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Demonstration and Interpretation of Bridging Vein Ruptures in Cases of Infantile Subdural Bleedings

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ABSTRACT: Report of two cases of lethal infantile subdural bleedings (SDB). Bridging vein (BV) ruptures were directly proven as the source of the (minimal) SDB by a postmortem X-ray. In the controversial discussion concerning the causes of infantile SDB, proof of the occurrence of several BV ruptures is seen as an important sign of a trauma of significant degree. Although infantile SDB undoubtedly can result from accidental as well as intentional injuries, and therefore, the SDB itself does not allow far-reaching conclusions as to the cause of injury, the presence of several BV ruptures combined with an SDB of insignificant volume, in an infant dead or in a deep coma on clinical presentation, is not compatible with the supposition of a minor fall as the cause. We have not observed such findings as the result of a minor accidental event for more than 15 years.

KEYWORDS: forensic science, forensic pathology, shaken baby syndrome, subdural hemorrhage, head injury, neurotraumatology, autopsy, postmortem X-ray

Since the publication of articles such as those by Caffey and Guthkelch (1,2), the combination of infantile subdural bleeding, retinal hemorrhages, the lack of external signs of a head injury and an inconsistent history of the event has been considered typical for abuse, especially for violent shaking—which was described as a wide-spread "method" of child abuse (shaken baby syndrome, SBS). This concept became widely accepted for many years. Many authors apparently agreed with the general rule that "the presence of severe neurologic injury in any child, especially if <3 years of age, without a history of significant trauma, should be considered as resulting from inflicted injury until proven otherwise" (3,4). Nevertheless, the discussion of accidental versus non-accidental infantile subdural bleedings (SDB) did not end (5–7). On the contrary, several reports emphasizing the existence of accidental injuries with alleged similar characteristics have been published in recent years (8–10), summarizing a position diametrically opposed to the statement cited above: "non-accidental injury is a less common cause of subdural hematomas than it is believed to be" (11). The situation once more became complicated because significant differences in the causes, the volumes, and the clinical signs between Caucasian and non-Caucasian infants were found (11).

Compared to the quantity of clinical data, relatively little information concerning the pathomorphological conditions are available. This is especially true for the cerebral bridging veins (BV); SDB and BV ruptures seem to be regularly linked without a direct demonstration of injuries of the latter, as if there were no other morphological source for subdural hemorrhages. Only a few remarks are available as to how to investigate the sources of such bleedings (3,13–15); statements like "more often than not, it is impossible to demonstrate the source of the bleeding" (3), or "it is almost never possible to identify the bleeding points" (12) are published. According to other investigators (16–18), these conclusions are obviously based on a technique for removal of the brain, which is inappropriate in cases of SBD. In our institute, the demonstration of the bleeding points in cases of SDB has been a standard element of the forensic investigation in lethal cases of intracranial hematomas for decades (16,18), and reports on lethal cases of shaken infant syndrome including the imaging of the injured BVs have been published (19,20). Recently it has been possible to improve and simplify the method using X-ray imaging of the cerebral veins (21). Morphologically detailed reports of infantile SDB as well as hints on how to investigate the intracranial cavity are rare; therefore, our methods, general experiences and the findings of two recent cases will be presented here.

Technical Note

During autopsy, after the skull is opened in the horizontal plane, the calvarium, together with the upper half of the brain, is cut with a long knife. Any subdural effusion is visible at this time, before the anatomical situation along the parasagittal region is artificially destroyed, and the further procedure can be adapted to the special intracranial findings. For X-ray imaging, contrast material (barium sulfate) is instilled into the dorsal opening of the superior sagittal sinus. X-rays in the axial direction show the superficial cerebral veins and the bridging veins, as is shown in Fig. 1. Figures 2 and 3 demonstrate the findings in the two cases presented here: in Case 1 (Fig. 2), the parasagittal veins were bilaterally torn, visible from the subdural extravasation of contrast material beginning immediately after instillation (Fig. 2*a;* volume of contrast material: 5 mL) and increasing with additional instillation (Fig. 2*b;* volume: 10 mL); note the absence of regularly filled venous vessels. In Case 2 (Fig. 3), the BVs of the left hemisphere were undamaged, whereas on the right side, the upper (parietal) veins were torn, resulting in subdural extravasation. In contrast to Case 1, this is not as diffuse but relatively well-shaped because of the longer survival time and the local healing process (adhesions between dura and arachnoidea). This procedure is possible and especially helpful even in cases with morphological destruction of the brain substance due to progressed brain death (Case 2; Figs. 3 and 6).

In cases of subdural hemorrhage, we always remove the upper half of the brain together with the dura mater, using a large curved

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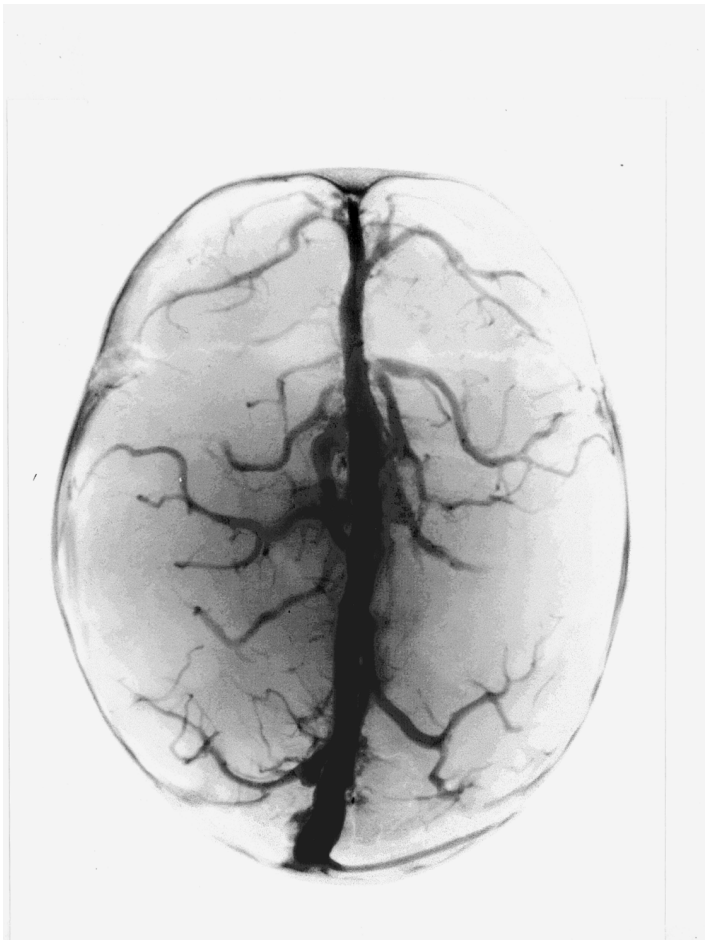

FIG. 1—*Axial postmortem X-ray of the calvarium containing the upper half of the brain after instillation of barium sulfate in the superior sagittal sinus. Control case (no head injury; age 12 month): extensive imaging of the superior cerebral veins and the bridging veins.*

spatula (for more details see (21)). In babies this is often difficult because the dura is firmly fixed along the sutures, but with some training it is generally possible to preserve the anatomical relation between brain and dura (Fig. 4), using a sharp knife to cut these connections. The sites of ruptures are visible from prominent thrombotic closure and/or from the lack of filling of the afferent veins with contrast material (Fig. 4). A histological examination of the ruptures identified can be made (Fig. 5) to prove the type of vessel and the absence of a previous pathological condition as well as to estimate the age of the injury.

Case 1

History: Nine-Month-Old Female Baby—During the final week the child was ill and febrile due to a respiratory infection, and vomited repeatedly. The last pediatric examination was on the evening before the event. At that time, the infant seemed to be ill, but presented no neurological alterations. Later in the night, according to the report of the parents, the infant was found with open eyes but non-responsive in its bed; they shook the child to wake it (without success), started cardiopulmonary resuscitation and alerted an emergency team. The child was found in cardio-respiratory arrest, with dilated and fixed pupils. Resuscitation was able to restore circulation, but the child remained apnoic. Cranial CT revealed mas-

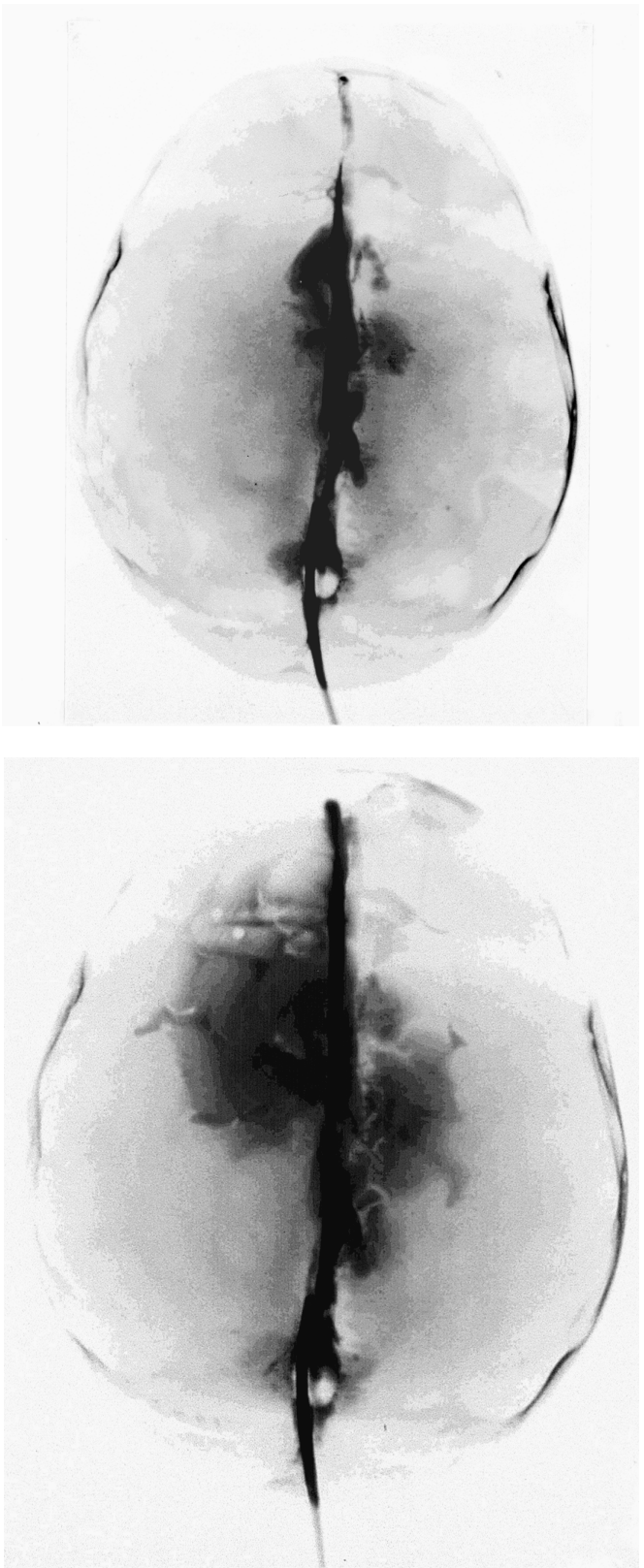

FIG. 2—*X-ray after instillation of 5 mL (Fig. 2*a) *resp. 10 mL (Fig. 2*b) *of barium sulfate. Case 1: obviously shaken baby syndrome. The diffuse subdural extravasation of the contrast material and the lack of regular venous filling of the cerebral veins demonstrate numerous ruptures of bridging veins.*

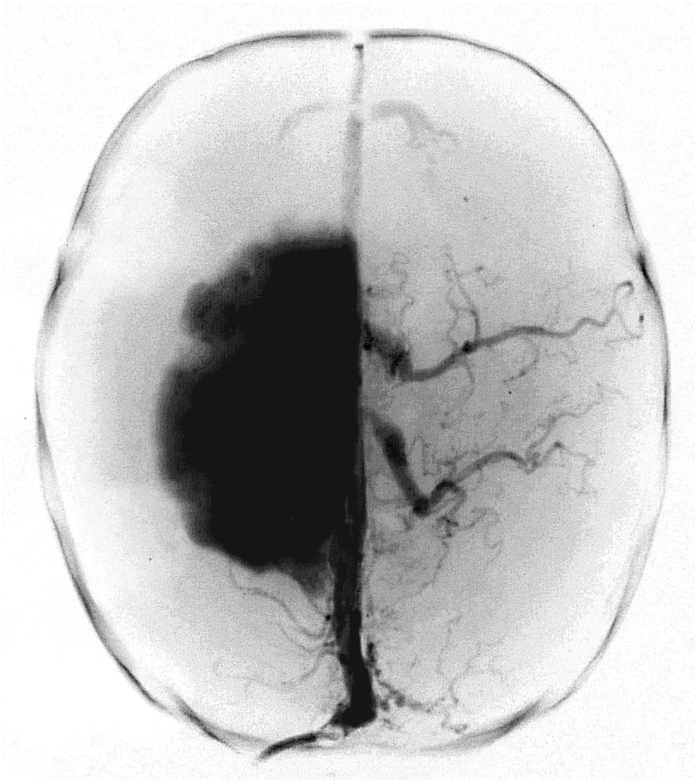

FIG. 3—*X-ray demonstration of a unilateral and well limited subdural extravasation of contrast material from parasagittal bridging vein ruptures. Case 2: probably shaken baby syndrome; survival time two weeks.*

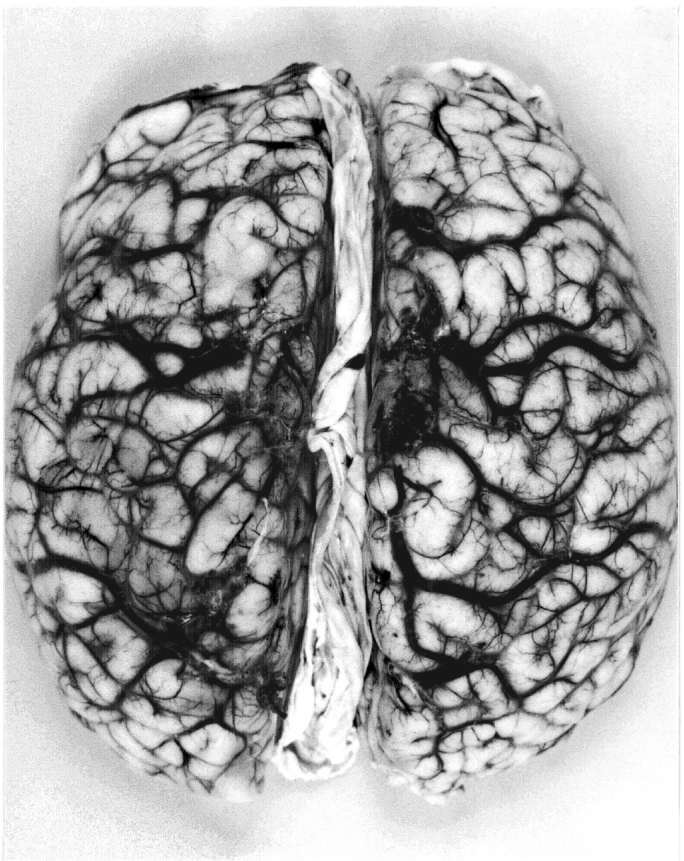

FIG. 4—*Upper half of the brain, which has been removed from the calvarium together with the dura matter; Case 1. Because nearly all bridging veins were torn, the dura could be folded to the midline, after some lateral incisions were made. Small but prominent subarachnoid hemorrhages surround the rupture sites, where thrombi have closed the vessels.*

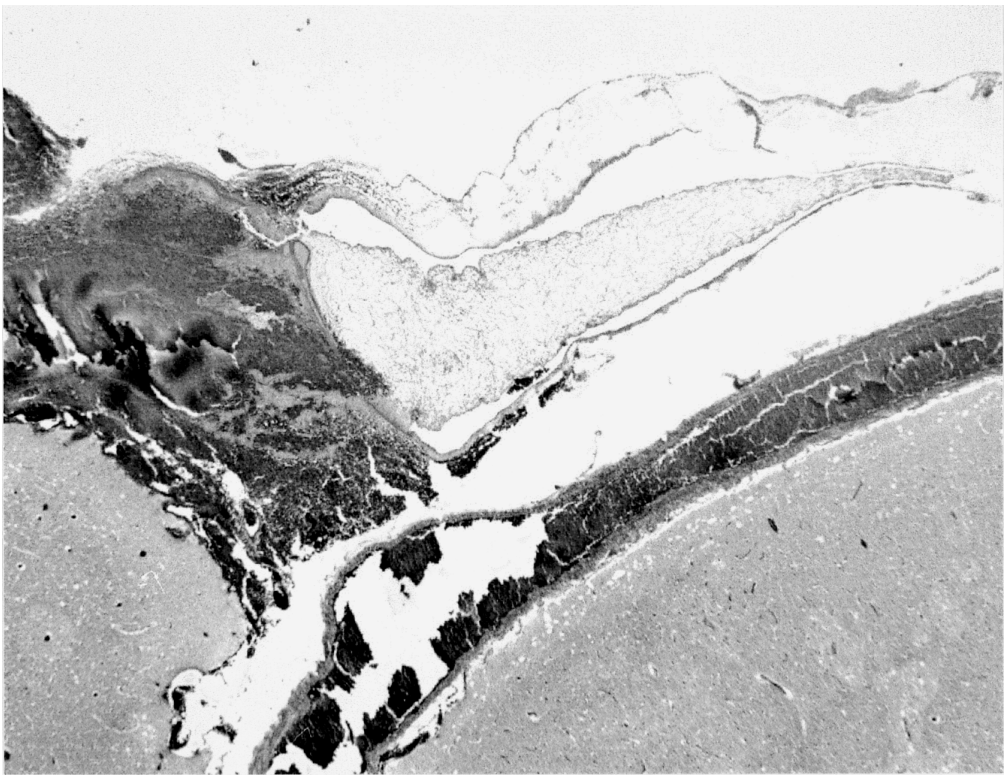

FIG. 5—*Microphoto of a bridging vein rupture, closed by thrombosis; Case 1.*

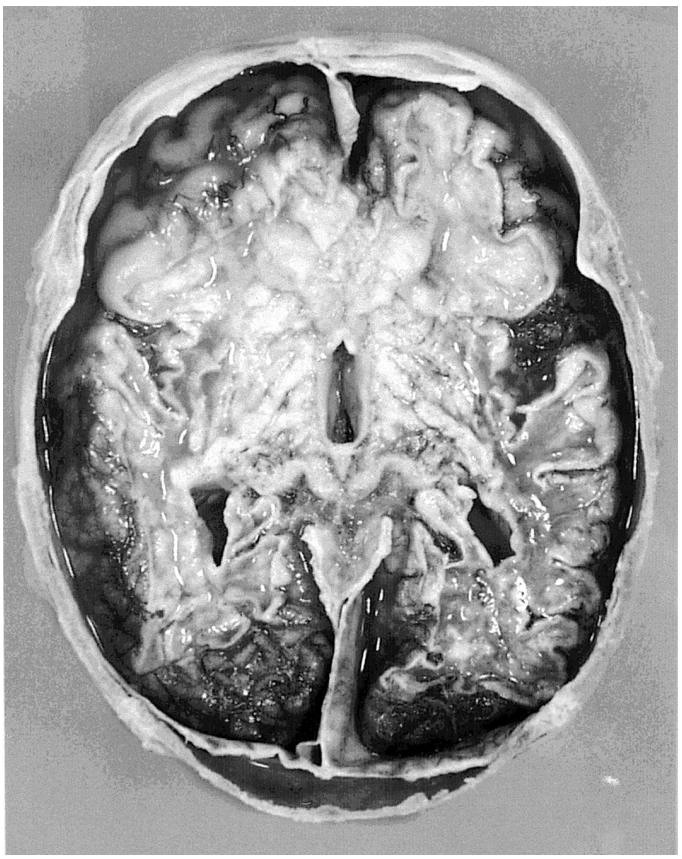

FIG. 6—*View at the horizontal plane of the upper half of the brain which has been cut together with the calvarium from the base; Case 2. The X-ray examinations shown in Figs. 1–3 were made in this stage of autopsy. There is a massive general destruction of the white matter and shrinking and general softening of the brain. Despite this condition, the X-ray method gave a good result (Fig. 3).*

sive brain swelling and minor subarachnoid hemorrhages over the upper frontal and occipital lobes, but no subdural bleeding and no focal lesion. No external injuries were seen. Normal coagulation studies—Lumbar puncture revealed bloody spinal fluid. Pre- and intraretinal hemorrhages in the peripheral parts of both retinae were found in the ophthalmological examination. The child remained in deep coma and brain death was certified four days later. No history of a recent trauma was presented by the parents.

Autopsy Findings: No Recent External Injury Marks—Internally, three insignificant hemorrhages of approximately 0.5 cm diameter were found in the left upper frontal and parietal region. Marked distension of the cranial sutures. Diffuse swelling and softening of the brain matter; prominent pressure marks of the cerebellar tentorium into base of the temporal lobes. Less than 5 mL of red blood bilaterally in the upper subdural space; in the left parietal region brownish adhesions between dura and arachnoidea were present. Nearly all parasagittal BVs were completely torn (Fig. 4); most of the peripheral sites of the ruptures were closed by prominent thrombi (Fig. 5), and the afferent veins were markedly congested (Fig. 4). After formalin fixation, no cortical or gliding contusions and no intracerebral clefts or bleedings were found. Histologically, one of the venous ruptures (corresponding to the small brownish subdural effusion) was clearly older than the majority of the other ruptures because of an advanced healing reaction including fibroblasts and numerous macrophages containing iron. The brain substance showed the typical signs of severe diffuse hypoxic damage, but no remarkable foci. Gross bleeding in the deep suboccipital neck muscles. No macroscopic injury of the cervical spinal cord. Additionally, a healed epiphyseal fracture of the right humerus with intensive formation of callus was present, and a healed rupture of the frenulum of the upper lip (according to the parents—accidental injuries).

Conclusions

The death of the child was medically attributed to a severe acceleration-deceleration injury of the head. The number of BV ruptures as well as the course excluded a minor injury such as an accidental fall from a low height; according to the other findings, a severe blunt impact against the head clearly did not occur. The injury must have been sustained during the few hours between the last medical investigation of the child and her hospital admission; a longer survival time without coma was held to be impossible. This was supported by the absence of any remarkable subdural bleeding, in spite of the number of torn vessels; this pointed to a severe disturbance of intracranial circulation. The other autopsy findings (older injuries) could only be interpreted as indicative of repeated child abuse. In conclusion, the final injury was related to abuse (shaking or shaking including soft impacts). Nevertheless, the proceedings were dropped by the criminal prosecutor prior to a formal indictment because it could not be determined who had abused the child. The parents, who were both present during the period in question, denied any physical abuse, as well as any recent accident; no other persons were implicated.

Case 2

History: Eight-Month-Old Male Baby—Several weeks prior to his death, an increasing head circumference was seen and enlarged external liquor compartments were diagnosed, without abnormalities in the child's behavior or general physical condition. At noon on the day in question, the child was presented to the pediatric physician by the mother because of sleepiness, but was medically found to be in good condition. That afternoon the father was alone with his child. According to his account, he fed his son and put him to bed; soon after (approximately 2 h after the child was seen pediatrically), he checked on him because of a suspicious noise, and found the infant unresponsive and apnoic. He alerted an emergency team and began to resuscitate. On clinical admission, a right subdural bleeding and a left subdural hygroma were diagnosed by CT. No neurosurgical intervention was undertaken because of the infaust prognosis. The child remained in a deep coma which was clinically attributed to severe hypoxic damage prior to hospital admission. Multiple small hemorrhages in both retinae were present. No other external or internal injury was found. The child died when the therapy was minimized two weeks later. The parents were confronted with the suspicion of child abuse by the treating physicians but denied any intentional or relevant accidental trauma.

Autopsy Findings:—Enlargement of the Head—No external or internal injuries except those caused by the medical treatment and the BV ruptures. Subdural extravasation of contrast material in postmortem X-ray into an apparently limited cavity (Fig. 3). Excessive destruction (softening and liquefaction) of nearly all the cerebral and cerebellar white matter with shrinking of the brain (Fig. 6). The ventricles and the external liquor compartments were widely enlarged. Extensive necrosis in the midbrain, pons and

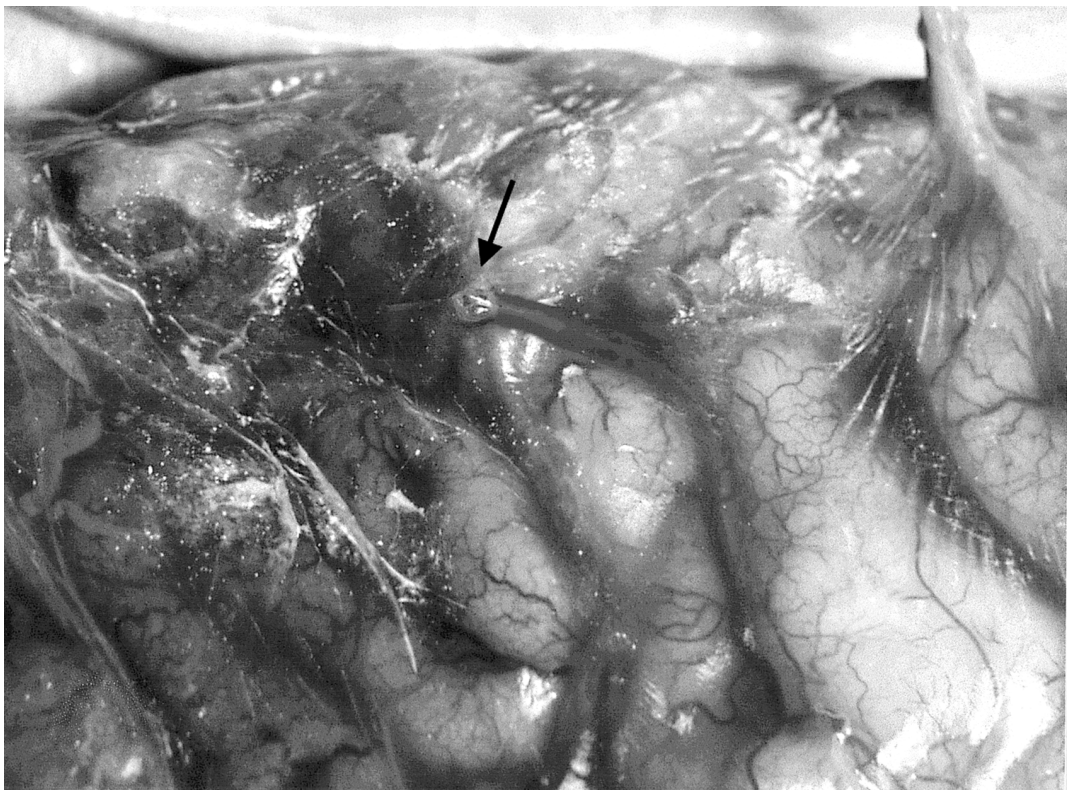

FIG. 7—*Detail of the parasagittal brain surface; Case 2. A ruptured bridging vein is visible near the center (arrow); near the upper margin on the right side a markedly thickened vein (result of an older injury?).*

medulla with demarcation. Covering the right parietal lobe and parasagittal region and stretching into the right side of the interhemispheric fissure, a brown-red subdural membrane (volume less than 30 mL), approximately 3 to 5 mm thick, was present. Small subarachnoid bleedings along the right parasagittal region, surrounding several ruptured (and thrombosed) bridging veins. Other vessels of that type were not interrupted, but thickened by connective tissue (Fig. 7). No gross injury to the surface of the brain; as far as it was possible to determine because of the condition of the tissue, no gross internal injury to the brain matter was present. No clear traumatic lesion older than the actual BV ruptures could be proven, despite extensive morphological investigation.

Conclusions

According to the history as well as to the autopsy findings, abnormalities of the central nervous system were present in this child prior to the fatal event. Neither a primary disease nor a trauma could be proven or ruled out; a morphological diagnosis was significantly restricted because of the degree of the secondary hypoxic damage. Nevertheless, thickening of some BVs could be the result of an older trauma; development of extracerebral fluid collections over the surface of the brain and cerebral atrophy in cases of survived shaking are known (19,22,23).

Despite the preceding brain lesions, the actual BV ruptures resulting in SDB were interpreted as resulting from a head injury, more likely without than with direct impact of the head, but the pathological anatomic condition could have made the BVs more susceptible to even minor injuries (22). Therefore, the conclusions reached from the findings as to the type and severity of the trauma in question were limited. In our opinion, however, repeated acceleration-deceleration injury (shaking) was the most likely cause of this course, but medical proof of this beyond doubt does not seem possible, and additional evidence is not currently available. Legal proceedings are pending.

Discussion

Acute subdural bleeding is one of the most common causes of death or significant morbidity in cases of child abuse (22–29). Although the syndrome has been named “shaken baby syndrome” (SBS), the lack of a confirmed/confessed history of shaking is one characteristic element of such cases: confession of abuse in the initial phase of medical treatment (25) is as untypical as in the later course of criminal proceedings: direct evidence from the perpetrators of the injury is almost never available, and when testimony as to the mechanism of injury is given, this is often of dubious veracity (30). In the 10 cases that we investigated in the last years, one perpetrator confessed violent shaking and another one shaking combined with blows to the face; in three cases, obscure histories were offered and in five cases all trauma was denied. Because most of the assaults are not witnessed by others, doubt must often remain as to the true mechanism of these injuries, and shaking can only be assumed in many of such cases (23,25,30,31). Despite that, from a clinical point of view, the SBS was stated as “a clearly definable medical condition” (23). The cause of intracranial damage is seen as periods of rapid and violent acceleration-deceleration of the head while the infants were shaken (mostly by caretakers or parents), resulting in shearing injuries of cerebral vessels as well as the cerebral matter. This mechanism of “pure” shaking without head impacts has been disputed (31), and the term “shaken impact syndrome” was introduced. The interpretation that shaking alone can-

not produce a life-threatening intracranial injury is confirmed by Elmer et al. (29,32) but contradicted by Gean et al. (22,23, 28,30,33–36); even injuries of the cervical spinal cord (28,35) and disruptions of atlanto-occipital ligaments (35) by this indirect type of trauma were found in some cases, underlining the injury potential of violent shaking.

One aspect of this discussion is the reliability of the diagnosis of the lack of external injury marks indicating a direct head impact, especially in clinical investigations. From forensic experience, it is well known that it is not uncommon for such marks—often injuries of insignificant degree—to be overlooked or not recorded in the hospital reports by clinicians, or that, in another relevant portion of cases, they are recognizable only at autopsy. However, the presence of external contusional marks on an infant's head in cases of intracranial bleeding does not necessarily mean that both injuries were suffered at the same time and/or by the same trauma. In cases of survival over several days, it may be impossible to prove the identical age of both types of injuries even by a histological examination. By contrast, the absence of external contusion marks does not prove a "pure" shaking mechanism, because even rough contacts against soft surfaces (e.g., body of the perpetrator, stuffing) may remain without any visible mark, even though deceleration of the head had been made more vigorous compared to a situation where the head swings freely. Therefore a reliable differentiation between pure shaking and shaking with such contact episodes is impossible in retrospective investigations, and may be impossible even in an existent case under careful examination. In our series, in four out of ten cases no external or internal injury mark on the face or head was present, although several BV ruptures were found. In one case, a detailed confession of violent shaking was made (other examples see (19)) and was consistent with the findings. From a practical point of view, if a child had died from such a trauma, the question of whether or not the head had contacted a (soft) surface during the action seems to be a purely academic one and not of forensic significance.

By contrast, the questions concerning the specific kind of injury (accident-abuse?), the severity of the trauma and the mechanisms resulting in death are of extreme importance to medical expertise and to legal proceedings. Surprisingly, the amount of generally accepted "hard data" with a solid scientific basis on these aspects is sparse, and significantly controversial statements are present in the literature. For the medical expert, this situation is unfortunate to a high degree; the description of Ref 34 seems still to be characteristic: "Proof of assault in suspected incidents of child abuse is a frustrating business. Extrapolation of data to determine mechanisms of injury from clinical evidence superimposes another degree of difficulty . . . therefore clinical syndromes and their interpretation require perpetual amendment."

The essential finding in SBS cases found in living as well as in deceased victims is a SDB. According to (1,2), infantile subdural hematomas are caused by trauma in practically all cases, and they are bilateral in more than 80%. Nevertheless, other possible causes exist: common causes of subdural hematoma in infants include birth trauma, bleeding diatheses and meningitis (22,37); spontaneous hemorrhages due to vitamin K deficiency have been reported (38). It is obvious that the proof of an actual mechanical trauma—such as the demonstration of bridging vein ruptures—provides a much better basis for expertise than an SDB alone, especially, if no other injury marks pointing to a traumatic event are present.

Generally, it is emphasized that closed head injuries in infants differ in several respects from those in later age groups (3,17,22,39,40); severe deformation alone is thought to be able to produce BV ruptures here (22). No adhesions within the subdural space between the dura and the arachnoidea are present—a situation that explains the typical extension of subdural bleedings over both hemispheres and into the interhemispheric fissure (22). Furthermore, infants from 6 to 12 months old have up to 5 mm of space between the cortex and calvarium, a condition that predisposes the BVs to maximal stretching (22). The white matter is poorly myelinated and the thinner fibers present are more vulnerable to shearing forces (39,40); this is the cause for the development of "gliding contusions" (slit-like tears in the parasagittal subcortical white matter) that are typically found in infants under the age of five months and are attributed predominantly to direct head impact traumas, whereas cortical contusions are uncommon in this age group (40,41). It has been possible to demonstrate such gliding contusions (in combination with subdural bleedings) not only in postmortems, but recently even in survivors by CT (35), MRI (27), and by ultrasound (42). As in other reports, in the presentation by Jaspan et al. (42), four of the six cases with such tears had skull fractures, pointing to an injury mechanism different from "pure" shaking. In our cases (those presented here as well as those investigated earlier (20)), contusional tears could not be found, but most of these victims were older than five months; in another postmortem series, white matter tears were found in only 4/13 cases (31). Other macroscopically visible brain injuries were uncommon in SBS cases and were not found in even one of our cases.

All reports agree that SDB in cases of shaking results from torn bridging veins. In surviving patients, these vessels are imaged only occasionally, by angiography (43) or MRI (22). In autopsy cases, injuries of the BVs are sometimes specifically mentioned (1,17,19,20,29,44) and sometimes not (2,25,28,31,35,40,41). Because of apparently widespread reserve in looking for such injuries, a comprehensive systematic study of the traumatology of these vessels—considering factors such as the age of the victim or the type, severity, and direction of the trauma, does not currently exist. Examining SDB in adults reveals important other sources of traumatic SDB (3,16,17,18,21,22), especially, cortical contusions (which are definitely not present in the majority of SBS cases) and isolated ruptures of cortical arteries (which have never been described in infants in the literature nor found in our own cases). Therefore, the significance of BVs for SDB in SBS cases seems to be obvious, but direct proof of such ruptures does not only facilitate the diagnosis of a mechanical cause of the SDB, but is probably also useful in the distinction between accidental and non-accidental trauma.

The exact intensity of force required to cause SDB (respectively BV ruptures) in SBS is not known and is impossible to reconstruct in an actual case. According to the reports of Aoki et al. (8,10,11,45,46), accidents of greater or lesser severity can lead to SDB. Insofar as details are mentioned, a relevant portion of these accidental SDB developed due to falls with an occipital impact, and the source of the bleeding is never described. According to our experiences in adults, the probability of a subdural hematoma occurring at all and the source of such bleeding strongly depends on the direction of the head injury (18): in occipital impacts, subdural hematomas are clearly less frequent than in lateral or frontal impacts, and are predominantly caused by cerebral contusions, whereas pure subdural hematomas due to ruptures of BVs or cortical arteries, without a significant focal brain injury, mostly occur in injuries from a frontal direction. One might therefore suspect that in infantile SDB after minor accidental trauma, a different pattern of intracranial injury would result than in SBS cases, but a comprehensive morphological analysis comparing those cases does not exist. In the majority of accidental cases reported, the infants did

not lose consciousness soon after the trauma, they were not in a life-threatening condition, there was enough time for an adequate clinical as well as radiological diagnosis, the SDB had a significant volume—"real" subdural hematomas, not uncommonly requiring operative intervention—and the prognosis was generally good (8,10,11,45,46). Although fatalities have been reported (47), "benign subdural hematoma is particularly common in infants between 6 and 12 months" (22). In the cases of non-accidental SDB, the condition of the infants is generally poor, many require resuscitation, the rate of death or significant disability is high, and the subdural bleedings mostly have no significant volume; often there is only "a 'smear' subdural" (27), not visible in CT, but only in MRI (22). Only a few reports deal with abuse cases with significant SDB: only 1 of 13 patients (31), and 2 of 28 patients (35) respectively had a hemorrhage warranting surgical intervention; volumes up to 50 mL could be removed by emergency subdural taps in the series of (25), 26 mL were evacuated in a case reported by Sargent et al. (48); 30 g were removed in a case presented by Aoki et al. (9); a space-occupying hematoma was imaged by Kleinmann (49). As far as reported, most of these hematomas were not the result of SBS. In accidents, in 13/28 infants the maximum thickness of SDB exceeded 0.5 cm (11); the volumes of SDH here were between 26 and 269 mL, with a mean volume of 122 mL; SDB with a mass effect were also reported by Aoki et al. (8,46). Although the volume of an SDB itself is not a reliable criterion for the distinction between an accidental and non-accidental event, the minimal volume of SDB in most of the reported abuse cases is striking.

In adult blunt head injuries, a situation remarkably similar to this can be seen: if the result of a head trauma is essentially restricted to lesions of superficial cerebral vessels (BVs or cortical arteries), the life-threatening effect is due to the space-occupying increase of the subdural hematoma's volume. The causes of such "pure" subdural hematomas are predominantly impacts of a likewise minor severity such as falls or assaults (3,12,16–18,50). On the other hand, there is a group of cases in which no severe focal injury of the brain occurs, but all, or a substantial portion of BVs rupture and no SDB develops even in cases with some survival (51,52). The events causing such injuries are severe impacts or deceleration injuries; the victims are predominantly passengers in high-velocity car accidents. Recently in a victim with a survival time of 15 h, a clinical-pathological investigation was able to demonstrate ruptures of nearly all parasagittal BVs, which did not result in any subdural bleeding despite the fact that no thrombosis of the ruptures had occurred (53). The cause for this striking finding has to be seen as a severe diffuse cerebral injury, which is followed almost immediately by a collapse of cerebral circulation, either due to a rapid increase of intracranial pressure, or a severe disturbance of circulation, or a combination of both factors. Ruptures of several BVs, a serious injury in other instances, can therefore remain an epiphenomenon, which has no essential influence on the course, which is lethal in most cases due to the severe diffuse brain injury. This scenario resembles the situation in many cases of SBS.

Obviously, most of the babies with SBS and some SDB do not die because of the SDB itself. In many reports (likewise in the two cases presented here), a situation of abnormal or stopped breathing, requiring resuscitation, and followed by seizures, precedes hospitalization; diffuse hypoxic alterations are often the predominant postmortem finding in fatalities with some survival (see Fig. 6). Typically the mechanism causing death is increased intracranial pressure with massive brain swelling (35,41,49). Rapid diffuse brain swelling may result from direct shearing injury of the white matter (35), which could be demonstrated in cases of subdural bleedings of young infants histologically (40,54) as well as using MRI (27,55); in the two cases presented here, a clear demonstration was not possible, perhaps because of the degree of the secondary hypoxic alterations. Disturbance of the vascular permeability and autoregulation (49) can also result in acute brain swelling; according to Brenner et al. (33,35,49), probable hypoxic brain injury as a result of an apneic period following the traumatic event is even more important for the poor outcome in non-accidental head trauma.

In such a situation, the timing of the primary injury is obviously closely linked to the onset of apnea (35); according to Bruce et al. (32), in the majority of severely injured children, unconsciousness occurs immediately after the non-accidental injury. In contrast to this, in accidental cases, delayed deterioration was frequently observed (11). Based on morphological findings, it is almost never possible to establish the time of injury with certainty, especially in cases with survival after resuscitation, but this aspect is often decisive for the legal proceedings. According to a literature review (56), very few cases in the medical literature provide sufficient detail to reconstruct lethal cases from the time of assault to the onset of symptoms. In the two cases presented here, the victims had a medical examination only hours prior to the situation requiring resuscitation. In Case 1, the severe intracranial injury was undoubtedly not present at the time of medical examination, and Case 2 probably resembles this. Our experiences with SBS as well as with subdural bleedings in adults (52,53) therefore point to the conclusions of Bruce et al. (32,35): if several BV ruptures without a significant SDB are found, the trauma causing this was of such a degree that a severely impaired neurological condition of the victim immediately after the trauma can be concluded with a high degree of probability.

In conclusion, in cases of SDB (mostly of insignificant volume), combined with several BV ruptures and an acute life-threatening condition of the patient, neither a minor fall nor other trivial injury can be accepted, rather, a trauma of an extraordinary degree has to be assumed as the cause. This point of view is supported by daily experience with such frequent infantile accidents, reported by several authors (24,32,57–59).

In our own institute, not a single case of a lethal accidental subdural bleeding of an infant resulting from a minor fall was observed in over 15 years, nor were any recorded in the official statistics of causes of death in our city (57); all accidental lethal head traumas in babies were due to massive events like traffic accidents or falls from a height. Even if severe car accident injuries were used for comparison, subdural bleedings, severe neurological complications and deaths were dramatically over-represented in the abuse group (4).

Although several statements have been published that the concept of SBS—in the sense of a vigorous and serious form of abuse, has to be modified, our own experiences agree with the conclusions of Duhaime et al. (31): "unless a child has predisposing factors, fatal cases of SBS are not likely to occur from the shaking that occurs during play, feeding or in a swing," and Sargent et al. (48): "diagnosis of acute or hyperacute SDH implies catastrophic forces acting upon the head and brain."

Retinal hemorrhages are obviously important findings for a morphological discrimination between accidental and non-accidental infantile SDB (2,25,28,29,31,36), although their specificity has been doubted because they could also be found in accidental cases (10,11,46). In our own bases, the eyes were not removed and investigated: Retinal hemorrhages had already been diagnosed clinically soon after admission to the hospital and the type of injury was defined by the BV ruptures. Furthermore, considering the survival

time (up to two weeks), the healing process and secondary hypoxic alterations, no additional findings supporting the diagnosis of the primary event could be expected.

"There is a spectrum of injury from accidental to non-accidental in which subtle and poorly understood forces at work, thus making the clear distinction between intentional and non-intentional injury difficult" (60). The specific findings of bridging vein ruptures in non-accidental compared to accidental cases have not been sufficiently investigated, which is in part caused by the scarceness of such cases: in our institute, only 10 infantile subdural bleedings were examined in the last 15 years, and the incidence in Germany is generally low (only 10 cases detected in five years (61,62)). Nevertheless, the currently known data and our own experiences seem to indicate that the proof of BV ruptures (especially if numerous vessels are torn) is important for the difficult discrimination in question.

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