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COMMENTARY

"Shaken baby syndrome" and forensic pathology

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The article by Byard [1] raises interesting points, and highlights issues faced by the forensic pathologist, and I will try to present a neuropathological perspective. An infant, by definition aged up to 12 months, is presented to the emergency services having suddenly collapsed. On admission the child is hypotensive and bradycardic, and, following resuscitation, investigations show bilateral thin film subdural hemorrhage (SDH), bilateral retinal hemorrhage, and ischemic encephalopathy. Life support is withdrawn at some point later and the forensic pathological investigations begin.

Much of true forensic pathology is relatively straightforward, at least in relation to cause of death (proving who caused that death can be much more challenging and really should be a function of the legal system rather than the pathologist)—fatal stab wounds, ballistic wounds etc. Even if we consider blunt force head trauma in the setting of an alleged assault, the actual cause of death is rarely challenged. A typical scenario would be punches or kicks causing focal (extradural or subdural hemorrhage) or diffuse (diffuse traumatic axonal injury) injury with subsequent death. However, this is rarely the case in sudden infant deaths with the triad-SDH, retinal hemorrhage, and encephalopathy. Why should this be? While we can all accept that adults regularly beat each other around the head, sometimes with fatal consequences, are we less willing to accept that an adult carer can inflict a fatal injury on a defenseless infant? However, that adults can, and do, inflict fatal injuries, including head injuries, on infants should not be in doubt and any forensic pathologist will have seen cases with severe injuries including broken bones, bruising, and other markers of abuse such as cigarette burns. However, these cases are less common. In the more typical case, the deceased has few marks of injury, possibly a healing or recent fracture or group of fractures, and the collapse is virtually always unwitnessed, the infant being in the sole care of one adult. Therefore, pathology is used to challenge the account of the collapse given by the adult carer, in essence saying that the carer is lying. That said, my experience from adult neurotrauma homicide cases is that the accused rarely admits to a given scenario, even when challenged by witnesses or video evidence.

The term "shaken baby syndrome" is not helpful. It implies a mechanism, and as a neuropathologist I am describing injuries rather than mechanisms. However, despite what some may claim, there is no scientific basis to exclude shaking as a possible mechanism of fatal head injury, and animal model data which would support this mechanism [2]. If we exclude mechanisms we are left with a description of pathologies, and we should focus on these pathologies.

Subdural hematoma is a consistent pathology in fatal inflicted infant head injury. It is my opinion that SDH is traumatic until otherwise proven, regardless of whether the case is an infant, child, or adult. That trauma causes SDH should not be in question. However, there are other potential causes of subdural bleeding to be considered [3], although the vast majority of these can be very quickly excluded and all other causes can be excluded by appropriate postmortem studies. Interestingly, to date I have never had either an inherited metabolic condition or rare coagulopathy offered as the cause for fatal SDH in an adult case of fatal head injury.

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Birth related trauma is an important cause of SDH and can be considered in two groups: in one group, following a difficult delivery, the neonate is very unwell from the outset; in the second group, the neonate is asymptomatic and SDH is only found as a result of imaging for some other reason or at postmortem examination. SDH is seen by imaging in up to 46 % of normal births [4], and has resolved in most cases by 1 month [5].

With this background we have to critically assess papers where authors have demonstrated SDH in neonates and assumed a causal association with hypoxia [6], whereas it is likely that what is actually being reported is the normal incidence of asymptomatic birth related traumatic SDH. If you do look at infant brains with clearly documented hypoxia (drowning, choking etc.) SDH is not a feature [7].

If we therefore accept that in the vast majority of cases SDH is due to trauma, where does the blood come from? In adult neurotrauma it is accepted that the bleeding is due to cortical contusional/laceration injury damaging superficial cortical veins or due to damage to the bridging veins passing from the cortical surface to the draining dural sinuses. Even a century ago, Cushing and his colleagues stated the bridging veins were the source of infantile SDH [8]. However, this concept has been challenged [9] despite anatomical [10], clinical [11], radiological [12], and pathological [13] evidence. The pathologist should also remember the complexity of the bridging vein system [14] and be aware that a naked eye assessment of the major parasagittal bridging veins draining into the superior sagittal sinus (SSS) does not exclude bridging veins as the source of subdural bleeding. Biomechanical studies have highlighted the interface between bridging vein and the SSS as the fulcrum of the force [15]. As such there is a body of evidence supporting SDH as being due to ruptured bridging veins with subsequent bleeding into the tissues at the interface between the dura and the arachnoid.

With regard to non-traumatic causes, very few of the documented potential causes such as infection with sepsis, coagulopathy, or genetic and metabolic causes require further discussion. Cerebral venous (sinovenous) thrombosis can be a radiological mimic of abusive head trauma [16], and is an important consideration as thrombosed cortical vessels are ubiquitous in infants who have been ventilated for a period of time irrespective of the cause of cardiorespiratory collapse. However, pathologically these lesions produce localized subarachnoid hemorrhage and are not associated with SDH or traumatic axonal injury, and therefore cerebral venous thrombosis is not a pathological mimic of childhood head trauma.

Traumatic axonal injury (TAI) is an important indicator of trauma. Infants are rarely exposed to the forces that produce diffuse TAI in the older age group, associated with road traffic accidents, assaults, or high-level falls. From my own experience when present, diffuse TAI is the result of a severe blunt force head injury, such as an infant being swung by the ankles and impacted against a solid surface such a table or wall, and is associated with extensive skull fractures. However, focal TAI, particularly in the cervicomedullary region, is an important indicator of trauma [17], and is not described in any other setting in the literature. Whether this lesion is, in itself, a universally fatal injury remains to be determined.

In summary, when faced with the sudden collapse of an infant in the sole care of one adult who, on postmortem examination, is found to have SDH, retinal hemorrhages, and encephalopathy, in the absence of any obvious natural disease such as cardiac malformation or sepsis secondary to infection, trauma must be the favored diagnosis. If cervicomedullary focal TAI is also present it really is the only reasonable diagnosis. Whether this injury is accidental or inflicted is not for the neuropathologist to determine, although an opinion may be offered in relation to certain scenarios such as short distance falls. However, other findings which may come to light, such as vitamin D levels and thrombosed veins, should be dismissed from a neuropathological perspective as they do not cause SDH or focal TAI.

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