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MINI-SYMPOSIUM: Forensic Pathology of Brain Trauma in Children

Forensic Pathology of Child Brain Trauma

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INTRODUCTION

The forensic approach to the autopsy differs rather significantly from the approach of the hospital pathologist to the autopsy. Hospital autopsies are done to determine why the patient has died and to evaluate the effects of therapeutic modalities and the accuracy of diagnostic procedures. Forensic autopsies are carried out on particular individuals who fall into the jurisdiction of the medical examiner or coroner system where the individual died. In a very broad sense these are individuals dying of unexpected or, possibly, unnatural causes of death. Other individuals who die from natural causes but because of age or circumstances are also included within the group of decedents of interest to society. The authority to autopsy without family consent is given by state statute to the medical examiner or coroner to investigate these deaths. The medical examiner or coroner system functions to safe guard public health.

Forensic examinations, aside from determining the cause of death, provide other functions, including: identification of the body; obtaining evidence and toxicological specimens from the body; documentation of disease states; documentation and evaluation of injury patterns, significance and mechanisms; protection of personal property; identification of unsafe practices, conditions and lifestyles; and provision of information regarding forensic issues such as time of death. The forensic autopsy is also performed to establish the manner of death that requires consideration of accessory information from death scene investigation, agency records and reports, and medical records. The manner of death describes how the cause of death came about and includes homicide, suicide, accident, natural or undetermined. Establishing the manner of death requires consideration of the circumstances surrounding the death. Of cases investigated by some type of examination of the body by the St. Louis County Medical Examiners Office in 2005, 26% were natural, 7% were homicide, 22% suicide, 39% accidents and 6% were undetermined (St. Louis County Health Department Annual Report; St. Louis, MO, 2005) In addition to those cases in which bodies were actually examined by a forensic pathologist, many other deaths were reviewed by inspection of medical records to establish the cause and manner of deaths for the purpose of death certification. Of these cases about 91% were natural deaths in the St. Louis County Medical Examiner's Office.

Forensic autopsies are directed toward the mission of the medical examiner or coroner system to provide the cause and manner of deaths as well as to carry out the related forensic functions noted earlier. The forensic pathologist may not be particularly interested in strictly medical issues of the deceased, and these medical issues may not be as fully investigated as would be done in a hospital autopsy. This is not to imply that the forensic pathologist does not appreciate the value of those medical issues, it is just that the law under which the forensic pathologist functions does not specify that purpose. Because the forensic autopsy is authorized by a legal statute for particular purposes, some other purposes may not be fully considered. There is no obligation for the forensic pathologist to investigate medical issues of interest to others or—in most states—to provide tissues or material for research purposes. It is not permissible in fact for autopsies authorized by medical examiner or coroner statues to provide research tissues, unless such permission is specifically granted by the next of kin.

The forensic pathologist may utilize a number of consultants to assist his investigations including forensic anthropologists, entomologists, toxicologists, odontologists and a variety of medical specialties including neuropathologists, cardiac pathologists, radiologists, pediatricians and psychiatrists. Forensic examinations provide a wealth of cases that are not available to hospital-based practices. However, the legal requirements that apply to the medical examiner or coroner concerning how tissues and bodies may be used also apply to these consultants. For the consultant neuropathologist, the opportunity to examine a wide variety of forensic materials is truly an educational experience. The purpose of those examinations remains to determine the cause and manner of death, and does not extend to other uses unless so granted by next of kin. A number of lawsuits have recently been initiated over such practices, as sending brains to a University center for neuropathologic examination and not informing the family that the brain was not buried with the body. Currently, medical examiners are behaving with caution in trying to utilize the consultants they need for their cases without becoming involved in litigation by families.

The forensic pathologist interacts with a variety of individuals and agencies who utilize forensic autopsy reports and findings including police, prosecuting and defense attorneys, insurance companies, news media, consumer protection agencies, hospitals, social services, juvenile services and families. Part of the role of the forensic pathologist is to assist those individuals and agencies in understanding the forensic issues in their reports. These interactions may sometimes be contentious with accusations of withholding facts or of biased opinions. Consultants who assist the forensic pathologist may encounter requests from these individuals and agencies for information or court appearances as part of their consultant duties.

Common cases of neuropathological interest in the medical examiner or coroner system include cases of traumatic brain injury, vascular diseases of the brain, brain tumors, brain malformations, seizure disorders and infectious diseases of the nervous system. In the traumatic brain injury cases, there is special interest in determining mechanisms of injury such as fall, impulse or direct impact to the head, consideration of causes of loss of consciousness or time to lose consciousness and aging of injuries. Vascular diseases of the brain are very common in forensic practice, and forensic interests include distinction between hypertensive and traumatic hemorrhages, recognition of aneurismal and vascular malformation bleeding, and identification of ischemic damage and patterns. Brain tumors are found occasionally as undiagnosed lesions in a small number of forensic cases, and these often provide insight into current or past clinical problems. Forensic pathologists autopsy a good number of individuals who die in state or custodial care facilities, and some of these individuals have congenital brain malformations or prenatal injury that explains their developmental problems. Medical examiner and coroner systems examine numerous individuals who die following a seizure. Often the brain examination fails to reveal either the cause of the existing seizure disorder or the reason for death although sometimes there are findings to explain both. Infectious diseases of the nervous system are seen in the medical examiner or coroner system in small numbers but represent an important public-health safeguard. Individuals who die after rapidly developing illnesses are sometimes found to have bacterial meningitis and it is important to identify the organisms responsible so that close contacts may be treated with antibiotics in appropriate cases.

The forensic pathologist routinely uses a number of autopsy procedures that are necessary for the full investigation of their cases but which may be unfamiliar to hospital pathologists. These procedures include removal of bones for documentation of injury and sectioning for histology, removal of digits, fingers or hands for printing for identification, extensive dissection of soft tissues, skeletonization of the face or body to examine wounds, removal of eyes and inner ears, retention of a body following autopsy for further re-examination at a later time and removal of the entire rib cage or spine. Each of these procedures is done when necessary for retrieval of information in a particular case but may require explanation to the funeral home or family of the need for such examination.

Cases involving pediatric neuropathology are common in medical examiner and coroner systems and represent an area in which forensic pathologists frequently need neuropatholgical assistance. This symposium will discuss three areas of pediatric neuropathology: inflicted traumatic brain injury or abusive head injury, non-inflicted or accidental traumatic head injury and perinatal or birth traumatic injury.

UNIQUE FEATURES OF THE YOUNG INFANT HEAD AND NECK

Understanding traumatic injuries in infants and young children requires an appreciation of the unique differences that exist anatomically and developmentally in the young child. The brain, skull and neck of a young child are in the process of maturing, and injuries during this maturation period differ somewhat from those that occur later in childhood and adult life. These distinctions exist up to the middle years of childhood but are most marked at the youngest ages.

The skull of the young child is very thin and pliable to allow the fetal head to move through the birth canal and to deform under pressure of the pelvic bones to leave the birth canal. Although that serves the birth of the fetus, the collapsible skull offers little protection against traumatic injury. The pliability of the skull is because of the unossified nature of the cranial bones. Over the first 2 years, the bone becomes thicker and develops the double diploe. The fontanels are fibrous connections between the unossified cranial bones and these offer little protection to impact. After the sutures are joined, the sutures offer much greater protection to impact. Impact can be readily transferred through the skull to affect the brain and adnexae, and the skull itself is more vulnerable to fracture.

The brain is a large organ from the time of birth and grows rapidly in infancy and early childhood. By 2 years of age, the brain will have obtained about 75% of the weight of an adult brain, although the young brain is still immature developmentally. The growth of the cerebrum is the primary influence of the growth of the calvarium. The weight of a young child's head is proportionally much heavier than that later in life. An infant's head weighs about 10% to 15% of the total body weight compared with 2% to 3% of the weight of an adult head relative to the body. The growth of the face of the child develops more slowly than the calvarium and is dependent upon the development of the maxillae and mandible—which develops in relationship to the growth of the permanent dentition and attains most of its growth and development after the fifth or sixth year of age (13). The brain has a softer consistency in infants and young children because of very high water content, the immaturity of the development of the glial cells, the immaturity of the myelination of the axons and the small size of the axons. The subarachnoid space of a young child is relatively thin but occupies a large surface area (2). The buttressing capacity of the subarachnoid space is thus much less in a young child than that later in life. Paucity of myelination would seem to facilitate axonal damage resulting from strain, as the site of axonal injury appears to be at the node of Ranvier where the myelin is normally discontinous (3, 6). Raghupathi and Margulies found that non-impact rotational velocities caused three times more axonal damage in the neonatal porcine brain and concluded that younger brain tissue may have a lower threshold for injury than the adult (10).

The neck muscles of the young child are quite undeveloped, and early in life these offer little support to the head. Acceleration–deceleration injuries of the head are dependent upon movement of the head that is facilitated by lack of neck strength. One of the principles of athletic conditioning to prevent acceleration–

deceleration injuries is neck strengthening. The athlete or any individual is at greater risk of head injury when the neck is limp and not rigid (1).

These anatomical and developmental features of the young child's head render young children more susceptible to shearing injury when subjected to acceleration–deceleration forces and also allow impact to readily be transmitted through the skull and subarachnoid space to be distributed into the brain itself. Ommaya notes that upon impact the infant skull undergoes large elastic deformation, and in some cases even plastic deformation, which may result in skull fracture, but furthermore, the changes in shape of the deformed skull may produce large strains throughout the cranium and its contents (9).

MECHANISMS OF TRAUMATIC BRAIN INJURY

Traumatic brain injury can be classified into static and dynamic injuries depending on the rate with which force is loaded to the head. Static injuries occur over longer periods of time—usually greater than 200 ms—and cause crushing head injury. Crushing head injuries occur rarely and result when a massive weight crushes the stationary head and results in comminuted fractures of the calvarium, facial skeleton and skull base with fracture contusions and fracture lacerations of the brain. These injuries will be discussed in the accompanying article on accidental head injuries. Dynamic head injuries account for the great majority of head injuries at all ages. Dynamic injuries occur when force is rapidly loaded to the head in less than 200 ms. Dynamic head injury can be caused by impulsive loading that causes the head to move either by direct impact to the head, which is free to move, or by an action to the body that causes the head to move. Impulsive loading will impart inertial movement of the brain within the cranial cavity. The unsupported head will rotate at some point where it joins the cervical spine and the rotational movement of the head will create differential movement of the brain and skull because of the different rigidities of the two structures. Because the dura is attached to the skull, differential movement between the skull and the brain may strain and tear bridging veins to the point of failure and bleeding into the subdural space. The inertial movement of the brain is maximal in the cortex but extends into the brain with greater forces. It is this inertial movement of the brain that results in traumatic diffuse axonal injury.

Impact loading produces several effects to the head. Impact causes contact injuries that include scalp laceration and skull fracture; creates pressure wave propagation into the cranial cavity and brain; and causes brain contusion. Impact also causes inertial brain movement that may be either translational or rotational (4, 5, 7, 8, 11, 12).

Head injury may be a focal or a diffuse injury or a combination of these two. Focal injuries result from direct impact to the head and can be seen grossly on inspection of the brain. Focal injuries include scalp laceration and contusion, skull fracture, epidural hemorrhage, subdural hemorrhage and brain contusions. Diffuse injuries result from inertial loading of the head and include interhemispheric subdural hemorrhage and traumatic diffuse axonal injury. Traumatic diffuse axonal injury results from the differential movement of the brain from the periphery inward and results in shearing of axons.

REFERENCES

1. Cantu RC (2000) Biomechanics of head trauma. In: *Neurologic Athletic Head and Spine Injuries*. RC Cantu (ed.), pp. 2–5. WB Saunders: Philadelphia.
2. Gean AD (1994) *Head Trauma*. Raven Press: New York.
3. Gennarelli TA, Tipperman R, Maxwell WL, Graham DI, Adams JH, Irvine A (1993) Traumatic damage to the nodal axolemma: an early secondary injury. *Acta Neurochir Suppl* 57:49–52.
4. Graham DI, McIntosh TK, Maxwell WL, Nicole AD (2000) Recent advances in neurotrauma. *J Neuropathol Exp Neurol* **59**:641–651.
5. Margulies SS, Thibault LE (1989) An analytical model of traumatic diffuse brain injury. *J Biomech Eng* **111**:241–249.
6. Maxwell WL, Watt C, Graham DI, Gennarelli TA (1993) Ultrastructural evidence of axonal shearing as a result of lateral acceleration of the head in nonhuman primates. *Acta Neuropathol (Berl)* **86**:136–144.
7. Ommaya AK (1985) Biomechanics of head injury: experimental aspects. In: *The Biomechanics of Trauma*. AK Ommaya (ed.), pp. 245–269. Appleton-Century-Crofts: Norwalk, CT.
8. Ommaya AK, Gennarelli TA (1974) Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain* **97**:633–654.
9. Ommaya AK, Goldsmith W, Thibault L (2002) Biomechanics and neuropathology of adult and paediatric head injury. *Br J Neurosurg* **16**:220–242.
10. Raghupathi R, Margulies SS (2002) Traumatic axonal injury after closed head injury in the neonatal pig. *J Neurotrauma* **19**:843–853.
11. Stahlhammer D (1986) Experimental models of head injury. *Acta Neurochir Suppl (Wien)* **36**:33–46.
12. Wilkins RH, Rengachary SS (1985) Biomechanics of head injury. In: *Neurosurgery*. RH Wilkins, SS Rengachary (eds), pp. 1531–1536. McGraw Hill: New York, NY.
13. Williams P (1995) Cranial characteristics at different ages. In: *Gray's Anatomy*, 38th edn. P Williams (ed.), pp. 607–609. Churchill Livingston: New York.