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EDITORIAL

The law and the laboratory

Roger W. Byard · Robert Vink

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Forensic pathologists are continually being asked in court to provide opinions on the possible causes, mechanisms, manifestations and effects of specific injuries. Although the pathological findings in some cases may not be of legal importance, the interpretation in others may have profound effects on the outcome of a particular trial by significantly influencing the decisions of judges and/or juries. For this reason pathologists must formulate opinions carefully. Unfortunately this may be difficult, as many traumatic conditions are not completely understood, the understanding of mechanisms may be in evolution, and individual pathologists may lack personal experience of particular types of cases [1, 2].

Blunt craniocerebral trauma is a good example of an injury type that may be associated with a wide range of outcomes. Unfortunately, the clinical manifestations may be nonspecific and there is often little experimental data to help with the evaluation of cases [3]. Histories may also be unreliable as they are often formulated in cases of inflicted head trauma to protect the perpetrator, rather than to provide a clear explanation of the events leading to the injury or death. This means that cases such as this are among the most challenging, particularly in terms of determining mechanisms, predisposing factors, time frames and the degree of force [4]. Involvement of forensic pathologists in experimental work in the laboratory may, however, be one way to answer some of these questions.

R. W. Byard (✉) · R. Vink
Discipline of Anatomy and Pathology, Adelaide Centre for Neuroscience Research, School of Medical Sciences, The University of Adelaide, Level 3 Medical School North Building, Frome Road, Adelaide 5005, Australia
e-mail: roger.byard@sa.gov.au; roger.byard@adelaide.edu.au

For a variety of reasons, forensic pathologists are not generally involved in laboratory work. This has the unfortunate effect of isolating pathologists from academic activities such as hypothesis testing using experimental methodology. Although standard and new techniques may be used to clarify or focus on certain diagnostic issues, for example with post-mortem toxicology [5–7], the following studies demonstrate how useful direct collaboration between forensic pathology and the laboratory may be in providing answers to other questions that potentially arise in court. Examples that will be discussed include i) the development of an anesthetized sheep model of traumatic brain injury that has enabled the monitoring of intracranial pressure (ICP) and brain oxygenation following closed head injury, and ii) the measurement of cerebral free magnesium concentrations after blunt cranial trauma in alcohol-intoxicated rats. Studies involving biomechanical testing also demonstrate other fruitful areas of laboratory activity [8].

The first study measuring ICP and brain oxygen levels in anesthetized sheep following blunt head trauma revealed that a dramatic increase in ICP occurred within minutes of trauma [9]. Although this was followed by a gradual decrease, and then a steady increase in ICP, at no stage after injury was the ICP ever normal. We propose that this biphasic response resulted from reactive vasodilation in the first instance, followed by developing cerebral edema. The latter was confirmed by positive staining of sections for albumin, which showed that leakage of serum proteins into tissues had occurred, a hallmark of vasogenic edema. The questions asked in court in this area are often centered around how rapidly manifestations of severe blunt head trauma arise and/or appear. In infants and young children this includes debate over the possibility of the child appearing completely normal following injury, i.e., having a "lucid interval." The

rapidity with which increased ICP occurred in the sheep model following blunt trauma would, however, be more in keeping with almost immediate clinical manifestations, generally making a "lucid interval" less likely. Rapid dilation of cerebral vessels after an impact could certainly explain post-mortem observations of gyral flattening and brain herniation [10] in decedents who have died very rapidly of other injuries, e.g., atlanto-occipital fracture dislocation.

In the second study, brain free magnesium levels were measured following trauma in intoxicated and non–intoxicated anesthetized rats [11]. Low magnesium levels in the brain are known to be associated with a worse outcome after head injury [12], and moderate to severe alcohol intoxication in this model caused a much greater fall in magnesium levels. The questions asked in court revolve around whether an individual who is intoxicated is more vulnerable to the effects of head trauma. This has certainly been demonstrated in isolated case reports [13, 14] and has been attributed to a synergistic depressant effect of alcohol and concussion on brainstem centers, in addition to possible vascular mechanisms [15]. The current model, however, provides an alternative biochemical explanation for this phenomenon.

The examples briefly discussed in this editorial demonstrate how experimental laboratory work may be extremely useful in providing data that can be used to answer questions that may arise in court. While it could be argued that the experimental results do not necessarily provide definitive answers to the questions asked, it has to be acknowledged that they have produced supportive evidence that may be very useful in attempting to unravel the underlying mechanisms responsible for the manifestations of certain injuries. Anesthetized rats and sheep, and mechanical models, are certainly not humans, however their responses in a controlled environment do shed light on possible mechanisms and outcomes. Given the difficulties that exist in attempting to retrospectively understand injuries in humans we would suggest that the laboratory is an underutilized resource that can be of great assistance in this area. Needless to say, animal experimental work can only be performed under strict ethical guidelines with approval from recognized multidisciplinary animal ethics committees that are following internationally accepted guidelines. However, the days of an opinion being proffered by an expert forensic pathologist in court based on unsubstantiated conjecture are hopefully nearing an end as science makes further inroads into forensic pathology. The importance of forensic institutes being closely affiliated with universities and research organizations cannot be emphasized enough, thus facilitating hypothesis generation and experimental testing around issues that complicate case interpretation. The laboratory clearly has a role in the law, and integration of forensic pathologists into research groups will only enhance our understanding of many complex issues that cannot be resolved by simple observational studies [16].

References

1. Byard RW, Rutty G, Blumbergs P, Sperhake J, Banner J, Krous HF. Lack of evidence for a causal relationship between hypoxic ischemic encephalopathy and subdural hemorrhage in fetal life, infancy and early childhood. Pediatr Develop Pathol. 2007;10:348–50.
2. Byard RW, Vink R. The second impact syndrome. Forensic Sci Med Pathol. 2009;5:36–8.
3. Byard RW, Bhatia K, Reilly P, Vink R. How rapidly does cerebral swelling follow trauma? Observations using an animal model and possible implications in infancy. Leg Med (Tokyo). 2009;11:S128–31.
4. Finnie JW, Blumbergs PC, Manavis J, Turner RJ, Helps S, Vink R, Byard RW, Chidlow G, Sandoz B, Dutschke J, Anderson RWG. Neuropathological changes in a lamb model of non-accidental head injury (the shaken baby syndrome). J Clin Neurosci. 2012;8:1159–64.
5. Gerostamoulos D, Beyer J, Staikos V, Tayler P, Woodford N, Drummer OH. The effect of the post-mortem interval on the redistribution of drugs: a comparison of mortuary admission and autopsy blood specimens. Forensic Sci Med Pathol. 2012;8:373–9.
6. Burke MP, O'Donnell C, Bassed R. The use of post-mortem computed tomography in the diagnosis of intentional medication overdose. Forensic Sci Med Pathol. 2012;8:218–36.
7. Byard RW, Butzbach DM. Issues in the interpretation of post-mortem toxicology. Forensic Sci Med Pathol. 2012;8:205–7.
8. Byard RW, Gabriellian L, Helps SC, Thornton E, Vink R. Further investigations into the speed of cerebral swelling following blunt cranial trauma. J Forensic Sci. 2012;57:973–5.
9. Byard RW. Patterns of cerebral and cerebellar herniation. Forensic Sci Med Pathol. doi:10.1007/s12024-012-9339-9.
10. Cory CZ, Jones MD, James DS, Leadbeatter S, Nokes LDM. The potential and limitations of utilizing head impact injury models to assess the likelihood of significant head injury in infants after a fall. Forensic Sci Int. 2001;123:89–106.
11. Vink R, Byard RW. Alcohol intoxication may exacerbate the effects of blunt cranial trauma through changes in brain free magnesium levels. J Forensic Sci. 2012;57:979–82.
12. McIntosh TK, Faden AI, Yamakami I, Vink R. Magnesium deficiency exacerbates and pretreatment improves outcome following traumatic brain injury in rats: 31P magnetic resonance spectroscopy and behavioural studies. J Neurotrauma. 1988;5:17–31.
13. Kodikara S. A death due to concussive brain injury augmented by alcohol. Forensic Sci Med Pathol. 2007;3:283–4.
14. Milovanovic AV, DiMaio VJ. Death due to concussion and alcohol. Am J Forensic Med Pathol. 1999;20:6–9.
15. Byard RW, Vink R. Speed of development of cerebral swelling following blunt cranial trauma J Forensic Leg Med. doi:10.1016/j.jflm.2012.12.004.
16. Byard RW. An approach to observational studies. Forensic Sci Med Pathol. 2007;3:239–40.