Journal of Biomechanics 47 (2014) 2578–2583

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech
www.JBiomech.com

Biomechanical studies in an ovine model of non-accidental head injury

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ARTICLE INFO

Article history:
Accepted 1 June 2014

Keywords:
Non-accidental head injury
Animal model
Biomechanics

ABSTRACT

This paper presents the head kinematics of a novel ovine model of non-accidental head injury (NAHI) that consists only of a naturalistic oscillating insult. Nine, 7-to-10-day-old anesthetized and ventilated lambs were subjected to manual shaking. Two six-axis motion sensors tracked the position of the head and torso, and a triaxial accelerometer measured head acceleration. Animals experienced 10 episodes of shaking over 30 min, and then remained under anesthesia for 6 h until killed by perfusion fixation of the brain. Each shaking episode lasted for 20 s resulting in about 40 cycles per episode. Each cycle typically consisted of three impulsive events that corresponded to specific phases of the head's motion; the most substantial of these were interactions typically with the lamb's own torso, and these generated accelerations of 30–70 g. Impulsive loading was not considered severe. Other kinematic parameters recorded included estimates of head power transfer, head–torso flexion, and rate of flexion. Several styles of shaking were also identified across episodes and subjects. Axonal injury, neuronal reaction and albumin extravasation were widely distributed in the hemispheric white matter, brainstem and at the craniocervical junction and to a much greater magnitude in lower body weight lambs that died. This is the first biomechanical description of a large animal model of NAHI in which repetitive naturalistic insults were applied, and that reproduced a spectrum of injury associated with NAHI.

1. Introduction

While non-accidental head injury (NAHI; or "shaken baby syndrome") is an important cause of death and severe neurological dysfunction in children under three years of age, the majority of cases occurring in the first 12 months of age, its pathogenesis and biomechanics are incompletely understood (Blumbergs et al., 2008). Early reports recognized subdural hemorrhage, retinal hemorrhages, and long bone fractures as being suggestive of inflicted head injury in infants and young children (Caffey, 1972, 1974). However, this concept has now evolved into a constellation of lesions (acute encephalopathy, and subdural and retinal hemorrhages) referred to as NAHI (Blumbergs et al., 2008; Krugman et al., 1993). In NAHI, death occurs in 10–40% of cases and many survivors are left with cognitive and behavioral disturbances, cerebral palsy, blindness and epilepsy (Blumbergs et al., 2008).

Many aspects of NAHI remain controversial and intermittently undergo revision (Donohoe, 2003) including whether shaking alone is sufficient to injure the brain or whether an additional head impact is required. This is due, in part, to varying mechanisms of brain injury between individual cases (Bandak, 2005) usually lack of a reliable history of the circumstances surrounding the suspected abuse (Leestma, 2005) and frequently denial of maltreatment by the perpetrator. Moreover, the absence of any external evidence of TBI does not necessarily preclude a diagnosis of NAHI and the lesions found in such cases are not pathognomonic (Blumbergs et al., 2008).

Very few animal models have been developed to study the biomechanics of NAHI and extrapolation of data from adult models to the pediatric population is frequently inaccurate (Gerber and Coffman, 2007; Margulies and Coats, 2010).

There have been several studies of NAHI in laboratory rodents (Bonnier et al., 2004; Smith et al., 1998), but the small, lissencephalic brain of these species does not satisfactorily replicate real-world human NAHI; the smooth lissencephalic brain surface may resist deformation after a traumatic insult more than brains possessing gyri, and since shearing forces and inertial loading are related to brain mass, small rodent brains can tolerate much greater angular acceleration forces than animals with larger gyrencephalic brains (Margulies and Coats, 2010).

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http://dx.doi.org/10.1016/j.jbiomech.2014.06.002
0021-9290/

We recently developed an ovine model of NAHI (Finnie et al., 2010, 2012). This species was selected because lambs have a relatively large, gyrencephalic brain and weak neck muscles resembling that of human infants. This study proved that manual shaking of a younger, lighter body weight subset of lambs could result in death, without an additional head impact being required (Finnie et al., 2012). Neuropathological examination of these lambs revealed mild, focal macroscopic subdural hemorrhage in three of nine shaken animals (the dura was not examined histologically) and, sometimes, microscopic subarachnoid hemorrhage. Axonal injury, neuronal reaction, and albumin extravasation was widely distributed in the brain and cervical spinal cord and of much greater magnitude than higher body weight shaken lambs that did not die. The eyes of shaken lambs showed damage to retinal inner nuclear layer neurons, mild, patchy ganglion cell axonal injury, widespread Muller glial cell reaction, and uveal albumin extravasation. It was suggested that mechanical deformation of the brain, rostral spinal cord and eyes was probably largely responsible for the observed pathology (Finnie et al., 2012). Pathological data has been reported previously and is summarized in Table 1.

This paper describes the biomechanical events that produced the reported neuropathological findings in this ovine model (Finnie et al., 2010, 2012). The objective of this study is to characterize the kinematics of lamb heads during shaking episodes, together with some general characterization of the relative motion of the head to the body.

2. Materials and methods

2.1. Experimental protocol

Nine anesthetized and ventilated lambs were manually grasped under the axilla and vigorously shaken for 20 s with sufficient force to move the head rapidly back and forth, similar to head motions believed to occur in human NAHI. There was no intentional head impact and the head moved freely during each episode. Each lamb was shaken in this manner 10 times over a 30-min period and then placed quietly in the sphinx position for 6 h under anesthesia. Four control lambs were not shaken, but were otherwise subjected to the same experimental protocol. Lambs were maintained under anesthesia for the full duration of the experiment, without ever regaining consciousness, until killed by perfusion fixation of the brain (Finnie et al., 2010, 2012).

The experimental protocol complied with the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes (National Health and Medical Research Council, 2013) and was approved by Animal Ethics Committees of the University of Adelaide and SA Pathology.

2.2. Biomechanical analysis

The acceleration of the head was acquired at 20,000 Hz using an 8 g triaxial accelerometer (Endevco©). The position and orientation of the head and torso were registered using the FASTRAK© system (Polhemus©): two 9.1 g motion sensors were used. The triaxial accelerometer and one motion sensor were mounted on the skull using plastic supports mounted in epoxy putty. A second motion sensor was sutured under the axilla of the right forelimb in order to measure the motion of the torso. This sensor was held under the hand of the operator during each shaking episode.

The position of the accelerometer and the head motion sensor was registered in an anatomical coordinate frame using a three-dimensional coordinate measuring arm. Sensor data were transformed into this consistent anatomical coordinate frame.

2.3. Signal processing

Acceleration and FASTRAK were synchronized using cross-correlation between the sensor data. The acceleration data could therefore be located both in time and in space, in order to determine which phases of the shaking motion high accelerations were occurring. Acceleration data were filtered forward and in reverse using a 500 Hz 8th order Chebychev digital filter, post-acquisition.

Severity was characterized by peak levels of head acceleration and the power transfer to and from the head. The Head Injury Criterion used in impact testing is similar to a power calculation (Hutchinson et al., 1998), and more than one power criterion has been proposed in the past (Neal-Sturgess, 2002; Newman et al., 2000). Power was estimated by taking the scalar product of the head acceleration vector and the head velocity vector; the power was expressed in the units of W/kg.

2.4. Brain injury evaluation

Full details of neuropathological findings may be found in Finnie et al. (2010, 2012) and are briefly highlighted in Section 1 of this paper and Table 1. A particular focus was on the amount of axonal and neuronal damage revealed by immunohistochemistry.

3. Results

3.1. Head kinematics—displacement

Three individuals manually shook animals over the course of the experimental series. Each animal was shaken at a frequency of about 2 Hz resulting in approximately 40 cycles per episode and about 400 per animal. The shaking input occurred generally in the sagittal plane. The motion of the axilla position sensor (at the hand of the shaker) was generally anterior–posterior, although there was cranial–caudal (vertical) displacement in some episodes. In response, the center of gravity of the head typically moved within or about the anterior–posterior plane of the animal.

Trajectories are shown below and in supplementary animated figures that are available electronically. Fig. 1 shows the trajectory of the head motion sensor and the axilla sensor in the laboratory space in the fourth shaking episode of Subject 3. The motion of the axilla sensor was cranial–caudal and anterior–posterior. In response, the head was propelled away from the shaker until it reached the lowest point in the laboratory space, after which the head rose vertically, closer to the shaker. An animation of this trajectory is shown in three orthogonal views in Supplementary Fig. S1.

Supplementary material related to this article can be found online at http://dx.doi.org/10.1016/j.jbiomech.2014.06.002.

The position of the axilla sensor represents the position of the torso of the subject and can be used to locate the head relative to the body (Fig. 2). In most episodes, this relative motion of the head was “C”-shaped trajectory.

3.2. Head kinematics—acceleration

Each shake was characterized by local acceleration peaks at various phases of the shaking cycle. An example of a single cycle (beginning at α and ending at ω) is shown in Fig. 3; the labeled points indicate the incidence of acceleration peaks. The acceleration history of this episode and the acceleration levels over the cycle α to ω are shown in Fig. 4. There were three acceleration peaks during the cycle (A, B and C). The first occurred as the head passed the summit of its arc and was being accelerated downwards (A; c.f. Fig. 3). A larger pulse was measured at the nadir of the arc as the head/neck reached the limit of motion (B) and short, sharp acceleration was recorded as the head suddenly reversed direction relative to the torso (C). This location corresponded to a point where the head interacted with the posterior aspect (dorsum) of the torso of the subject.

Local peak acceleration levels and their associated locations in the head trajectory, across the entirety of Episode 4 of Subject 3 are shown in the top left panel of Fig. 5, and in a real-time animation on three orthogonal views in Supplementary Fig. S2. The peak acceleration level recorded in this episode was 67 g.

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