomori, Grossman, Goldberg, Zimmerman, & Billaniuk, 1985; Han et al., 1984; Langfitt et al., 1986; Sato et al., 1989; Snow, Zimmerman, Gandy, & Deck, 1986). Consultation with an experienced pediatric radiologist or neuroradiologist is strongly recommended.

On the other hand, the CT appearance of focal or multifocal, high-density, acute hemorrhage(s) in association with a moderate or large, low-density, subdural collection provides compelling evidence of subdural rehemorrhage or re-injury. Additional radiological manifestations of rehemorrhage or re-injury include septations, loculations, areas of varying density or intensity, and layering within the subdural collection (Kleinman & Barnes, 1998).

Intracranial hemorrhage or rebleeding may occur as a complication of an acquired coagulopathy—a frequent and potentially severe complication of traumatic brain injury in both adults (Kaufman et al., 1984; Olson et al., 1989; Stein, Young, Taluchi, Greenbaum, & Ross, 1992) and children (Hymel, Abshire, Luckey, & Jenny, 1997; Miner, Kaufman, Graham, Haar, & Gildenberg, 1982; Stein & Spettell, 1995). Tissue factor released from damaged brain cells may bind to factor VII and initiate activated coagulation, resulting in prothrombin time (PT) prolongation and occasionally disseminated intravascular coagulation (DIC) with ongoing or recurrent hemorrhage. More simply stated, the injured brain may protectively activate blood clotting throughout the body, ultimately consuming excessive blood clotting factors and leaving the patient vulnerable to rebleeding. In young victims of inflicted head trauma, PT prolongation and activated coagulation are strongly related to the presence of parenchymal brain damage (Hymel, Abshire, et al., 1997).

Because so many conditions may be confused with bleeding and rebleeding on CT or MR cranial imaging, it is highly likely that subdural bleeding with rebleeding has been overdiagnosed in the past. If this is true, then current opinions regarding the frequency, consequences, or forensic significance of subdural rebleeding should be considered speculative.

Under What Circumstances Do Subdural Hemorrhages Rebleed?

Nonacute subdural collections can rebleed spontaneously. As we have discussed previously, the transformation of a delayed, space-filling, posttraumatic, subdural hygroma into a chronic subdural hematoma with potential mass effect requires rebleeding. Nonacute subdural collections also rebleed as a consequence of minor cranial impact.

Consider this case example. An 11-month-old boy was admitted to the hospital with nausea, recurrent vomiting, and irritability. He was treated with intravenous fluids and then discharged. He was readmitted one month later with persistent, chronic irritability. His neurological examination upon readmission was nonfocal and his parents denied any knowledge of closed head trauma. Cranial CT scan at that time revealed a nonacute subdural hematoma overlying the right cerebral hemisphere anteriorly, swelling of the right cerebral hemisphere with midline shifting,

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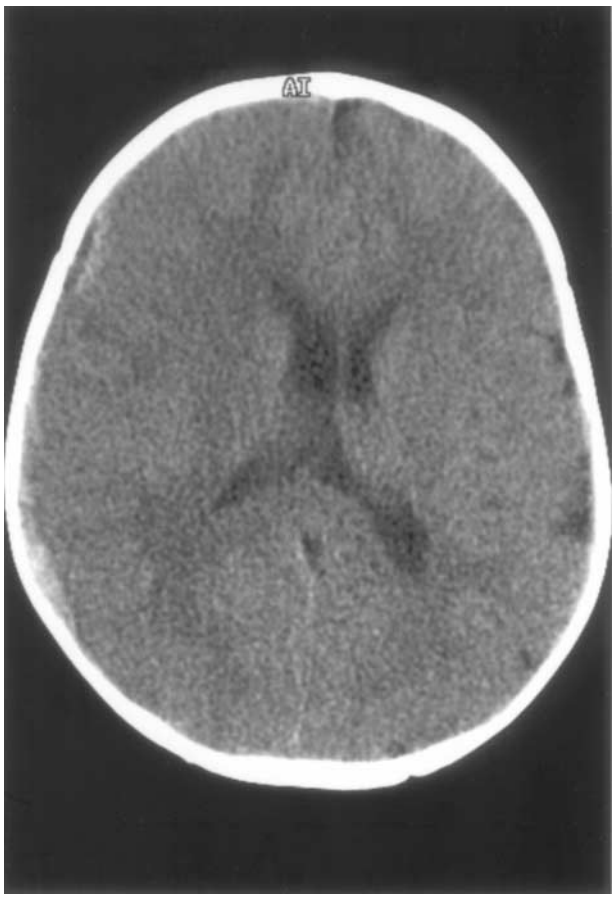

FIGURE 4: **This 11-Month-Old Boy Was Hospitalized and Treated With Intravenous Fluids for His Recurrent Vomiting and Irritability. He Was Readmitted One Month Later With Persistent Clinical Signs. Neurological Exam at That Time Was Nonfocal and His Caregivers Denied Knowledge of Closed Head Trauma. Nevertheless, CT Scan Revealed a Nonacute Subdural Hematoma Overlying the Right Cerebral Hemisphere, Swelling of the Right Cerebral Hemisphere With Midline Shifting, and Probable Atrophy of the Left Cerebral Hemisphere.**

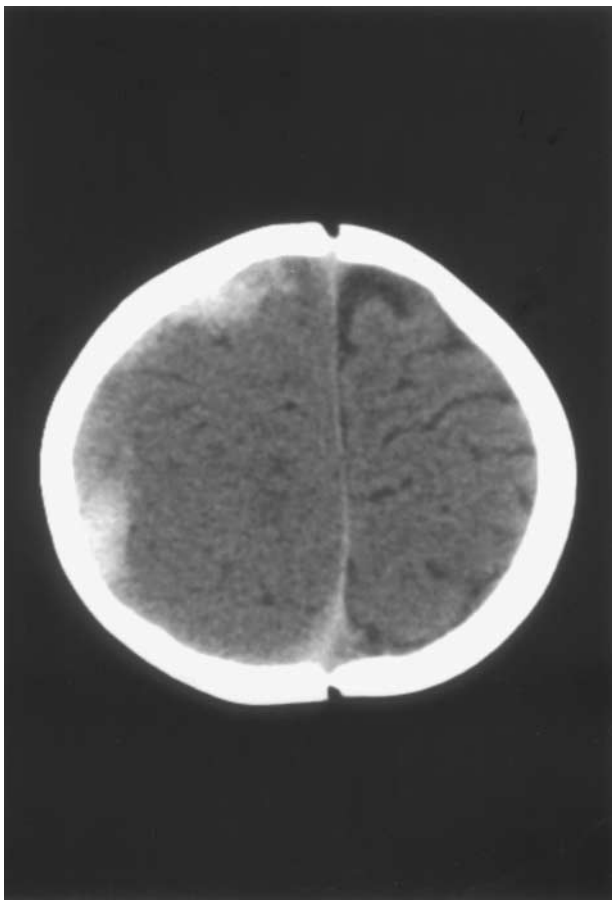

FIGURE 5: **On the Evening His Nonacute Subdural Collection Was Identified, This 11-Month-Old Child Fell From His Hospital Bed. Although He Suffered No Acute Loss of Consciousness, He Appeared Less Active Following the Fall and Became More Lethargic Over the Next Few Hours. Late That Evening, He Manifested Intermittent Periodic Breathing and Bradycardia. Urgent Cranial CT Scan Revealed Increased Swelling of the Right Hemisphere, Acute Subdural Rebleeding on the Right, and Worsening Mass Effect With Increased Midline Shifting. Neurosurgical Intervention Confirmed Acute Subdural Rebleeding.**

and probable atrophy of the left cerebral hemisphere (see Figure 4).

On the evening of admission, his nurse forgot to lower his top bed railing. Although his fall was not directly witnessed, his nurse found the child on his back on the floor next to his bed. He suffered no acute loss of consciousness and was consolable but appeared overall less active following the fall. Over the next few hours, he became more lethargic and late that evening manifested intermittent periodic breathing and bradycardia. An urgent cranial CT scan revealed increased swelling of the right hemisphere, increased volume of the acute, right-sided subdural collection, and worsening mass effect with increased midline shifting (see Figure 5). The child underwent acute neurosurgical intervention. A large, acute subdural hematoma was evacuated, along with an older component of subdural hematoma. Ophthalmologic examination revealed scattered intraretinal hemorrhages. After his neurosurgery, this young child improved clinically and had a favorable long-term outcome.

We postulate the following explanation for the clinical findings in this case. At one year of age, skull and brain maturation had facilitated his older subdural collection to begin to act with mass effect. Clinically, these changes manifested as persistent irritability. Acute subdural rebleeding triggered by his minor fall on the evening of his hospitalization accelerated these adverse mass effects and resulted in clinical deterioration over several hours. Because the fall

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from his hospital bed did not represent a clinically significant cranial deceleration event, this young child did not suffer immediate loss of consciousness, apnea, seizure, hypotension, or prolonged traumatic coma.

To date, no prospective, comparative studies have measured the frequency of subdural rebleeding—or its clinical consequences, specifically in young children with known chronic subdural collections. Acute rebleeding within a chronic subdural collection during infancy may represent inflicted re-injury. Because the history in such cases is frequently unreliable, a prospective study of this type may be impossible to accomplish (Kleinman & Barnes, 1998).

CONCLUSIONS

To conclude that an infant's intracranial hemorrhage or rebleeding resulted from inflicted cranial injury or re-injury may have serious forensic consequences. How can we objectively make this determination? We recommend the following approach.

1. Thoroughly image the affected child's extra-axial collection(s). In most cases, a combination of CT and MR cranial imaging will be required.
2. In consultation with the pediatric radiologist or neuroradiologist, objectively characterize the extra-axial collection as an expansion of the subdural space, the subarachnoid space, or both.
3. If applicable, consider the differential diagnosis for expansion of the subarachnoid space (see Table 1).
4. If applicable, consider the differential diagnosis for subdural hemorrhage (see Table 2).
5. Exclude nontraumatic causes for subdural bleeding by appropriate history, physical examination, radiological imaging, and/or laboratory evaluation (see Table 2).
6. In consultation with the pediatric radiologist or neuroradiologist, objectively estimate the age(s) of the child's subdural collection(s).
7. In cases demonstrating heterogeneity in the radiological appearance of a subdural collection, consult with a pediatric radiologist or neuroradiologist to differentiate between hyperacute subdural hemorrhage, subdural rebleeding (occurring spontaneously or induced by re-injury), or other potential explanations for the heterogeneity (see Table 6).
8. If applicable, attempt to differentiate between subdural hygroma (acting as a space-filling lesion) and chronic subdural hematoma (potentially acting with mass effect) (see Table 3).
9. If neurosurgical intervention is required, make sure that the drained subdural fluid or clot, and the surrounding tissues, are carefully collected for biochemical and histopathological analysis. Review these results to ascertain the composition of the subdural collection and to verify the presence or absence of a neomembrane.
10. Finally, attempt to differentiate between subdural rebleeding and re-injury by considering the child's acute clinical presentation (see Table 5 and Figure 3). Was the acute clinical presentation predictable? In the majority of cases, severe cranial re-injury is associated with acute neurological deterioration, brain swelling, and/or retinal hemorrhages—findings not otherwise associated with spontaneous rebleeding within a chronic subdural hematoma (Kleinman & Barnes, 1998).

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