The Biomechanics of the Perinatal, Neonatal and Pediatric Cervical Spine: Investigation of the Tensile, Bending and Viscoelastic Response

by

Jason Frederick Luck

Department of Biomedical Engineering
Duke University

Date:_______________________

Approved:

___________________________

Barry S. Myers, Supervisor

___________________________

Cameron R. ‘Dale’ Bass

___________________________

John E. Dolbow

___________________________

Donald P. Frush

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Roger W. Nightingale

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Lori A. Setton

Dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Biomedical Engineering in the Graduate School of Duke University

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ABSTRACT

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Abstract

Pediatric cervical spinal injuries are associated with high morbidity and mortality. Cervical injuries observed in the pediatric population appear to be age dependent with younger children experiencing more upper cervical level injuries compared to increased lower level cervical injury patterns to older children. The majority of pediatric cervical spinal injuries are motor vehicle crash related. Current progress in child occupant protection, including increased and proper restraint usage continues to reduce serious injury and fatalities to child occupants. However, improper restraint usage and incorrect child seating location, especially with children transitioning from rear-facing child restraints to forward-facing restraints is still a concern. Continued reductions in serious injury and fatalities to child occupants in survivable motor vehicle crashes will be based on continued education and improvements in child anthropometric test devices, child computational injury models and child restraint system design. Improvements in all of these categories are dependent on an improved understanding of the developmental biomechanics of the human cervical spine. Currently, limited data exist on human child neck biomechanics and none of the current cadaveric work has evaluated the biomechanical response over the entire age spectrum from birth to young adulthood. Numerous surrogate studies exist and have formed the basis of child injury criteria and developmental biomechanics,
but have not been assessed in relation to the response of the pediatric human cervical spine. The current work investigates the biomechanics of the osteoligamentous human cervical spine from birth to young adulthood under tensile and bending loading environments. Tensile low-load and load-to-failure stiffness, load-to-failure, and flexion-extension bending stiffness increased with age. Tensile normalized displacement at failure and total bending low-load range of motion decreased with age. Viscoelastic rate effects are present in the pediatric cervical spine and are modeled with quasi-linear viscoelasticity. Peak load and loading energy increases with increased loading rate, while hysteresis energy is rate insensitive at lower loading rates, but increases at higher rates of loading. These data establish structural response behavior and injury thresholds for the osteoligamentous cervical spine by age. Additionally, they provide human data to assess the appropriateness of current surrogate models and current scaling techniques associated with these models. Finally, these data provide human response by age useful in progressing the biofidelity of computational and physical models for child occupant protection.
Dedication

This dissertation representing the work of many years is dedicated to my family in recognition of the support they always provided, from the beginning to the end of the journey. To my mom, dad and sister; I thank you for always being there to support this journey in just the right way. You’ve helped in so many ways, both seen and unseen and I’m forever grateful. To my daughters, Genevee and Emelia Jean, you’ve taught me so much in such a short time and always provided a deeper perspective during this journey and I’m forever blessed by the experiences we’ve had and will have in the future. To my wife, Vera, you’ve seen this journey from beginning to end and one thing has always been constant – your unwavering support and encouragement. Thank you for never doubting me and for always helping to see the bigger picture.

Additionally, this work is dedicated to the donor parents for making the gracious and uncommon choice to gift their children’s remains to science. A more thorough and comprehensive understanding of the biomechanics of the child neck is the initial result of this work, but the application of the work towards helping generations of children to come will be a gift to society that is forever.
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1. Introduction

Pediatric cervical spine injury continues to be an important public health issue due largely to high mortality and morbidity rates compared with adult cervical spinal injury. The economic and societal impact of these injuries, particularly the prospect of long-term disability and care, is magnified by the youth of the victims (Nitecki and Moir 1994; Dietrich et al. 1991; Anderson and Schutt 1980; Hartunian et al. 1980). Unintentional injury including motor-vehicle crashes (MVC) are the leading cause of pediatric cervical spine injuries (Nitecki and Moir 1994; McGrory et al. 1993; Dietrich et al. 1991).

Recent improvements in child occupant restraint systems, increased usage, successful public-awareness initiatives and mandatory child restraint legislation have reduced child injury and fatalities associated with MVCs. However, further improvements are constrained by limited research on pediatric biomechanics. Current child occupant protection is evaluated using simple anthropometric test devices (ATDs) or dummies to simulate the physical response of a child in a restraint system. A fundamental assumption of this process is that the biomechanical response of the ATDs and the injury metrics used to assess the response of the ATDs are in a word, ‘child-like’. The reality is that these dummies are largely based on adult human cadaveric and animal surrogate responses scaled in most cases by geometrical relationships between the surrogate and the desired child anthropometric age range. While these methods
have produced models of human pediatric biomechanical response and have allowed the development of protective measures that have saved numerous lives; in the end, these methods provide approximations of unknown validity to the biomechanical responses of the pediatric human.

Furthering our understanding of the biomechanical response of the pediatric cervical spine is of obvious importance in designing neck injury countermeasures. However, improvements in injury prevention as a result of this effort are not limited to the neck. The cervical spine is the delivery device for the head; especially for restrained vehicle occupant kinematics, and serves an important component in determining head motion and subsequent contact with objects or inertial loading. Improvements in characterizing the response of the child neck in tensile and bending loading will more appropriately define head motion and thus potential injurious contact and non-contact loading.

Though a small number of human cadaveric studies over the past 135 years have investigated various aspects of pediatric cervical spine response and injury (e.g. Duncan, 1874; Kallieris et al. 1976; Wismans et al. 1979; Dejeannes et al. 1984; Ouyang et al. 2005), full elucidation of pediatric cervical spinal biomechanics has been hampered by the limited availability of pediatric cadavers. In lieu of human cadaveric tissue, a number of research initiatives using animal surrogates were undertaken to approximate the response of the child neck to load (Prasad and Daniel 1984; Mertz et al. 1982; Pintar
et al. 2000; Hilker et al. 2002; Ching et al. 2001; Nuckley et al., 2005; Nuckley and Ching 2006). While these studies have served a critical role in advancing child occupant protection there remains a significant void in our basic understanding of the biomechanics of the pediatric cervical spine. This dissertation characterizes the biomechanical response of the human pediatric cervical spine, both whole and segmental, under multiple modes of loading from birth to young adulthood. This study significantly augments the few data points that currently exist, expands the understanding of neck response over the pediatric age range, and provides insight and direction into future investigations.

1.1 Hypotheses

Historically, the ligamentous supporting structure of the pediatric cervical spine was thought to be ‘lax’ in comparison to the mature spine. Morphological differences, weak supporting musculature and the large head mass of the young child in relation to overall body mass have also been routinely asserted as factors contributing to differences between the response of the immature and mature neck. These assertions have only weakly been validated biomechanically, typically through radiographic examination and anecdotal clinical and injury associated observations. To assess these concepts, this study examines the following hypotheses:

- Under tensile loading, the structural stiffness of the cervical spine increases with age.
• It is proposed that the structural tolerance or load-to-failure of the cervical spine increases with age. The relationship between the structural and load-to-failure response of the cervical spine as a function of age is hypothesized to be non-linear in form.

• Under flexion-extension bending loads, the structural stiffness of the cervical spine increases with age, allowing for increased range of motion at low-loads in the youngest ages.

• The tensile viscoelastic structural response of the pediatric cervical spine may be modeled with quasi-linear viscoelasticity (QLV).

• The full strain history (FSHSE) approach, which models the relaxation of the spine with a spectral model and considers the actual displacement-controlled loading history during parameter estimation, is hypothesized to model the response of the pediatric spine more effectively than the other QLV implementations investigated. The different QLV implementations will be assessed quantitatively by comparing the root mean squared error and difference in peak load, loading and hysteresis energy between model predictions and constant velocity experimental tests spanning three orders of loading frequency.
1.2 Dissertation Organization

The dissertation is organized around five manuscripts focusing on the fundamental biomechanics of the pediatric cervical spine. These manuscripts are either published in the peer-reviewed literature (2), are under review for a scholarly journal (1) or are in the final stages of preparation for submission (2).

Chapter 1 introduces the fundamental problem and hypotheses. A review of cervical spine anatomy and growth and development is presented in Chapter 2. Chapter 3 provides a background on the scope of pediatric cervical spine injury including the importance of motor vehicle crashes (MVC), typical injury patterns observed, pediatric occupant protection and review of pediatric cervical spinal biomechanics.

Chapters 4 through 8 include the central manuscripts of this dissertation. Chapter 4 focuses on the development of the experimental testing apparatus and is currently published in the Journal of Biomechanics (2012, Volume 45, Number 2, pages 386-389) as “An apparatus for tensile and bending tests of perinatal, neonatal, pediatric and adult cadaver osteoligamentous cervical spines”. Chapter 5 focuses on the quasi-static tensile stiffness and load-to-failure data from birth to 14 years of age and is currently published in the Stapp Car Crash Journal (2008, Volume 52, pages 107-134) as “Tensile mechanical properties of the perinatal and pediatric PMHS osteoligamentous cervical spine”. Chapter 6 presents the tensile load-to-failure data of all twenty-four perinatal, pediatric and young
adult specimens with a focus on load-to-failure structural stiffness and tolerance of the upper and lower cervical spine. Functional relationships between the biomechanical parameters and age are presented. This work has been submitted and is currently under review in *Spine* (2012, SPINE-S-11-01039) as “Tensile failure properties of the perinatal, neonatal, pediatric cadaveric cervical spine”. Chapter 7 focuses on the viscoelastic response of the 6-year-old whole, upper and lower cervical spine. Multiple implementations of quasi-linear viscoelasticity are compared to model the time-dependent response of the pediatric spine. Chapter 8 focuses on the flexion and extension bending response of the pediatric cervical spine with the intent of submission to the *Journal of Bone and Joint Surgery* as “Flexion and extension bending of the perinatal, neonatal and pediatric cadaveric cervical spine”.

Overarching themes and conclusions of the entire pediatric cervical spine testing initiative are discussed in Chapter 9. This discussion includes the contributions of this dissertation and future research directions.
2. Anatomy, Growth and Pediatric Considerations

This study provides a biomechanical analysis of the osteoligamentous cervical spine from birth to young adulthood. The analysis was conducted on tissue from 20 weeks gestation to 18 years. This chapter includes a description of the general anatomy of the human cervical spine and the differences between the pediatric and adult or mature cervical spine that is representative of the age range of the tissue sample investigated.

2.1 General Spinal Anatomy

The cervical spine is an inherently flexible structure allowing for varying degrees of compression, tension, flexion, extension, lateral bending and torsion (Figure 1). The cervical spine extends from the base of the skull to the thoracic or chest region and contains seven independent vertebrae. Adjacent vertebrae, the intervertebral disc that separates the vertebral bodies and all associated ligamentous connections between the adjacent vertebrae are typically referred to as either motion segments or functional spinal units (FSU) as they represent the smallest unit of the spine with biomechanical characteristics representative of the ligamentous spine (Junghanns 1977; White and Panjabi 1978). The joint space in the upper portion of the cervical spine that interacts with the occipital region of the base of the skull is typically further delineated as the atlanto-occipital joint (first cervical vertebra to base of skull; AO or OA joint) or craniocervical junction, the atlanto-axial joint (first to second cervical vertebrae; AA
joint) and the entire complex as the atlantoaxial-occipital joint (second cervical vertebra
to base of skull (O-C2); AAO or OAA joint). The current experimental investigation will
focus on the whole cervical spine (WCS or WS; head and intact craniocervical junction to
the first thoracic vertebra), the upper cervical spine (UCS; including O-C2) and its
interaction with the base of the skull, and lower cervical motion segments (LCS;
including C3-C4, C5-C6, C4-C5 and C6-C7).

**FIGURE 1:** Cervical spine (Gray 1918)

### 2.1.1 Vertebra

The cervical spine is composed of both bony and soft tissue components that
interact to form the fundamental joint structure and allow for physiological movements.
The bony vertebrae are the fundamental building blocks that give shape and create the
scaffold for this region. Normative measurements of the size of the different anatomical
features of the adult vertebrae have been well characterized (Nissan and Gilad 1984; Gilad and Nissan 1986; Panjabi et al. 1991a; Doherty and Heggeness 1994). The seven vertebrae of the cervical spine are typically divided into the two upper vertebrae and the five lower vertebrae. This sub-division is predicated on the distinct morphological differences between the vertebrae in these regions.

The five lower vertebrae are similar in that they each contain a vertebral body of roughly a cylindrical to ellipsoid shape, as viewed in the transverse plane, positioned in the anterior half of the vertebra (Figure 2). The vertebral arch attaches bilaterally in a posterior-lateral aspect to the vertebral body and forms a posterior bony boundary for the vertebra as a whole. The vertebral foramen formed by the boundary of the posterior surface of the vertebral body and the inner surface of the vertebral arch provide a canal for the spinal cord to traverse the length of the spinal column and thus provides in addition to axial structural support a protective structure to the central nervous system of the body. Two transverse and one spinous process, extend laterally and posteriorly, respectively, from the vertebra and are landmarks associated with muscle attachment points. All of the cervical vertebrae with the exception of the seventh have a bifid process as opposed to a spinous process. Four articular processes on the superior and inferior faces of the vertebral arch provide surfaces allowing adjacent vertebral movement while limiting excessive movement between vertebrae. The section of the
vertebral arch that connects the articular processes with the spinous process is the lamina.

**Figure 2:** Typical lower cervical vertebra - superior view (Gray 1918)

The two upper or more cephalic vertebrae are differentiated from the five lower in the following areas. The first cervical vertebra, C1, also termed atlas, is an osseous interface between the inferior portion of the skull at the occipital condyles and the introduction of the cervical spine (Figure 3). Unlike the remaining cervical vertebrae, C1 does not have a vertebral body. It is often described as a ring, bounded anteriorly by the anterior arch that extends laterally to the transverse processes. These regions contain superior and inferior articulating processes that engage the occipital condyles of the skull and the superior articular surface of the second cervical vertebra inferiorly. A posterior arch extends from the lateral masses of C1 posteriorly terminating at the posterior tubercle.
Inferior to C1 is C2, also called the axis, which exhibits a distinct morphology when compared to both C1 and the five lower cervical vertebrae. The axis resembles the lower vertebrae with a vertebral body, but unlike these vertebrae, an osseous element projects upwards from the vertebral body and terminates at the level of where a vertebral body would exist for C1 if it was present. This structure is typically referred to as the odontoid process or dens and it most notably serves as an ‘axis’ allowing the skull and C1 to rotate about the dens and thus the remainder of the cervical spine. The remaining morphology of C2 is similar to that of the lower vertebra with transverse processes, lateral masses with superior and inferior articular surfaces and the bifid process (Figure 4).
2.1.2 Ligaments, Membranes and Intervertebral Disc

The vertebrae of the cervical spine are connected to the base of the skull, adjacent vertebrae and more distal vertebrae through connective soft tissue structures that include ligaments, membranes and the intervertebral discs (IVD). While the bony vertebrae establish overall form for the cervical region, the soft tissue components allow for physiological movement of the cervical spine while establishing constraints against excessive movement. Putz (1992) provides an extensive review of the ligaments of the vertebral column.

The ligaments of the cervical spine typically fall into one of two categories; those that connect adjacent vertebrae only or those that connect both adjacent and multiple levels over their length. Running along the anterior face of each cervical vertebra and originating from the base of the skull is the anterior longitudinal ligament (ALL) (Figure 5). This structure is composed of crossed collagen fibers and is multi-layered connecting adjacent vertebrae by way of the innermost ligamentous fibers, while outer layers
connect more distal vertebrae to one another. A similar ligamentous structure, the posterior longitudinal ligament (PLL) runs along the posterior surface of the vertebral bodies along the length of the spine (Prestar and Putz 1982). Cephalically, this multi-layered ligament integrates with the tectorial membrane in the AA joint and while extending caudally connects adjacent vertebrae with the inner fibers through integration with the annulus fibrosis of the IVD and connection to the vertebral endplates. The inner fibers of the ALL and PLL have been reported to be continuous structures on the lateral aspects of the vertebral body (Hayashi et al. 1977).

**FIGURE 5:** Ligaments of the lower spine (Gray 1918)

In contrast to the ALL and PLL that connect adjacent and more distal vertebrae, a number of other ligaments connect only adjacent vertebrae. Laterally, each adjacent vertebra is connected to one another at the facet or zygapophyseal joints with the facet capsular ligaments. The posterior portion of adjacent vertebrae is connected through multiple ligamentous structures attached to the vertebral arch and posterior process and
include the interspinous, supraspinous and ligamenta flava (LF) (Figure 5). The interspinous and supraspinous ligaments connect adjacent spinous processes and are primarily composed of collagen fibers. The LF is primarily composed of elastic connective fibers; with collagen fibers interspersed, arranged longitudinally and tightly oriented. The LF connects along the entire length of the laminae of adjacent vertebral arches from the posterior aspect of an inferior vertebra to the anterior aspect of the superior lamina (Ramsey 1966).

**FIGURE 6:** Ligaments of the craniocervical junction (Gray 1918)

The AO and AA joints have additional ligamentous and membrane support structures not observed caudally (Figure 6). The alar ligaments connect bilaterally in an oblique orientation at the tip of the odontoid process and extend to the medial aspect of the occipital condyles. The apical ligament connects between the centerline of the tip of the odontoid and extends to the basion. The cruciform ligament extends axially and in the transverse plane forming a cross-like pattern posterior to the region of the odontoid...
process. The axially oriented section extends from the vertebral body of C2 and attaches to the base of the skull in the lower clivus. The transverse oriented portion, the transverse ligament (TL), attaches bilaterally to the medial boundary of the C1 lateral masses and provides a band of tissue confining the upper portion of the odontoid process in a region defined along its posterior face by the TL and along its anterior face by the posterior surface of the anterior arch of C1 (Figure 7). The accessory ligament connects bilaterally to the medial portion of atlas and to the base of the odontoid bilaterally. Aside from the apical ligament that is primarily composed of elastic fibers, the remaining ligaments are dominated by collagen fibers (Saldinger et al. 1990; Tubbs et al. 2000). The entirety of these ligamentous structures, along with the AOM and atlanto-axial membrane (AAM), in the AA and AO joint secure the UCS to the skull while also securing C2 and the odontoid relative to the skull and C1 (Figure 8).

![Figure 7: Transverse ligament as related to C1 and the odontoid process of C2 (Gray 1918)](image)

While the upper cervical spine is dominated by unique ligamentous structures not seen in the lower segments, from C2 through the remaining vertebrae, the IVD is the
connective soft tissue junction between adjacent vertebral bodies. The IVD follows the endplate geometry of the vertebral bodies and contains a central core, nucleus pulposus, surrounded by the annulus fibrosis. These structures are integrated together to form the overall structure of the IVD.

![Diagram of vertebral structures](image)

**FIGURE 8:** AOM and AAM of the craniocervical junction (Gray 1918)

### 2.2 Age-Related Terminology

The progression of the perinatal or fetal cervical spine to that of the neonate, through infancy, childhood and eventually a developmentally mature structure generally follows the overall growth of the body. As the child grows, the cervical spine and its components increase in size and change morphologically. Additionally, the rate of growth of the human body and its constitutive components varies between sexes, between individuals within the same population and between populations (Garn et al. 1966; Brodeur et al. 1981; Maresh 1970; Miller et al. 1991). Unlike the developmentally mature spine that is represented by a relatively static structure the immature spine is
continually changing throughout the growth process. The continual structural and material changes with age accompanied by the inherent lack of pediatric cadaveric tissue are complicating factors in assessing the biomechanical response of the cervical spine. In lieu of sufficiently large age-specific sample sizes at all ages of development, the biomechanical response of the developing cervical spine may be characterized by developing age-related trends.

Age may be defined in a number of ways, including chronological or numerical, skeletal and dental. While skeletal and dental age are important metrics for comparing something of unknown age to a known standard, chronological or numerical age are the primary metric associating time from birth to overall human growth. Chronological age is routinely used to track the growth of children based on height and weight and a number of studies have exhaustively characterized the growth of numerous anthropometric measurements of the growing child (Weber et al. 1985; Schneider et al. 1986; Snyder et al. 1977; Snyder et al. 1975; U.S. Centers for Disease Control & Prevention 2009). Historically, child anthropometric test devices (ATDs) were designed to roughly approximate in size, shape and response children of different chronological age from 6 months to 10 years.

The specimens of the current investigation are defined by chronological age and routinely sub-divided based on broader terms that encompass bands of chronological age from around birth to older childhood. The time frame prior to birth is defined as
the prenatal period while that after birth is the postnatal period. A more narrowed time frame that bounds birth on both sides is termed the perinatal period. Similar to subdivisions within the prenatal period, the postnatal period also is typically divided based on time scales post birth. The perinatal period associated with the time prior to and just after birth leads to the neonatal period that is typically defined as the first four weeks after birth (1 month). These periods are enclosed by a larger time scale termed the infant which extends from birth to the end of the first year chronologically. At the conclusion of the first chronological year of life numerous terms are used until adult life to define the individual and may include terms such as child, pediatric, juvenile and adolescence, although the latter two terms are typically associated with the period after childhood and before adulthood, or more specifically from the time of puberty to adult life (Ascádi and Nemeskéri 1970; Ferembach et al. 1980).

2.3 Developmental Cervical Spine Anatomy

The developing cervical spine is observed to change in overall size, composition and morphology from birth to maturity (Scheuer and Black 2004; Bailey 1952). An understanding of these differences with age is important as they help to describe the underlying structural changes in the cervical spine that are potential contributors to the biomechanical response and injury patterns of the neck.
2.3.1 Overall Neck Growth

The growth of the neck on the whole from birth to maturity has been quantified using metrics such as neck circumference and length (Nafiu et al. 2010; Weber et al. 1985; Schneider et al. 1986; Snyder et al. 1977; Snyder et al. 1975). These measurements, taken both externally and radiographically, often provide a means for scaling between ages. External measurements provide a bulk measure of the growth of both the cervical column and accompanying musculature. Weber and colleagues compiled the anthropometric observations of three studies conducted at the University of Michigan by Schneider et al. and Snyder et al. and found that neck circumference increased from 21.6 ± 2.3 to 28.7 ± 1.8 cm in children from 0-3 months up to 10 years (mean ± standard deviation). Snyder et al. 1977 reported neck circumference from children 2 years of age to youths of approximately 18 years and found that neck circumference increased from 23.8 ± 1.2 to 34.5 ± 3.3 cm. Prenatal or fetal neck circumference and neck area are reported to increase with gestational age (14 to 40 weeks) and are highly correlated to a number of anatomical measurements of the fetus including biparietal diameter and head circumference (Hata et al. 1988; Sherer et al. 2007).

2.3.2 Osteo-cartilaginous Development

The development of the osteo-cartilaginous structures of the cervical spine that eventually form the bony vertebrae is well documented (Ogden 1984; O’Rahilly et al. 1983; Bailey 1952). The presence and eventual obliteration of these cartilaginous regions
during growth is a potential contributor to differences in the biomechanical response of
the spine with age and is also commonly the site of injury, especially in the UCS. The
pediatric cervical spine is thought to reach a more adult morphology around eight years
of age (Ogden 1984; Bailey 1952). The development of the cervical vertebral column
over the age range of the current study is discussed to provide perspective of the
structural changes in relation to age and biomechanical response.

**FIGURE 9:** Typical perinatal C3-C7 (Scheuer and Black 2004)

The axial and lateral growth of the cervical spine occurs within the confines of
each vertebra through the interplay of bone ossification centers and adjoining
cartilaginous regions (O’Rahilly et al. 1983). The primary ossification centers of the
lower cervical spine (C3-C7) (Figure 9), located at the centrum (vertebral body) and two
neural arches (vertebral arches), appear in the prenatal period and are present at all ages
of the current study (Misawa et al. 1994; Chen et al. 1991; Ford et al. 1982; Bagnall et al.
1977). The cartilaginous structures of the individual vertebrae that allow for growth are
synchondroses composed of hyaline cartilage. These structures may also be termed a physis as they are the location allowing for overall growth of the vertebrae. Closure or obliteration of these synchondroses over the length of the entire vertebral column is reported to occur anywhere between 2 to 16 years (Zhang et al. 2009; Rajwani et al. 2002; Bailey 1952).

The five lower cervical vertebrae are similar in both morphology and the presence and distribution of synchondroses. Lateral to the primary ossification center associated with the vertebral body and adjacent to the pedicles are the neurocentral synchondroses. These regions connect the ‘centrum’ to the ‘neural’ arch. The two neural arches are bound together posteriorly at the point of the eventual spinous or bifid process by the posterior synchondrosis and typically fuse by 2 years of age (Scheuer and Black 2004). Initiation of and closure of the neurocentral synchondroses are reported to occur by 6 years of age and predominately in the 3 to 4 years of age range (Scheuer and Black 2004; Rajwani et al. 2002).

**FIGURE 10:** Atlas (C1) in perinatal development (Scheuer and Black 2004)
Atlas and axis unlike the lower five cervical vertebrae have distinctive cartilaginous patterns representative of their unique morphologies. Three primary ossification centers, the body and two neural arches, generally form the basis for the development of atlas although in some cases the body center is absent (Figure 10) (Bailey 1952). The posterior arch generally fuses by 3 to 5 years followed by fusion of the anterior central body at the neurocentral synchondroses by age 5 to 8 years (Scheuer and Black 2004; Bailey 1952). Axis, unlike atlas and the lower cervical vertebrae, has a more extensive developmental framework including four primary ossification centers and at least six commonly referenced synchondrotic joints (Figure 11) (Sherk et al. 1978; Ewald 1971; Bailey 1952). The four ossification centers include two neural arches, the centrum and the odontoid process (dens). The dens is initially composed of two separate ossification centers separated by the intradental synchondrosis that fuses by approximately the 28th gestational week (Bailey 1952). The cartilaginous plate between the dens and the body of C2 (subdental/dentocentral synchondrosis), between the lateral aspect of the dens and the neural arches (dentoneural synchondrosis), and between the body of C2 and the neural arches (neurocentral synchondrosis) do not ossify until a child is 3 to 6 years old (Scheuer and Black 2004; Bailey 1952). The neural arches close at the location of the posterior synchondrosis by the age of 2 or 4 years (Scheuer and Black 2004; Bailey 1952). The cartilaginous epiphysis at the tip of the odontoid, the chondrum terminale, may or may not appear as a secondary ossification center around 2 to 6 years
and typically fuses to the remainder of the odontoid process by 12 years (Scheuer and Black 2004; Bailey 1952).

**FIGURE 11:** Axis (C2) in perinatal development (Scheuer and Black 2004)

### 2.3.3 Ligamentous and IVD Development

The ligaments and intervertebral discs of the cervical spine begin to take form during the prenatal period and have some level of definition at all ages of the current sample, including at the youngest age of 20 weeks gestation. Limited literature is available on the characteristics of growth from around birth to maturity in these structures. However, it is clear that the ALL, PLL, LF, alar ligaments, transverse ligament and IVD are all present in some form for all of the cervical spines in the sample (Abe et al. 2011; Misawa et al. 1994; O’Rahilly et al. 1983; Hayashi et al. 1977; Ramsey 1966).

### 2.3.4 Morphological Development

In addition to the cervical spine changing in size and composition during the growth process, differences in morphology have also been observed that are typically
suggested to potentially increase the mobility of the spine. Changes in the orientation of the facets and general shape of the vertebral bodies have been reported throughout the cervical spine. The superior articular surface of atlas progresses from a more flat to curved contour with age that by approximately 8 years has reached ninety-percent of the curvature observed in adulthood (Hallgren et al. 2011). The facets of the C2-C7 vertebrae show variation in angle by level, while the joint orientation progresses from a more horizontal to vertical alignment up until approximately 10 years of age at which point minimal change is observed (Kasai et al. 1996). Kasai and colleagues also quantified a ‘sliding’ motion between adjacent vertebrae and found that as the joint orientation became more vertical with age the amount of sliding also decreased. The vertebral bodies of the lower cervical spine typically go through a time where they are wedge shaped, tapering anteriorly, but tend to lose this wedged appearance as growth occurs (Bailey 1952; Swischuk et al. 1993). Oval shaped vertebral bodies, as viewed in the sagittal plane, are persistent up to about 3 years of age at all levels of the cervical spine, while a more rectangular profile with a minimal rounded corner anteriorly is observed from birth, but tends to be more prevalent in the 4-7 year old age range (Swischuk et al. 1993). Normal anatomical variants including pseudosubluxation as well as the prevalence of spinal trauma in young children at the higher cervical levels is often predicated on the idea of increased instability at these ages based on morphology and
these work support at the very least the existence of these morphological differences in a quantitative form.
3. Background

3.1 Pediatric Cervical Spine Injury

3.1.1 General Epidemiology of Pediatric Cervical Spine Injury

Injuries to the pediatric spine and cervical spine account for approximately 3% and 1-2%, respectively, of pediatric trauma admissions (Cirak et al. 2004; Patel et al. 2001; Brown et al. 2001; Patrick et al. 2000; Dietrich et al. 1991). The most often injured location of the spine in children is the cervical region (Bilston and Brown 2007; Eleraky et al. 2000; McPhee 1981). Pediatric cervical spine injuries occur in approximately 1.9 – 9.5% of all cervical injuries (Hamilton and Myles 1992a). An incidence of 7.41 pediatric cervical spine injuries per 100,000 individuals per year has been suggested (McGrory et al. 1993). Older children are typically reported to sustain the greatest overall number of cervical injuries when compared to younger children, although the severity and likelihood of these severe injuries in the younger ages tends to be greater (Bilston and Brown 2007; McGrory et al. 1993).

Child cervical spine injury is associated with a high rate of mortality. Overall mortality rates for pediatric spinal trauma approach 4% (Cirak et al. 2004), while pediatric cervical spine related mortality rates have been reported between 8 and 32% (Dogan et al. 2006; Patel et al. 2001; Brown et al. 2001; Patrick et al. 2000; Nitecki and Moir 1994; Dietrich et al. 1991; Anderson and Schutt 1980). Fatalities are typically associated with upper level cervical injuries (Cirak et al. 2004; Nitecki and Moir 1994;
Moreover, cervical spine related fatalities are generally younger than those that survived with an associated cervical injury (Patel et al. 2001).

The relative incidence of upper versus lower cervical spine injuries in all children is debatable, while consensus exists on differences in level of injury based on age. Lower cervical spine injuries have been observed in up to 73% of all child cervical spine injuries, with injuries to C7 (22%) and C2 (15%) observed most often (ages 1 to 17), while others have noted that 68% of child cervical spine injuries at all ages occurred in the upper cervical spine (Brown et al. 2001; Nitecki and Moir 1994). However, delineations in injury pattern within children on the whole have been observed with increased upper cervical injuries in children less than 7 to 13 years compared with older children, in which lower cervical injuries are more prevalent (Bilston and Brown 2007; Patel et al. 2001; Nitecki and Moir 1994; McGrory et al. 1993; Sherk et al. 1978; Klimo et al. 2007; Dietrich et al. 1991; Mann and Dodds 1993). Upper cervical injuries in younger children are estimated to be as high as 90% (68-90%) of the cervical injuries observed in this age group (Brown et al. 2001; Nitecki and Moir 1994; McGrory et al. 1993). Moreover, ligamentous injuries, dislocations, subluxation, severe or complete spinal cord injury are characteristic of the younger ages compared to fracture associated injuries in the older ages (Klimo et al. 2007; Nitecki and Moir 1994; McGrory et al. 1993; Mann and Dodds 1993).
Differences in cervical spinal injury patterns between younger and older children are attributed most often to anatomical and material differences between the immature developing child and the physically more mature older child (Myers and Winkelstein 1995; Fuchs et al. 1989; McGrory et al. 1993; Klimo et al. 2007). The ligamentous supporting structure of the younger neck is often characterized as having increased ‘laxity’ in comparison to older children. The facet joints are described as shallow or flat from the base of the skull to C7, while vertebral bodies are described as wedged anteriorly, allowing for increased mobility in both rotation and translation between adjacent vertebrae. The ring apophyses located on the superior and inferior surfaces of the vertebral bodies and the location of later appearing secondary ossification centers are postulated as a potential site of weakness. The head size of younger children relative to overall body proportions, in comparison to older children, is larger introducing decreased stability of the head-neck structure. The underdeveloped musculature of younger children may also limit the protective capacity of the overall head-neck structure to load, while also allowing excessive head and neck motion. While, the fulcrum for flexion and extension of the neck is hypothesized to change with age with changes in overall body proportions, progressively moving more caudally. The fulcrum in the infant is suggested to be around C2-C3, C3-C4 by age 5, C4-C5 at 10 years and C5-C6 by 15 years of age. These elements are often suggested as the underlying physical and material differences that result in different injury patterns, however, aside from the
anthropomorphically and morphologically observed differences much of the structural and material response differences have yet to be substantiated.

Spinal cord injury, head trauma and the unique developmental characteristics of the immature spine and their association with pediatric spinal injury is an important consideration in the discussion on morbidity, mortality and the accurate identification of the true extent of pediatric spinal injury. Pediatric spinal cord injury is routinely associated with spinal structural injuries, with rates of association between 5.6 and 38% (Bilston and Brown 2007; Dogan et al. 2006; Fassett et al. 2006; Mann and Dodds 1993; McGrory et al. 1993; McPhee 1981). Spinal cord injury without radiographic abnormality (SCIWORA) has been reported over a wide range from 1 to 50% of child spinal injuries (Bilston and Brown 2007; Cirak et al. 2004; Pang and Wilberger 1982). Younger children have a greater propensity for these injuries and higher mortality rate; while older children are less likely than younger children to fully recover neurologically (Mann and Dodds 1993; McGrory 1993; Pang and Wilberger 1982; McPhee 1981). These injuries are often associated with the upper cervical spine and mortality rates range from 25 to 53% depending on the level of cervical injury and extent of neurological sequela (Patel et al. 2001; Farley et al. 1992).

Head trauma is routinely associated with pediatric spinal injury with association rates ranging from 24 to 37% (Dogan et al. 2006; Cirak et al. 2004; Mann and Dodds 1993). Moreover, head trauma associated with spinal injury has been observed in over
one half of pediatric spinal injury cases that are motor vehicle crash (MVC) related (Brown et al. 2001; McPhee 1981). The cervical spine is often observed as the site of spinal injury in combination with head trauma (Dogan et al. 2006; Dietrich et al. 1991). Multiple studies have noted that greater than 90% of those with combined spinal and craniocerebral injuries were fatally injured (Cirak et al. 2004; Brown et al. 2001). Brown et al. (2001) observed a strong correlation between pediatric cervical spine injury and closed head injuries (CHI) with a 38% association rate and 49% mortality rate. This was even more pronounced in the younger ages with a 66% association and 52% mortality rate.

Identification of cervical spine injury in children can be complicated as a result of the unique developmental characteristics of the immature spine. Most notably, the numerous cartilaginous growth regions and the morphology of the vertebrae coupled with the apparent laxity of the supporting ligamentous structures create an environment where diagnosis of fractures, traumatic subluxation and dislocation and spinal cord injury may be more difficult to assess as compared to their adult counterparts. Synchondroses may be mistaken for fractures (Smith et al. 1993; Swischuk et al. 1979), pseudo-subluxation (Shaw et al. 1999; Papavasiliou 1978; Townsend and Rowe 1952) and spread (Suss et al. 1983) may be mistaken for traumatic subluxation and dislocations and normal radiographs void of osseous abnormalities may hide the fact that traumatic spinal cord injury associated with spontaneous spinal reduction has occurred.
High mortality rates, strong association with head and spinal cord injuries and unique anatomical characteristics are critical characteristics associated with pediatric spinal trauma that further the challenge of quantifying the true extent of these injuries. Fatalities involving head trauma may lead to underreporting of underlying spinal injuries. Additionally, there exists a vast nomenclature dealing with spinal related injuries and the reporting of these injuries. Spinal injury, spinal cord injury, cervical spine injury, spinal related injury, upper and lower cervical injury and SCIWORA are all routinely used when discussing injuries to the neck of children and in some instances are used interchangeably. Moreover, depending on the inclusion criteria and definitions utilized a given sample of pediatric trauma patients may yield differing spinal injury rates. One notable example is the variability in the definition of the upper cervical spine (UCS). Six different definitions of UCS including occiput to C4 (Cirak et al. 2004), occiput to C3 (McGrory et al. 1993), C1-C4 (Brown et al. 2001), C1-C3 (Patrick et al. 2000), occiput to C2 (Finch and Barnes 1998) and no definition provided (Eleraky et al. 2000) were used in six different retrospective reviews of pediatric spine injury. For these reasons among others, the true quantitative impact of pediatric spine and specifically cervical spine injury may not be as clearly delineated as other injury patterns.

3.1.2 Typical Pediatric Cervical Spine Injuries

A number of comprehensive clinical reviews on pediatric spine trauma have delineated the most common injuries afflicting the immature spine (Reilly 2007; McPhee
Clinically relevant pediatric spinal-associated injuries include atlanto-occipital dislocations (AOD/OAD) (Collalto et al. 1986; El-Khoury et al. 1984), occipital condyle fractures (Caroli et al. 2005; Capuano et al. 2004; Momjian et al. 2003; Wasserberg and Bartlett 1995), atlas (C1) bony and synchondroses fractures (Vilela and Peterson 2009; Thakar et al. 2005; Suss et al. 1983; Marlin et al. 1983; Jefferson 1920; Reilly and Leung 2005; Kapoor et al. 2004; Judd et al. 2000; Mikawa et al. 1987), atlanto-axial instability (Grogono 1954) or complete AA dislocation (AAD), fractures of axis, including odontoid fractures (Sanderson and Houten 2002; Blauth et al. 1996; Schippers et al. 1996; Sherburn et al. 1996; Seimon 1977), fractures of the pedicular region of axis (Hangman’s type) (Grisoni et al. 2003; Pizzutillo et al. 1986), dislocations (Papavasiliou 1978) and subaxial injuries as well as thoracic and lumbar related spine injuries. Cervical cord injuries associated with birth (Goetz 2010; Jones and Hensinger 1981; Abroms et al. 1973; Leventhal 1960), non-accidental trauma (Thomas et al. 1995) and SCIWORA have also been reported (Pang and Wilberger 1982).

Specifically, many of these injuries observed generally within the pediatric population are also seen routinely in MVC-related incidents. Axis-related (C2) injuries (Mann and Dodds 1993; Sumchai and Sternbach 1991; Gaufin and Goodman 1975; Hadley et al. 1995; Conry and Hall 1987; Pizzutillo et al. 1986; Bodenheim et al. 1992; Weiss and Kaufman 1973; Vining et al. 1992; Papavasiliou 1978; Marshall et al. 1998; Willis et al. 1996), including odontoid synchondrosis fractures (Panczykowski et al. 2010;
Fassett et al. 2006; Garton et al. 2002; Sherk et al. 1978; Blauth et al. 1996; Schippers et al. 1996; Claper and Pailthorpe 1995; Steele and Aks 1995; Keller and Mosdal 1990; Fuchs et al. 1989; Conry and Hall 1987; Bhattacharyya 1974; Ewald 1971; Anderson and D’Alonzo 1974), similar to the traditional Type I-III fractures observed in adults (Yoganandan and Pintar 2005; Adams 1992b; Anderson and D’Alonzo 1974; Skold 1973; Schatzker et al. 1971) are among the more commonly observed injuries. Traumatic dislocation type injuries of the upper cervical spine including AOD (Salinsky et al. 2007; Saveika and Thorogood 2006; Violas et al. 2006; Vera et al. 2005; Angel and Ehlers 2001; Houle et al. 2001; Bailey et al. 2000; McCaffrey et al. 1999; Cooper et al. 1998; Morrison et al. 1998; Bulas et al. 1993; Mann and Dodds 1993; Farley et al. 1992; Fuchs et al. 1989; Traynelis et al. 1986; Dietrich et al. 1991) and AAD (Hammerstein et al. 2007; Violas et al. 2006; Cooper et al. 1998; Swoboda et al. 1995; Adams 1992b; Floman et al. 1991; El-Khoury et al. 1984) have also been reported (Bulas et al. 1993; Koller et al. 2006; Guigui et al. 1995; Adams 1992a and 1992b; Maiman and Cusick 1982).

3.2 Significance of MVCs to Pediatric Mortality, Injury, Occupant Protection and Mitigation of Pediatric Cervical Spine Injury

MVCs are the leading cause of death for children from 3 to 14 years of age (U.S. Department of Transportation 2009c). The leading cause of death from 1999 to 2006 for teenagers, 12 to 19 years, was unintentional accidents accounting for 48% of deaths. The majority of these fatalities, 73% were associated with MVCs (U.S. Centers for Disease Control and Prevention 2010). An average of four children less than 14 years of age is
fatally injured in MVCs daily as of 2009. MVC related pediatric spinal injury has been reported as the cause of injury in 21 to 46% of pediatric spinal injury cases (Bilston and Brown 2007; Cirak et al. 2004; Mann and Dodds 1993; McPhee 1981; Anderson and Schutt 1980). Moreover, spinal injuries resulting from MVC related incidents have been reported to result in serious injury 50% of the time (Bilston and Brown 2007). The most common causes of cervical spine injury in children are typically MVC related or falls (Dogan et al. 2006; Patel et al. 2001; Brown et al. 2001; Eleraky et al. 2000; Patrick et al. 2000; McGrory et al. 1993; Dietrich et al. 1991; Sherk et al. 1978). For children less than 8 years of age the leading cause of cervical spine injuries are MVC related mechanisms (Patel et al. 2001; Nitecki and Moir 1994). The leading cause of immediate and complete spinal cord injury at the cervical level has been reported to be MVC related (Farley et al. 1992). Dietrich et al. (1991) reported that 88% of the cases reviewed in their study on pediatric cervical spine injury were fatally injured as a result of an MVC related incident, while others noted that 95% of those fatally injured with associated cervical spine injury were MVC-related, while the mortality rate for MVC-occupants with a cervical spine injury was 25% of all fatalities (Brown et al. 2001). Whether the site of injury is the overall spine or more specifically the cervical region or neurological deficits associated with cord compromise, MVC association is prominent in the discussion.

The economic and societal costs associated with childhood morbidity and long-term disability is historically a critical component in defining the impact of MVCs on
society on the whole (Hartunian et al. 1980; Agran et al. 1990). In 2005, the total lifetime costs associated with MVC-related fatal and non-fatal injuries for all ages were estimated at $99 billion and included approximately $21 billion in costs for children from birth to 19 years (Naumann et al. 2010). Approximately $15 of the $21 billion in estimated lifetime costs was associated with pediatric MVC-occupant injuries. Agran and colleagues reported that the years of potential life lost as a result of pediatric mortality data from 1987 was approximately 500,000 years, while over 600,000 injuries were incurred during the same year for all pediatric ages. Along with monetary costs and years lost, the effects of pediatric trauma have also been observed in the form of detrimental emotional and behavioral outcomes for both the victims and their relatives (Harris et al. 1989). Pediatric mortality, morbidity and the economic and societal costs associated with MVCs stress the importance of a continued focus on interventions that can mitigate these costs in the future.

Occupant protection and specifically restraint use and air bags have played a critical role in the reduction in both fatality and injuries rates (U.S. Department of Transportation 2009c, 2012). While restraint usage continues to rise throughout the US, helped in part by state associated primary enforcement laws, restraint usage nationally remains below 90%, increasing from 58 to 84% from 1994 to 2011 (U.S. Department of Transportation 2011b). In the infant category (less than 1 year), child restraint usage is estimated to reduce the risk of fatal injury by 71%, while a 54% reduction is observed for
the 1 to 4 year old age range (U.S. Department of Transportation 2009d). Child restraint usage, since 2002, in children from birth to 7 years has been constant and estimated to be 87% (U.S. Department of Transportation 2009b). Infants were the most often restrained (99%) children in 2008 with decreasing restraint usage with increasing age (1-3 YO, 92%; 4-7 YO, 89%; 8-12 YO, 85%; 2008 National Survey of the Use of Booster Seats (NSUBS)).

Proper child restraint usage based on height, weight and age of the child, along with overall usage numbers, is an important consideration in progressing child occupant protection. While infant restraint use is high, roughly 21% of children less than 1 year of age or under 20 lbs are not restrained in a rear-facing restraint or rear-facing child safety seat [RFCSS] (U.S. Department of Transportation 2009a). In the 20 to 40 lb weight group that is typically associated with forward-facing child seats, 44% of these children were not restrained in this type of restraint. Approximately 30% had graduated to either a booster seat or adult restraint with an additional 9% unrestrained. For children that were under 12 years old and between 37 and 53 inches, 49% were using adult restraints or unrestrained as opposed to being in a child booster seat. These data are indicative of premature graduation of infants, into forward-facing child restraints, and younger children, from forward-facing child restraints to booster seats and then to adult three-point restraints. The possible implications of inertially loaded cervical spine injury as a result of the premature progression of younger occupants into forward facing restraint systems is of interest based on field data associated with restrained younger occupants.
Odontoid fractures are one injury that has been documented as occurring to restrained younger aged children from 16 months to 3 years of age in forward facing systems in frontal collisions (Panczykowski et al. 2010; Garton et al. 2002; Blauth et al. 1996; Clasper and Pailthorpe 1995; Keller and Mosdal 1990). AOD and AAD type injuries have also been described in restrained children where head contact was not indicated (Hammerstein et al. 2007; Houle et al. 2001). While it should be stressed that current occupant protection is overwhelmingly a fundamental and critical component in saving lives and preventing injuries, these benefits do not eliminate the possibility that the unique biomechanics of the developing child accompanied by improper restraint usage may place the cervical spine at risk of injury due to distractive or tensile forces, potentially as a result of inertial loading of the child’s head.

Age appropriate seating position, similar to proper restraint usage, is also an important element in child occupant protection. The vast majority of infants (99%) and children 1 to 3 years of age (98%) in child restraints were placed in the rear seat. However, similar levels of rear seating were not observed with older children from 4 to 7 years of age (88% rear placement). In 2008, although the vast majority of children were in the rear seat and restrained, 2% of child passengers in RFCSS and 14% of unrestrained children from birth to 7 years were in the front passenger seat (U.S. Department of Transportation 2009b). Improper seating position may have a number of implications to occupant protection, not the least of which is possible out-of-position (OOP) air bag
interaction. During the late 1980’s and throughout the 1990’s, a small number of cervical spine injuries and fatalities associated with low-to-moderate severity frontal collisions in which air-bag restraint systems deployed and interacted with an OOP victim began to surface in the literature. While accounting for a small number of the overall air bag deployments, these injuries and fatalities were alarming in that children and small adults, specifically female drivers, appeared to be most affected, prompting extensive review and investigation of the biomechanics and circumstances of these injuries (Nightingale et al. 1998; Kleinberger and Summers 1997; Winston and Reed 1996). Older child occupants (not including those in RFCSS) sustained head or cervical injuries in 98% of the cases reviewed compared to just 38% of the adult cases (Nightingale et al. 1998). Moreover, 65% of the children experienced some form of cervical spine injury, while 66% of those with this type of injury experienced the lesion in the upper cervical spine. The mortality rate of these injuries was quite high, with 76% of the child cases resulting in death. AOD and C1/C2 subluxation were commonly observed injuries and have been consistently detailed in case reports as air bag related and have been associated with children from 7 days to 17 years (Saveika and Thorogood 2006; Angel and Ehlers 2001; Bailey et al. 2000; McCaffery et al. 1999; Marshall et al. 1998; Morrison et al. 1998; Cooper et al. 1998; Willis et al. 1996; Perez and Palmatier 1996) and adults (Gossman et al. 1999; Maxeiner and Hahn 1997; Blacksin 1993).
While the impact of these injuries and fatalities cannot be overstated, it is important to keep the scope of these injuries in perspective with the benefits afforded both children and adults by occupant protection. Kleinberger and Summers (1997) note that just 4 fatalities occurred for every 100,000 air bag deployments and that for the 63 fatalities associated with an air bag deployment that they reviewed there were an estimated 1,900 lives saved during the same period. While, proliferation of air bags into the fleet have occurred since these early reports during the 1990’s, OOP related low-to-moderate severity injuries and fatalities to occupants have decreased significantly over the years (U.S. Department of Transportation 2009e). These decreases are commensurate with significant educational efforts resulting in age appropriate rear seating of infants and children, but as recent data show, while the youngest restrained occupants are almost exclusively properly positioned in the rear seats, over 10% of children from 4 to 7 years and greater than 10% of unrestrained children from birth to 7 years are still found in the front seat of vehicles.

While improvements in occupant protection, restraint laws and educational efforts have played a critical role in reducing the number of MVC related fatalities and injuries, additional research is needed to continue these positive trends. Within the child restraint appropriate age range it is interesting to note that differences in the effectiveness of these systems in reducing fatalities is observed between the under one year and one to four year age group, which corresponds to different child restraint
systems. This gap in effectiveness may indicate that improvements in older child restraints and improved understanding of the biomechanics of injury in these older ages compared to infants are important steps in progressing overall child occupant protection.

3.3 Pediatric Neck Biomechanics

3.3.1 Animal and Surrogate Juvenile Cervical Spine Biomechanics

Early efforts to better characterize the biomechanical response of the pediatric human to injurious environments have relied heavily on the use of animal surrogates. Although limitations exist in the use of non-human tissue, in lieu of actual pediatric human cadavers, animal surrogates filled the void and allowed for extensive progress in characterizing pediatric neck biomechanics. These studies have focused on defining the biomechanical response of the immature neck, guiding anthropometric test device (ATD) development, establishing injury criteria and tolerance levels and evaluating the biofidelity of child ATDs and automotive restraint and occupant protection systems.

During the early eighties, General Motors and Ford Corporation researchers using living juvenile porcine tissue as a surrogate for the three-year-old child investigated interactions with a passenger deployed airbag system under development. These investigations compared airbag inflicted injuries to the porcine tissue to the biomechanical response of a 3 YO child dummy (ATD) through paired testing (Mertz and Weber 1982; Mertz et al. 1982; Wolanin et al. 1982; Prasad and Daniel 1984). These
data were critical first steps in relating injuries of varying severity as defined by the Abbreviated Injury Scale (AIS) to quantitative responses of a child ATD and thus providing an outline of injury metrics for the child neck. While Mertz and Weber (1982) estimated the probable occurrence of severe injury in piglets and then inferred similar injuries in a 3 YO child by combining the qualitative injury experience of the piglets to the quantitative loading response of the ATD, Prasad and Daniel (1984) provided insight into response characteristics and their relation to possible severe injury in the piglet without discussion on a probability derived metric. Noted findings included that severe to fatal neck injuries were typically associated with increased average upper spine accelerations with increasing duration. Increased neck loads were observed with increasing peak upper spine accelerations. But most important was the inference that axial load alone may not predict neck injury as well as a metric that combines both axial load and flexion-extension bending moments.

A number of studies have sought to address the changes in biomechanical response and tolerance with age of the cervical spine and have focused on the use of animal models in lieu of pediatric human tissue (Pintar et al. 2000; Hilker et al. 2002; Ching et al. 2001; Nuckley and Ching 2006; Elias et al. 2006). These studies using goat (Pintar et al. 2000; Hilker et al. 2002) and baboon (Ching et al. 2001; Nuckley and Ching 2006; Elias et al. 2006) models investigated bending and tension responses. Both investigative efforts assigned human-equivalent ages for their animal surrogates based
on evaluation of the skeletal maturity of the cervical vertebrae and depending on the
study evaluated the biomechanical response of animals from 1 to 26 years (human-
equivalent). Age groups were selected based on current child ATDs with the intent of
proposing scaling relationships suitable for the creation of injury assessment reference
values (IARVs) to the various ages. Functional spinal units (FSU) of the upper and lower
cervical spine were evaluated in both institutional efforts. Tensile and bending stiffness
and load-to-failure in tension increased with age for all segments in both animal studies,
while differences in stiffness and load-to-failure were observed between spinal units at a
given age. Scaling relationships for each of the biomechanical parameters was
determined by normalizing by the adult animal responses. These scaling ratios
provided initial estimates for tolerance and stiffness of the child neck.

3.3.2 Human Pediatric Cervical Spine Biomechanics

The study of the biomechanics of the cervical spine in the pediatric human is
bounded by data obtained from two distinct physiologic models. These include
cadaveric, post mortem human subjects (PMHS) models and living children or volunteer
models. Cadaveric based models have included various levels of anatomical
complexity; whole body cadavers, osteoligamentous head-neck complexes and
osteoligamentous spinal motion segments. These cadaveric models have provided an
important platform for investigation of both functional biomechanics, without muscle
involvement, of the cervical spine and tolerance and injury related biomechanics that are
unattainable from volunteer based models. Volunteer models have provided important information on the passive and active response of the cervical spine, both osteoligamentous and muscular, in a low-load environment.

3.3.2.1 Post Mortem Human Subject (Cadaveric) Cervical Spine Biomechanics

Cadaveric pediatric human biomechanical studies associated with furthering our understanding of cervical spine biomechanics are limited in the peer-reviewed literature to a total of five institutional studies (Duncan 1874; Kallieris et al. 1976; Wismans et al. 1979; Dejeammes et al. 1984; Ouyang et al. 2005). Each of these studies varies in the biomechanical responses, the sample sizes and the level of anatomical complexity investigated. Duncan, Kallieris, Wismans and Dejeammes provide whole body data, while Ouyang provides cervical spine osteoligamentous response data. Kallieris, Wismans and Dejeammes investigated whole body kinematics in high speed decelerations applicable to child restraint development with limited quantitative cervical spine response data. Duncan focused on the tensile response of the whole body cadaver of fetal and neonate tissue, while Ouyang examined tensile and bending responses from 2 to 12 years of age. Duncan examined whole spine response, while Ouyang examined whole osteoligamentous cervical spine response. Kallieris and Duncan studied the response of four cadavers each, Wismans and Dejeammes studied even smaller sample sizes and Ouyang examined the response of 10 cadavers. Each of these studies has
unique characteristics that provide differing perspectives and insights into the biomechanical response of the cervical spine from birth to adulthood.

To date the only whole body pediatric human cadaveric work associated with progressing our understanding of occupant protection in frontal collisions and cervical neck injury mechanisms are the work from five studies at three institutions that encompass a total of nine cadaveric subjects (Kallieris et al. 1976; Wismans et al. 1979). The largest of these studies was conducted by Kallieris et al. (1976) with a total of four pediatric cadavers from 2.5 to 11 years. These three institutional studies provided kinematic comparisons between child ATDs and cadavers, while also identifying minimal ligamentous injuries in the upper cervical spine that are characteristic of injuries to the pediatric population.

Duncan (1874) was the first to investigate the structural tolerance of the pediatric human neck. Duncan tested five specimens, including four stillborn infants and one infant that died two weeks after birth. The cadavers were fully intact prior to testing with all soft tissue retained including the musculature and outer skin layers. The tensile failure load of the four stillborn infants was 471 ± 79 N (average ± standard deviation). The two-week old infant had a tensile strength of 654 N. Failures in Duncan’s sample were distributed throughout the lower cervical region from the C3-C4 joint to the C6-C7 joint, in contrast to epidemiological observations of predominately upper cervical spine injuries in the neonate.
Ouyang et al. (2005) investigated the structural responses of whole pediatric PMHS cervical spines in sagittal plane bending and in tensile loading to failure. Ten cadaveric specimens of mixed gender ranging in age from two to twelve years were used. All testing, including failure tests, were conducted on whole cervical spines. Maximum moments applied during the bending protocol ranged from –2.4 Nm (extension) to 2.4 Nm (flexion) and followed methodology similar to previous studies (Camacho et al. 1997; Nightingale et al. 2002). The tension protocol was similar to the experiments by Van Ee et al. (2000b). The mean bending stiffness of all the specimens was 0.041 N-m/degree. The average tensile failure load was 726 ± 171 N and the displacement at failure was reported to be 20.2 ± 3.2 mm. The average tensile stiffness of the whole cervical spine specimens was 35 ± 6 N/mm. Load-to-failure was observed to increase with age \( F = a \ln (x+1) + b \), while no clear trends were observed in the displacement at failure. The majority of the failures occurred in the lower cervical spine, similar to the observations of Duncan (1874), and were end-plate fractures occurring at the site of fixation.

3.3.2.2 Fetal and Infant Autopsy Findings

A small number of studies associated with pediatric autopsy reports; all of which lack a clear definition of the structures tested or loads applied, have been published that provide minimal information on the behavior of the human spine to externally applied loads (Leventhal 1960; Abroms et al. 1973; Aufermaur 1974; Ewald 1971). While
lacking in the quantitative rigor and description of the information reported these reports do provide limited data in a scientific area where little data exists.

3.3.2.3 Volunteer Human Cervical Spine Biomechanics

Child volunteer testing has been limited to low level loading environments and numerous range-of-motion (ROM) studies. Although these data are limited in progressing our understanding of cervical spine injury and tolerance they do provide insight into the general kinematics of the cervical spine in children.

Arbogast et al. (2009) investigated the kinematic responses of the head and spine of children, 6 to 14 years, and adults in low-speed non-injurious frontal sled tests; while Balasubramanian et al. (2009) analyzed a sub-set of ages from 9-12 years of the Arbogast study. The kinematic response of the volunteers was quantified by tracking external markers positioned at landmarks on the head and spine. These data provide an important component in furthering our understanding of the response of the child head and spine in low-severity frontal impact and establishes kinematic corridors for child response to low-speed decelerations. Combined with pediatric computational models these data sets provide biofidelic bounds for assessing the biofidelity of both computational and physical models.

Child cervical spine bending ROM have been investigated with volunteers from three years to adult ages (Arbogast et al. 2007; Lewandowski and Szulc 2003; Sforza et al. 2002; Feipel et al. 1999; Youdas et al. 1992). Quantifying the ROM of child and
adolescent volunteers have typically fallen into two categories, active (Arbogast et al. 2007; Sforza et al. 2002; Fiepel et al. 1999; Youdas et al. 1992) and passive (Seacrist et al. 2009) ROM studies. Active ROM studies quantified bending as defined by the user ‘actively’ flexing their neck to a maximum tolerable level, while passive ROM studies quantified bending where the subject allowed their neck to flex without effort on their part. These studies provide ROM boundaries of the entire human cervical structure including the effects of musculature, but do not provide quantitative loading data to accompany the ROM observed. Active cervical spine flexion ROM was observed to increase with age (Arbogast et al. 2007), while others have reported reductions in flexion and extension ROM with age in children (Lewandowski and Szulc 2003; Sforza et al. 2002; Feipel et al. 1999; Youdas et al. 1992). Active cervical spine flexion ROM has been observed to be less than extension ROM over all ages, although these differences were observed to lessen with increasing age (Arbogast et al. 2007; Sforza et al. 2002; Youdas et al. 1992); while other studies have indicated that flexion ROM was greater than extension ROM (Feipel et al. 1999). Passive cervical spine flexion was observed to be greater in children compared to adults (Seacrist et al. 2009). Differences in trends with age with ROM with different studies are in part a direct result of differences in measurement techniques and establishment of consistent initial or neutral positions.
3.3.3 Child ATD Neck Development, Biofidelity and Injury Metrics

Due to the lack of human pediatric cervical tissue over the years, the biofidelity and injury metrics of child ATDs have relied heavily on adult cadaveric and animal responses that were subsequently scaled based on geometrical and material property relationships to approximate the response of the child (Mertz and Patrick 1967, 1971). Irwin and Mertz (1997) outlined the development and basis for the biofidelic impact response of the CRABI (50th percentile 6, 12, 18 M) series of infant dummies and the Hybrid III (50th percentile 3 and 6 YO) child dummies. The 3 YO Hybrid III and CRABI necks were based on the GM modified VIP-3C (Dummy 2 Neck) neck detailed in Wolanin et al. (1982). The biofidelity corridors defining neck performance were based on the 50th percentile mid-size adult male corridors developed for the Hybrid III dummy (Mertz and Patrick 1967, 1971). These corridors were established based on scaling procedures that considered the geometric and material property differences between the adult and child (McPherson and Kriewall 1980; Hubbard 1971; Irwin and Mertz 1997; Mertz et al. 1989). While this methodology provided a structured basis to develop corridors potentially representative of the child neck, it is important to consider that none of the biomechanical response data is based on child neck structural data.

Quantifying injury risk for the child population as a result of MVC related mechanisms and then relating this risk to observed injury and quantifiable loads and moments observed in the mechanical surrogates is of similar importance to designing a
biofidelic neck. Mertz and colleagues created injury risk curves and Injury Assessment Reference Values (IARVs) for use with the child dummies and adult family of Hybrid III dummies based on the original injury risk curves established for the 3 YO ATD (GM Dummy Neck 2) during the Mertz and Weber (1982) piglet and child ATD study (Mertz et al. 1997; Mertz and Prasad 2000; Mertz et al. 2003). Progression of the initial risk curves were undertaken based on combining the experience of the two manufacturer associated piglet studies (Mertz and Weber 1982; Mertz et al. 1982; Prasad and Daniel 1984). Neck tension, extension moment and a combination of tension and extension moment ($N_j$) measured at the occipital condyles of the 3 YO ATD were proposed as indicators for neck injury severity. Both studies were in agreement that neck tension was the best indicator of AIS $\geq 3$ neck injury with no AIS $\geq 3$ neck injury occurring in any of the piglets below a neck tension load of 1160 N.

IARVs and injury risk curves for the remaining child and adult ATDs were developed from the 3 YO ATD response using scaling methods. Neck circumference was used to characterize neck size for geometric scaling (E-1), while human calcaneal tendon failure data was used for material property scaling (E-2) (Melvin 1995; Yamada 1970).

\[
\text{Geometric Neck Scale Factors (tolerance)} = \lambda_c = \frac{\text{Neck Circumference (child or adult)}}{\text{Neck Circumference (3 YO)}} \quad (E-1)
\]

\[
\text{Material Neck Scale Factors (tolerance)} = \lambda_\sigma = \frac{\text{Failure Stress (child or adult)}}{\text{Failure Stress (3 YO)}} \quad (E-2)
\]
Neck tension and moment scale factors based on a relationship between the failure stress level and geometry provided the ability to relate the 3 YO injury response to the response of interest (E-3 and E-4).

\[
\lambda_T = \lambda_\sigma \lambda_C^2 \tag{E-3}
\]

\[
\lambda_M = \lambda_\sigma \lambda_C^3 \tag{E-4}
\]

The authors proposed a similar injury criterion \( N_i \) based on the combined effects of tension and extension, to represent the AOD type injuries observed in the piglet studies. Mertz and Prasad (2000) revisited the injury risk curves first proposed in Mertz and Weber (1982) and then updated in Mertz et al. (1997) by updating the percent of muscle tone and associated variations in ligamentous failure stress with age, which was initially considered to be independent of age in the formulations put forth initially. The updated values for peak neck tension, peak extension moment and combined tension and extension moment at the 3, 5 and 2% AIS\( \geq 3 \) risk levels respectively were the criteria set forth for use with Federal Motor Vehicle Safety Standard (FMVSS) 208 for OOP occupant testing of airbag restraint systems.

The impact of cervical spine injuries on the pediatric population, coupled with the paucity of pediatric human cervical spine biomechanical response data is the catalyst for the investigation detailed in this dissertation. Developing relationships that describe the tensile and bending response of the cervical spine from birth to young adulthood is a fundamental step towards the future of child occupant protection.
4. An Apparatus for Tensile and Bending Tests of Perinatal, Neonatal, Pediatric and Adult Cadaver Osteoligamentous Cervical Spines


4.1 Abstract

Investigations of biomechanical properties of pediatric cadaver cervical spines subjected to tensile or bending modes of loading are generally limited by a lack of available tissue and limiting sample sizes; both per age and across age ranges. It is therefore important to develop fixation techniques capable of testing individual cadavers in multiple modes of loading to obtain more biomechanical data per subject. In this study, an experimental apparatus and fixation methodology was developed to accommodate cadaver osteoligamentous head-neck complexes from around birth (perinate) to full maturation (adult) [cervical length: 2.5 – 12.5 cm; head breadth: 6 – 15 cm; head length: 6 – 19 cm] and sequentially test the whole cervical spine in tension, the upper cervical spine in bending and the upper cervical spine in tension. The experimental apparatus and fixation methodology provided a rigid casting of the head during testing and did not compromise the skull. Further testing of the intact skull and sub-cranial material was made available due to the design of the apparatus and fixation
techniques utilized during spinal testing. The stiffness of the experimental apparatus and fixation technique are reported to better characterize cervical spine stiffness data obtained from the apparatus. The apparatus and fixation technique stiffness was 1986 N/mm. This experimental system provides a stiff and consistent platform for biomechanical testing across a broad age range and under multiple modes of loading.

4.2 Introduction

Determining biomechanical properties of adult cadaver cervical spines under multiple loading modes can be challenging. This is exacerbated when testing pediatric cadavers. While adult cohorts typically consist of a sample of similar dimensions, pediatric testing may involve a large range of head and cervical spine sizes depending on the ages investigated. This variability, not typically observed in adult studies, must be accounted for in the test design. Furthermore, adult cadaver spines are available in sufficient numbers that test designs can more frequently focus on single modes of loading. While some adult studies have focused on test designs capable of quantifying multiple modes of loading, in a sequential fashion, on a single specimen (Goel et al. 1988; Moroney et al. 1988), many have focused on a single mode of loading (McElhaney et al. 1983; Nightingale et al. 2002; Van Ee et al. 2000b; Yoganandan et al. 1996). Unlike adults, pediatric cadaver spines are scarce; therefore it is essential to maximize the biomechanical data obtained from each specimen through innovative experimental apparatus and fixation methodology.
Few biomechanical studies of the pediatric human head-neck complex exist (Duncan 1874; Ouyang et al. 2005). Duncan (1874) focused exclusively on tension of the spine in the whole body perinate, while Ouyang et al. (2005) investigated osteoligamentous whole cervical spine (WCS) tension and bending in specimens from 2 to 12 years. Recently, the authors published results from pediatric cadaver cervical spines from the perinate to 14 years in WCS and motion segments in tension (Luck et al. 2008). We present the experimental apparatus and fixation methodology utilized by Luck et al. (2008) and subsequent work for two modes of loading; bending and tension, completed on specimens in the WCS and upper cervical spine (UCS) configuration. The experimental apparatus and fixation technique accommodates specimens from around birth to adulthood and does not compromise the skull, leaving it suitable for further biomechanical testing of the intact head.

4.2 Methods and materials

An apparatus was designed to test structural properties of the cervical spine under multiple modes of loading using perinatal to adult size head-neck complexes. The apparatus maintained cranial integrity and was designed to accommodate tension and bending without cranial repositioning; controlling variability potentially introduced when using multiple testing arrangements with the same sample. Schematics of the apparatus are provided for three loading phases, WCS tension and UCS bending and tension (Figs. 12–15).
4.2.1 Design and implementation for whole spine tension tests

The apparatus rigidly attached osteoligamentous head-neck complexes to a servo-hydraulic actuator (MTS, Eden Prairie, MN). The apparatus and cranium of the specimen attached to the upper platen while the caudal vertebra attached to the actuator enabling force and displacement controlled translation of the spine.

The cranium was attached to the apparatus using an adjustable wedge-plate-platen system to position the neck along the line of action of the displacement actuator. This design accommodated a range of head anthropometries (breadth: 6 – 15 cm; length: 6 – 19 cm) and cervical spine lengths (2.5 – 12.5 cm) and provided anterior and posterior bearing surfaces for attaching the cranium to the apparatus (Fig. 16).
FIGURE 12: Experimental pediatric cadaver testing apparatus in the osteoligamentous whole spine configuration within the MTS servo-hydraulic testing frame. Quasi-static tensile tests of the whole spine were administered in this arrangement. Polymethyl-methacrylate (PMMA) applied during the curing process around the cranium and against the testing apparatus established a rigid fixation between the test specimen and apparatus.
FIGURE 13: Experimental pediatric cadaver testing apparatus in the upper cervical spine configuration with sagittal plane bending eccentricity bar attached to specimen. Discrete pure moment loading in flexion and extension of the upper cervical spine was administered in this arrangement. A linear bearing system capable of translating in two axes maintained a vertical upward force during rotation of the loading bar.
FIGURE 14: The upper cervical spine fixation under extension bending. Removable regions in the posterior section of the apparatus allowed for greater extension rotation as a result of increased clearance.
FIGURE 15: Experimental pediatric cadaver testing apparatus in the osteoligamentous upper cervical spine configuration within the MTS servo-hydraulic testing frame. Quasi-static and failure tensile tests of the upper cervical spine were administered in this arrangement.

The anterior of the cranium was attached along the maxilla, inferiorly and anteriorly, and along the surface of the frontal and nasal bones, anteriorly, using a plate and platen that adjusted to complement cranial size variability. The plate established a boundary along an anterior and vertical plane; adjacent to the frontal and nasal bones
that was easily adjusted for maxilla length variability. The posterior of the cranium was attached along an oblique plane established with dual adjustable rigid wedges which freely translated along a removable posterior platen of the apparatus. Similar to the anterior plate and platen, the posterior wedge system provided ample adjustability to accommodate a range of occipital bone geometries and cranial lengths.

The cranium was constrained in all directions by applying polymethyl-methacrylate (PMMA) at the contact points between the specimen and apparatus and across the top of the head. The PMMA was in a partially cured form and easily manipulated to fill the space between the cranium and apparatus. PMMA was attached to the apparatus by packing it around screws in the anterior and posterior wedge-plate-platen locations; and screws were embedded in the curing PMMA to provide connection points for subsequent PMMA applications. Additional PMMA was applied in a criss-cross pattern across the top of the head with end points terminating at the previously cured PMMA locations.
FIGURE 16: The experimental pediatric cadaver testing apparatus was designed to accommodate cranial sizes ranging from perinatal to adult. Adjustability in the fore and aft areas of the apparatus was designed to allow for accommodation of multiple size cadavers while providing adequate surface area to rigidly fixate the cranium without structural compromise. Multiple elements, including the removable platens were designed to follow a one-time fixation protocol allowing for WCS tension, UCS bending and UCS tension/failure with zero cast-recast.
4.2.2 Design and implementation for upper cervical spine bending tests

After WCS tension testing, the apparatus and attached specimen were removed as a single unit from the loading frame. Preparation for bending of the UCS and lower cervical spine included separation of the WCS at C3-C4, C5-C6 and C7-T1. The UCS segment (cranium to C3) was cast at C2-C3 and prepared for flexion-extension bending. Throughout the transition from WCS tension, dissection, and then to UCS bending, the cranium was rigidly attached to the apparatus at all times.

The apparatus and specimen were inverted and secured to a six-axis load cell at the base; similar to previously published methods of UCS bending (Nightingale et al. 2002; Ouyang et al. 2005). An eccentric bar was designed to load the UCS in extension and flexion with a pure moment; due to initial tests indicating rotations up to 40 degrees could be expected for applied moments of less than 0.5 N-m. A canal was designed into the posterior wedges that allowed additional rotation of the eccentric bar. A plate on the posterior side of the apparatus and the platen under the dual wedges were removable and provided additional clearance for the rotation of the bar (Fig. 14). A linear bearing system capable of translating in two-axes was positioned superior to the apparatus and allowed for the upward force to be held vertical at each load step. The downward force emanated from a freely hanging mass and followed the movement of the bar.
4.2.3 Design and implementation for upper cervical spine tension and failure tests

Tensile structural, viscoelastic and failure testing of the UCS followed the completion of UCS bending tests. To transition from the bending to tension set-up, the posterior supporting platen and support plate were reattached and the eccentric loading bar assembly removed from the caudal casting. The apparatus was mounted upright in the loading frame and the C2-C3 fixation attached to the actuator.

4.2.4 Determination of loading apparatus compliance

Compliance of the experimental test apparatus and fixation was determined to compensate for total compliance of the experimental design. A non-compliant test surrogate was fashioned out of a block of hardwood (red oak) in place of the specimen. Casting procedures consistent with those utilized in cadaveric testing were applied to the oak surrogate. The surrogate was pre-conditioned and thirty ramp-and-return waveforms were applied to a level of 1800 N. Stiffness for each test was determined by regressing the loading phase of the force-displacement results. An average stiffness was obtained from the 30 trials.

4.3 Results and discussion

Twenty-three cadaveric osteoligamentous head-neck complexes were tested using the tension-bending apparatus. These specimens ranged in age from birth to adulthood. Tensile properties of 17 specimens using this apparatus were previously
reported (Luck et al. 2008). After neck testing, the intact cranium was removed from the apparatus and biomechanical testing of the pediatric skull occurred (Prange et al. 2004).

Though apparatus and fixation compliance may be important in quantifying biomechanical response, it is not routinely reported. Tracking bone to bone movement in spinal segments directly with optical markers does not require compensation for potential fixation compliance. However, previous studies have shown that the compliance of the apparatus and fixation can have a significant effect on the measured responses of spine segments that are not directly tracked. Stiffness models of WCS and motions segments that included frame compliance were stiffer by 6 to 41 percent compared to similar models that did not factor in the compliance of the testing apparatus (Nightingale et al. 2004).

The average stiffness of the apparatus was 1986 ± 13 N/mm (n = 30). The linear regression used for quantifying the stiffness was well correlated ($R^2 = 0.999$). The stiffness increased by less than one percent when the stiffness of the hardwood surrogate was taken into consideration. The current apparatus and fixation was over twice as stiff as the testing apparatus reported by Nightingale et al. (2004) that yielded corrections of between 6 and 20 percent in WCS and UCS tensile stiffness, respectively, once apparatus stiffness was taken into account.
This report details an apparatus and fixation technique that maximizes the information attainable from multiple testing modes in perinatal to adult cadaveric head-neck complexes. Apparatus design and fixation techniques rigidly attached the head without impinging upon the soft tissue and bony framework of the skull. This design enabled individual cadaveric head-neck complexes to be tested in two modes of loading and then removed intact for future biomechanical testing of both the whole skull and sub-cranial material. Moreover, apparatus and fixation stiffness were reported to evaluate biomechanical data obtained from the testing apparatus. The apparatus and fixation technique provides a platform for furthering the understanding of pediatric cervical spinal mechanics in an environment where limited availability of pediatric cadaveric tissue is a significant obstacle to understanding and quantifying the biomechanics of these structures.

4.4 Acknowledgements

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5. Tensile Mechanical Properties of the Perinatal and Pediatric PMHS Osteoligamentous Cervical Spine


5.1 Abstract

Pediatric cervical spine biomechanics have been under-researched due to the limited availability of pediatric post-mortem human subjects (PMHS). Scaled data based on human adult and juvenile animal studies have been utilized to augment the limited pediatric PMHS data that exists. Despite these efforts, a significant void in pediatric cervical spine biomechanics remains. Eighteen PMHS osteoligamentous head-neck complexes ranging in age from 20 weeks gestational to 14 years were tested in tension. The tests were initially conducted on the whole cervical spine and then the spines were sectioned into three segments that included two lower cervical spine segments (C4-C5 and C6-C7) and one upper cervical spine segment (O-C2). After non-destructive tests were conducted, each segment was failed in tension. The tensile stiffness of the whole spines ranged from 5.3 to 70.1 N/mm. The perinatal and neonatal specimens had an ultimate strength for the upper cervical spine of 230.9 ± 38.0 N and for the lower cervical
spine of 212.8 ± 60.9 and 187.1 ± 39.4 N for the C4-C5 and C6-C7 segments, respectively. The lower cervical segments were significantly weaker and stiffer than the upper cervical spine segments in the older cohort. For the entire cohort of specimens, the stiffness of the upper cervical spine ranged from 7.1 to 199.0 N/mm. The tolerance ranged from 173.6 to 2960 N for the upper cervical spine and from 142 to 1757 N for the lower. There was a statistically significant increase in stiffness and strength with age. The results also suggest that juvenile animal surrogates estimate the stiffness of the human cervical spine fairly well; however, they may not provide accurate estimates of pediatric cervical spine strength.

5.2 Introduction

Spinal trauma remains a prominent issue that results in debilitating social, economic, and physical outcomes (Harrop et al. 2001; Sekhon and Fehlings 2001). It has been reported that over half of all spinal cord injuries occur in the cervical spine and that the leading mechanism of injury to the spinal cord are motor vehicle crashes which account for 47.5% of these injuries (Brown et al. 2001; Eleraky et al. 2000; Kokoska et al. 2001; Roche and Carty 2001). According to NHTSA’s National Center for Statistics and Analysis, in 2003, motor vehicle crashes were the leading cause of death for the age group of 4 to 34 years of age (U.S. Department of Transportation 2006). For children from one to four years, automobile crashes ranked among the top ten causes of death. Motor vehicle crashes also rank as the most common cause of spinal injuries in the
pediatric population (Babcock 1975; Bonadio 1993). Cervical spine trauma accounts for approximately 2-10% of all pediatric spinal injuries (Brown et al. 2001; Eleraky et al. 2000; Roche and Carty 2001; Schippers et al. 1996; Thakar et al. 2005). The mortality rate amongst pediatric spinal trauma victims is approximately 25-32%, demonstrating the vulnerability of the maturing neck (Roche and Carty 2001).

Reviews of injuries to restrained occupants provides evidence that neck injury can occur by forces and moments generated by the inertia of the head in moderate to severe frontal crashes (Huelke et al. 1993). Although this mechanism occurs throughout the adult and pediatric populations, it has been hypothesized that the anatomy of the developing child makes children particularly vulnerable. The relatively large head mass, ligamentous laxity, decreased facet angle, and slender cervical spine, all suggest a heightened risk of neck injury due to inertial loading of the neck by the child’s head (Eleraky et al. 2000; Orenstein et al. 1994; Roche and Carty 2001). Tensile neck injuries are also observed due to airbag interactions with out-of-position occupants (Braver et al. 1997; Brown et al. 1995; Dalmotas et al. 1995; Giguere et al. 1998; Traynelis and Gold 1993). In the period prior to airbags being depowered, (Graham et al. 1998) observed that air bags increased the mortality risk for children younger than 12 years.

The majority of pediatric neck injuries are reported to occur in the upper cervical spine (Klinich et al. 1996), which makes this region of particular interest. The upper cervical spine consists of the first two vertebrae, also known as the atlas and axis. At
birth, the atlas contains two cartilaginous regions, one anterior and one posterior connecting two lateral masses (Bailey 1952; Ogden 1984a; Ogden 1984b). The axis exhibits a more complicated growth pattern with a total of seven synchondroses (Fesmire and Luten 1989; Ogden 1984a). Similar to the lower cervical spine, the axis has a posterior synchondrosis and two neurocentral synchondroses (Figure 17). Additionally, the axis has a dentocentral synchondrosis that runs superior to the centrum and inferior to the dens. Three additional synchondroses are associated with the dens and lateral masses. The dentoneural synchondroses are positioned superior to the line of the dentocentral synchondrosis and intersect with the neurocentral synchondroses bilaterally. The intradental synchondrosis resides between the two dental ossification centers and typically fuses prior to birth. These cartilaginous growth regions are often reported to be the site of injury (Adams 1992; Bhattacharyya 1974; Clasper and Pailthorpe 1995; Ewald 1971; Keller and Mosdal 1990; Schatzker et al. 1971; Seimon 1977; Smith et al. 1993).

Despite its importance, few studies of PMHS have investigated the biomechanical behavior of the pediatric cervical spine owing to a lack of human pediatric donors. Moreover, the studies in the literature vary significantly in design and often have small sample sizes. Duncan (1874) investigated the strength of the pediatric PMHS cervical spine in tension to better understand the loads that could be tolerated in
the neonate during breech delivery. Cervical spine failure was reported to occur over a range from 400 to 654 N, followed by subsequent decapitation at higher loads.

**Figure 17:** Developmental ossification and cartilaginous regions of axis. Seven cartilaginous regions allow for growth of the enlarging axis vertebra. These cartilaginous regions or synchondroses are often the site of injury in the child as a result of trauma to the neck and are thus important anatomical landmarks of the maturing child. (adapted from Scheuer and Black 2004)

This study did not investigate structural properties of the cervical spine, including the stiffness of the whole spine or individual segments. Additionally, the study was limited to a sample comprised solely of neonatal PMHS. Moreover, the loading of the PMHS occurred in a discrete manner with approximately thirty-second intervals between consecutive load steps. Ouyang et al. (2005), in the most comprehensive study to date, reported on the structural response of the pediatric PMHS whole cervical spine to tension under both non-destructive and failure loads. Failure of the whole cervical spine was reported to occur over a range from 493.7 to 918.1 N.
Tensile stiffness of the whole cervical spine was reported as the ratio of the ultimate failure load and displacement at failure. The stiffness or tolerance of individual cervical segments was not reported. The destructive tests were conducted under displacement control at 5 mm/s until failure occurred. The test sample ranged from 2 to 12 years. These studies introduce structural and failure properties for the whole spine. However, both studies used a very small sample of specimens and neither evaluated the structural and failure properties of individual segments.

The difficulty of acquiring tissue from human pediatric donors has fostered research on juvenile animals. Whole body porcine studies were conducted to examine the effect of airbag inflation on child-size surrogates and compare the injuries to load responses in an instrumented three year-old child test dummy (Aldman et al. 1974; Mertz et al. 1982; Prasad and Daniel 1984). Based on these tests, injury assessment reference values (IARV) for the CRABI 6-month dummy were estimated based on scaling techniques applied to previous animal, dummy, fetal neck and calcaneal tendon data (Melvin 1995). More recently, osteoligamentous caprine cervical spines were tested non-destructively in bending and to failure in tension and scaling relationships were determined between pediatric and adult caprine specimens for tensile tolerance and stiffness (Pintar et al. 2000). The scaling relationships of Pintar et al. (2000) were further refined for the pediatric caprine model by normalizing the pediatric strength data by a mid-sized adult caprine model to determine updated scale factors at specific pediatric
ages (Hilker et al. 2002). Similar studies in post-mortem baboons have been used to
determine tensile tolerance and stiffness scaling relationships between pediatric and
adult baboon specimens (Ching et al. 2001; Nuckley and Ching 2006). The underlying
hypothesis in these models is that the age-property relationships in the animal model
may be used to scale adult human data to pediatric data. While reasonable, the validity
of this hypothesis has yet to be tested.

Accordingly, the purpose of the current study is to provide data on the structural
and failure responses of pediatric PMHS from donors aged 0 to 14 years. An additional
purpose is to give a preliminary assessment of the utility of animal based age-property
relationships in predicting human response.

5.3 Methods

Eighteen whole cervical spines from unembalmed pediatric post-mortem human
subjects (PMHS) were tested in a tensile mode of loading (Table 1). The pediatric PMHS
specimens ranged in age from 20 weeks gestation to 14 years old and consist of eleven
perinatal and neonatal specimens (20 weeks gestation to 24 days old) and seven infant to
young adult specimens (5 month to 14 years old). A battery of tensile tests, including
stiffness, rate sensitivity and stress relaxation tests, was conducted on the whole cervical
spine (head to T1). The specimens were then transected and a similar battery of tensile
tests was conducted on individual cervical spinal segments (O-C2, C4-C5, and C6-C7).
The results of the viscoelastic testing are still being analyzed and will be presented in a future manuscript.

**Figure 18:** Schematic of the fixture used for pediatric PMHS tension tests. The cranial end of the PMHS was fixated at the maxilla and occipital bone to the fixture without intrusion of the skull. The caudal end of the spine was attached to the hydraulic actuator. An LVDT recorded linear displacement of the actuator while a six-axis load cell recorded load and moment data.

A testing frame was designed to accommodate the wide range of specimen sizes (Figure 18). The caudal end of the spine was coupled to a hydraulic actuator (MTS Systems Corporation, Eden Prairie, MN). A six-axis load cell (2554A, Denton, Inc., Rochester Hills, MI) was attached to the test frame above the PMHS to quantify the loads. An LVDT (227, MTS Systems Corporation, Eden Prairie, MN) was attached to the
hydraulic actuator to quantify the axial displacement of the ram along the loading path. Data were collected using a digital acquisition system (LabView 7.0, PCI 6071E, National Instruments, Austin, TX). A digital camera documented the test set-up and recorded the motions of the cervical segments during the non-destructive and destructive tests (Phantom v4.2 camera, Vision Research, Inc., Wayne, New Jersey). The camera data was used to assist in assessing when failure occurred and what cervical structures were involved in the failures.

Specimen preparation and handling was conducted adhering to CDC guidelines (Cavanaugh and King 1990). Testing was completed in accordance with all protocols outlining the ethical use of PMHS. Institutional Review Board (IRB) exemption and approval was granted for all PMHS work. Each pediatric PMHS was dissected to isolate the osteoligamentous spine. All neck musculature, subcutaneous fatty tissue, and skin were removed. The mandible was disarticulated in order to allow for more rigid head fixation and visualization of the upper cervical spine. The Frankfort plane was marked from the superior point of the external auditory meatus to the infraorbital foramen.

The specimen was secured to the frame at the maxilla and about the occipital region with the Frankfort plane aligned horizontal. In order to minimize damage to the heads, molded polymethylmethacrylate (PMMA) (Dentsply International; York, PA) was used to secure the skull and maxilla to the testing frame. K-wires and pedicle loops were used to rigidly wire the T1 vertebrae for casting with PMMA. Reinforced polyester
resin was added to a cup attached to the hydraulic actuator about the K-wire and PMMA casting to rigidly affix the caudal end of the specimen. The specimen was then placed under load control to zero Newtons.

It was felt that a controlled stress rate would also make comparisons with existing adult data (Dibb et al. 2009; Chancey et al. 2003; Van Ee et al. 2000b) more relevant. The loading rates used in the prior tests on stiffer adult specimens and the 14 year old donor would have resulted in much higher strain rates in the smaller and more compliant specimens and were potentially damaging. Therefore a scale factor based on the area of the T1 vertebral body was used to determine load rates that would maintain a constant stress rate in the cervical spine throughout the size range. The major and minor axes of each T1 vertebral body was used to approximate the area as an ellipse. The scale factor was determined by dividing this area by the adult area of 308.8 mm\(^2\) (Panjabi et al. 1991a). This scale factor was then multiplied by 50 N/sec to determine the scaled loading rate (SLR) to be utilized for a given pediatric PMHS.

A series of fixed-fixed force-displacement tests was conducted under load control at the scaled loading rate to approximately 10% of the estimated failure load. The whole cervical spine specimens were preconditioned under load control at 1 Hz for sixty cycles in a fixed-fixed end condition. This was followed by a series of tension tests at scaled load rates.
### TABLE 1: Pediatric PMHS Anthropometric Data

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age</th>
<th>Sex</th>
<th>CO2</th>
<th>Whole Body</th>
<th>Head</th>
<th>Spine</th>
<th>Scale Factor</th>
<th>Seated Loading Rate (kN)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mass (kg)</td>
<td>Height (cm)</td>
<td>Mass (kg)</td>
<td>Breadth (cm)</td>
<td>Length (cm)</td>
</tr>
<tr>
<td>C6F</td>
<td>20 WKG</td>
<td>F</td>
<td>Heart Failure</td>
<td>-</td>
<td>0.094</td>
<td>5.9</td>
<td>7.1</td>
<td>2.46</td>
</tr>
<tr>
<td>13P</td>
<td>29 WKG [0 MN]</td>
<td>F</td>
<td>-</td>
<td>-</td>
<td>0.422</td>
<td>7.1</td>
<td>10.2</td>
<td>3.40</td>
</tr>
<tr>
<td>C9F</td>
<td>33 WKG [0 MN]</td>
<td>M</td>
<td>Fetal Demise</td>
<td>2.04</td>
<td>43.2</td>
<td>-</td>
<td>9.1</td>
<td>3.61</td>
</tr>
<tr>
<td>C7F</td>
<td>33 WKG [0 MN]</td>
<td>M</td>
<td>-</td>
<td>-</td>
<td>0.434</td>
<td>8.2</td>
<td>10.1</td>
<td>3.66</td>
</tr>
<tr>
<td>13P</td>
<td>35 WKG [0 MN]</td>
<td>M</td>
<td>Fetal Demise</td>
<td>-</td>
<td>-</td>
<td>9.1</td>
<td>10.0</td>
<td>4.12</td>
</tr>
<tr>
<td>C8P</td>
<td>37.5 WKG [0 MN]</td>
<td>M</td>
<td>Pulmonary Hypoplasia</td>
<td>-</td>
<td>50.8</td>
<td>9.4</td>
<td>11.4</td>
<td>4.21</td>
</tr>
<tr>
<td>C5F</td>
<td>1 DY [0.03 MN]</td>
<td>F</td>
<td>Diaphragmatic Hernia</td>
<td>2.75</td>
<td>-</td>
<td>0.065</td>
<td>9.1</td>
<td>11.7</td>
</tr>
<tr>
<td>C8P</td>
<td>3 DY [0.1 MN]</td>
<td>M</td>
<td>Ischemic Encephalopathy; Central Infection</td>
<td>-</td>
<td>-</td>
<td>0.402</td>
<td>8.5</td>
<td>10.3</td>
</tr>
<tr>
<td>06P</td>
<td>11 DY [0.57 MN]</td>
<td>F</td>
<td>Non-immune Hydrops Fetalis &amp; Intercostal Hemorrhage</td>
<td>2.02</td>
<td>44.5</td>
<td>0.702</td>
<td>10.4</td>
<td>11.2</td>
</tr>
<tr>
<td>11P</td>
<td>16 DY [0.63 MN]</td>
<td>F</td>
<td>Anencephaly</td>
<td>2.27</td>
<td>-</td>
<td>0.93</td>
<td>6.3</td>
<td>6.0</td>
</tr>
<tr>
<td>C4P</td>
<td>24 DY [0.68 MN]</td>
<td>F</td>
<td>Dandy-Walker Syndrome</td>
<td>2.72</td>
<td>45.7</td>
<td>1.152</td>
<td>10.5</td>
<td>17.5</td>
</tr>
</tbody>
</table>

**Pediatric PMHS > One Month (Older Cohort)**

| 12P | 5 MN | M | Respiratory Failure | - | - | 1.071 | 12.3 | 13.2 | 5.22 | 20.45 | 8.00 | 0.468 | 23.4 |
| 14P | 9 MN | M | COPOC | 7.00 | - | 1.060 | 11.5 | 15.0 | 5.26 | 20.00 | 8.28 | 0.420 | 21.0 |
| 15P | 11 MN | F | SIDS | 6.16 | 71.1 | 1.570 | 11.9 | 14.8 | 6.12 | 21.70 | 10.05 | 0.299 | 29.9 |
| 16P | 16 MN | M | Thrombosis | 11.60 | 81.3 | SC | 15.5 | 15.2 | 7.17 | 21.30 | 12.20 | 0.660 | 34.0 |
| 17P | 22 MN | F | Non-Hodgkin Lymphoma | - | - | 12.5 | 16.4 | 6.73 | 21.30 | 11.50 | 0.623 | 31.2 |
| 18P | 9 YR [100 MN] | M | End-stage Renal Disease; Hypokalemia | - | - | 2.440 | 13.1 | 16.3 | 8.66 | 23.60 | 15.10 | 0.906 | 45.3 |
| C1P | 14 YR [168 MN] | F | Brain Aneurysm | 61.20 | 165.0 | - | - | - | 11.73 | 1.73 | 50.0 |

**WKG** - weeks gestation; **WP8** - weeks post birth; **DY** - days; **MN** - months; **YR** - years; **SC** - skull compromise

Equation for area of an ellipse: \( A = \pi a b \) used to approximate area of T1 endplate for seated loading rate
Upon completion of the whole spine cervical testing battery, the specimens were transected into three segments: O-C2, C4-C5 and C6-C7. The vertebrae of each spinal segment were cast into aluminum cups using the casting techniques described earlier. The upper cervical spinal segment, O-C2, was mounted with the head in the testing fixture and C2 cast in an aluminum cup attached to the hydraulic actuator. The two lower motion segments were mounted with both the superior and inferior vertebra cast in aluminum cups attached to the load cell and the hydraulic actuator, respectively.

The spinal segments were again preconditioned under load control at 1 Hz for sixty cycles in a fixed-fixed end condition. A series of fixed-fixed force-displacement tests were conducted to approximately 10% of the estimated failure load. Fixed-fixed tensile failure tests were then conducted on each spinal segment. Because of the wide range of specimen stiffnesses, issues of instability arose in the control algorithm at high loading rates. It was not possible to tune the algorithm with the specimens in place for fear of destroying the specimen. Therefore, the protocol for the failure testing was changed from load control to displacement control part way through the series. The specimens were failed at the following rates: 14 year PMHS at 1000 N/s; perinatal/neonatal & 5 month PMHS – scaled loading rate; infant to 9 years PMHS – 230 mm/s).

Tensile stiffnesses were calculated by obtaining a linear regression from 50-100% of the loading portion of the fixed-fixed force-displacement test. An initial and ultimate
tensile failure load was determined for each failure test. Initial failure was defined by a
decrease in load with increasing displacement in the force-displacement response.
Ultimate failure was defined as the maximum load in the force-displacement response.
All perinatal donors were designated zero months of age for data analysis. All neonatal
donor ages were converted to a fraction of a month based on 30 days/month. All fixed-
fixed force-displacement data were filtered at 100 Hz. All failure tests were filtered at
450 Hz.

The perinatal and neonatal donors were grouped to assess differences in tensile
stiffness and tolerance by level. This was done using a one-way ANOVA (α = 0.05) with
a post-hoc Tukey-Kramer HSD multiple comparison test. In order to evaluate
differences in tolerance and stiffness by age, a two-way ANOVA (α = 0.05) of age and
level was computed for all pediatric specimens that had a full complement of stiffness
and tolerance data at all levels. All means are given in the form: mean ± standard
deviation.

A regression analysis was completed to compare the current pediatric stiffness
data to previously published animal studies. A power law function was fit to both the
current stiffness data and the work of Nuckley et al. (2006). The power law functions
representing both data sets were then populated over an age range that included ages
present in both the current study and previous animal study at equal intervals
throughout the range. The mean absolute deviation was determined to assess the similarity in stiffness between the current study and previous animal study.

The neck length of each donor was measured from the apex of the dens of axis to the anterior and superior edge of the T1 vertebral body from high resolution CT scans (Table 1). Head breadth measurements were taken from the temporal bone region. Head length measurements were taken from the nasion to the occipital protuberance. Head mass was obtained upon the completion of a subsequent head testing protocol. Some head masses were not obtainable (T09P, T10P, T11P, T16P) due to a compromised skull case associated with cranial pathologies or post-mortem procedures prior to specimen procurement. The T1 vertebral body breadth (major axis) and length (minor axis) were measured.

5.4 Results

The donors ranged in age from 20-week gestation (pre-term) to 14 years of age (Table 1). Cervical spine neck length, head breadth, and head length measurements increased with increasing age from the perinatal and neonatal donor population to the adolescent ages. None of the specimens included in the study showed evidence of pathology or trauma that would affect the mechanical properties of the cervical spines.

The force-displacement response of the whole spines and motion segments showed a non-linear stiffening behavior characteristic of biological tissue (Figure 19). Non-destructive force-displacement plots of all ages are presented in Appendix A.
Tensile stiffness and low-load displacement are reported for the neonatal cohort (Table 2) and older cohort (Table 3).

**Figure 19:** The force-displacement response of the upper cervical spine (O-C2) of the 9 year (108 months) PMHS (T18P). The tensile stiffness (N/mm) was determined by linear regression of the loading portion of the response from 50 to 100% of the applied load.

Tensile stiffness increased with age for the whole cervical spine and for each of the motion segment levels tested (p < 0.001, two-way ANOVA, Figure 20). The average whole spine stiffness from the perinatal/neonatal cohort to the 14 year old increased by approximately ten fold. An even greater increase in stiffness was observed in the upper cervical spine where a twenty fold increase existed between the perinatal/neonatal cohort and the 14 year old. Smaller increases in stiffness were observed in the lower motion segments. A comparison of the perinatal/neonatal cohort to the 22 month PMHS yielded an increase in tensile stiffness of approximately 3.25 in the C4-C5 segment. An
approximately six fold increase in stiffness was observed between the perinatal/neonatal cohort and the 9 year old C6-C7 segment. The stiffness of the whole spine and upper cervical spine were significantly lower than either of the lower cervical segments (p < 0.001, one-way ANOVA, Table 2). The lower cervical spine stiffness of the perinatal/neonatal cohort was approximately 4.5 times greater than the stiffness of the upper cervical spine of the perinatal/neonatal cohort. Post-hoc comparison did not reveal any significant differences between the C4-C5 motion segments and the C6-C7 motion segments.

**Table 2:** Tensile Stiffness and Low-Load Displacement for Perinatal and Neonatal Cohort

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>LCS</th>
<th>O-C2</th>
<th>C4-C5</th>
<th>C6-C7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>LLD (mm)</td>
<td>Stiffness (N/mm)</td>
<td>LLD (mm)</td>
<td>Stiffness (N/mm)</td>
</tr>
<tr>
<td>02P</td>
<td>0</td>
<td>0.02</td>
<td>5.4 (^1)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>1.28</td>
<td>7.7</td>
<td>0.29</td>
<td>12.2</td>
</tr>
<tr>
<td>09P</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>07P</td>
<td>0</td>
<td>0.69</td>
<td>7.9 (^2)</td>
<td>0.61</td>
<td>11.9</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>08P</td>
<td>0</td>
<td>1.98</td>
<td>6.0</td>
<td>0.23</td>
<td>10.5</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>1.27</td>
<td>5.3</td>
<td>0.68</td>
<td>7.4</td>
</tr>
<tr>
<td>03P</td>
<td>0.1</td>
<td>0.49</td>
<td>7.7</td>
<td>0.72</td>
<td>11.2</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>0.33</td>
<td>6.8</td>
<td>1.45</td>
<td>7.1</td>
</tr>
<tr>
<td>11P</td>
<td>0.53</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>0.34</td>
<td>7.3</td>
<td>0.18</td>
<td>9.3</td>
</tr>
<tr>
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<td>SD</td>
<td></td>
<td>0.65</td>
<td>1.0</td>
<td>0.44</td>
<td>2.1</td>
</tr>
</tbody>
</table>

\(^1\) - Donor 02P tested from occiput to C6 (excluded from mean calculation)

\(^2\) - Donor 07P tested from occiput to C7 (excluded from mean calculation)

- - Specimen not available; Grayed areas indicate PMHS used in two-way ANOVA
TABLE 3: Tensile Stiffness and Low-Load Displacement for Pediatric PMHS > 1 Month

Tensile failure tests showed evidence of a structural failure prior to ultimate failures (Figure 21). Force-displacement plots of failure tests for all ages are presented in Appendix B. Initial sub-catastrophic and ultimate force-to-failure for all specimens are presented in Tables 4 and 5. Ultimate strength increased monotonically with age (Figure 22, two-way ANOVA, p < 0.001). The upper cervical spine of the 14 year old was approximately thirteen times stronger than the average strength observed in the upper cervical spines of the perinatal/neonatal cohort.

A more modest increase in strength was observed in the lower segments. A comparison of the perinatal/neonatal cohort to the 22 month PMHS showed an increase of approximately four fold in C4-C5 strength. A similar comparison between the perinatal/neonatal cohort and the 9 year PMHS showed an increase of approximately nine fold in C6-C7 strength. No significant differences in the ultimate tensile strength by level were found within the neonatal cohort (p = 0.2334, one-way ANOVA) whereas the
upper cervical spine was significantly stronger than the lower cervical spine in the older cohort (p < 0.001, two-way ANOVA).

**FIGURE 20:** Comparison of tensile stiffness of the whole spine and all cervical segments. Tensile stiffness increases with age across all spinal levels (two-way ANOVA; p < 0.001).

Failure testing produced clinically observed injuries including physeal/endplate disruptions and cartilaginous synchondrotic disruptions (Table 6). The lower cervical spine segments exhibited physeal or endplate disruptions about one of the endplate locations. Fractures at the neurocentral synchondroses and through the posterior synchondrosis were also observed. The upper cervical spine exhibited a spectrum of injuries. These included an occipito-atlantal dislocation with associated atlas and occipital condyle fracture; fracture-dislocations through the neurocentral synchondroses and dentocentral synchondrosis of axis with associated lamina fractures resulting in a C2-C3 dislocation; fracture-dislocation through the dentoneural synchondroses and
dentocentral synchondrosis of axis, similar in the adult population to the Type III dens fracture; atlanto-axial dislocation with chondrum terminale disruption in the apex of the dens; and, atlanto-axial dislocation with ossiculum terminale dislocation in the apex of the dens.

![Graph of force vs. deflection](image)

**Figure 21:** Representative failure test data for the upper cervical spine (OC2). These data are taken from PMHS T18P, a 9 year-old (108 month). The O-C2 segment exhibits an initial failure followed by a catastrophic ultimate failure.

### 5.5 Discussion

The primary limitations of this study are related to the sample size, the large variability in physical size and stiffness of the donors, the need to safeguard against accidental damage to the specimen, and the lack of live muscle effects. In our experiments, acquisition of pediatric PMHS occurred over the decade prior to testing. This procurement effort resulted in a large cohort of perinatal and neonatal donors and a more sparse sample of donors older than one year. Accordingly, we chose to report
these data in two cohorts recognizing that age related changes occur throughout development. The quality of pediatric donors was also a limitation of the current study. Although we did not observe cervical spine pathology in our donor population that would alter the mechanical response, we did encounter donors that limited the scope of our testing. Due to cranial deformities, we were unable to test the whole cervical spine and upper cervical spine of certain donors (T09P, T10P, T11P, T16P). Additionally, we worked with one donor (T16P) with post-mortem damage to the cervical spine that necessitated testing of the C3-C4 and C5-C6 lower spinal segments as opposed to our protocol of the C4-C5 and C6-C7 segments. By choosing to study the isolated osteoligamentous cervical spine, we have removed the effects of the musculature which have been shown to significantly impact the kinetics and failure properties of the adult cervical spine in tension (Chancey et al. 2003; Van Ee et al. 2000b).

Given the importance of each donor, it was necessary to safeguard the structural integrity of our specimens by using especially low loads during non-destructive testing. Following a similar methodology to Van Ee et al. (2000b), all non-destructive tests were performed at approximately 10% of estimated failure. Estimates of tolerance in the perinatal ages were derived from juvenile animal surrogate studies (Ching et al. 2001; Hilker et al. 2002; Pintar et al. 2000). These data indicated that under a tensile load, the estimated failure of the neonate was likely to be under 200 N. Upon conducting the initial perinatal test (02P), a whole spine failure occurred at 57.2 N. Taking the age and
size of this specimen into consideration, and comparing it with previous animal data, we chose a level of 150 N for the estimated strength of the perinatal specimens, resulting in a non-destructive load level of 15 N.

**TABLE 4: Perinatal & Neonatal Strength**

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>O-C6 Ultimate (N)</th>
<th>O-C6 Initial (N)</th>
<th>O-C2 Ultimate (N)</th>
<th>O-C2 Initial (N)</th>
<th>C4-C5 Ultimate (N)</th>
<th>C4-C5 Initial (N)</th>
<th>C6-C7 Ultimate (N)</th>
<th>C6-C7 Initial (N)</th>
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</thead>
<tbody>
<tr>
<td>02P</td>
<td>0</td>
<td>57.2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>274.8</td>
<td>334.3</td>
<td>194.9</td>
<td>204.1</td>
<td>—</td>
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<td>09P</td>
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<td>—</td>
<td>196.9</td>
<td>168.1</td>
<td>148.8</td>
<td>154.1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>07P</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>241.9</td>
<td>188.4</td>
<td>207.5</td>
<td>209.7</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>208.9</td>
<td>165.8</td>
<td>182.7</td>
<td>142.0</td>
<td>—</td>
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</tr>
<tr>
<td>08P</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>241.9</td>
<td>167.3</td>
<td>182.9</td>
<td>191.4</td>
<td>—</td>
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</tr>
<tr>
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<td>0.03</td>
<td>—</td>
<td>—</td>
<td>188.3</td>
<td>225.5</td>
<td>172.6</td>
<td>181.3</td>
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</tr>
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<td>—</td>
<td>257.6</td>
<td>139.3</td>
<td>142.0</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>—</td>
<td>—</td>
<td>173.6</td>
<td>140.2</td>
<td>151.9</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>11P</td>
<td>0.53</td>
<td>—</td>
<td>—</td>
<td>167.3</td>
<td>182.9</td>
<td>191.4</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>—</td>
<td>—</td>
<td>260.0</td>
<td>237.2</td>
<td>250.4</td>
<td>262.2</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Mean: 224.2 230.9 206.5 212.8 172.9 187.1
SD: 50.7 38.0 61.4 60.9 39.9 39.4

— - Specimen not available; Grayed areas indicate PMHS used in two-way ANOVA
X - Data loss

The demands of the experimental protocol required a stiff, robust test frame to measure a large range of loads for a large range of specimen sizes and stiffnesses. The resonant frequency of the apparatus is dictated by the combined mass of the head and frame and the stiffness of the load frame and load cell. Since sensitive load cells are relatively compliant, the resonant frequency of the apparatus was lower than ideal.
An alternative would have been to place the load cell on the caudal end of the specimen, but this would have made testing under load control potentially unstable due to the accelerations of the load cell. A great deal of effort was put into reducing the mass of the frame while keeping it as stiff as possible; however, this resulted in a lowest resonant frequency of 150 Hz in tests using the head. As a result the resonant frequency of the apparatus was excited by the high frequency content of the failures in some of the failure tests. This was not a problem for the lower cervical spine motion segments because the overall mass of the system was much less – in those tests the resonant frequency was approximately 500 Hz. In order to reduce the noise from the 500 Hz mode, the results were filtered at 450 Hz. While this removed the oscillation from the motion segment tests, it did not do so for the tests owing to the lower natural frequency.
FIGURE 22: Comparison of tensile tolerance of the upper cervical spine (O-C2) and two lower (C4-C5/C6-C7) cervical segments from the current pediatric PMHS study. Tensile tolerance monotonically increases with age across all spinal levels.

The results of this study compare well with the only other currently published biomechanical data on the pediatric cervical spine. Ouyang et al. (2005), conducted non-destructive tensile stiffness tests on whole pediatric cervical spines, reporting a single mean stiffness for the entire sample age range from 2 to 12 years. The linear tensile stiffness of 34.7 ± 5.7 N/mm reported by Ouyang falls within the range of stiffnesses determined in this study for the whole cervical spine (9.7 to 70.1 N/mm). However, the Ouyang testing fixture used a free cranial boundary condition as opposed to the fixed boundary condition present in this study, which could result in a substantially more compliant structure, depending on the eccentricity of the tensile load and the potential for head rotation. In addition, their stiffnesses are reported over a greater load range.
### Table 6: Injury Descriptions Associated with Failure Tests

<table>
<thead>
<tr>
<th>PHMS ID</th>
<th>Age</th>
<th>Sex</th>
<th>Segment</th>
<th>Injury Descriptions</th>
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</thead>
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<tr>
<td>02P</td>
<td>20 WKG</td>
<td>F</td>
<td>WC5 (CC-00)</td>
<td>Physi failure (superior physi failure of C5)</td>
</tr>
<tr>
<td></td>
<td>[0 MN]</td>
<td></td>
<td>CC2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C45</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td></td>
</tr>
<tr>
<td>13P</td>
<td>29 WKG</td>
<td>F</td>
<td>[5 WPB]</td>
<td>Atlanto-axial dislocation with Type III dens fracture through fixation (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior])</td>
</tr>
<tr>
<td></td>
<td>[0 MN]</td>
<td></td>
<td>CC2</td>
<td>Physi failure (superior physi failure at C5)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>C45</td>
<td>(superior physi failure at C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>Physi failure (superior physi failure at C7)</td>
</tr>
<tr>
<td>09P</td>
<td>33 WKG</td>
<td>M</td>
<td>[0 MN]</td>
<td>Atlanto-axial dislocation with Type III dens fracture (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior])</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC2</td>
<td>Physi failure (superior physi failure of C7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C46</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td>07P</td>
<td>33 WKG</td>
<td>M</td>
<td>[0 MN]</td>
<td>Atlanto-axial dislocation with Type III dens fracture (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior])</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>CC2</td>
<td>Physi failure (superior physi failure of C6)</td>
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<td>C45</td>
<td>Physi failure (superior physi failure of C5)</td>
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</tr>
<tr>
<td>10P</td>
<td>36 WKG</td>
<td>M</td>
<td>[0 MN]</td>
<td>Atlanto-axial dislocation with Type III dens fracture (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior])</td>
</tr>
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<td>CC2</td>
<td>Physi failure (superior physi failure of C6)</td>
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<td>C46</td>
<td>Physi failure (superior physi failure of C6)</td>
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<td></td>
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<td>C67</td>
<td>Physi failure (superior physi failure at C7)</td>
</tr>
<tr>
<td>05P</td>
<td>37.5 WKG</td>
<td>M</td>
<td>[0 MN]</td>
<td>C2-C3 dislocation with associated C2 fractures (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior], fx through right lateral mass, inferior-superior oblique fx line exiting at superior articulating process; bilateral fx through C5 lamina proximal to posterior synchondrosis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC2</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>C45</td>
<td>(superior physi failure at C6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>(superior physi failure at C6)</td>
</tr>
<tr>
<td>05P</td>
<td>1 DY</td>
<td>F</td>
<td>[0.03 MN]</td>
<td>Physi failure (superior physi failure of C2) - C3 fractures through right neurocentral and posterior synchondroses</td>
</tr>
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<td></td>
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<td></td>
<td>CC2</td>
<td>Physi failure (superior physi failure of C4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C46</td>
<td>Physi failure (superior physi failure of C4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>Physi failure (superior physi failure of C7)</td>
</tr>
<tr>
<td>03P</td>
<td>3 DY</td>
<td>M</td>
<td>[0.1 MN]</td>
<td>Physi failure (superior physi failure of C4) with bilateral C4 neurocentral synchondroses fractures, bilateral C5 lamina fractures</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC2</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C45</td>
<td>Physi failure (superior physi failure of C4) with unilateral right C6 neurocentral synchondrosis fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td>08P</td>
<td>11 DY</td>
<td>F</td>
<td>[0.37 MN]</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
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<td></td>
<td>CC2</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>C45</td>
<td>Physi failure (superior physi failure of C4)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td>11P</td>
<td>16 DY</td>
<td>F</td>
<td>[0.33 MN]</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
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<td></td>
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<td>CC2</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>C45</td>
<td>Physi failure (superior physi failure of C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td>04P</td>
<td>24 DY</td>
<td>F</td>
<td>[0.8 MN]</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC2</td>
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<td></td>
<td>C45</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C67</td>
<td>Physi failure (superior physi failure of C6)</td>
</tr>
</tbody>
</table>
Pediatric PMHS tensile tolerance increased with age for each motion segment tested. This increase in strength is particularly rapid during the first year of life. Two previous studies reported tolerance in the pediatric PMHS; although both study designs differed from the current study in that Duncan (1874) distracted whole PMHS neonates to failure while Ouyang et al. (2005) used different boundary conditions. Recognizing these differences, it is not surprising that the failure loads reported by Ouyang et al. (2005) are substantially lower than those reported here. Interestingly though, in the youngest of the specimens tested by Ouyang et al. (2005) the failure loads were similar
to the current study. By contrast, the failure loads of Duncan’s study are more than two
standard deviations higher when compared to the mean of the current studies perinatal
and neonatal cohort. Prior whole PMHS studies from adult donors have reported
similar differences suggesting that soft tissue structures in the neck significantly affect
strength. Computational modeling of the pediatric neck that includes muscle effects will
be useful in examining the effects of the soft tissues.

**FIGURE 23:** Tensile stiffness (N/mm) of the upper cervical spine (O-C2) segment from the current
pediatric PMHS study compared to three previous juvenile animal surrogate studies (Pintar et al.
2000, Ching et al. 2001 and Nuckley et al. 2006). The stiffness trends for the juvenile animal
studies compare well with the human.

Prior to the occurrence of ultimate failure a number of cervical segments
experienced an initial sub-catastrophic failure event. These sub-catastrophic failures
were relatively common in the lower cervical segments of the perinatal/neonatal cohort;
however, fewer than 50% of the upper cervical segments in the same cohort experienced
such failures. Initial failure in the C4-C5 and C6-C7 segments occurred at 97% and 92% of ultimate failure, respectively. Within the older cohort, initial sub-catastrophic failures occurred in 75% or more of the specimens tested in both the upper and two lower cervical spine segments.

**Figure 24:** Tensile tolerance of the upper cervical spine (O-C2) compared to previous juvenile animal surrogate studies scaled to the human and FMVSS 208 (Hilker et al. 2002, Ching et al. 2001, Eppinger et al. 2000). Tensile tolerances are higher than the scaled data reported by each of the juvenile animal surrogate studies and FMVSS 208. The juvenile animal studies used an age estimation protocol to relate each of their animal donors with a corresponding human equivalent age. An underestimation of tolerance by the animal studies exist that may imply an improved basis for human equivalency is necessary for using animal surrogates to predict pediatric human tolerance.

PMHS stiffness versus age relationships for the upper cervical spine was accurately described by previously published animal studies (Figure 23). Regression of the baboon and caprine data sets against the human data, revealed correlation coefficients greater than 0.84. Mean absolute deviation between the animal models and
the PMHS data was 3% for donors between the ages of 1-14 years, supporting the use of the animal models.

Neither the studies on juvenile animals nor FMVSS 208 correctly predicted the tolerance of the human pediatric cervical spine. An adult value of 2417 N for human tensile tolerance (Dibb et al. 2009) was used with the scale factors from the caprine study of Hilker et al. (2002), the baboon study of Ching et al. (2000), and from FMVSS 208 (Eppinger et al. 2000). Both the animal models and FMVSS 208 underestimated the tolerance of the pediatric PMHS spine (Figure 24). This was particularly true for ages less than two years. The human appears to strengthen dramatically in the first two years of life compared to the animal models. While FMVSS 208 predicts the one year PMHS tolerance better than the caprine model, it still underestimates the human by almost 100%. It is noteworthy that the nine year PMHS had 80% of the adult strength (Dibb et al. 2009; Chancey et al. 2003) and that the fourteen year PMHS was 20% stronger than the mean adult. The average age of the donors in the adult tests was 58 years, which suggests that the older population may be significantly weaker than young adults in tension. The adult animals in the Hilker and Ching studies were young adults. Accepting this assertion, it may be appropriate to use a value larger than 2417 for scaling, though this will require additional data from teenage donors or older animals to assess with confidence. In addition, the scale factors reported by Hilker and Ching were based on the average strength of all cervical segments at a given age. The current
pediatric PMHS data presented in Figure 24 is from the O-C2 segment, which is the strongest of the cervical segments.

Review of the work of Hilker and Ching suggests that if the scale factors were based on their O-C2 data rather than the averaged data, the predictive ability of the animal models would be even worse.

Absent PMHS data, we have relied on animal surrogates to develop stiffness and tolerance versus age relationships. The results of this study suggest that juvenile animal surrogates are appropriate models for estimating cervical spine stiffness in the pediatric human population; however, these models do not provide accurate estimates of pediatric osteoligamentous spine tolerance. The experiments using caprine and baboon models employed an age estimation protocol to relate their respective models to a human equivalent age. These protocols relied on matching the developmental anatomy of the animal surrogates to known developmental anatomy of the maturing child. A comparison of the work of Pintar et al. (2000), Ching et al. (2001), and Nuckley et al. (2006) showed a high correlation between the juvenile caprine and baboon surrogate studies estimated stiffness-age relationship in the upper cervical spine. By contrast, when scaled to human, these models underestimate pediatric upper cervical spine tolerance. Based on the current data, it is unclear if the lack of predictive ability is the result of differences in the structural properties or a result of the age-estimation techniques.
5.6 Conclusion

The results of the current study indicate that previously published cervical spine stiffness data from juvenile animal models compare well to pediatric PMHS stiffness in the upper cervical spine. Juvenile animal models, scaled to the human, under-estimate upper cervical spine tolerance when compared to the PMHS upper cervical spine strength in the current study. Pediatric PMHS cervical spine stiffness and strength are observed to increase with age. The perinatal/neonatal whole spine and upper cervical spine are significantly more compliant than the lower cervical segments of the same cohort. Tensile strength of the perinatal/neonatal cohort showed no significant differences by cervical level. The older cohort had significant differences in tensile strength across levels.

5.7 Acknowledgments

This study was supported by the NHTSA Cooperative Agreement No. DTNH22-94-07133 and DTNH22-08-H-00187. The authors wish to thank Dr. Don Frush, Rachael Brady, Dr. V.C. Chancey, Laura Tran and Danielle Ottaviano for their assistance in the completion of this study.
6. Tensile Failure Properties of the Perinatal, Neonatal and Pediatric Cadaveric Cervical Spine


6.1 Acknowledgments

The authors wish to thank Steven J. Owen and John H. Goodfellow for assisting in machining and construction of the experimental testing device. The authors wish to thank the following for experimental assistance through the course of this work: Danielle Ottaviano, Lucy Fronheiser, Laura Tran, V. Carol Chancey, Michael Prange and Alan Dibb.

6.2 Mini Abstract

The tensile failure properties of the cervical spine were assessed from birth to young adulthood using cadaveric osteoligamentous head-neck complexes. Tensile stiffness and load-to-failure increased non-linearly by age, while normalized axial displacement decreased non-linearly by age. Increased ligamentous laxity in children and SCIWORA is supported quantitatively by the current findings.
6.3 Structured Abstract

6.3.1 Study Design

Biomechanical tensile testing of perinatal, neonatal and pediatric cadaveric cervical spines to failure.

6.3.2 Objective

To assess the tensile failure properties of the cervical spine from birth to adulthood.

6.3.3 Summary of Background Data

Pediatric cervical spine biomechanical studies have been few owing to the limited availability of pediatric cadavers. Therefore, scaled data based on human adult and juvenile animal studies have been used to augment the limited pediatric cadaver data. Despite these efforts, substantial uncertainty remains in our understanding of pediatric cervical spine biomechanics.

6.3.4 Methods

A total of 24 cadaveric osteoligamentous head-neck complexes, 20 weeks gestation to 18 years, were sectioned into segments (O-C2, C4-C5, and C6-C7) and tested in tension to determine axial stiffness, displacement at failure and load-to-failure.
### 6.3.5 Results

Tensile stiffness-to-failure (N/mm) increased by age (O-C2: 23-fold, neonate - 22 ± 7, 18 years - 504; C4-C5: seven-fold, neonate - 71 ± 14, 18 years - 509; C6-C7: seven-fold, neonate - 64 ± 17, 18 years - 456). Load-to-failure (N) increased by age (O-C2: 13-fold, neonate - 228 ± 40, 18 years - 2888; C4-C5: nine-fold, neonate - 207 ± 63, 18 years - 1831; C6-C7: 10-fold, neonate - 174 ± 41, 18 years - 1720). Normalized displacement at failure (mm/mm) decreased by age (O-C2: six-fold, neonate – 0.34 ± 0.076, 18 years – 0.059; C4-C5: three-fold, neonate – 0.092 ± 0.015, 18 years – 0.035; C6-C7: two-fold, neonate – 0.088 ± 0.019, 18 years – 0.037).

### 6.3.6 Conclusions

Cervical spine tensile stiffness-to-failure and load-to-failure increased non-linearly, while normalized displacement at failure decreased non-linearly, from birth to adulthood. Pronounced ligamentous laxity observed at younger ages in the O-C2 segment quantitatively supports the prevalence of spinal cord injury without radiographic abnormality (SCIWORA) in the pediatric population. This study provides important and previously unavailable data for validating pediatric cervical spine models, for evaluating current scaling techniques and animal surrogate models, and for the development of more biofidelic pediatric crash test dummies.
6.3.7 Key words

Spine biomechanics, Pediatric, Tension, Stiffness, Load-to-failure, Neonate, SCIWORA, Pseudosubluxation

6.4 Key Points

1. Tensile stiffness derived from failure tests increases non-linearly with age, from birth to young adulthood, in O-C2, C4-C5 and C6-C7 cervical motion segments.

2. Tensile load-to-failure increases non-linearly with age, from birth to young adulthood, in O-C2, C4-C5 and C6-C7 cervical motion segments.

3. Normalized axial displacement at failure decreases non-linearly with age in the upper and lower cervical spine, most notably in the O-C2 segment and provides quantitative support to the idea of increased ligamentous laxity in children as seen clinically in non-pathologic pseudosubluxation and traumatic spinal cord injuries associated with SCIWORA.

4. Yielding behavior at sub-catastrophic failure levels in the lower cervical segments and stiffening behavior in the upper cervical spine should be considered when estimating tensile stiffness-to-failure of motion segments and whole pediatric cervical spines.

5. Tensile loading produced clinically observed injuries in the upper and lower cervical spine representative of injury patterns observed in children and to maturity.
6.5 Introduction

Biomechanical studies of the pediatric cervical spine have been limited in number, yet this critical anatomical structure when injured leads to relatively high morbidity and mortality. Moreover, the cervical spine governs the kinematics of the head-neck structure. The biomechanics of the pediatric cervical spine are important in both neck injury prevention and the understanding of head injury. Motor vehicle crashes are the most common cause of spinal injuries in children (Platzer et al. 2007; Brown et al. 2001; Kokoska et al. 2001; Eleraky et al. 2000; Hamilton and Myles 1992a; Bonadio 1993; Babcock 1975). Pediatric spinal trauma accounts for between 1-12% of all spinal injuries (Hamilton and Myles 1992a; Hadley et al. 1988; Aufdermaur 1975). The overall mortality rate among victims of pediatric spinal trauma is approximately 16-41% but is considerably higher for the youngest ages (Platzer et al. 2007; Brown et al. 2001; Kokoska et al. 2001; Eleraky et al. 2000; Givens et al. 1996; Orenstein et al. 1994; Hamilton and Myles 1992b).

Advances in occupant protection technologies over the last three decades dramatically reduced serious injuries and fatalities for all motor vehicle occupants (Viano 1995; Huelke et al. 1981; Hartemann et al. 1977). However, the ability to survive large decelerations creates an increased potential for neck injuries from inertial loading (Huelke et al. 1978; Huelke et al. 1992; Huelke et al. 1993; Huelke et al. 1995). Greater head mass in relation to overall body proportions, ligamentous laxity of the cervical
spine, decreased facet angle and a relatively slender cervical spine, are all potential factors that may increase the risk of cervical spine injury in children (Kasai et al. 1996; Swischuk 1977; Cattell and Filtzer 1965; Bailey 1952).

Few studies have investigated the biomechanical behavior of the cadaveric pediatric cervical spine owing, in large measure, to a lack of donors. The four studies to date vary significantly in design and typically have small sample sizes. Duncan (1874) investigated the force required to inflict injury to the neonate cervical spine during a breech delivery. Ouyang et al. (2005) reported the tensile responses of the pediatric whole cervical spine (PWCS) under both non-destructive and failure loads. Luck et al. (2008) reported the quasi-static tensile stiffness and ultimate load-to-failure of a subset of the data used in the current study. Kallieris et al. (1976) and Wismans et al. (1979) investigated the response of whole body pediatric cadavers to simulated automotive collisions. Owing to limited human data, numerous animal studies were used as human surrogates (Nuckley and Ching 2006; Hilker et al. 2002; Ching et al. 2001; Pintar et al. 2000; Mertz and Weber 1982; Mertz et al. 1982; Prasad and Daniel 1984; Nuckley et al. 2002; Nuckley et al. 2005).

No studies have evaluated both the structural and failure properties of individual pediatric cervical segments from birth through maturity, and current studies are insufficient to evaluate animal scaling techniques widely used to characterize pediatric force response. The goal of the current study is to provide additional
biomechanical response data on pediatric cervical spines across all ages. These data will be useful in evaluating current scaling techniques for developing biofidelity requirements and injury assessment reference values for pediatric dummies. They also provide additional insights into the mechanisms of cervical spine injuries in children.

6.6 Materials and Methods

Twenty-four whole unembalmed cervical spines from 20 weeks gestation to 18 years were tested (Table 7). Specimens included eleven perinatal and neonatal (younger cohort: 20 weeks gestation - 24 days) and thirteen infant to young adults (older cohort: 5 month - 18 years). Specimens were transected to obtain three segments (Occiput-C2 [O-C2], C4-C5, C6-C7) that were loaded to failure. Neck musculature, subcutaneous tissue, and skin were removed, and the mandible was disarticulated enabling rigid head fixation and visualization of O-C2. O-C2 was secured to the testing apparatus at the maxilla and the occipital region with the Frankfort plane aligned horizontally (Figure 25-1A). The caudal end of the spine (C2/C3) was potted in polymethyl-methacrylate (Dentsply International; York, PA) and epoxy acrylic resin (Acsys Orthopedic; Vista, CA) and was attached to an actuator (MTS Systems Corporation, Eden Prairie, MN) (Luck et al. 2008; Luck et al. 2012a). The two lower segments were mounted with the cranial and caudal vertebra cast in aluminum cups (Figure 25-1B). Details of quasi-static testing and the rate of loading for each load-to-failure test are described in Luck et al. (2008); with the exception of specimens T19P-T24P that were loaded at 230 mm/sec.
<table>
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<th>Age (MN)</th>
<th>Sex</th>
<th>COD</th>
<th>Whole Body</th>
<th>Head</th>
<th>Spine</th>
<th>Scaled Loading Rate (N/s)</th>
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<td></td>
<td></td>
<td></td>
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<td>Height (cm)</td>
<td>Mass (kg)</td>
<td>Breadth (cm)</td>
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<td>10.2</td>
<td>3.40</td>
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<tr>
<td>09P</td>
<td>0</td>
<td>M</td>
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<td>43.2</td>
<td>SC</td>
<td>9.1</td>
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<tr>
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</tr>
<tr>
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<td>Fetal Demise</td>
<td>-</td>
<td>SC</td>
<td>7.9</td>
<td>10.0</td>
</tr>
<tr>
<td>08P</td>
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<td>M</td>
<td>P-Hyp</td>
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<td>11.4</td>
</tr>
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<td>F</td>
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<td>HE; cerebral infarction</td>
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<td>F</td>
<td>DWS</td>
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<td>45.7</td>
<td>SC</td>
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<td>12P</td>
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<td>F</td>
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<td>F</td>
<td>NHL</td>
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<td>-</td>
<td>12.5</td>
<td>16.4</td>
</tr>
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<td>-</td>
<td>14.2</td>
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<td>F</td>
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<td>-</td>
<td>14.9</td>
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<td>-</td>
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<td>160.0</td>
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<td>Overdose</td>
<td>-</td>
<td>-</td>
<td>SC</td>
<td>-</td>
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</tbody>
</table>

**Equation for area of an ellipse**: \( \frac{1}{2} \pi \cdot W \cdot D \) used to approximate area of T1 endplate for scaled loading rate

**PMHS - Post-Mortem Human Subject**

**COD - Cause of Death**

**MN - months; SC - skull compromise (PMHS acquired in this state)**

**P-Hyp - Pulmonary Hypoplasia; COPD - Chronic Obstructive Pulmonary Disease; SIDS - Sudden Infant Death Syndrome**

**NIHF - Non-immune hydrops fetalis; ICH - Intercranial Hemorrhage; NHL - Non-Hodgkin's Lymphoma; GCT - Germ Cell Tumor (Carcinoma)**

**ESRD - End-stage Renal Disease; ↑K+ - Hyperkalemia; HE - Hypoxic Ischemic Encephalopathy; DWS - Dandy-Walker Syndrome**
FIGURE 25: (1A) Upper cervical spine (O-C2) cast in the experimental apparatus with rigid fixation of the cranium and C2-C3 cup fixation caudally enabling axial distraction of the spine. (1B) Lower cervical spine (C4-C5 and C6-C7) cast with a cup-cup fixation.

Six-axis load (2554A, Denton, Inc., Rochester Hills, MI) and axial displacement (Schaevitz Sensors, Hampton, VA) was recorded (National Instruments, Austin, TX). A high-rate video camera recorded cervical segment motion (Vision Research, Inc., Wayne, NJ) and determined the time and location of failure. Post failure dissection was completed on all segments to document injuries.

All axial displacement measurements were adjusted for frame-cup compliance (O-C2) and cup-cup compliance (C4-C5 and C6-C7). This adjustment removed displacement associated with compliance of the fixation techniques and testing apparatii (Luck et al. 2012a; Nightingale et al. 2004).
Minor, major and ultimate tensile load-to-failure was determined for each test. Minor failure was defined by a decrease in load with increasing displacement in the force-displacement response, followed by an increase in load with increasing displacement. Major failure was defined by a decrease in load and/or stiffness of the specimen with continued loading and coincident with evidence of tissue damage. Ultimate failure was defined as the maximum load in the force-displacement response. Tensile stiffness was determined from the slope of a linear regression from 20-80% of the loading portion up to the major failure point of the force-displacement response. This stiffness estimation technique attempted to capture the structural characteristics of the segment after the low-load region and prior to yielding associated with major failure. Analysis of the effects of yielding on the structural response of the specimens prior to the defined major load-to-failure point was evaluated by examining the stiffness defined from 20-50% of major failure. The two stiffness estimates were compared over all specimens by examining the ratio of these quantities.

Displacements at minor, major and ultimate tensile load-to-failure were determined for each failure test. The displacement was normalized by the cervical spine length, defined as the distance between the anterior tubercle of atlas (C1) and the anterio-inferior mid-point of the C7 vertebral body.

The biomechanical properties were regressed by age with a power-law function 

\[ E^{-5}; Y, \text{ biomechanical property}; \ \text{AGE (months)}; A, B \text{ and } C \text{ are model parameters}; i, \]
biomechanical property; $j$, motion segment; $k$, failure-type). Goodness-of-fit was assessed using the root mean square error and coefficient of determination ($R^2$). Statistical significance of the regressions was assessed using a single-factor ANOVA ($P<0.05$).

\[ Y_{i,j,k} = A_{i,j,k}[AGE]^{B_{i,j,k}} + C_{i,j,k} \]  \hspace{2cm} (E-5)

Statistical analysis of the biomechanical responses of the three segments of the younger cohort were compared using a single-factor ANOVA, followed by a post-hoc Tukey-Kramer HSD multiple-comparison test ($P<0.05$). Tensile stiffness estimation methods were compared using a Student’s t-test ($P<0.01$).

### 6.7 Results

**6.7.1 Perinatal, Neonatal and Pediatric Cervical Spine Tensile Failure and Stiffness**

Major and ultimate load-to-failure increased for all cervical levels by approximately an order of magnitude from the perinate to the young adult (Table 8). A thirteen-fold increase was observed in O-C2, while C4-C5/C6-C7 saw 9-12 fold increases. No significant differences were found in the major or ultimate load-to-failure by level within the younger cohort (major: $P=0.13$, $n=24$; ultimate: $P=0.22$, $n=24$).
### Table 8: Tensile Load-to-Failure for Perinatal, Neonatal and Pediatric PMHS

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<td>57</td>
<td>-</td>
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<td>-</td>
<td>-</td>
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<td>-</td>
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<td>N/O 183</td>
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<td>-</td>
<td>-</td>
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</table>

#### Average
- O-C2
  - Minor: 228
  - Major: 231
  - Ultimate: 207
- C4-C5
  - Minor: 207
  - Major: 213
  - Ultimate: 174
- C6-C7
  - Minor: 187
  - Major: 187
  - Ultimate: 174

#### SD
- O-C2
  - Minor: 40
  - Major: 38
  - Ultimate: 63
- C4-C5
  - Minor: 61
  - Major: 61
  - Ultimate: 41
- C6-C7
  - Minor: 39
  - Major: 39
  - Ultimate: 39

### Additional Notes
- ¥ - O-C6 segment; † - C3-C4 segment; ‡ - C5-C6 segment; Σ - Occ-C1-C2 re-cast test
- - Specimen not available; N/O - Not Observed; N/A - Not Applicable; DAP - Data Acquisition Problem
Table 9. Displacement at Failure for Perinatal, Neonatal and Pediatric PMHS

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>O-C2 Minor (mm)</th>
<th>O-C2 Major (mm)</th>
<th>O-C2 Ultimate (mm)</th>
<th>C4-C5 Minor (mm)</th>
<th>C4-C5 Major (mm)</th>
<th>C4-C5 Ultimate (mm)</th>
<th>C6-C7 Minor (mm)</th>
<th>C6-C7 Major (mm)</th>
<th>C6-C7 Ultimate (mm)</th>
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</thead>
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<td>8.00</td>
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<td>4.13</td>
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<tr>
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<td>17.09</td>
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<td>4.13</td>
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</tr>
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<td>—</td>
<td>—</td>
<td>N/O</td>
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<td>3.90</td>
<td>N/O</td>
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<td>4.17</td>
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<td>1.22</td>
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<tr>
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<td>14.31</td>
<td>17.92</td>
<td>N/O</td>
<td>4.96</td>
<td>4.96</td>
<td>N/O</td>
<td>4.47</td>
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<tr>
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<td>N/O</td>
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<td>2.22</td>
<td>9.01</td>
<td>13.12</td>
<td>2.94</td>
<td>4.76</td>
<td>6.61</td>
<td>N/O</td>
<td>4.53</td>
<td>7.27</td>
</tr>
<tr>
<td>16P † ‡</td>
<td>18</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>N/O</td>
<td>5.23</td>
<td>7.91</td>
<td>N/O</td>
<td>2.28</td>
<td>4.44</td>
</tr>
<tr>
<td>17P</td>
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<td>11.26</td>
<td>14.13</td>
<td>2.31</td>
<td>2.83</td>
<td>5.76</td>
<td>N/O</td>
<td>4.99</td>
<td>8.58</td>
</tr>
<tr>
<td>24P</td>
<td>72</td>
<td>10.35</td>
<td>11.60</td>
<td>11.60</td>
<td>1.98</td>
<td>3.33</td>
<td>5.07</td>
<td>N/O</td>
<td>2.29</td>
<td>5.20</td>
</tr>
<tr>
<td>24P Σ</td>
<td>84</td>
<td>N/O</td>
<td>6.12</td>
<td>6.62</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>19P</td>
<td>108</td>
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<td>3.85</td>
<td>5.19</td>
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<tr>
<td>18P</td>
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<td>N/O</td>
<td>10.71</td>
<td>13.14</td>
<td>1.64</td>
<td>7.30</td>
<td>7.30</td>
<td>N/O</td>
<td>4.31</td>
<td>5.93</td>
</tr>
<tr>
<td>01P</td>
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<td>6.70</td>
<td>8.06</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>21P</td>
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<td>N/O</td>
<td>7.73</td>
<td>10.16</td>
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<tr>
<td>22P</td>
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<td>7.83</td>
<td>13.16</td>
<td>N/O</td>
<td>3.30</td>
<td>6.11</td>
<td>N/O</td>
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<td>7.72</td>
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<tr>
<td>23P</td>
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<td>7.35</td>
<td>9.05</td>
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<td>6.87</td>
<td>N/O</td>
<td>4.59</td>
<td>6.99</td>
</tr>
</tbody>
</table>

¥ - O-C6 segment; † - C3-C4 segment; ‡ - C5-C6 segment; Σ - Occ-C1-C2 re-cast test
— - Specimen not available; N/O - Not Observed; N/A - Not Applicable; DAP - Data Acquisition Problem
### Table 10: Tensile Stiffness for Perinatal, Neonatal and Pediatric PMHS (N/mm)

**Pediatric PMHS < 1 Month**

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Tensile Stiffness from Major Failure Point (20-80%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>02P</td>
<td>¥</td>
<td>7.56 0.93</td>
</tr>
<tr>
<td>07P</td>
<td>0</td>
<td>23.7 1.08</td>
</tr>
<tr>
<td>08P</td>
<td>0</td>
<td>17.0 0.97</td>
</tr>
<tr>
<td>09P</td>
<td>0</td>
<td>62.9 0.90</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
<td>61.3 0.90</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>34.1 1.57</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>20.7 1.07</td>
</tr>
<tr>
<td>03P</td>
<td>0.1</td>
<td>26.4 1.10</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>14.4 1.09</td>
</tr>
<tr>
<td>11P</td>
<td>0.53</td>
<td>75.8 1.01</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>19.6 1.00</td>
</tr>
</tbody>
</table>

**Average**

<table>
<thead>
<tr>
<th>O-C2</th>
<th>Ratio</th>
<th>C4-C5</th>
<th>Ratio</th>
<th>C6-C7</th>
<th>Ratio</th>
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</thead>
<tbody>
<tr>
<td>22.3</td>
<td>-</td>
<td>70.9</td>
<td>-</td>
<td>63.5</td>
<td>-</td>
</tr>
<tr>
<td>6.6</td>
<td>-</td>
<td>14.3</td>
<td>-</td>
<td>16.7</td>
<td>-</td>
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</table>

**Pediatric PMHS > 1 Month**

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Tensile Stiffness from Major Failure Point (20-80%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12P</td>
<td>5</td>
<td>35.2 1.06</td>
</tr>
<tr>
<td>14P</td>
<td>9</td>
<td>83.7 1.08</td>
</tr>
<tr>
<td>15P</td>
<td>11</td>
<td>114.0 0.92</td>
</tr>
<tr>
<td>16P</td>
<td>† ‡</td>
<td>18</td>
</tr>
<tr>
<td>17P</td>
<td>22</td>
<td>130.0 1.06</td>
</tr>
<tr>
<td>24P</td>
<td>72</td>
<td>195.3 1.09</td>
</tr>
<tr>
<td>24P</td>
<td>Σ</td>
<td>423.9 1.14</td>
</tr>
<tr>
<td>19P</td>
<td>84</td>
<td>181.3 0.95</td>
</tr>
<tr>
<td>18P</td>
<td>108</td>
<td>263.0 0.90</td>
</tr>
<tr>
<td>20P</td>
<td>144</td>
<td>305.0 0.92</td>
</tr>
<tr>
<td>01P</td>
<td>168</td>
<td>531.0 1.28</td>
</tr>
<tr>
<td>21P</td>
<td>192</td>
<td>298.2 0.97</td>
</tr>
<tr>
<td>22P</td>
<td>Δ</td>
<td>204</td>
</tr>
<tr>
<td>23P</td>
<td>Γ</td>
<td>216</td>
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**Average**

<table>
<thead>
<tr>
<th>O-C2</th>
<th>Ratio</th>
<th>C4-C5</th>
<th>Ratio</th>
<th>C6-C7</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
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<td>59.3</td>
<td>0.90</td>
</tr>
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<td>188.9</td>
<td>1.06</td>
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<td>N/A</td>
<td>N/A</td>
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<td>455.6</td>
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</tbody>
</table>

¥ - O-C6 segment; † - C3-C4 segment; ‡ - C5-C6 segment; Δ - front half of skull not present (maxilla and anterior half of skull modeled for fixation purposes); Γ - craniotomy, skull cap rigidly fixated to base of skull and maxilla; Σ - Re-cast, base of skull test

- Specimen not available; DAP - Data acquisition problem; N/A - Not applicable

Ratio - (20-80% Stiffness)/(20-50% Stiffness)
Displacement at failure, within the younger cohort, was significantly different by level and approximately 3-4 times greater in the O-C2 segment compared to the C4-C5/C6-C7 segments (Table 9, major and ultimate: $P<0.01$; $n=24$). There were no significant differences between C4-C5 and C6-C7 (major: $P=0.99$; ultimate: $P=0.82$).

Tensile stiffness based on the major failure point increased for all cervical levels by seven-fold and up to 23-fold in the O-C2 segment from the perinate to the young adult (Table 10). Within the younger cohort, the O-C2 segment was three times less stiff than the C4-C5/C6-C7 segments ($P<0.01$). There were no significant differences in stiffness between C4-C5 and C6-C7 ($P=0.59$).

**Figure 26:** Power law models of the major and ultimate load-to-failure for O-C2, C4-C5 and C6-C7 from birth to young adulthood. Load-to-failure increased with increasing age in a non-linear form. A rapid increase in the load-to-failure with increasing age occurs in the early years of life followed by a continual but less rapid increase to young adulthood.
### Table 11: Age Based Pediatric Regression Models for Axial Load-to-Failure, Displacement at Failure, Normalized Displacement and Stiffness

<table>
<thead>
<tr>
<th>Segment Level</th>
<th>Failure Type</th>
<th>Dependent Variable (Y)</th>
<th>Non-Linear Regression Models</th>
<th>Goodness of Fit</th>
</tr>
</thead>
<tbody>
<tr>
<td>O-C2</td>
<td>Ultimate Axial Load</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>$A$</td>
<td>$B$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>175.24</td>
</tr>
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<td>-0.02</td>
</tr>
<tr>
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<td></td>
<td>Normalized O-C2 Disp Axial Load</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
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<tr>
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<td>Displacement</td>
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<tr>
<td></td>
<td>Major</td>
<td>Normalized O-C2 Disp 20-80% Stiffness</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>-5.54E-02</td>
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<tr>
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</tr>
<tr>
<td>C4-C5</td>
<td>Ultimate Axial Load</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>$A$</td>
<td>$B$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>19.36</td>
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<tr>
<td></td>
<td></td>
<td>Normalized C4-C5 Disp Axial Load</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>-2.51E-02</td>
</tr>
<tr>
<td></td>
<td>Displacement</td>
<td>Normalized O-C2 Disp 20-80% Stiffness</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>26.35</td>
</tr>
<tr>
<td></td>
<td>Major</td>
<td>Displacement</td>
<td></td>
<td>5.78E-04</td>
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<tr>
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<td>Normalized C4-C5 Disp 20-50% Stiffness</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>$A$</td>
<td>$B$</td>
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<td>-2.42E-02</td>
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<tr>
<td>C6-C7</td>
<td>Ultimate Axial Load</td>
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<td>$B$</td>
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<td>Displacement</td>
<td>Normalized C6-C7 Disp Axial Load</td>
<td>$Y_{i,j,k} = A_{i,j,k}(\text{AGE})^{B_{i,j,k}} + C_{i,j,k}$</td>
<td>-8.16E-01</td>
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<tr>
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<td></td>
<td>Displacement</td>
<td></td>
<td>-2.04E-02</td>
</tr>
<tr>
<td></td>
<td>Major</td>
<td>Normalized C6-C7 Disp 20-80% Stiffness</td>
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<td>20-50% Stiffness</td>
<td></td>
<td></td>
<td>-0.23</td>
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<tr>
<td>LCS Combined</td>
<td>Ultimate Axial Load</td>
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<td>$B$</td>
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<td></td>
<td></td>
<td></td>
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<td>30.53</td>
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<tr>
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<td>Displacement</td>
<td>Normalized LCS Disp Axial Load</td>
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<td>Normalized LCS Disp Displacement</td>
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<td>-2.20E-02</td>
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<tr>
<td></td>
<td>Major</td>
<td>Normalized LCS Disp 20-80% Stiffness</td>
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<td></td>
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<td>-1.67E-02</td>
</tr>
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</table>

Model subscripts: (i) biomechanical property, (j) segment level, (k) failure type; LCS - Lower cervical spine

Single-Factor ANOVA significance levels: ¥ - $(P < 0.05)$; § - $(P < 0.01)$; • - statistically insignificant

$R^2 = 1 - \frac{\text{SSE}}{\text{SST}}$
6.7.2 Age Based Regression Models

Load-to- major and ultimate failure in O-C2 was well correlated and statistically significant with age (Figure 26 and Table 11). The regression captures the rapid increase in load-to-failure in the early years of life. Load-to-failure continues to increase although more slowly as cervical maturity is attained. Load-to-failure in C4-C5 and C6-C7 were also well correlated and statistically significant with age (Figure 26 and Table 11).

**FIGURE 27:** Normalized axial displacement at failure defined at the major load level for the O-C2, C4-C5 and C6-C7 segments. Normalized displacement decreases at all levels with age, with a steeper descent associated with the O-C2. By early adulthood the normalized displacement observed at failure in all levels of the cervical spine are estimated to be the same.

Displacement at failure was not well correlated or statistically significant with age for the majority of segment and load levels (Table 11). However, normalized displacement with age was well correlated and statistically significant at all levels (Figure 27 and Table 11). O-C2 normalized displacement at failure was six-fold greater
at birth compared to the young adult. Normalized displacement in O-C2 at birth was four-fold greater than the lower cervical spine (LCS), but approached the values of the LCS by approximately 12-14 years of age. Although a decrease in normalized displacement in the LCS was observed from the perinate to young adult (two-to-three-fold), the overall change was less than observed in the O-C2.

Tensile stiffness (20-80%) by age for O-C2 is well correlated and statistically significant (Figure 28 and Table 11). Similarly good correlation was found for the LCS segments (Figures 28 and Table 11). Tensile stiffness was observed to increase with age for all segment levels.

Injuries during the failure tests included ligamentous, cartilaginous and bony fractures of the vertebra and associated soft tissue complexes (see Appendix C, Table, Supplement Digital Content 1, Injury Descriptions). O-C2 injuries included basilar skull and occipital condyle fractures, and occipito-atlantal and atlanto-axial dislocations. C4-C5/C6-C7 injuries included complete ligamentous disruptions without fracture, vertebral fractures, physeal endplate ruptures and cartilaginous disruptions in many of the younger specimens.
**FIGURE 28:** Cervical spine tensile stiffness, based on major load-to-failure, from birth to young adulthood increases non-linearly with age in the O-C2, C4-C5 and C6-C7 segments. A more rapid increase in stiffness at the younger ages, more pronounced in the C4-C5 and C6-C7 segments, is followed by a less rapid continual increase in stiffness to young adulthood.

### 6.8 Discussion

This work substantially adds to the limited existing data on tensile stiffness and load-to-failure of the perinatal, neonatal and pediatric cadaveric cervical spine. Moreover, power-law models relating age and failure properties in the cervical spine are introduced to better approximate the biomechanical behavior of the pediatric neck.

Limitations of the current study included a limited sample size and large variability in the physical size and stiffness of the cadaveric head-neck complexes. Preservation of the head for separate biomechanical testing was also a limiting factor (Luck et al. 2012a; Prange et al. 2004). Although cervical spine pathology was not observed; some specimens had iatrogenic cranial defects that limited testing of O-C2 (T09P, T10P, T11P, T16P) and harvest damage (T16P) that necessitated testing of adjacent segments. All regressions were assessed over the age range in the current study. Extrapolating properties for ages greater than those bounded by the current study are not appropriate and may not be defined by the current models.
FIGURE 29: Yielding behavior prior to catastrophic joint failure was observed in the lower cervical spine. This behavior was observed in both younger (T14P – 9 month) and older ages (T20P – 144 month). Tensile stiffness was determined based on a catastrophic major failure point that encapsulated both a portion of the early stiff joint response as well as the more compliant upper load region (20-80% regression – black dashed line). Stiffness calculations based on a lower load, pre-yielding region, produces a stiffer overall joint response (20-50% regression – gray solid line).

Estimates of O-C2 stiffness were similar when using the two approximation techniques (20-80% vs. 20-50%), while the C4-C5 and C6-C7 segments were significantly different (O-C2: $P=0.1174$; C4-C5 and C6-C7: $P<0.01$). While, yielding in the force-displacement response of the lower segments was typical (Figure 29), a stiffening response, although not statistically significant was observed in O-C2. Yielding behavior is not uncommon in cervical segments and isolated components loaded-to-failure (Winkelstein et al. 2000; Yoganandan et al. 1989). However, estimating stiffness based
solely on loads approaching catastrophic levels may not capture the behavior at sub-
catastrophic levels. This suggests that modeling at the segmental level should account
for changes in stiffness during load-to-failure. Moreover, PWCS models based solely on
stiffness approaching catastrophic failure may not appropriately model the overall
stiffness of the spine prior to failure unless the sub-catastrophic response is accounted
for in the model.

The pediatric spine is hypothesized to be more compliant than the adult spine.
This ligamentous laxity has been postulated to be an important factor in its behavior,
Radiographic studies of children have illustrated hypermobility, including
pseudosubluxation, of the upper cervical spine (UCS) and SCIWORA (Cattell and Filtzer
1965; Pang and Wilberger 1982). SCIWORA in particular, may occur because the
cervical spine can withstand larger displacements than the spinal cord. Although
displacement at failure was only weakly correlated to age, normalized displacement at
failure was well correlated and statistically significant with age. At younger ages, O-C2
joint displacement, normalized by spinal length, required to cause catastrophic joint
injury is up to five times greater than what is required at older ages. As children
mature, the displacement they can withstand while avoiding catastrophic joint injury
does not increase at the same rate, but rather stays the same or decreases. These data
suggest that at younger ages the spine is able to withstand greater normalized
displacements without structural injury, yet the spinal cord has been documented to be unable to withstand comparable distractions. Leventhal (1960) reported on a cadaveric infant spine distracted two inches, while the spinal cord ruptured at a quarter of an inch. Although these data are from a poorly defined whole spine, making direct comparisons difficult, it is interesting to note that if all of the spinal cord distraction reported occurred in the O-C2 joint, the cord would rupture at approximately 17% normalized displacement, over 13% less than what the neonatal spine can withstand before catastrophic joint injury. Spinal cord tensile displacement to failure in the adult has been documented to be 8.9 mm (Yoganandan et al. 1996). Spinal cord rupture would occur at approximately 7% normalized displacement in the adult spine, whereas the current data indicate that catastrophic joint injury would occur between 3.5-5.5% at all spinal levels in the adult. These data support the theory that in children as compared to adults, the spine is able to withstand greater distractions without joint injury, while sustaining spinal cord injuries. Moreover, the ‘slack’ observed in younger children supports the theory that the child spine has the capacity to experience hypermobility without joint injury that may contribute to pseudosubluxation and other normal variants that may be mistaken for injury.
Figure 30: Lower cervical segment load-to-failure in the current study compared well to whole spine ultimate load-to-failure reported by Ouyang et al. (2005) at younger ages. However, at older ages, load-to-failure was greater in the current study. Whole body load-to-failure reported by Duncan (1874) was greater than LCS load-to-failure in the current study.

Current load-to-failure results are consistent with the limited previously reported human pediatric data that focused on whole body cadavers and whole spines. Duncan (1874) reported two failure loads with the four fetal and one neonatal specimens tested (initial load-to-failure: 507 ± 107 N, n=5; decapitation load: 577 ± 120 N, n=5). The initial load-to-failure was greater by approximately two-and-a-half times the load-to-failure (major) of the current study \( (P<0.01) \). This difference may partially be explained by the fact that Duncan’s work included full cadavers and the methodology used to identify load-to-failure. The inclusion of musculature and whole body tissues compared to the osteoligamentous spine is consistent with increased load-to-failure. Yoganandan et al. (1996) reported whole body cervical spine load-to-failure over two times greater than load-to-failure observed in osteoligamentous whole spines. The load-to-failure
reported by Duncan (1874) and Ouyang et al. (2005) are generally consistent with the current findings for ultimate failure of the LCS (Figure 30). One exception from Ouyang et al. (2005) is a 12-year-old specimen that was approximately 45% weaker than the current study (T20P: C4-C5, 1732 N; C6-C7, 1570 N; Ouyang: 918 N). Ouyang et al. (2005) reported stiffness and displacements from whole spines which are not directly comparable to the current study.

The structural properties of the older cohort are similar to previously reported values for the adult neck, indicating that load-to-failure may peak in young adulthood and decline with age. Dibb et al. (2009) reported the ultimate load-to-failure in adult O-C2 segments was 2-2.4 kN. Pediatric O-C2 specimens 12 years and older failed at an ultimate load-to-failure of 2771 ± 263 N (15.4 ± 2.4 years; 12-18 years; n=5) and were 36% stronger than the adult loads (2032 ± 249 N; 58.9 ± 7.1 years; 47-68 years; n=8) (P<0.01). Dibb et al. (2009) reported LCS ultimate load-to-failure between 1.7-1.9 kN which are consistently lower, although not significantly (P=0.17), than those observed in the older segment of our sample (2013 ± 315 N; C4-C5 and C6-C7). Similarly, Yoganandan et al. (1996) reported an average load-to-failure of 1.6 kN in adult whole spines that typically failed in the C6-C7 region.

Tensile O-C2 stiffness at older ages compared well to previous studies on adults. O-C2 stiffness of 400-425 N/mm from 16-18 years was calculated from the power-law model. Dibb et al. (2009) reported between 400-454 N/mm for adults, Liu et al. (1982)
reported an average of 423 N/mm and Shea et al. (1991) reported between 157-433 N/mm.

Tensile LCS stiffness was greater than adult values and attained adult levels by an early age. By age four the C4-C5 and C6-7 stiffness is 270 and 255 N/mm, respectively, while at age 18, stiffness has increased to 481 and 410 N/mm. Dibb et al. (2009) reported adult stiffness between 243-360 N/mm, which is considerably less than the stiffness estimated for the young adult.

Failure testing produced clinically observed injuries (Smith et al. 2010; Hammerstein et al. 2007; Swoboda et al. 1995; Ewald 1971). Moreover, these injuries are representative of cervical spine trauma documented in pediatric patients involved in motor vehicle crashes (Fuchs et al. 1989; Panczykowski et al. 2010; Schippers et al. 1996; Clasper and Pailthorpe 1995; Keller and Mosdal 1990; Bhattacharyya 1974; Blauth et al. 1996).

This study presents heretofore unavailable quantitative biomechanical data useful in understanding, treating, and modeling injury to the pediatric cervical spine. UCS and LCS load-to-failure and stiffness-to-failure were determined and found to increase non-linearly with age. Normalized displacement in the UCS was observed to decrease significantly with age and approach the value of the LCS by young adulthood. These data may provide a quantitative assessment for the etiology of SCIWORA and support for increased ligamentous laxity under distractive loading in the pediatric
cervical spine. Although segmental stiffness-to-failure is reported, they should be used with caution because of the yielding behavior observed in many of the lower segments. Further research is needed to determine how best to model this behavior and use the current data to estimate stiffness-to-failure properties for the PWCS.
7. The Quasi-Linear Viscoelastic Response of the Pediatric Osteoligamentous Human Cervical Spine in Tension

The following work is in the final manuscript formatting process with intended submission to the Journal of Biomechanics: Luck, J.F., R.W. Nightingale, M. Panzer, B.S. Myers, and C.R. Bass. 2012. The quasi-linear viscoelastic response of the pediatric osteoligamentous human cervical spine in tension.

7.1 Introduction

Pediatric spinal injuries, most commonly caused by motor vehicle crashes, have a relatively high morbidity and mortality (Babcock 1975; Hamilton and Myles 1992a; Bonadio 1993; Eleraky et al. 2000; Brown et al. 2001; Kokoska et al. 2001; Platzer et al. 2007). A limited number of studies have investigated the low-load and failure responses in tension and flexion-extension bending of the developing human cervical spine from birth to maturity (Duncan 1874; Ouyang et al. 2005; Luck et al. 2008; Luck et al. 2012a; Luck et al. 2012b; Luck et al. 2012c). These studies have shown that the pediatric cervical spine has a nonlinear force-displacement response, similar to that observed in the adult spinal column under various external loads (e.g. White and Panjabi 1978; Myers et al. 1991; McElhaney et al. 1988). Though the viscoelastic response of the adult cervical spine has been investigated (McElhaney et al. 1988; Myers et al. 1991), there are no available studies that characterize the structural viscoelastic behavior of the pediatric human cervical spine. Understanding this viscoelastic behavior is a crucial component
in improving the biofidelity of both computational and mechanical models to elucidate the etiology of spinal injuries in children.

Quasi-linear viscoelasticity (QLV) (Fung, 1967) is widely used to model biological soft tissues for a range of strain levels. Existing studies using QLV have investigated ligaments, tendons, cartilage, cardiac and skeletal muscle, aortic valve, and plantar tissue (Woo et al. 1980; Woo 1982; Best et al. 1994; Carew et al. 1999; Funk et al. 2000; Abramowitch et al. 2004; Doehring et al. 2004; Ledoux et al. 2004; Duenwald et al. 2009). QLV has been used to model the adult cervical spine structural response for compression, torsion and combined loading (McElhaney et al. 1983; McElhaney et al. 1988; Myers et al. 1991). Isolated adult cervical spine ligaments under tensile loading have been modeled using both quasi-linear theory (Moller et al. 1992; Lucas et al. 2008) and fully nonlinear viscoelasticity (Troyer and Puttlitz 2011; Troyer and Puttlitz 2012). However, the characterization of the tensile viscoelastic structural response of the adult whole or motion segment level cervical spine using QLV has yet to be reported.

QLV assumes that the instantaneous displacement response of the tissue is separable from the time-dependent relaxation response so that, \( G(\delta, t) = F^e(\delta)G_r(t) \), where \( F^e(\delta) \) represents the generally nonlinear instantaneous elastic force-displacement response and \( G_r(t) \) is the reduced relaxation function describing the temporal response. The force response of a tissue, \( F(t) \), subjected to a displacement history, \( \delta(t) \), can be determined from QLV using a convolution integral,
\[ F(t) = \int_{0}^{t} G_r(t - \tau) \frac{\partial F_e(\delta)}{\partial \delta} d\tau, \]  
\[ (E-6) \]

where the reduced relaxation function may be defined as,

\[ G_r(t) = \frac{1+C[E(t/\tau_2)-E(t/\tau_1)]}{1+C\ln(\tau_2/\tau_1)}, \]  
\[ (E-7) \]

and \( E \) is the exponential integral function and \( C, \tau_1 \) and \( \tau_2 \) are material constants.

The instantaneous nonlinear elastic function can be determined using the loading (displacement) portion of a stress relaxation test and is often represented by a polynomial or exponential function. The time constants that bound the continuous relaxation spectrum, \( \tau_1 \) and \( \tau_2 \), can be obtained using both the loading and constant strain level holding portion of stress relaxation tests (Neubert 1963; Dortmans et al. 1984).

The goals of this study are to characterize the viscoelastic structural response of the pediatric whole, upper and lower cervical spine using QLV and to assess QLV as implemented using several methods previously published in the biomechanics literature. Models developed using these methods will be validated against constant velocity tests over three orders of magnitude.

### 7.2 Methods and materials

#### 7.2.1 Specimen Preparation and Experimental Protocol

The whole cervical spine (skull to T1, [WCS]), upper cervical spine (Occiput-C2, [O-C2]) and two lower cervical segments (C4-C5 and C6-C7) were isolated from an
unembalmed six-year-old pediatric human cadaver. Neck musculature, subcutaneous tissue, and skin were removed, and the mandible was disarticulated enabling rigid head fixation and visualization of the cervical spine from the O-C2 joint space to T1.

Additional specimen preparation methodology and testing apparatus details are presented in previous work (Luck et al. 2008, Luck et al. 2012a, Luck et al. 2012b). WCS and O-C2 tests were secured to the testing apparatus at the maxilla and the occipital region with the Frankfort plane aligned horizontally. The caudal end of the spine (T1 in WCS and C2/C3 in the O-C2 test) was potted in polymethyl-methacrylate (Dentsply International; York, PA) and epoxy acrylic resin (Acsys Orthopedic; Vista, CA) and were attached to a hydraulic force test machine (MTS Systems Corporation, Eden Prairie, MN). The two lower segments were mounted with the cranial and caudal vertebra cast in aluminum cups. Six-axis load (2554A, Denton, Inc., Rochester Hills, MI) and axial displacement (Schaevitz Sensors, Hampton, VA) were recorded (National Instruments, Austin, TX). Axial displacement measurements were adjusted for frame-cup compliance (WCS and O-C2) and cup-cup compliance (C4-C5 and C6-C7).

The WCS, O-C2, C4-C5 and C6-C7 were first preconditioned to 5% of the expected tensile failure load using a 1 Hz sine for 60 cycles under a load controlled signal. After preconditioning, the peak displacement for the subsequent viscoelastic test battery was the axial displacement to 10% of the expected tensile failure load (Luck et al. 2008 and Luck et al. 2012b). This battery was performed on the WCS, O-C2, C4-C5 and
C6-C7 segments, using a finite ramp (0.1 sec) and hold (125 sec) relaxation test followed by eight constant velocity tests at 10, 5, 1, 0.5, 0.2, 0.1, 0.05 and 0.01 Hz. Relaxation tests and constant velocity tests were separated by a minimum of 3 min and a maximum corresponding to twice the duration of the previous test to allow for stress relaxation from the previous loading.

7.2.2 Quasi-linear Viscoelastic Model Generation, Validation and Comparison

Three categories of QLV methods, based on underlying approach, were used to assess viscoelastic response of the pediatric WCS, O-C2, C4-C5 and C6-C7 segments. These are termed Best (similar to the method of Best et al. 1994), Myers (similar to the method of Myers et al. 1991) and the full strain history (FSH) (similar to direct-fit methods undertaken by: Abramowitch and Woo (2004); Lucas et al. (2008)). For consistency, the instantaneous non-linear elastic response in all models considered of the WCS and cervical segments was represented by the function, \( F^e(\delta) = A(e^{B\delta} - 1) \), commonly used to represent soft biological tissues.

The Best method was based on Fung (1967). The finite rate ramp was assumed to be instantaneous and the relaxation normalized by the maximum load at the end of the finite ramp to establish the reduced relaxation function. Best et al. (1994) originally used a fourth-order polynomial for the instantaneous nonlinear elastic function. The Myers method used an extrapolation deconvolution technique to account for the relaxation that occurs during the ramp. The full strain history method (FSH), used a gradient-based
constrained non-linear multivariate minimization algorithm (\textit{fmincon} function, MATLAB) to minimize the root-mean-squared (RMS) error between the experimental and predicted force derived directly from the time history of the experimental strain. One advantage to this methodology is that potential overshoot during loading or settling during the hold phase of the displacement profile is incorporated into parameter estimation through direct integration of the displacement history in the convolution integral (Gimbel et al. 2004). The FSH category included four relaxation models of differing complexity. These are a spectral model (FSHSE), similar to that used by Best et al. and Myers et al., and a single (FSH1DE), double (FSH2DE) and triple (FSH3DE) branch generalized Maxwell-Weichert model, similar to the multi-branch model used by Lucas et al. (2008) and represented as follows,

\[ G_r(t) = G_\infty + \sum_{i=1}^{n} G_i e^{-t/\tau_i} \]  

(E-8)

\[ G_\infty + \sum_{i=1}^{n} G_i = 1 \]  

(E-9)

Where \( n = 1, \) (FSH1DE); \( n = 2, \) (FSH2DE) and \( n = 3, \) (FSH3DE)

Quality assessments of the QLV models included predicted peak load (PL), loading energy (LE) calculated as the integral of the load-displacement curve, hysteresis energy (HE) as a percentage of LE, and two estimates of the goodness-of-fit for the entire loading curve. Goodness-of-fit was estimated between the experimental force-time response and predicted response for the loading portion of the constant velocity tests.
(RMSE-L) and for the entire loading and unloading portion of the constant velocity tests (RMSE-LU) using RMS error.

At each loading frequency, each QLV model was assessed using LE, HE, PL, RMSE-L and RMSE-LU. The five metrics were linearly combined using a weighting scheme based on the standard deviation of the results. The three metrics, LE, HE and PL were weighted based on the standard deviation of the absolute difference between a given model and the experimental response normalized by the maximum absolute difference observed over all methods. The two metrics, RMSE-L and RMSE-LU were weighted based on the standard deviation of the RMSE of a given method normalized by the maximum RMSE observed over all methods and is represented in the following algorithm:

\[ \text{SCORE}_{QLV Method.freq} = \frac{1}{\sum_{i=1}^{5} \text{SD}_i} \left( \sum_{j=1}^{3} \text{SD}_j \cdot M_{(D-N),j} + \sum_{k=1}^{2} \text{SD}_k \cdot M_{(N),k} \right) \]  \hspace{1cm} (E-10)

Where \( i \) represents the five metrics, \( j \) represents metrics LE, HE and PL, \( k \) represents metrics RMSE-L and RMSE-LU, \( \text{SD} \) is the standard deviation, \( M_{(D-N),j} \) and \( M_{(N),k} \) are as follows:

\[ M_{(D-N),j} = \frac{|M_j - M_{\text{experiment}}|}{\max(|M_j - M_{\text{experiment}}|)} \]  \hspace{1cm} (E-11)

\[ M_{(N),k} = \frac{M_k}{\max(M_k)} \]  \hspace{1cm} (E-12)
A cumulative score for a given QLV method, taking into consideration all five metrics over all eight loading frequencies was determined by equally weighting the scores determined from the aforementioned analysis. QLV methods were also assessed over all loading frequencies for each individual metric using a similar methodology using the standard deviation to weight the results.

7.3 Results

7.3.1 Pediatric WCS, O-C2, C4-C5 and C6-C7 Viscoelastic Experimental Response

The PL increased for the pediatric cervical spine at all loading frequencies for the WCS and O-C2, while increasing for C4-C5 and C6-C7 from 0.01 to 0.5 Hz. The average PL was 222 ± 14 N and 161 ± 9 N for the WCS and O-C2, respectively. PL increased 21% (WCS) and 20% (O-C2) over three decades of loading. Lower cervical spine average PL was 176 ± 14 N and 189 ± 12 N for C4-C5 and C6-C7, respectively. PL increased 12% (C4-C5) and 10% (C6-C7) over approximately two decades of loading from 0.01 to 0.5 Hz, while PL decreased at 1, 5 and 10 Hz for both lower segments. However, the peak applied displacement for these three constant velocity tests progressively declined for both lower segments to approximately 82% (C4-C5) and 84% (C6-C7) of the applied displacement controlled in the lower rate tests, indicative of PL reduction observed at higher loading frequencies. In comparison, over the 0.01 to 0.5 Hz range, WCS and O-C2 PL increased by 11% and 13%, respectively, comparable to PL increases observed in the lower cervical spine.
Pediatric cervical spine LE generally increased with loading frequency, similar to PL, although a monotonic increase at all frequencies was only observed in the WCS. LE increased 19% (WCS) over three orders of loading and averaged $442 \pm 25$ N-mm. O-C2 LE increased monotonically from 0.01 to 1 Hz (16%), while seeing no additional increase in LE at 5 and 10 Hz. Peak actuator displacement was consistently controlled (97-100% controlled displacement) in the 0.01 to 1 Hz tests, but fell to 92% and 91% in the 5 and 10 Hz O-C2 tests. Although PL at 10 Hz for O-C2 was greater than at all other loading frequencies, the maximum O-C2 LE was observed at 1 and 10 Hz. Average O-C2 LE was $145 \pm 7$ N-mm over all loading frequencies. LE increases were more modest in the lower cervical spine and peaked at 10% (1 Hz) and 9% (0.1 Hz) in C4-C5 and C6-C7, respectively. Average LE was $50 \pm 4$ (C4-C5) and $64 \pm 5$ (C6-C7) N-mm over all loading frequencies. LE reductions at 5 and 10 Hz were observed for both segments, similar to PL reductions at these frequencies that corresponded to reductions in the controlled peak displacement compared to the lower rate tests. Due to the reduction in inputted displacement at the higher frequencies when compared to the remaining constant velocity tests, strict interpretations of LE response over the entire tested frequency range are limited, although observed increases in LE from 0.01 to roughly 1 Hz are clearly present.

The HE of the pediatric cervical spine was relatively insensitive to loading rate in the lower frequency domain, while higher rates of loading resulted in greater variations
in HE, most notably in O-C2 (Figure 31). From 0.01 to 1 Hz, HE variations were less than 24% (O-C2) in all segments; 15% (WCS), with minimal variation in the lower segments (3%, C4-C5; 2%, C6-C7). Average HE was 23 ± 3% (WCS), 25 ± 5% (O-C2), 32 ± 5% (C4-C5) and 37 ± 6% (C6-C7) over three decades of loading. At 5 and 10 Hz, the variation in HE compared to the 0.01 Hz test were more apparent, with increases in HE of 41% (WCS) and 63% (O-C2); while the lower segments increased by more modest amounts, 33% (C4-C5) and 23% (C6-C7).

**FIGURE 31:** The energy dissipation of the pediatric cervical spine at all levels is observed to be relatively rate insensitive at low loading frequencies, while increases were observed at all levels with the highest loading rates. C4-C5 and C6-C7 energy dissipation was observed to be greater than observed in O-C2 and the whole spine.
7.3.2 Pediatric WCS, O-C2, C4-C5 and C6-C7 QLV Model Response

The Myers, FSHSE and Best QLV models ranked the highest in predicting pediatric cervical spine structural response (Table 12). All models were highly correlated to the experimental stress relaxation tests. Individual instantaneous nonlinear elastic functions were also well correlated with correlation coefficients greater than 0.98 in all models.

**Table 12:** Pediatric cervical spine quasi-linear viscoelastic (QLV) models for axial tensile loading

<table>
<thead>
<tr>
<th>Segment Level</th>
<th>QLV Approach</th>
<th>Instantaneous Non-linear Elastic Function</th>
<th>Reduced Relaxation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Fe(δ) = A(e^Bδ - 1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCS</td>
<td>Myers</td>
<td>37.26, 0.334</td>
<td>19.6, 0.97</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0849, 0.0042</td>
</tr>
<tr>
<td></td>
<td></td>
<td>124.87, 0.012</td>
<td>0.99</td>
</tr>
<tr>
<td>O-C2</td>
<td>Myers</td>
<td>48.30, 0.609</td>
<td>22.1, 0.96</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0708, 0.0040</td>
</tr>
<tr>
<td></td>
<td></td>
<td>124.86, 0.017</td>
<td>0.98</td>
</tr>
<tr>
<td>C4-C5</td>
<td>FSHSE</td>
<td>69.94, 1.8</td>
<td>24.1, 0.95</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0905, 0.0093</td>
</tr>
<tr>
<td></td>
<td></td>
<td>124.73, 0.027</td>
<td>0.96</td>
</tr>
<tr>
<td>C6-C7</td>
<td>Best</td>
<td>62.70, 1.59</td>
<td>24.1, 0.95</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0905, 0.0093</td>
</tr>
<tr>
<td></td>
<td></td>
<td>124.73, 0.027</td>
<td>0.96</td>
</tr>
</tbody>
</table>

WCS - Whole cervical spine; † - Goodness of fit metrics for the FSHSE method are listed in the Reduced Relaxation section and represent the entire loading and relaxation fit.

The pediatric WCS structural response was modeled most effectively with the Myers model over three decades of loading (Appendix D: WCS and Myers Model). The cumulative score of the Myers model was greater than a standard deviation less than the average score for all six models (Table 13). Average PL, LE and HE predicted by the Myers model was 267 ± 22 N (20% overestimation (OE)), 535 ± 42 N-mm (21% OE) and
20 ± 1 % (12% underestimation (UE)), respectively. PL and LE increased with loading rate, consistent with experiment, by 28% and 26%, respectively, but both metrics were overestimated. The Myers model was rate insensitive, indicative of the spectral relaxation model, resulting in improved HE estimations from 0.01 to 1 Hz compared to the higher loading rates where experimental WCS HE increased. The average RMSE-LU and RMSE-L for the Myers model was 14 ± 4 N and 19 ± 5 N, respectively. Correlation coefficients at all loading rates for the Myers model exceeded 0.99. Although the Myers model captured the experimental response of the WCS when all metrics were considered over all frequencies of loading, the FSH1DE model ranked the highest for LE, PL, RMSE-LU and RMSE-L when each metric was independently analyzed over all loading frequencies (Table 14).

Similar to the WCS, the Myers model predicted the O-C2 structural response and outperformed the remaining models (Appendix D: O-C2 and Myers Model). The Myers model cumulative score was greater than a standard deviation less than the average model score (Table 13). The Myers model also ranked the highest for LE, PL, RMS-LU and RMS-L when each metric was independently analyzed over all loading frequencies (Table 14). Average PL, LE and HE predicted by the Myers model was 164 ± 9 N (2% OE), 164 ± 10 N-mm (13% OE) and 18 ± 1 % (28% UE), respectively. Increases in PL were modeled up to 1 Hz (22%), but PL decreases were predicted at higher frequencies and included an underestimation of PL at 10 Hz. Model LE increased by 22% with loading
rate up to 1 Hz and was consistent with experimental increases, although slight overestimation was present. Less relaxation was predicted by the Myers model for O-C2 and resulted in greater deviation between the modeled HE and that observed experimentally. The average RMSE-LU and RMSE-L for the Myers approach was 7 ± 2 N and 8 ± 2 N, respectively. Correlation coefficients at all loading rates for the Myers approach exceeded 0.99.

The FSHSE and Best models most effectively modeled the structural response of the C4-C5 and C6-C7 segments, respectively (Appendix D: C4-C5 and FSHSE Model; C6-C7 and Best Model). The FSHSE and FSH2DE models each performed well in predicting C4-C5 response with cumulative scores greater than a standard deviation less than the average model score and the FSHSE receiving the lowest overall score amongst all models (Table 13). The FSHSE model also ranked high when predicting PL, RMSE-LU and RMSE-L when each metric was independently analyzed over all loading frequencies (Table 14). Although FSH2DE ranked the highest for LE over all frequencies, FSHSE also performed well, predicting a response that was greater than a standard deviation less than the average model response. The Best model yielded a cumulative score greater than one standard deviation less than the average model score and outperformed all other models (Table 13). The Best model had the lowest cumulative score for all metrics over all loading frequencies with the Myers model also performing well although its cumulative score was within a standard deviation of the
The Best model ranked the highest for LE, PL and RMSE-L when each metric was independently analyzed over all loading frequencies (Table 14).

**Table 13**: Pediatric WCS, O-C2, C4-C5 and C6-C7 QLV Method Comparison and Validation by Frequency of Loading

<table>
<thead>
<tr>
<th>Segment</th>
<th>Method</th>
<th>Constant Velocity Loading Frequency (All Metrics: LE, HE, PL, RMS-LU, RMS-L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.01 Hz</td>
<td>0.05 Hz</td>
</tr>
<tr>
<td>Best</td>
<td>0.66</td>
<td>0.54</td>
</tr>
<tr>
<td>Myers</td>
<td>0.42 †</td>
<td>0.51 †</td>
</tr>
<tr>
<td>FSH1DE</td>
<td>0.67</td>
<td>0.34 †</td>
</tr>
<tr>
<td>FSH2DE</td>
<td>0.58</td>
<td>0.79 †</td>
</tr>
<tr>
<td>FSH3DE</td>
<td>0.76 †</td>
<td>0.84 †</td>
</tr>
<tr>
<td>FSHSE</td>
<td>0.51</td>
<td>0.53</td>
</tr>
<tr>
<td>Average</td>
<td>0.60</td>
<td>0.59</td>
</tr>
<tr>
<td>SD</td>
<td>0.12</td>
<td>0.19</td>
</tr>
</tbody>
</table>

**O-C2**

| Best    | 0.48   | 0.60   | 0.66   | 0.69   | 0.68   | 0.67   | 0.41   | 0.51 † | 0.59 |          |
| Myers   | 0.32 † | 0.50 † | 0.55 † | 0.60 † | 0.59 † | 0.62 † | 0.41 † | 0.49 † | 0.51 † |
| FSH1DE  | 0.80 † | 0.61   | 0.53   | 0.80   | 0.61   | 0.96 † | 0.79 † | 0.73 † | 0.73 |          |
| FSH2DE  | 0.68 † | 1.00 † | 0.97 † | 0.92 † | 0.92 † | 0.70 † | 0.82 † | 0.63 | 0.79 † |
| FSH3DE  | 0.59 † | 0.76   | 0.72 † | 0.60 † | 0.47 † | 0.52 † | 0.73 † | 0.74 | 0.64 |          |
| FSHSE   | 0.64   | 0.81   | 0.88   | 0.89 † | 0.82 † | 0.82 † | 0.68   | 0.81 | 0.80 † |
| Average | 0.58   | 0.71   | 0.72   | 0.75   | 0.63   | 0.72   | 0.64   | 0.65 | 0.68 |          |
| SD      | 0.17   | 0.19   | 0.18   | 0.14   | 0.12   | 0.15   | 0.18   | 0.13 | 0.12 |          |

**C4-C5**

| Best    | 0.87 † | 0.73   | 0.70   | 0.62   | 0.70   | 0.62   | 0.85 † | 0.89 † | 0.75 |          |
| Myers   | 0.87   | 0.73   | 0.70   | 0.63   | 0.69   | 0.60   | 0.79 † | 0.84 | 0.73 |          |
| FSH1DE  | 0.45 † | 0.69 † | 0.87 † | 0.90 † | 0.92 † | 0.95 † | 0.77 † | 0.73 † | 0.79 † |
| FSH2DE  | 0.57   | 0.63   | 0.59   | 0.40 † | 0.27 † | 0.50 † | 0.47 † | 0.63 | 0.51 † |
| FSH3DE  | 0.73 † | 0.83   | 0.77 † | 0.60   | 0.59 † | 0.41 † | 0.36 † | 0.45 † | 0.59 † |
| FSHSE   | 0.72   | 0.55   | 0.52 † | 0.43 † | 0.43 † | 0.40 † | 0.37   | 0.38 † | 0.47 † |
| Average | 0.70   | 0.69   | 0.69   | 0.60   | 0.60   | 0.58   | 0.60   | 0.65 | 0.64 |          |
| SD      | 0.17   | 0.10   | 0.13   | 0.18   | 0.23   | 0.20   | 0.23   | 0.21 | 0.13 |          |

**C6-C7**

| Best    | 0.73   | 0.61   | 0.43   | 0.38   | 0.41   | 0.48 † | 0.43 † | 0.47 † | 0.49 † |
| Myers   | 0.85 † | 0.70   | 0.49   | 0.37   | 0.40   | 0.45 † | 0.45 † | 0.49 † | 0.53 † |
| FSH1DE  | 0.67 † | 0.90 † | 1.00 † | 1.00 † | 1.00 † | 0.94 † | 0.78   | 0.73 | 0.88 † |
| FSH2DE  | 0.64   | 0.68   | 0.54   | 0.55   | 0.48   | 0.71 † | 0.90 † | 0.93 † | 0.68 † |
| FSH3DE  | 0.71   | 0.62   | 0.50   | 0.50   | 0.44   | 0.71 † | 0.91 † | 0.94 † | 0.66 † |
| FSHSE   | 0.71   | 0.53   | 0.38   | 0.39   | 0.42   | 0.62   | 0.70   | 0.81 | 0.57 † |
| Average | 0.71   | 0.67   | 0.56   | 0.53   | 0.53   | 0.65   | 0.69   | 0.73 | 0.63 |          |
| SD      | 0.08   | 0.13   | 0.22   | 0.24   | 0.23   | 0.18   | 0.21   | 0.21 | 0.14 |          |

QLV method with the lowest weighted score assessing all five metrics (0.00 score: corresponds to no difference between QLV method and experiments; 1.00 score: corresponds to the maximum theoretical value, a QLV method with the greatest normalized absolute deviation from the experimental data for all five metrics)

Cumulative Score: Equally weighted cumulative score for all metrics over all loading frequencies

† - QLV Method ≥ 1 SD predictive improvement when compared to the average of all QLV methods

‡ - QLV Method ≥ 1 SD diminishment in predictive ability when compared to the average of all QLV methods

* - QLV method is within one standard deviation of the average of all QLV methods
Average C4-C5 PL, LE and HE predicted by the FSHSE model was 156 ± 12 N (11% UE), 47 ± 4 N-mm (4% UE) and 22 ± 1 % (30% UE), respectively. Increases in PL were modeled with increasing loading rate up to 1 Hz (24%) followed by PL decreases at 5 and 10 Hz that were also present in the experimental response as a result of the reduced peak input displacement. Model LE increased by 26% with loading rate up to 1 Hz and was generally consistent with the experimental response of increased LE with increased rate. Furthermore, although at 5 and 10 Hz, 100% peak actuator displacement was not attained, the FSHSE model accurately predicted the LE at these rates (< 2% absolute deviation). Similar to WCS and O-C2, C4-C5 HE was consistently underestimated at all loading rates with the FSHSE model. The average RMSE-LU and RMSE-L for the FSHSE approach was 7 ± 3 N and 9 ± 4 N, respectively. Correlation coefficients at all loading rates for the FSHSE approach exceeded 0.98.

Average C6-C7 PL, LE and HE predicted by the Best model was 185 ± 13 N (2% UE), 65 ± 6 N-mm (2% OE) and 21 ± 6 % (41% UE), respectively. C6-C7 PL model response was similar to C4-C5 with increases in PL with increasing loading rate up to 1 Hz (22%) followed by PL decreases at 5 and 10 Hz that were also present in the experimental response. Model LE increased by 24% with loading rate up to 1 Hz and was consistent with experimental behavior. C6-C7 HE was underestimated with the Best model, although when compared to the highest performing models for HE prediction (FSHSE (31% UE) and Myers (35% UE)) the underestimation was relatively
small. The average RMSE-LU, -L for the Best model was 9 ± 2 N and 7 ± 2 N, respectively. Correlation coefficients at all loading rates for the Best model exceeded 0.98.
Table 14: Pediatric WCS, O-C2, C4-C5 and C6-C7 QLV Method Comparison and Validation by Metric

<table>
<thead>
<tr>
<th>Segment</th>
<th>QLV Method</th>
<th>Comparison Metrics (All Frequencies of Loading: 10 to 0.01 Hz)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>WCS</td>
<td>Best</td>
<td>0.70 • 0.29 • 0.70 • 0.76 • 0.69 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myers</td>
<td>0.67 • 0.16 • 0.66 • 0.71 • 0.66 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH1DE</td>
<td>0.43 † 0.88 † 0.17 † 0.68 † 0.49 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH2DE</td>
<td>0.70 • 0.54 • 0.45 • 0.75 • 0.63 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH3DE</td>
<td>1.00 † 0.17 † 1.00 † 1.00 † 1.00 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSHSE</td>
<td>0.84 • 0.16 • 0.69 • 0.81 • 0.79 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>0.72 0.37 0.61 0.79 0.71</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.19 0.28 0.28 0.12 0.17</td>
<td></td>
</tr>
<tr>
<td>O-C2</td>
<td>Best</td>
<td>0.54 † 0.74 • 0.43 • 0.62 • 0.58 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myers</td>
<td>0.51 † 0.59 • 0.33 † 0.58 † 0.57 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH1DE</td>
<td>0.89 • 0.71 • 0.39 • 0.93 † 0.95 †</td>
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</tr>
<tr>
<td></td>
<td>FSH2DE</td>
<td>0.91 • 0.61 • 0.74 • 0.90 † 0.92 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH3DE</td>
<td>0.68 • 0.26 † 0.99 † 0.62 • 0.67 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSHSE</td>
<td>0.91 • 0.61 • 0.79 • 0.84 • 0.90 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>0.74 0.59 0.61 0.75 0.76</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.19 0.28 0.27 0.16 0.18</td>
<td></td>
</tr>
<tr>
<td>C4-C5</td>
<td>Best</td>
<td>0.81 • 0.44 • 0.98 † 0.69 • 0.96 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myers</td>
<td>0.84 • 0.32 † 0.98 † 0.70 • 0.97 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH1DE</td>
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<tr>
<td></td>
<td>FSH2DE</td>
<td>0.20 † 0.62 • 0.61 • 0.65 • 0.68 •</td>
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<tr>
<td></td>
<td>FSH3DE</td>
<td>0.67 • 0.41 • 0.59 • 0.56 † 0.71 †</td>
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</tr>
<tr>
<td></td>
<td>FSHSE</td>
<td>0.26 † 0.46 • 0.59 • 0.56 † 0.65 †</td>
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<tr>
<td></td>
<td>Average</td>
<td>0.58 0.52 0.73 0.69 0.84</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.28 0.19 0.19 0.15 0.15</td>
<td></td>
</tr>
<tr>
<td>C6-C7</td>
<td>Best</td>
<td>0.38 † 0.61 • 0.37 † 0.59 • 0.53 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myers</td>
<td>0.45 • 0.52 • 0.45 • 0.57 • 0.57 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH1DE</td>
<td>0.66 † 0.93 ¥ 0.98 † 0.99 † 0.98 †</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH2DE</td>
<td>0.60 • 0.56 • 0.74 • 0.73 • 0.84 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSH3DE</td>
<td>0.59 • 0.55 • 0.71 • 0.71 • 0.81 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSHSE</td>
<td>0.58 • 0.50 ¥ 0.44 • 0.63 • 0.74 •</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>0.54 0.61 0.61 0.70 0.74</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.11 0.16 0.23 0.15 0.17</td>
<td></td>
</tr>
</tbody>
</table>

QLV method with the lowest weighted score assessing all five metrics (0.00 score: corresponds to no difference between QLV method and experiments; 1.00 score: corresponds to the maximum theoretical value, a QLV method with the greatest normalized absolute deviation from the experimental data for all five metrics)

† - QLV Method ≥ 1 SD predictive improvement when compared to the average of all QLV methods
‡ - QLV Method ≥ 1 SD diminishment in predictive ability when compared to the average of all QLV methods
¥ - QLV Method ≥ 2 SD diminishment in predictive ability when compared to the average of all QLV methods
• - QLV method is within one standard deviation of the average of all QLV methods
7.4 Discussion

Pediatric whole, upper and lower cervical spine responses to axial tension were modeled with quasi-linear viscoelastic models that predicted the loading and unloading response over three decades of loading frequency. Inclusion of viscous effects provided an improvement over purely nonlinear elastic models of the pediatric cervical spine. Increases in PL, LE and HE were observed experimentally for all cervical structures. As few studies exist on the biomechanics of the pediatric human cervical spine, development of increasingly robust biomechanical models that include rate effects is an important step in progressing computational and physical models focused on investigating these structures under real-world conditions.

7.4.1 Viscoelastic Modeling & Comparison of Models

A single QLV model was unable to approximate the loading/unloading and energy dissipation characteristics of the entire pediatric cervical spine, WCS and segments, better than any other single model also evaluated. Although the Myers model ranked the highest for both the WCS and O-C2, the FSHSE and Best models, ranked the highest for C4-C5 and C6-C7, respectively. The Myers, FSHSE and Best models ranked as generally robust predictive models for all metrics over all frequencies of loading, but this result is not necessarily the best overall predictive model for any one metric at all frequencies of loading. The FSH1DE model scored the highest for predicting WCS LE, PL, RMSE-LU and RMSE-L at all frequencies, when each metric was evaluated
independently. Conversely, the Myers method ranked the highest only for WCS HE, while the remaining metrics were within a standard deviation of the average overall response of all the models. The O-C2 segment was effectively modeled by the Myers model not only when evaluating all metrics over all frequencies, but also when evaluating individual metrics independently. The Myers method ranked the highest for O-C2 LE, PL, RMSE-LU and RMSE-L, while the FSH3DE method scored the highest in predicting HE over all frequencies. At the C4-C5 level, the FSH2DE and Myers methods modeled LE and HE, while FSHSE modeled RMSE-L and both FSHSE and FSH3DE modeled PL and RMSE-LU equally well at all frequencies, when all metrics were evaluated independently. The Best method predicted LE, PL and RMSE-L, while Myers and FSHSE predicted RMSE-LU and HE, respectively, when all metrics were evaluated independently over all frequencies for the C6-C7 segment. Modeling a comprehensive biomechanical model of the pediatric cervical spine is ideal, but as these data indicate, depending on the application, robust models that approximate multiple response characteristics over multiple loading rates are somewhat limited in predicting any one response optimally.
The spectral relaxation models of the Myers and FSHSE approaches accurately modeled the constant hysteresis energy at low load rates but were unable to model the increases in hysteresis observed at higher loading rates.

Spectral relaxation models were typically sufficient in modeling the rate insensitivity observed experimentally at low loading rates, but were unable to model the increases in HE seen at the higher rates. The Myers and FSHSE models outperformed the discrete time constant models (FSH1DE, FSH2DE, and FSH3DE) and Best method in predicting HE in WCS (Figure 32) and C6-C7 (Figure 33) response, while the Myers model outperformed all other methods for C4-C5 (Figure 34) response when considering all loading frequencies. The improved performance of these two models relative to the discrete models was based on the improved prediction in the lower loading frequencies where HE was observed to be rate insensitive. At the higher loading frequencies the Myers and FSHSE models routinely underestimated HE as energy dissipation was

**FIGURE 32**: The spectral relaxation models of the Myers and FSHSE approaches accurately modeled the constant hysteresis energy at low load rates but were unable to model the increases in hysteresis observed at higher loading rates.
observed to increase experimentally at 5 and 10 Hz. While the FSH1DE and FSH2DE models overwhelming failed to capture the energy dissipation response, the FSH3DE model outperformed all other models at predicting O-C2 HE (Figure 35) over all loading frequencies (14% absolute deviation, -12% relative deviation). The three time constant discrete model was effective in modeling the increases in HE observed in the mid to high loading rate (0.5 to 5 Hz; 3% absolute deviation) in comparison to the top performing spectral model (Myers: 0.5 to 5 Hz; 38% absolute deviation) while producing results similar to these methods at the lowest frequencies (0.01 to 0.5 Hz absolute deviations; FSH3DE: 15%; Myers: 18%). This was similarly observed in the WCS and C4-C5 segments, although the cumulative response of the FSH3DE model lagged in comparison to the spectral models. While the spectral models performed well in representing the rate insensitivity at the lower loading frequencies, these methods overall underestimated HE in the spine. With the exception of the WCS where the mean absolute deviation, from 0.01 to 0.5 Hz, was 3.5% (Myers) and 4.1% (FSHSE), the top performing spectral models for predicting HE over all loading frequencies typically underestimated HE in the segments by 12 to 32% (mean absolute deviation) from 0.01 to 0.5 Hz. Myers et al. (1991) reported a mean absolute and relative deviation of 17% and -0.1%, respectively, in HE when comparing the Myers model to constant velocity experimental data on whole and isolated cervical spines subjected to torsion and loaded between 0.02 and 2 Hz. While the underestimation was minimal, this represents the
cumulative relative deviation of 36 tests of the whole cervical and segmental spine at three loading rates, while the current work is based on a single sample tested as the whole spine and three cervical segments. While the segments showed consistent underestimation the predicted HE response of the WCS using a similar modeling approach to Myers et al. (1991) performed well in predicting low loading rate behavior.

**FIGURE 33:** The Best and FSHSE approaches appropriately modeled the constant energy dissipation observed at low loading rates in the C6-C7 segment, but underestimated the hysteresis at all loading rates.
FIGURE 34: The Myers and FSHSE approaches appropriately modeled the constant energy dissipation observed at low loading rates in the C4-C5 segment. The Myers approach tended to predict the energy dissipation at lower loading rates better than the FSHSE approach. However, the FSHSE approach modeled the higher loading rate effects better than Myers, although both methods underestimated the increases in hysteresis energy observed at the higher rates of loading.

The FSHSE model relative to the Myers model yielded improvements in HE prediction at higher loading rates due to differences in short time constant estimation between the two methods. The FSHSE and Myers model produced similar distributed relaxation constants, $C_{Myers} = 0.0849$ and $C_{FSHSE} = 0.0848$, when modeling the WCS, but the FSHSE model produced a lower earlier time constant than Myers ($\tau_{1,Myers} = 0.0042$ sec and $\tau_{1,FSHSE} = 0.0008$ sec). The lower time constant broadened the frequency range of the relaxation effects and improved HE predictions at higher frequencies (Sauren and Rousseau 1983). For the 5 and 10 Hz loading rates, the FSHSE model underestimated.
HE in the WCS by 37%, while the Myers model underestimated by 41%. Although the relaxation constants obtained for the C4-C5 segment for these two methods varied ($C_{Myers} = 0.1187$ and $C_{FSHSE} = 0.0930$) the FSHSE short time constant was less than the Myers model ($\tau_{1,Myers} = 0.024$ sec and $\tau_{1,FSHSE} = 0.0009$ sec) and resulted in extended relaxation at 5 and 10 Hz yielding improvements in HE prediction by approximately 17%. While both the FSHSE and Myers models attempt to take into account relaxation that may occur during loading, these results indicate that the methodology employed in the FSHSE model was able to capture the earlier relaxation better than the Myers model.

**FIGURE 35:** The FSH3DE and Myers models predicted hysteresis energy in the O-C2 segment better than all other models. The FSH3DE model was better able to represent the increases in hysteresis energy observed in the mid to high range of loading.

Improvements in PL estimation over all loading frequencies were observed with the top overall QLV models when compared to purely nonlinear elastic models without
viscoelastic effects. PL estimation in the WCS improved from a 56% to 20% overestimation with the Myers model. Similarly the Myers model improved PL estimation from 29% to 2% in the O-C2 segment, while improvements were also observed with the FSHSE and Best models for the C4-C5 (-11% vs. 27%) and C6-C7 (-2% vs. 26%) segments, respectively.

7.4.2 Experimental Viscoelastic and Rate Effects

The experimentally observed viscous effects of the pediatric cervical spine varied by level, with increased energy loss observed in C4-C5 and C6-C7 compared to the WCS and O-C2. Average HE for the adult cervical spine, reported as a percentage of the maximum stored strain energy, was reported as 37% under torsional loading from 0.02 to 2 Hz (Myers et al. 1991) and 17% (O-C2) and 37% (C4-C5 and C6-C7) under low rate tensile loading (Dibb et al. 2009). Average HE in the current study covered a range from 23 ± 3% (WCS) to 37 ± 6% (C6-C7) over three decades of loading and 21 ± 1% (WCS) to 34 ± 5% (C6-C7) over two decades of loading (0.01 to 1 Hz). Myers et al. reported that the adult cervical spine in torsion is relatively rate insensitive with less than an 18% variation in HE over a two decade variation in loading frequency. Similar rate insensitivity was observed in the pediatric cervical spine over a similar two decade variation in loading frequency (0.01 to 1 Hz) with maximum variation occurring in O-C2 (24%). Increased rate sensitivity was observed in all pediatric cervical structures over three decades of loading (0.01 to 10 Hz), although it is unclear if similar variations exist.
in the adult cervical spine at higher loading rates. McElhaney et al. (1988) reported minimal changes in hysteresis with increases in loading frequency on human cervical segments subjected to combined loading from 0.01 to 5 Hz. Isolated human adult alar and transverse ligaments showed increases in hysteresis energy with increasing displacement rates (Moller et al. 1992). In contrast, others have observed that HE decreased with increasing loading rate in isolated cervical ligaments (Troyer et al. 2011; Yoganandan et al. 1989). In comparing the isolated ligament studies to the current and past cervical structural level work it is important to consider that the structural level studies encompass the response of multiple ligaments, bones, cartilage, intervertebral discs and soft tissue interacting to provide an overall response. Differences in the rate dependent behavior of different adult cervical ligaments have been observed (Lucas et al. 2008). It is unclear when a cervical segment or whole spine are loaded how the differences in time of loading for individual components in addition to the underlying differences in baseline material level response of each of these structures may play into the observed differences between the current work and isolated material studies.

LE and PL were observed to increase with loading rate for the WCS and all segments. LE increased 17% and 14% from 0.01 to 10 Hz in the WCS and O-C2, respectively. Increases of 8% and 5% from 0.01 to 1 Hz in the C4-C5 and C6-C7 segments, respectively, were also observed. Increases in the energy absorbed by isolated adult spinal ligaments under tensile loading have been reported (Neumann et al. 1994;
PL increased by 12, 15, 10 and 8% in the WCS, O-C2, C4-C5 and C6-C7 segments, respectively from 0.01 to 1 Hz. While overall increases in PL of 21% and 20% in the WCS and O-C2, respectively, were observed up to 10 Hz loading. Numerous studies on the cervical spine have reported increases in the load at failure with increasing loading rate (Sances et al. 1981). While others have reported similar increases of load with loading rate in isolated adult spinal ligaments (Neumann et al. 1994; Yoganandan et al. 1989). Increases in LE and PL within the pediatric cervical spine with accompanying increases in loading rate highlight the importance of considering rate effects at sub-catastrophic load and distraction levels. While this indicates that these structures absorb additional energy and reach higher loads as rates of loading are increased, similar increases in energy dissipation, especially at the highest rates of loading may represent sub-catastrophic protective mechanisms in the pediatric cervical spine that appear to not be present in the limited adult cervical spine literature regarding loading rate and energy dissipation.

7.4.3 Limitations

Limitations of the current study were primarily associated with the limited sample size that is typical of any pediatric human cadaveric based work. Investigation of the biomechanics of the pediatric human cervical spine is rare (Duncan 1874; Ouyang et al. 2005; Luck et al. 2008; Luck et al. 2012a; Luck et al. 2012b; Luck et al. 2012c) and similar investigations of the viscoelastic response of these structures have yet to be
reported. In lieu of available pediatric tissue to augment the current sample for statistical robustness, the current investigation represents a first approximation at the viscoelastic and rate dependent response of the pediatric cervical spine.

Ramp overshoot in the stress relaxation tests occurred to varying degrees as a result of the wide range of displacements required for the different cervical structures and the fast ramp durations employed (0.1 sec). Overshoot may limit the effectiveness of viscoelastic parameter estimation and methods including extrapolation and slower ramp rates may mitigate this behavior and improve parameter estimation (Abramowitch and Woo 2004; Dortmans et al. 1994; Myers et al. 1991). Five of the six approaches employed for parameter estimation in the current study contained elements to mitigate overshoot (Myers, FSHSE, FSH1DE, FSH2DE, and FSH3DE).

Creep occurred between the stress relaxation test and the initial constant velocity test in the WCS. The complete test battery included both displacement and load controlled tests, including multiple load controlled rate tests. The no-load displacement prior to the 0.01 Hz constant velocity test compared to prior to the stress relaxation test was greater than zero indicating that during load controlled test and hold periods, viscoelastic creep may have occurred in the WCS. Creep was accounted for by assuming that the nonlinear elastic response predicted in the stress relaxation test be shifted by the creep displacement amount. Due to the strain stiffening response of the nonlinear elastic function, this predicted a WCS structure in a region stiffer than experienced at the
onset of the stress relaxation test. While a similar protocol was employed for O-C2, C4-C5 and C6-C7; although negligible creep was observed in these segments, it is unclear if the response predicted by this shifted nonlinear response is wholly representative of the WCS response at displacement levels greater than those controlled in the stress relaxation test. Overestimation of PL and LE for Myers and other models in the WCS may be a result of this attempt at correcting for creep. Further investigation of the creep behavior of the pediatric WCS may be required to tune this adjustment and therefore improve the comparisons between the QLV models and the experimentally observed constant velocity tests. It is likely that further understanding of these viscoelastic phenomena may lead to improved agreement between model and experimental response.

Relaxation rate as a function of strain magnitude is not reported, while an underlying assumption of QLV theory is that relaxation rate is independent of strain magnitude. Relaxation rate of individual adult human cervical ligaments have been shown to be rate dependent (Troyer et al. 2011). However, previous work on the structural response of the adult cervical spine, similar to the current study, has shown that relaxation rate is independent of both the rate and level of loading (Myers et al. 1991). It is unclear whether the relaxation behavior of the pediatric cervical spine is independent of strain magnitude, although its response is expected to be more similar to
the structural experiments of Myers et al. as opposed to isolated ligamentous studies (Provenzano et al. 2001; Troyer et al. 2011).

Comparisons between the QLV models based on the cumulative scoring metric (E-10 to E-12) may be limited due to differences between the individual metrics and the applicability of combining these metrics without introducing more robust statistical techniques to compensate for these differences. Two of the five metrics were based on energy approximations, two on the RMSE and a single metric based on peak loading. Weighting of each of the five metrics as a component of the cumulative score for a given model response at a given loading frequency was based solely on examining the standard deviation associated with the six model responses for a given metric at a given loading frequency. This process provided increased weighting for metrics where larger differences were observed between models, while reducing the weight of metrics where model responses were similar. However, this process does not fully take into account fundamental differences in the weight of a given parameter used to create a given metric. Specifically, while the peak load metric was based on a single data point, the other four metrics were based on either the entire loading or unloading response. The current cumulative scoring metric does not address these a priori differences. Further investigation of the robustness of the current cumulative scoring metric and further development may be appropriate.
This study provides the first viscoelastic characterization of the pediatric human cervical spine in tension for the WCS, O-C2, C4-C5 and C6-C7 cervical segments. Energy dissipation was different between levels with more dissipation in lower segments than the O-C2 and WCS at all loading rates. Moreover, the cervical spine at the WCS and segmental level was generally rate insensitive at low loading rates, while at more dynamic rates of loading, increased energy dissipation was observed at all levels, although greater rate effects were observed in the O-C2 and WCS. The dissipative capacity of the WCS behaved similar to the O-C2 segment at low loading rates, while at higher rates more deviation existed between these structures with the O-C2 segment dissipating more energy compared to the WCS. PL and LE also increased with loading rate in the pediatric cervical spine. The Myers, FSHSE and Best models tended to outperform the FSH discrete time constant models in predicting the viscoelastic structural response of the pediatric cervical spine over three decades of loading.

7.5 Acknowledgments

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8. Flexion and Extension Bending of the Perinatal, Neonatal and Pediatric Cadaveric Cervical Spine

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8.1 Introduction

Biomechanical studies of the pediatric cervical spine have been limited in number, yet injuries to this critical anatomical structure lead to relatively high morbidity and mortality. Moreover, the cervical spine connects the torso to the head, controlling the kinematics of the head-neck structure. As such, the biomechanics of the pediatric cervical spine are not only important in understanding neck injury prevention, but also in improving the understanding of injury mechanisms of the head and skull. Research on pediatric head and neck injury has focused, in large part to injury prevention in the motor vehicle environment. Motor vehicle related crashes rank as the most common cause of spinal related injuries in the pediatric population (Platzer et al. 2007; Brown et al. 2001; Kokoska et al. 2001; Eleraky et al. 2000; Hamilton and Myles 1992a; Bonadio 1993; Babcock 1975). Pediatric spinal trauma accounts for between 1-12% of all spinal injuries (Hamilton and Myles 1992a; Hadley et al. 1988; Aufdermaur 1974). The overall mortality rate amongst victims of pediatric spinal trauma is approximately 4-41% but is

With the advances in occupant protection devices that have occurred over the last three decades, there has been a dramatic reduction in serious injuries and fatalities for all motor vehicle occupants (Viano 1995; Huelke et al. 1981; Hartemann et al. 1977). However, a small number of neck injuries attributed to inertial loading (non-contact neck injuries) in restrained occupants in severe collisions have been reported (Hammerstein et al. 2007; Houle et al. 2001; Huelke et al. 1978, 1992, 1993, 1995). While these injuries have been reported throughout the population, the anatomy of developing children may make them more susceptible. Mechanical factors that may heighten the risk of cervical spine injury in young children include greater head mass relative to neck strength and neck cross section than in the adult, laxity of the ligamentous components of the cervical spine, and decreased facet angle (Kasai et al. 1996; Swischuk 1977; Cattell and Filtzer 1965; Bailey 1952).

Despite its importance, few studies have investigated the biomechanical behavior of the human pediatric cervical spine owing in large measure to a lack of tissue donors. The existing studies vary significantly in design and typically have small sample sizes (Duncan 1874; Ouyang et al. 2005; Luck et al. 2008; Luck et al. 2012a; Luck et al. 2012b; Kallieris et al. 1976; Wismans et al. 1979). Ouyang et al is the only human
based study to-date that has focused on the bending response of the pediatric population. They examined the flexion and extension response of the whole cervical spine in pediatric cadavers from two to twelve years of age. Numerous animal surrogate studies assisted our understanding of the biomechanics of the pediatric cervical spine owing to limited human pediatric data (Nuckley and Ching 2006; Hilker et al. 2002; Ching et al. 2001; Pintar et al. 2000; Mertz and Weber 1982, Mertz et al. 1982, Prasad and Daniel 1984; Nuckley et al. 2002; Nuckley et al. 2005). The mechanical responses of individual pediatric motion segments have not been investigated previously. Similarly, the bending responses of the perinatal and neonatal population have not been investigated.

The primary goal of this study is to investigate the biomechanics of the pediatric cervical spine in bending. We tested the hypothesis that the trends in pediatric bending properties are grossly similar in those previously seen in the adult, the range of motion of the osteoligamentous cervical spine in flexion and extension at low loads decreases with age, and the range of motion of the upper cervical spine (O-C2) is greater than the range of motion of the lower cervical spine (C4-C5 and C6-C7) at all ages. The flexion and extension bending stiffness of the cervical spine at all cervical levels increases with age and the stiffness in extension is greater than the stiffness in flexion bending.
### Table 15: Perinatal, Neonatal, Pediatric and Young Adult Post-Mortem Human Subject (PMHS) Anthropometry

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (MN)</th>
<th>Sex</th>
<th>COD</th>
<th>Whole Body</th>
<th>Head</th>
<th>Spine</th>
</tr>
</thead>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mass (kg)</td>
<td>Height (cm)</td>
<td>Mass (kg)</td>
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<tr>
<td>13P</td>
<td>0</td>
<td>F</td>
<td>-</td>
<td>0.492</td>
<td>7.1</td>
<td>10.2</td>
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<tr>
<td>09P</td>
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<td>Fetal Demise</td>
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<td>SC</td>
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<td>-</td>
<td>0.434</td>
<td>8.2</td>
<td>10.1</td>
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<tr>
<td>10P</td>
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<td>M</td>
<td>Fetal Demise</td>
<td>-</td>
<td>-</td>
<td>SC</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>F</td>
<td>Diaphragmatic Hernia</td>
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<td>-</td>
<td>0.665</td>
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<tr>
<td>03P</td>
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<td>M</td>
<td>HIE; cerebral infarction</td>
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<td>-</td>
<td>0.492</td>
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<tr>
<td>06P</td>
<td>0.37</td>
<td>F</td>
<td>NIHF; ICH</td>
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<td>44.5</td>
<td>0.702</td>
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<td>11P</td>
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<td>Anencephaly</td>
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<td>-</td>
<td>SC</td>
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<tr>
<td>04P</td>
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<td>F</td>
<td>DWS</td>
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</table>

#### Pediatric PMHS > One Month [Older Cohort]

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<tr>
<th>PMHS ID</th>
<th>Age (MN)</th>
<th>Sex</th>
<th>COD</th>
<th>Whole Body</th>
<th>Head</th>
<th>Spine</th>
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<td></td>
<td></td>
<td>Mass (kg)</td>
<td>Height (cm)</td>
<td>Mass (kg)</td>
</tr>
<tr>
<td>12P</td>
<td>5</td>
<td>M</td>
<td>Respiratory Failure</td>
<td>-</td>
<td>-</td>
<td>1.071</td>
</tr>
<tr>
<td>14P</td>
<td>9</td>
<td>M</td>
<td>COPD</td>
<td>7.00</td>
<td>-</td>
<td>1.950</td>
</tr>
<tr>
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<td>11</td>
<td>F</td>
<td>SIDS</td>
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<td>F</td>
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<td>F</td>
<td>GCT</td>
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<td>18P</td>
<td>108</td>
<td>M</td>
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<td>-</td>
<td>-</td>
<td>2.440</td>
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<td>-</td>
<td>SC</td>
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<tr>
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<td>-</td>
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<td>M</td>
<td>Gunshot</td>
<td>66.68</td>
<td>160.0</td>
<td>SC</td>
</tr>
<tr>
<td>23P</td>
<td>216</td>
<td>F</td>
<td>Overdose</td>
<td>-</td>
<td>-</td>
<td>SC</td>
</tr>
</tbody>
</table>

MN - months; SC - skull compromise (PMHS acquired in this state); DWS - Dandy-Walker Syndrome

COPD - Chronic Obstructive Pulmonary Disease; SIDS - Sudden Infant Death Syndrome

NIHF - Non-immune hydrops fetalis; ICH - Intercranial Hemorrhage

ESRD - End-stage Renal Disease; ↑K+ - Hyperkalemia; HIE - Hypoxic Ischemic Encephalopathy

NHL - Non-Hodgkin's Lymphoma; GCT - Germ Cell Tumor (Carcinoma)
8.2 Materials and Methods

Twenty-two unembalmed cervical spinal (head to T1) cadaver specimens from 29 weeks gestation to 18 years old were tested (Table 15). These specimens included nine perinatal and neonatal specimens (29 weeks gestation to 24 days old) and thirteen infant to young adult specimens (5 month to 18 years old). All specimen handling was performed in compliance with CDC guidelines (Cavanaugh and King, 1990). All neck musculature, subcutaneous fatty tissue, and skin were removed, and the mandible was disarticulated to enable rigid head fixation and allow visualization of the upper cervical spine. The spines were sectioned into 58 spinal segments including the upper (O-C2) and lower (C4-C5 and C6-C7) cervical spine.

Because of their high scientific value, the heads and necks of these pediatric specimens were the subject of a large testing program designed to maximize the knowledge gained. Full details of the apparatus and associated tests are presented elsewhere (Luck et al. 2008; Luck et al. 2012a; Luck et al. 2012b; Prange et al. 2004). The caudal vertebrae of the lower cervical segments were cast into aluminum cups. Fixation of the cephalic vertebrae of the lower cervical segments included k-wire and polymethyl-methacrylate (PMMA) application (Dentsply International; York, PA, U.S.A.). A polycarbonate plate with anchoring screws was inserted into the PMMA during the curing process to form an attachment point for the eccentric loading bar. The upper cervical spinal segment was mounted with the head inverted in the testing
fixture. The specimen was secured to the testing fixture at the maxilla and about the occipital region with the Frankfort Plane aligned horizontally. The C2/C3 vertebrae were secured to one another using k-wires and associated fixation techniques. Otherwise, the fixation was similar to that for the lower cervical spine segments (Figure 36).

An eccentric loading bar applied pure moments to the upper and lower segments due to the large rotations expected in the younger cohort and to limitations associated with the necessity to rigidly fixate the head in the experimental apparatus. Details of the fixation technique of the head and the associated experimental apparatus are available in Luck et al. 2012a. A counterweight was attached to the eccentric loading bar at a predetermined center of gravity using a precision spherical bearing. The counterweight and its position along the eccentric bar were designed to counteract any compression and bending moment that the bar might impart on the specimen. As the vertebrae size increased with age, the amount of PMMA used to fix the superior vertebrae was reduced to maintain a constant mass and center of gravity for the entire eccentric bar and superior vertebrae complex.
FIGURE 36: Upper cervical spine (O-C2) cast in the experimental apparatus with rigid fixation of the cranium and C2-C3 fixation caudally enabling pure moment application. A pure moment was applied through an eccentric loading bar. A counterweight (C) system enabled continuous vertical alignment of the off-set load for the eccentric bar through its center-of-gravity. A loading carriage (L) enabled continuous vertical alignment of the upward force with sagittal and coronal plane translation.
Figure 37: O–C2 flexion resulted in large rotations at small moments specifically within the perinatal and neonatal age group. The experimental apparatus and eccentric loading bar system allowed for these extreme angles to be quantified. The downward line-of-action (LoA) from the loading disk proceeded through an opening designed into the posterior section of the loading frame; allowing for additional rotation by increasing the horizontal clearance in the experimental set-up.
**FIGURE 38:** O-C2 extension resulted in large rotations at small moments specifically within the perinatal and neonatal age group. These large rotations required the loading bar to move through the profile of the experimental apparatus.

The specimens were manually preconditioned with 30 cycles at approximately 10% of the expected maximum moment applied during testing. Each segment was loaded in flexion and extension with the peak applied moment determined by one of
two criteria (Figure 37 and 38). The first was if the flexion or extension rotation was large enough to cause binding of either the eccentric loading bar or the superior and inferior casting. The second criterion was if the segment or fixation reached a point where damage to either was imminent. After load application, the segment was allowed to creep for 30 seconds prior to data acquisition and the load was released after each loading step (Nightingale et al. 2007, 2002). A six-axis load cell (2554A, Denton, Inc., Rochester Hills, MI) was mounted at the base of the fixation to monitor the applied moment and all off-axis channels. All six channels of data were acquired with a PC based data acquisition system (National Instruments, Austin, TX). An imaging system recorded the motions of the cervical segments during the tests (Phantom camera system, Vision Research, Inc., Wayne, New Jersey) and was used to determine the angular displacement data. Optical markers located on the superior casting and eccentric loading bar were tracked and the angular displacements were computed using the TEMA Motion Analysis software package (Image Systems, North Hollywood, CA).

The angle and moment data were correlated using a least squares regression of a non-linear function of the following form: \( \theta = A \ln(BM + 1) \), where \( \theta \) is the angle, \( M \) is the moment and \( A \) and \( B \) are model constants. This flexibility function has been used in describing the non-linear behavior of soft tissues (Fung, 1972; Simon et al., 1984; Nightingale et al. 2002; Nightingale et al. 2007), including modeling of the adult cervical spine in bending (Nightingale et al. 2002; Nightingale et al. 2007). Individual flexibility
functions were determined for each spinal segment for both flexion and extension. The individual flexibility functions are defined over a range of moment from approximately a peak moment of ± 0.1 N-m to ± 2.5 N-m depending on the segment and age. While individual flexibility functions provide the moment-angle response over the entire loading of an individual specimen, it is difficult to compare the stiffness of different ages by comparing these functions. A linear estimate of flexion and extension stiffness for each specimen was determined by calculating the stiffness at 0.1 and 0.05 N-m from the individual flexibility functions and determining the slope defined by these two points. This linear stiffness approximates the low-load response of the cervical spine and was used for this analysis as it is the maximum moment applied to all of the specimens tested.

Non-linear models of the flexion and extension total range of motion and stiffness with age were determined with a least squares regression to ascertain changes in these parameters from birth to early adulthood. All perinatal donors were designated as zero months of age and all neonatal donor ages were converted to fractions of a month for the purposes of this analysis. The statistical significance of the non-linear regressions compared to the regressed data was assessed using a single-factor ANOVA (p < 0.01). The younger cohort (perinates and neonates (~ 1 month)) was grouped and the biomechanical responses for the three segment levels (O-C2, C4-C5 and C6-C7) were
analyzed using a single-factor ANOVA, followed by a post-hoc Tukey-Kramer HSD multiple-comparison test (p < 0.01).

### 8.3 Results

#### 8.3.1 Perinatal, Neonatal and Pediatric Cervical Spine Flexion and Extension Response

The flexion and extension bending response for each segment was defined by a non-linear flexibility function between moment and angle. At the younger ages, the maximum applied moments were smaller than those in the older cohort as a result of increased rotation at younger ages. These increases in rotation led to the potential for specimen-fixture binding and the onset of ligamentous injury and therefore halted further loading. Therefore, although some of the mechanical responses represented by the flexibility functions, especially in the older cohort, may be applicable to quasi-static load levels in the range of ± 2.5 N-m; this is not necessarily the case in the younger ages where loads typically peaked at ± 0.1 N-m. These data are grouped into two cohorts, an older cohort consisting of data from pediatric and young adult cadavers at five months and older in age and a younger cohort consisting of data from perinatal and neonatal cadavers from 29 weeks gestation to 24 days (Tables 16-18).
### Table 16: Flexion and Extension Bending Coefficients for Perinatal, Neonatal and Pediatric O-C2 Segments

\[ \theta = A[\ln(BM+1)] \]

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Perinatal and Neonatal PMHS &lt; One Month [Younger Cohort]</th>
<th>Overall ROM</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td>O-C2 Extension Max Angle</td>
<td>Max Moment</td>
</tr>
<tr>
<td>07P</td>
<td>0</td>
<td>-3.31 -463749 -34.9</td>
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<tr>
<td>09P</td>
<td>0</td>
<td>-     -     -</td>
<td>-</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
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<td>-</td>
</tr>
<tr>
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</tr>
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<tr>
<td>11P</td>
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<td>-</td>
</tr>
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<td>0.8</td>
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<td>-0.09</td>
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<table>
<thead>
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<th>Age (months)</th>
<th>Pediatric PMHS &gt; One Month [Older Cohort]</th>
<th>Overall ROM</th>
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<td>-</td>
</tr>
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θ = A[ln(BM+1)]; A (degrees) and B (1/Nm) are model coefficients determined from regression of experimental data

- Segment not available; Max Angle and Moment - Maximum angle and moment observed during test

Overall ROM - Flexion and extension range of motion combined [Maximum moment observed is not the same for all specimens and should be considered when comparing specimen results]

Individual bending response models are valid through the maximum moment and angles observed during testing. Caution should be used when extrapolating the response to larger moments [outside of the maximum moments indicated]
Table 17: Flexion and Extension Bending Coefficients for Perinatal, Neonatal and Pediatric C4-C5 Segments

\( \theta = A \ln(BM+1) \): relationship relating measured angle (\( \theta \)) and measured moment (\( M \))

### Perinatal and Neonatal PMHS < One Month [Younger Cohort]

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
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<th>Max Angle</th>
<th>Max Moment</th>
<th>Flexion</th>
<th>Max Angle</th>
<th>Max Moment</th>
<th>Overall ROM</th>
</tr>
</thead>
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### Pediatric PMHS > One Month [Older Cohort]

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<th>Max Angle</th>
<th>Max Moment</th>
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<th>Max Angle</th>
<th>Max Moment</th>
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</table>

\( \theta = A \ln(BM+1) \): A (degrees) and B (1/Nm) are model coefficients determined from regression of experimental data

- Segment not available; † - C3-C4 segment; Max Angle & Moment - Maximum angle and moment observed during test

Overall ROM - Flexion and extension range of motion combined [Maximum moment observed is not the same for all specimens and should be considered when comparing specimen results]

Individual bending response models are valid through the maximum moment and angles observed during testing. Caution should be used when extrapolating the response to larger moments [outside of the maximum moments indicated]
### Table 18: Flexion and Extension Bending Coefficients for Perinatal, Neonatal and Pediatric C6-C7 Segments

The table below presents the flexion and extension bending coefficients for Perinatal, Neonatal and Pediatric C6-C7 segments. The coefficients are calculated using the equation $\theta = A \times \ln(BM+1)$, where $\theta$ is the measured angle, $A$ and $B$ are model coefficients determined from regression of experimental data.

**Perinatal and Neonatal PMHS < One Month [Younger Cohort]**

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Extension A</th>
<th>Max Angle</th>
<th>Max Moment</th>
<th>Flexion A</th>
<th>Max Angle</th>
<th>Max Moment</th>
<th>Overall ROM</th>
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**Pediatric PMHS > One Month [Older Cohort]**

<table>
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<th>PMHS ID</th>
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<th>Extension A</th>
<th>Max Angle</th>
<th>Max Moment</th>
<th>Flexion A</th>
<th>Max Angle</th>
<th>Max Moment</th>
<th>Overall ROM</th>
</tr>
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<td>—</td>
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<td>4.88</td>
<td>5.21</td>
<td>11.9</td>
</tr>
<tr>
<td>23P</td>
<td>216</td>
<td>-3.88</td>
<td>-4.55</td>
<td>-8.7</td>
<td>-2.07</td>
<td>5.34</td>
<td>8.81</td>
<td>15.8</td>
</tr>
</tbody>
</table>

$\theta = A \times \ln(BM+1)$: A (degrees) and B (1/Nm) are model coefficients determined from regression of experimental data. Overall ROM - Flexion and extension range of motion combined [Maximum moment observed is not the same for all specimens and should be considered when comparing specimen results]. Individual bending response models are valid through the maximum moment and angles observed during testing. Caution should be used when extrapolating the response to larger moments [outside of the maximum moments indicated].

Total range of motion (flexion and extension) was observed to decrease with age from the perinate to the young adult for all cervical levels at low-load (Tables 19 and 20). For the younger cohort, statistically significant differences were found in total range of motion.
motion by level at low load (P < 0.0001, n=21, single-factor ANOVA). Post-hoc comparisons indicated significant differences in the range of motion between the upper cervical spine and both lower cervical spine segments (P < 0.0001, Tukey-Kramer HSD). This analysis did not reveal any significant differences between the two lower motion segments, C4-C5 and C6-C7 (P = 0.9908; Tukey-Kramer HSD).

**TABLE 19: Age Based Pediatric Regression Models for Flexion and Extension Range of Motion**

<table>
<thead>
<tr>
<th>Segment Level</th>
<th>Dependent Variable (Y)</th>
<th>Non-linear Regression Models</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>R²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>O-C2</td>
<td>Range of Motion (± 0.1 N-m load range)</td>
<td>Y = A(age)^B + C</td>
<td>-26.07</td>
<td>0.192</td>
<td>101.01</td>
<td>0.84</td>
<td>§</td>
</tr>
<tr>
<td>C4-C5</td>
<td>Range of Motion (± 0.1 N-m load range)</td>
<td></td>
<td>-9.82</td>
<td>0.203</td>
<td>30.75</td>
<td>0.87</td>
<td>§</td>
</tr>
<tr>
<td>C6-C7</td>
<td>Range of Motion (± 0.1 N-m load range)</td>
<td></td>
<td>-11.91</td>
<td>0.177</td>
<td>32.38</td>
<td>0.91</td>
<td>§</td>
</tr>
<tr>
<td>WCS</td>
<td>Range of Motion (± 0.1 N-m load range)</td>
<td></td>
<td>-82.32</td>
<td>0.204</td>
<td>283.42</td>
<td>0.92</td>
<td>§</td>
</tr>
</tbody>
</table>

**Single-Factor ANOVA:** § - statistically significant (p < 0.01)

The range of motion for the whole spine was estimated using the low-load response of the individual segments (Table 21). Whole spine range of motion was observed to decrease with increasing age. The whole spine was not directly tested, rather, the bending response of the segments for a specific specimen were used to approximate the total range of motion from the base of skull to T1. This estimation represents an upper bound at low load of the magnitude of rotation possible for a given age, but based on the physical anatomy of the child, this upper bound may not be attainable.
**Table 20:** Flexion and Extension Range of Motion for Perinatal, Neonatal and Pediatric PMHS (± 0.1 N-m load range)

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Perinatal and Neonatal PMHS &lt; One Month [Younger Cohort]</th>
<th>Pediatric PMHS &gt; One Month [Older Cohort]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>O-C2</td>
<td>C4-C5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexion (degrees)</td>
<td>Extension (degrees)</td>
</tr>
<tr>
<td>07P</td>
<td>0</td>
<td>57.0</td>
<td>-35.6</td>
</tr>
<tr>
<td>09P</td>
<td>0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>60.4</td>
<td>-35.5</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>86.9</td>
<td>-27.5</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>50.9</td>
<td>-41.0</td>
</tr>
<tr>
<td>11P</td>
<td>0.53</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>42.1</td>
<td>-36.1</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td>59</td>
<td>-35</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>17</td>
<td>5</td>
</tr>
</tbody>
</table>

Range of motion determined for low load (± 0.1 N-m) for all ages using individual bending response models [Tables 16, 17 and 18] for each segment. \( \theta = A \{ \ln(BM+1) \} \): M is the moment (± 0.1 N-m) and \( \theta \) is the angle presented in the table.

— - Segment not available; † - C3-C4 segment; ‡ - C5-C6 segment

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Table 21: Range of Motion (ROM) Estimation for the Whole Spine of Perinatal, Neonatal and Pediatric PMHS (± 0.1 N-m load range)

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Flexion (degrees)</th>
<th>Extension (degrees)</th>
<th>ROM (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>07P</td>
<td>0</td>
<td>199.7</td>
<td>-72.3</td>
<td>271.9</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>157.6</td>
<td>-102.7</td>
<td>260.3</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>224.5</td>
<td>-72.6</td>
<td>297.1</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>121.9</td>
<td>-119.8</td>
<td>241.7</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>173.2</td>
<td>-59.8</td>
<td>233.0</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>175</td>
<td>-85</td>
<td>261</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>39</td>
<td>25</td>
<td>25</td>
</tr>
</tbody>
</table>

Pediatric PMHS > One Month

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>Flexion (degrees)</th>
<th>Extension (degrees)</th>
<th>ROM (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12P</td>
<td>5</td>
<td>83.3</td>
<td>-63.2</td>
<td>146.5</td>
</tr>
<tr>
<td>14P</td>
<td>9</td>
<td>94.3</td>
<td>-36.6</td>
<td>130.8</td>
</tr>
<tr>
<td>15P</td>
<td>11</td>
<td>80.6</td>
<td>-41.2</td>
<td>121.9</td>
</tr>
<tr>
<td>17P</td>
<td>22</td>
<td>49.4</td>
<td>-56.6</td>
<td>106.1</td>
</tr>
<tr>
<td>24P</td>
<td>72</td>
<td>46.9</td>
<td>-45.1</td>
<td>91.9</td>
</tr>
<tr>
<td>19P</td>
<td>84</td>
<td>27.3</td>
<td>-16.8</td>
<td>44.1</td>
</tr>
<tr>
<td>18P</td>
<td>108</td>
<td>60.6</td>
<td>-20.5</td>
<td>81.1</td>
</tr>
<tr>
<td>20P</td>
<td>144</td>
<td>34.1</td>
<td>-16.6</td>
<td>50.7</td>
</tr>
<tr>
<td>21P</td>
<td>192</td>
<td>23.6</td>
<td>-30.6</td>
<td>54.3</td>
</tr>
<tr>
<td>22P</td>
<td>204</td>
<td>29.1</td>
<td>-16.8</td>
<td>45.9</td>
</tr>
<tr>
<td>23P</td>
<td>216</td>
<td>27.3</td>
<td>-41.5</td>
<td>68.8</td>
</tr>
</tbody>
</table>

Range of motion determined for low load (± 0.1 N-m) using individual segmental bending responses [Table 20]. Whole spine estimated by adding the angular response observed at the segment level: O-C2 + C4-C5 + C6-C7 + 4(AVE(LCS)). Where 4(AVE(LCS)) represents the C2-C3, C3-C4, C5-C6 and C7-T1 response. AVE(LCS) is the average of the experimentally determined C4-C5 and C6-C7 responses.
### Table 22: Bending Stiffness for Perinatal, Neonatal and Pediatric PMHS (Nm/degree)

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>O-C2 Flexion</th>
<th>O-C2 Extension</th>
<th>C4-C5 Flexion</th>
<th>C4-C5 Extension</th>
<th>C6-C7 Flexion</th>
<th>C6-C7 Extension</th>
</tr>
</thead>
<tbody>
<tr>
<td>07P</td>
<td>0</td>
<td>0.0033</td>
<td>0.0218</td>
<td>0.0053</td>
<td>0.0754</td>
<td>0.0159</td>
<td>0.0299</td>
</tr>
<tr>
<td>09P</td>
<td>0</td>
<td>─</td>
<td>─</td>
<td>0.0154</td>
<td>0.0220</td>
<td>0.0216</td>
<td>0.0273</td>
</tr>
<tr>
<td>10P</td>
<td>0</td>
<td>─</td>
<td>─</td>
<td>0.0071</td>
<td>0.0233</td>
<td>0.0083</td>
<td>0.0356</td>
</tr>
<tr>
<td>13P</td>
<td>0</td>
<td>0.0030</td>
<td>0.0050</td>
<td>0.0115</td>
<td>0.0164</td>
<td>0.0176</td>
<td>0.0155</td>
</tr>
<tr>
<td>05P</td>
<td>0.03</td>
<td>0.0012</td>
<td>0.0069</td>
<td>0.0142</td>
<td>0.0236</td>
<td>0.0451</td>
<td>0.0121</td>
</tr>
<tr>
<td>06P</td>
<td>0.37</td>
<td>0.0180</td>
<td>0.0062</td>
<td>0.0101</td>
<td>0.0237</td>
<td>0.0249</td>
<td>0.0195</td>
</tr>
<tr>
<td>11P</td>
<td>0.53</td>
<td>─</td>
<td>─</td>
<td>0.0141</td>
<td>0.0233</td>
<td>0.0149</td>
<td>0.0215</td>
</tr>
<tr>
<td>04P</td>
<td>0.8</td>
<td>0.0058</td>
<td>0.0034</td>
<td>0.0135</td>
<td>0.0274</td>
<td>0.0072</td>
<td>0.0301</td>
</tr>
</tbody>
</table>

**Average:**
- 0.0063 | 0.0087
- 0.0114 | 0.0294
- 0.0194 | 0.0251

**SD:**
- 0.0068 | 0.0075
- 0.0036 | 0.0188
- 0.0120 | 0.0067

---

<table>
<thead>
<tr>
<th>PMHS ID</th>
<th>Age (months)</th>
<th>O-C2 Flexion</th>
<th>O-C2 Extension</th>
<th>C4-C5 Flexion</th>
<th>C4-C5 Extension</th>
<th>C6-C7 Flexion</th>
<th>C6-C7 Extension</th>
</tr>
</thead>
<tbody>
<tr>
<td>12P</td>
<td>5</td>
<td>0.0084</td>
<td>0.0180</td>
<td>0.0302</td>
<td>0.0236</td>
<td>0.0314</td>
<td>0.0224</td>
</tr>
<tr>
<td>14P</td>
<td>9</td>
<td>0.0084</td>
<td>0.0105</td>
<td>0.0212</td>
<td>0.0465</td>
<td>0.0176</td>
<td>0.0403</td>
</tr>
<tr>
<td>15P</td>
<td>11</td>
<td>0.0080</td>
<td>0.0103</td>
<td>0.0243</td>
<td>0.0599</td>
<td>0.0219</td>
<td>0.0291</td>
</tr>
<tr>
<td>17P</td>
<td>22</td>
<td>0.0091</td>
<td>0.0316</td>
<td>0.0263</td>
<td>0.0512</td>
<td>0.0290</td>
<td>0.1642</td>
</tr>
<tr>
<td>24P</td>
<td>72</td>
<td>0.0122</td>
<td>0.0174</td>
<td>0.0294</td>
<td>0.0421</td>
<td>0.0279</td>
<td>0.0610</td>
</tr>
<tr>
<td>19P</td>
<td>84</td>
<td>0.0111</td>
<td>0.0223</td>
<td>0.0621</td>
<td>0.1388</td>
<td>0.0531</td>
<td>0.1518</td>
</tr>
<tr>
<td>18P</td>
<td>108</td>
<td>0.0076</td>
<td>0.0226</td>
<td>0.0306</td>
<td>0.1204</td>
<td>0.0339</td>
<td>0.0778</td>
</tr>
<tr>
<td>20P</td>
<td>144</td>
<td>0.0131</td>
<td>0.0234</td>
<td>0.0397</td>
<td>0.1963</td>
<td>0.0477</td>
<td>0.0790</td>
</tr>
<tr>
<td>01P</td>
<td>168</td>
<td>0.0145</td>
<td>0.0221</td>
<td>─</td>
<td>─</td>
<td>─</td>
<td>─</td>
</tr>
<tr>
<td>21P</td>
<td>192</td>
<td>0.0138</td>
<td>0.0266</td>
<td>0.0570</td>
<td>0.2029</td>
<td>0.0727</td>
<td>0.2824</td>
</tr>
<tr>
<td>22P</td>
<td>204</td>
<td>0.0150</td>
<td>0.0174</td>
<td>0.0463</td>
<td>0.1836</td>
<td>0.0545</td>
<td>0.1128</td>
</tr>
<tr>
<td>23P</td>
<td>216</td>
<td>0.0189</td>
<td>0.0164</td>
<td>0.0351</td>
<td>0.0896</td>
<td>0.0351</td>
<td>0.0758</td>
</tr>
</tbody>
</table>

---

**Notes:** Specimen not available; Stiffness calculated using flexibility functions for each PMHS and determining the angle at (±) 0.05 and 0.1 Nm and determining the slope associated with these end-points.
Low-load flexion and extension stiffness was observed to increase with age from the perinate to the young adult for all cervical levels (Table 22). For the younger cohort, the flexion and extension stiffness of O-C2 were not significantly different from one another, as was the case with C6-C7 where no differences were observed (P = 0.6087 [O-C2]; P = 0.2698 [C6-C7]). C4-C5 extension was different, but not significantly from the flexion response (P = 0.0308). Flexion stiffness of the O-C2 segment was less than both of the lower segments, but not statistically different (P = 0.0349). Similarly, extension stiffness of the O-C2 segment was less than both lower segments, but not statistically different (P = 0.0333). However, if the extension stiffness of 07P was removed as an outlier, as it is 2.5 times greater than the average C4-C5 extension stiffness, then statistically significant differences are found in extension stiffness by level (P < 0.001, n=20, single-factor ANOVA). Post-hoc comparisons indicated significant differences in the extension stiffness between the O-C2 and both lower cervical spine segments (P < 0.01, Tukey-Kramer HSD). This analysis did not reveal any significant differences between the two lower motion segments, C4-C5 and C6-C7 (P = 0.7472; Tukey-Kramer HSD).

8.3.2 Age Based Regression Models

Age and range of motion observed at ± 0.1 N-m in the upper cervical spine was well correlated and statistically significant when the data was regressed with a power law type relationship (Figure 39: O-C2 ROM, r²=0.84, p < 0.01). The non-linear
regression accurately represents the rapid decrease in range of motion from birth to less than five months. Decreasing range of motion with increasing age is observed at ages greater than five months although the decrease in range of motion is less at older ages. A comparison of the five-month-old response to the average of the perinatal and neonatal cohort shows that the range of motion decreased by 44%, whereas a similar comparison between the average range of motion of the older cohort and the younger cohort reveals a reduction in range of motion of only 58%.

**FIGURE 39:** Range of motion in the upper cervical spine at low load (± 0.1 N-m; flexion and extension).
Figure 40: Range of motion in the lower cervical spine (C4-C5) at low load (± 0.1 N-m; flexion and extension).

Figure 41: Range of motion in the lower cervical spine (C6-C7) at low load (± 0.1 N-m; flexion and extension).
Range of motion in the lower motion segments were also well correlated and statistically significant with age when regressed with a similar power law relationship (Figures 40 and 41: C4-C5 ROM, $r^2=0.87$, $p < 0.01$; C6-C7 ROM, $r^2=0.91$, $p < 0.01$). Similar non-linear decreases in range of motion in the lower cervical spine with age are observed. Both lower segments show large decreases in range of motion in the first months of life (42 to 46%; reduction in range of motion when comparing the five-month-old ROM to the average ROM for the younger cohort). However, unlike the upper segment where the majority of the reduction in range of motion is observed in the first few months; in the lower segments continued large reductions in range of motion are observed in the older age cohort (73 to 75% reduction in range of motion when comparing the average ROM for the older cohort to the average ROM for the younger cohort).

Estimated range of motion in the whole cervical spine (OC-T1) was well correlated and statistically significant with age when regressed with a power law relationship (Figures 42: Whole Spine Estimated ROM, $r^2=0.92$, $p < 0.01$). Range of motion in the whole spine rapidly decreased from birth to five-months-old (44% decrease) and was followed by additional decreases in range of motion with age in the older cohort, although the decrease was not as large in the older cohort as observed with the lower segments (67% reduction in range of motion when comparing the average ROM for the older cohort to the average ROM for the younger cohort).
FIGURE 42: Range of motion in the whole cervical spine at low load (± 0.1 N-m; flexion and extension). Whole spine range of motion is estimated based on the response of the upper and lower cervical spine.

Low-load flexion and extension stiffness in all segments was well correlated and statistically significant when the data was regressed with a power law type relationship (Figures 43-45: O-C2 Flexion, $r^2=0.51$, $p < 0.01$; O-C2 Extension, $r^2=0.37$, $p < 0.05$; C4-C5 Flexion, $r^2=0.75$, $p < 0.01$; C4-C5 Extension, $r^2=0.74$, $p < 0.01$; C6-C7 Flexion, $r^2=0.57$, $p < 0.01$; C6-C7 Extension, $r^2=0.47$, $p < 0.01$). Increases in low-load extension stiffness of greater than 100% were observed in all segments between the average younger and older cohort response. Smaller increases in flexion stiffness approaching 100% were observed in the O-C2 and C6-C7 segments while greater than a 200% change in stiffness with age was observed in the C4-C5 segment.
FIGURE 43: Flexion and extension stiffness in the upper cervical spine (O-C2) at low load (± 0.1 N-m; flexion and extension).

Modeling parameters associated with each of the non-linear regressions are provided in Tables 19 and 23. All regressions were carried out over the range of the sample included in the current study. Extrapolating structural properties for ages greater than those bounded by the current study are not appropriate and may not be defined by the current non-linear models. Moreover, the regressions are based on flexion and extension range of motion data at quasi-static low loads (± 0.1 N-m) and are not intended to portray the behavior of the cervical spine under dynamic loading conditions associated with high loads.
8.4 Discussion

This work adds to the limited existing data on the flexion and extension bending response of the perinatal, neonatal and pediatric cadaveric cervical spine. Individual bending response models for ages from 29 weeks gestation to 18 years are provided for flexion and extension that model the low-load bending behavior and in the case of many of the older ages the loading behavior up to ± 2.5 N-m. Moreover, correlated models relating age to bending range of motion and flexion and extension stiffness in the developing cervical spine are introduced that may be used to better approximate the biomechanical behavior of the pediatric neck.
This study had a number of limitations. Perinatal, neonatal and pediatric cadaveric tissues are exceedingly rare. While the ages under one month were combined into a younger cohort and provided additional statistical confidence, the older ages were limited to a single specimen per age. The lack of load-to-failure testing and limitations in the peak applied moment was directly associated with additional biomechanical investigations including cervical spine tensile testing and evaluation of the biomechanics of the head that occurred after this investigation and thus created additional constraints to the current experimental design (Prange et al., 2004; Luck et al. 2008; Luck et al. 2012b). Although we did not observe cervical spine pathology in our donor population that would alter the mechanical response, there were specimens with cranial defects or
deformities that limited testing in the upper cervical spine (T09P, T10P, T11P, and T16P) and specimens with local post-mortem damage (T16P) that required testing of adjacent spinal segments (C3-C4 and C5-C6).

**Table 23:** Age Based Pediatric Regression Models for Flexion and Extension Stiffness

<table>
<thead>
<tr>
<th>Segment Level</th>
<th>Rotation</th>
<th>Dependent Variable (Y)</th>
<th>Non-linear Regression Models</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Y = A(age)^B + C</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>O-C2</td>
<td>Flexion</td>
<td>3.99E-03</td>
<td>0.183</td>
<td>3.46E-03</td>
</tr>
<tr>
<td></td>
<td>Extension</td>
<td>2.63E-03</td>
<td>0.306</td>
<td>9.39E-03</td>
</tr>
<tr>
<td>C4-C5</td>
<td>Flexion</td>
<td>7.92E-03</td>
<td>0.290</td>
<td>9.26E-03</td>
</tr>
<tr>
<td></td>
<td>Extension</td>
<td>4.82E-03</td>
<td>0.634</td>
<td>2.67E-02</td>
</tr>
<tr>
<td>C6-C7</td>
<td>Flexion</td>
<td>1.16E-03</td>
<td>0.634</td>
<td>1.94E-02</td>
</tr>
<tr>
<td></td>
<td>Extension</td>
<td>1.34E-02</td>
<td>0.413</td>
<td>2.10E-02</td>
</tr>
</tbody>
</table>

**Single-Factor ANOVA:** † - statistically significant (p < 0.05); § - statistically significant (p < 0.01)

Range of motion decreased with age for all cervical levels with greater reductions in range of motion seen in the youngest ages. These reductions are not unexpected based on engineering beam theory and the natural growth of the cervical spine with age. Beam theory predicts that given the same material properties, but differences in length and second moment of area the deflection of two beams will differ given that they are loaded similarly. The length of the cervical spine from birth to young adulthood was observed to increase non-linearly with age in the current work, while the second moment of area approximated by the C7 and T1 cross-section was also observed to increase non-linearly with age. Material properties being equal at one year and 18 years and applying only the differences in length and second moment of area it is expected
that reductions in range of motion by simple geometry can be expected on the order of three fold. The results of the current study indicated that WCS ROM reduced by roughly four fold. While the approximations of length and second moment of area could be refined these results are consistent with the majority of the reduction in ROM resulting from anatomical growth, while a smaller reduction in ROM is due to differences in the structural stiffness between ages.

The authors are unaware of any studies that have evaluated the segmental bending responses of the cervical spine in perinatal, neonatal and pediatric osteoligamentous tissue. Ouyang et al. investigated the flexibility of the whole spine by evaluating the rotation at the T1 and C2 levels referenced to a fixed skull in specimens from 2 to 12 years of age. Pure moments of \( \pm 2.4 \) N-m were applied in extension and flexion at three load steps in each direction and absolute rotations of T1 and C2 and the relative rotation of T1 to C2 were measured. Flexibility functions for the individual ages were not provided. Greater ROM in the upper cervical spine was typically observed in the current study when compared to Ouyang. The 22-month-old in the present study exhibited a ROM nine-fold greater than the 2 year old in the Ouyang study at roughly 40\% of the moment (\(~0.65\) N-m to \(~1.6\) N-m). Moreover, the ROM from 2.5 to 5 years in the present study ranged from being equivalent to nine-fold the ROM exhibited in the five specimens from Ouyang (2.5 to 5 years) at roughly 13\% of the moment (\(~0.2\) N-m to \(~1.60\) N-m). For the oldest age range coincident with both studies (6 to 12 years), our
ROMs were typically 1.6 to 2.7 times greater than those of Ouyang et al. at roughly 93 to 97% of the moment. ROM in the whole spine at ± 0.1 N-m in the current study was typically 2 to 10 times greater at 1/8th the moment when compared to Ouyang.

Differences between the current findings and those of Ouyang may be attributed in part to differences in the testing methodology. While the current study, especially at younger ages acquired moment-angle responses less than 0.8 N-m, Ouyang acquired their first data point at 0.8 N-m. The current findings suggest that large rotations are possible in the upper cervical spine at low-loads, especially in younger ages and it is therefore likely that the highly non-linear characteristics of the spine were not observed by Ouyang.

The limited availability of pediatric tissue has led to alternative models for the investigation of the biomechanical behavior of the human pediatric neck. Pediatric models based on caprine necks have been used as a surrogate for pediatric human spines (Pintar et al. 2000). Upper and lower motion segments were evaluated in flexion and extension bending with a pure moment of 2 N-m. The upper cervical segments of the caprine model had such large rotations with small moments that the data was disregarded. Flexion and extension stiffness defined as a linear regression between 0.5 and 2 N-m were reported for the 1, 3, 6 and 12 year-old human equivalent spine. Flexibility functions and range of motion were not directly reported. However, estimations of range of motion at 0.5 N-m and 2.0 N-m are possible based on the linear
stiffness data provided. The estimates for total flexion and extension range of motion in
the human surrogate one-year-old at 0.5 N-m was approximately 31% of the range of
motion seen in the human at 0.1 N-m. The caprine surrogate consistently
underestimated the range of motion in the lower cervical spine of the human at low load
(3 YO, 37%; 6 YO, 12%; 12 YO, 21%).

Ligamentous laxity of the pediatric cervical spine has been suggested as one
factor contributing to such normal anatomical variants as pseudosubluxation and
hypermobility in the atlanto-axial joint (Cattell and Filtzer, 1965; Swischuk, 1977; Bailey
1952). Pseudosubluxation of the C2 vertebra on C3 has been documented as a normal
variant in children and noted as a confounding factor in identifying injury.
Additionally, increased distances between the odontoid process and the anterior arch in
flexion and overriding of the anterior arch on the odontoid in extension have been
observed. These normal variants have been well documented radiographically, but a
quantitative evaluation providing additional support at the osteoligamentous level of
the extent to which the perinatal, neonatal and pediatric cervical spine is able to rotate in
flexion and extension at low loads has yet to be documented. Morphological changes in
facet angle with age and reduction in adjacent vertebral sliding have been reported in
children from radiographs and suggest that increasing facet angle with age reduces
sliding between adjacent vertebrae (Kasai et al. 1996). Range of motion at low-loads was
observed to reduce significantly with age, while flexion and extension stiffness at low-
load was observed to increase with age. While the results of the current study do not differentiate between changes in stiffness that may result from only morphological or ligamentous changes it is clear that at younger ages especially where pseudosubluxation has been observed the cervical spine at all levels is less stiff and more mobile than at older ages. These results support the suggestion that the immature cervical spine has greater ligamentous laxity than the older spine at low loads and may be a contributing component to the observation of pseudosubluxation.

The current study investigated the biomechanical flexion and extension bending response in perinatal, neonatal, pediatric and young adult human cadavers. Upper and lower cervical spine flexibility functions for flexion and extension, and total range of motion at low load were determined. Non-linear regression models relating total flexion and extension range of motion to age were presented. Total range of motion in bending was observed to decrease in all segments with increasing age. The structural responses obtained from these data are an important set of biomechanical data that will be useful in increasing the understanding of the bending biomechanics of the pediatric human cervical spine.

8.5 Acknowledgments

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9. Discussion

9.1 Contribution and Relevance to the Progress of Pediatric Biomechanics

The tensile and bending biomechanics of the osteoligamentous human cervical spine from birth to young adulthood are characterized in the current study and represents the first and only study to date to investigate these responses over the entire pediatric age spectrum. Age-associated relationships describing the changes in stiffness and load-to-failure are introduced from birth to 18 years of age. This study provides the first investigation of the load-to-failure response of the pediatric human upper and lower cervical spine. The flexion-extension bending response of the upper and lower cervical spine with age is also presented and stands as the only study to-date to evaluate this response in the pediatric human. Tensile viscoelasticity of the pediatric WCS, UCS and LCS is characterized and provides insight into rate dependent effects. While others, including Duncan (1874) and Ouyang et al. (2005) have investigated aspects of the tensile and bending cervical spine response of the human during childhood; neither study encompassed the entire age range from birth to young adulthood. Additionally, both studies limited their investigations to the WCS, while the current work evaluated the response of the WCS, followed by investigations of the response of the UCS and LCS.

Computational models of the pediatric cervical spine will benefit from improvements in the biofidelity and tolerance data of the current study. Tensile and
bending stiffness responses for the motion segment level provide age-appropriate response characteristics suitable for modeling the cervical spine at all ages. UCS and LCS tensile tolerance by age provides injury levels for the osteoligamentous portion of the spine. Inclusion of the effects of muscle in varying states of activation have been reported in adult cervical spine models and contributed to furthering our understanding of cervical injuries in the field compared to those observed experimentally (Van Ee et al. 2000b; Chancey et al. 2003). It is unclear at the youngest ages how important neck musculature, including activation and pre-impact awareness are to overall cervical response, while at older pediatric ages the affect of musculature in conjunction with the response of the osteoligamentous spine may be of more importance to overall cervical kinematics and loading as they are in the adult (Siegmund et al. 2003a, 2003b; Ono et al. 2003). One component of elucidating the effect of musculature with age on the loading response of the cervical spine is an appropriate underlying osteoligamentous response and injury criteria; both of which are provided from the current study.

Physical models of the cervical spine, including the family of CRABI (6, 12 and 18 month) and child Hybrid III ATDs (3, 6, 10 years), may be enhanced based on the data from the current study. Current biofidelity corridors for the pediatric ATDs are based on scaling from adult volunteer and cadaver testing (Mertz and Patrick 1967, 1971). Similarly, IARVs for loads in the cervical spine for the entire family of ATDs, child and adult, are based on the paired piglet and 3 year old child ATD studies (Mertz and Weber
Scaling of the biofidelity corridors and IARVs are based on geometric and material property differences between the ages of ATDs of interest. The material property scaling is currently based on human calcaneal tendon data and the current load-to-failure data may provide improvements in this scaling methodology (Melvin 1995).

The tensile and bending responses of the current study provide human pediatric cervical spine response data appropriate for benchmarking existing juvenile animal cervical spine studies. The paucity of cadaveric tissue for future investigations of the human pediatric cervical spine necessitates increased understanding of the current response of the animal models in comparison to the human responses of the current study. While continued development and improvements in the biofidelity of computational and physical models will arise from the current human data, it is plausible that future juvenile animal studies will be important in the on-going progression of these modeling efforts. The design, applicability and interpretation of future juvenile animal studies will benefit from comparisons to the current work.

The injury patterns observed in the UCS and LCS were consistent with pediatric cervical spine injuries reported in the literature. UCS level injuries were consistent with the small number of reported cases of MVC-related non-head contact and OOP air-bag interaction injuries that are suggested to be associated with distractive or tensile type loading of the neck. Axis and typically odontoid associated fractures were observed in
the younger ages, including: 24 DY, 5, 9, 11, 22 MN and three perinatal specimens and was similar to multiple cases of restrained younger child occupants from 16 months to 3 years with odontoid fracture patterns (Panczykowski et al. 2010; Garton et al. 2002; Blauth et al. 1996; Clasper and Pailthorpe 1995; Keller and Mosdal 1990). An AAD injury with associated chondrum terminale disruption was observed in a 9 year old in the current study, while a restrained 6 year old involved in an MVC suffered the same injury pattern (Hammerstein et al. 2007). AODs were observed in the 12 and 17 year old matching a similar injury to a restrained 10 year old occupant involved in a MVC (Houle et al. 2001). LCS injuries at the younger ages were predominantly physeal or endplate type failures of the inferior or superior interface with the vertebral bodies. Complete ligamentous and intervertebral disc disruptions along with vertebral body fractures were observed in the older specimens including the 6, 7, 12, 16 and 18 year olds which are in accordance with older aged fracture patterns.

The pediatric cervical spine biomechanical responses and trends with age determined in the current work will have significant impact in a multitude of areas of study. While a strong emphasis has been placed on the biomechanics of the pediatric cervical spine in association with child occupant protection, it is important to emphasize that the utility of this investigation is not limited to only this arena of study. MVC-related events dominate the cause of injury and fatality during the pediatric years and are heavily associated with cervical spine injury, but falls and sports-related injuries are
also prominent contributors in this discussion. Many other research areas that rely on an improved understanding of the biomechanics of the developing cervical spine may also benefit from this investigation. The biomechanics of birth, specifically with regard to spinal and cord injuries, is one area of utilization. Similarly, the phenomena of spinal cord injury without radiographic abnormality and pseudosubluxation, both somewhat unique to the pediatric spine may also be further understood and modeled. Likewise, the extensive debate on non-accidental trauma (NAT) in infants, historically referred to as ‘shaken baby syndrome’, may benefit from further understanding of the biomechanics of the immature neck. In all of these cases, as well as with regard to furthering the discussion on head injury and its relationship to spine kinematics, the investigation of the pediatric cervical spine provides foundational data to progress the current discussion, while also enhancing computational and physical models important to ongoing investigations.

9.2 Conclusions

The current investigation of the biomechanical response of the pediatric cervical spine characterized the tensile and bending behavior from birth to young adulthood and provided age based relationships applicable to defining the biomechanical response of the cervical spine at all ages of development. In association with the currently reported data, additionally acquired data of the spine under compressive loading and the tensile viscoelastic and rate dependent response at other ages are still to be evaluated. While
additional insight and understanding will be obtained from continued interpretation of this study, a number of conclusions and observations are applicable:

1. Quasi-static low-load tensile stiffness of the WCS, UCS and LCS increases with age in a non-linear form. Increases in stiffness at the earliest ages are more rapid than at older pediatric ages. WCS and UCS stiffness in the younger cohort was significantly less than LCS stiffness, while WCS and UCS stiffness and C4-C5 and C6-C7 stiffness were not different from one another within the youngest cohort.

2. Load-to-failure tensile stiffness of the UCS and LCS increases with age in a non-linear form. Increases in stiffness at the earliest ages are more rapid than at older pediatric ages. UCS load-to-failure stiffness in the younger cohort was significantly less than LCS load-to-failure stiffness.

3. Load-to-failure of the UCS and LCS increases with age in a non-linear form. Increases in load-to-failure at the earliest ages are more rapid than at older pediatric ages. UCS and LCS load-to-failure was not significantly different in the younger cohort.

4. Displacement at failure of the UCS and LCS were weakly correlated to age, while normalized displacement at failure decreased with age in a non-linear form for both the UCS and LCS. Reductions in normalized displacement at failure with age in the UCS were more dramatic than those observed in the LCS and may provide additional insight into structural and spinal cord related injury with age.
5. Clinically observed pediatric cervical spine injuries were observed under tensile loading to failure. UCS injuries were commonly associated with axis level synchondrotic disruptions or AOD/AAD, while LCS injuries in the younger ages included endplate failures that progressed to vertebral fractures and ligamentous dislocations at the older ages.

6. Viscoelastic rate effects were observed in the WCS, UCS and LCS of the pediatric cervical spine. Peak load and loading energy were observed to increase with loading rate for all levels of the cervical spine. While, hysteresis energy was constant at the lower loading rates, but increased at the highest rates of loading at all levels of the spine.

7. Viscoelastic rate effects for the pediatric WCS, UCS and LCS were successfully modeled and validated over three orders of loading frequency using QLV. No single formulation of QLV provided an overall response superior to all other formulations for all levels of the spine. However, the Myers model provided the most robust modeling response for the WCS and UCS segments, while the FSHSE and Best models provided the most predictive ability for the C4-C5 and C6-C7 segments, respectively.

8. Quasi-static low-load bending range of motion of the UCS and LCS decreased with age in a non-linear form. Range of motion decreased more rapidly at the earliest ages compared to the older pediatric ages.
9. Quasi-static low-load flexion and extension bending stiffness of the UCS and LCS increased with age in a non-linear form. Extension bending stiffness was greater than flexion bending stiffness in the older age cohort. Extension and flexion stiffness were statistically different in the UCS and C6-C7 segments at the younger ages, while no statistical difference was observed in the C4-C5 segment.
9.3 Recommendations for Future Work

The investigation of the biomechanics of the cervical spine from around birth to adulthood included a number of areas of study, including those presented in the current work, however, a number of additional studies resulting in additional quantitative data were obtained during the duration of the overall investigation. A number of further investigations based on these data are currently being analyzed or will in the near future. These include evaluation of the low-load compressive stiffness response of the WCS, UCS and LCS with age. The quasi-static low-load tensile stiffness of the WCS, UCS and LCS will be refined to account for frame compliance in the testing fixtures, similar to the procedures outlined in Chapter 4 and 6 for the load-to-failure responses. Centers of rotation for the UCS joint in flexion and extension bending will be evaluated similar to the work of Chancey et al. (2007) and evaluated for differences in age.

Cadaveric cervical spine work focused on quantifying spine stiffness has historically focused on the whole spine or motion segments. In the current work as well as previous adult work, whole spine and then sub-section motion segments were tested at the motion segment level to determine stiffness and load-to-failure response. In our experience, in both the adult and pediatric work, reconstruction of the whole by way of the segments has consistently underestimated the whole spine stiffness determined experimentally. Interestingly, aside from the 14 year old pediatric specimen, all of the pediatric tissue of the current study was tested in a frame different than used for the
adult work of our institution (Dibb et al. 2009). Dissection and fixation procedures used in both the pediatric and adult test series were similar. Differences between the experimental and reconstructed stiffness may be attributed to superficial ligamentous disruptions, viscoelastic rate effects or motion segment alignment in the whole spine versus potential alignment differences in individual motion segment tests. Determining the origin of this difference is an important next step in further evaluation of the motion segment level load-to-failure response and progression of these observations to overall whole spine failure response.

The rate- and time-dependent response of the pediatric cervical spine was presented for the six year old, but in all pediatric specimens, stress relaxation tests were acquired. Based on the current modeling experience, the remaining data will be analyzed to assess these properties by age and similar to the stiffness, load-to-failure and bending responses be reviewed for age dependence. Similar to the six year old these models will be validated with constant velocity tests. Furthermore, while QLV was utilized successfully to model the viscoelastic response of the six year old over three orders of loading frequency the applicability of QLV compared to a fully non-linear viscoelastic model was not addressed, although stress relaxation tests at 10, 25, 50 and 75% of the maximum loading amplitude were acquired. These data will be evaluated to further evaluate the utility of QLV at all ages.
Scaling ratios and relationships associated with current juvenile animal models and child ATD biofidelity and IARVs should be assessed in reference to the current pediatric human cervical response data. The utility of current scaling ratios relating tensile stiffness and load-to-failure and bending stiffness from animal models should be evaluated. Relating the human to animal response is important in interpreting future juvenile animal studies that may investigate developmental biomechanics that are unable to be investigated with human cadaveric tissue. Furthermore, current animal models are typically represented in human-equivalent years based on skeletal maturity of the animals and these approximations may be further refined based on comparisons to the current pediatric human responses. Current scaling relationships utilized to develop child ATD biofidelity corridors from the mid-size adult response and similar relationships used to scale neck IARVs from the three year-old ATD response may be evaluated specifically with regard to the material property scale factor currently based on human calcaneal tendon data. Moreover, the current tensile tolerance data by age provides a conservative lower-bound injury level based solely on the osteoligamentous cervical spine. Additional assessment of the current family of child ATD neck responses under tensile and bending loading following the methodology of the current work will also be of benefit in evaluating current differences in biofidelity.

Structural and material property characterization of the cartilaginous synchondroses of the immature spine are of importance as these regions both clinically
and within our own experimental experience were a typical site of injury. The current work included a pilot study focusing on evaluating the biomechanical response of these structures in a single perinatal specimen. The structural response of the bone-cartilage-bone interface was evaluated for load-to-failure and load-to-failure stiffness. These properties will be of use for approximating injury in computational models that include the bone-cartilage-bone morphology. Additionally, characterizing the material response of the cartilaginous region is useful in augmenting finite element models focused on evaluating the biomechanical response of these often injured regions. Insight gained from the pilot study will direct a more comprehensive investigation of the synchondroses, structurally and materially, that will include tissue from the perinatal to sub-infant age range. Similar to the current work on the response of the spine as a whole, this work will characterize the response of these isolated bone-cartilaginous interfaces during growth.
Appendix A

Non-Destructive Force Displacement Plots

Force displacement data is organized as follows: Individual Pediatric PMHS – Row (increase in age as you progress downward through data plots). Individual Cervical Segment – Column (Whole Spine, O-C2, C4-C5, C6-C7 as you progress left to right). Empty Cells – Specimen not available for testing.

The whole spine data is plotted on a 6 mm by 60/200 N grid for comparison purposes. The cervical segments data are plotted on a 4 mm by 60/200 N grid for comparison purposes. T02P and T16P are arranged at the end of the data set and not in the order of age. The cervical segments tested for T02P and T16P were not consistent with the remainder of the testing sample (T02P – O-C6; T16P – C3-C4 and C5-C6). Stiffness – calculated from 50-100% of curve, LLD – low load displacement.
PMHS ID: T13PWS
Segment: WS
Age: 0 MN (perinatal)
Stiffness: 7.71 N/mm
LLD: 1.28 mm

PMHS ID: T13PO2
Segment: O-C2
Age: 0 MN (perinatal)
Stiffness: 12.2 N/mm
LLD: 0.269 mm

PMHS ID: T13PS5
Segment: C4-C5
Age: 0 MN (perinatal)
Stiffness: 61.4 N/mm
LLD: 0.066 mm

PMHS ID: T13P67
Segment: C6-C7
Age: 0 MN (perinatal)
Stiffness: 39.4 N/mm
LLD: 0.103 mm

PMHS ID: T09PO7
Segment: O-C7
Age: 0 MN (perinatal)
Stiffness: 7.93 N/mm
LLD: 0.694 mm

PMHS ID: T09PO2
Segment: O-C2
Age: 0 MN (perinatal)
Stiffness: 11.90 N/mm
LLD: 0.608 mm

PMHS ID: T09P45
Segment: C4-C5
Age: 0 MN (perinatal)
Stiffness: 50.6 N/mm
LLD: 0.033 mm

PMHS ID: T09P67
Segment: C6-C7
Age: 0 MN (perinatal)
Stiffness: 36.7 N/mm
LLD: 0.252 mm

PMHS ID: T07PO7
Segment: O-C7
Age: 0 MN (perinatal)
Stiffness: 7.33 N/mm
LLD: 0.684 mm

PMHS ID: T07PO2
Segment: O-C2
Age: 0 MN (perinatal)
Stiffness: 11.90 N/mm
LLD: 0.608 mm

PMHS ID: T07P45
Segment: C4-C5
Age: 0 MN (perinatal)
Stiffness: 46.1 N/mm
LLD: 0.117 mm

PMHS ID: T07P67
Segment: C6-C7
Age: 0 MN (perinatal)
Stiffness: 44.4 N/mm
LLD: 0.075 mm

PMHS ID: T10P45
Segment: C4-C5
Age: 0 MN (perinatal)
Stiffness: 35.8 N/mm
LLD: 0.576 mm

PMHS ID: T10P67
Segment: C6-C7
Age: 0 MN (perinatal)
Stiffness: 37.1 N/mm
LLD: 0.168 mm
PMHS ID: T06PWS  
Segment: WS  
Age: 0.37 MN  
Stiffness: 6.83 N/mm  
LLD: 0.328 mm

PMHS ID: T06PO2  
Segment: O-C2  
Age: 0.37 MN  
Stiffness: 7.10 N/mm  
LLD: 1.45 mm

PMHS ID: T06P45  
Segment: C4-C5  
Age: 0.37 MN  
Stiffness: 50.5 N/mm  
LLD: 0.061 mm

PMHS ID: T06P67  
Segment: C6-C7  
Age: 0.37 MN  
Stiffness: 50.8 N/mm  
LLD: 0.058 mm
PMHS ID: T11P45
Segment: C4-C5
Age: 0.53 MN
Stiffness: 35.5 N/mm
LLD: 0.014 mm

PMHS ID: T11P67
Segment: C6-C7
Age: 0.53 MN
Stiffness: 61.5 N/mm
LLD: 0.020 mm

PMHS ID: T04PWS
Segment: WS
Age: 0.8 MN
Stiffness: 7.30 N/mm
LLD: 0.342 mm

PMHS ID: T04PO2
Segment: O-C2
Age: 0.8 MN
Stiffness: 9.29 N/mm
LLD: 0.179 mm

PMHS ID: T04P45
Segment: C4-C5
Age: 0.8 MN
Stiffness: 42.8 N/mm
LLD: 0.045 mm

PMHS ID: T04P67
Segment: C6-C7
Age: 0.8 MN
Stiffness: 34.2 N/mm
LLD: 0.043 mm

PMHS ID: T12PWS
Segment: WS
Age: 5 MN
Stiffness: 9.71 N/mm
LLD: 0.475 mm

PMHS ID: T12PO2
Segment: O-C2
Age: 5 MN
Stiffness: 14.5 N/mm
LLD: 0.726 mm

PMHS ID: T12P45
Segment: C4-C5
Age: 5 MN
Stiffness: 58.2 N/mm
LLD: 0.016 mm

PMHS ID: T12P67
Segment: C6-C7
Age: 5 MN
Stiffness: 43.5 N/mm
LLD: 0.021 mm

PMHS ID: T14PWS
Segment: WS
Age: 9 MN
Stiffness: 28.5 N/mm
LLD: 1.16 mm

PMHS ID: T14PO2
Segment: O-C2
Age: 9 MN
Stiffness: 41.5 N/mm
LLD: 1.66 mm

PMHS ID: T14P45
Segment: C4-C5
Age: 9 MN
Stiffness: 58.2 N/mm
LLD: 0.300 mm

PMHS ID: T14P67
Segment: C6-C7
Age: 9 MN
Stiffness: 103.0 N/mm
LLD: 0.262 mm
PMHS ID: T02P06
Segment: C3-C4
Age: 18 MN
Stiffness: 130.9 N/mm
LLD: 0.210 mm

PMHS ID: T16P06
Segment: C5-C6
Age: 18 MN
Stiffness: 171.5 N/mm
LLD: 0.034 mm

PMHS ID: T02P06
Segment: O-C6
Age: 0 MN (perinatal)
Stiffness: 5.41 N/mm
LLD: 0.017 mm
Appendix B

**Destructive Force Displacement Plots**

Force displacement data is organized as follows: Individual Pediatric PMHS – Row (increase in age as you progress downward through data plots). Individual Cervical Segment – Column (O-C2, C4-C5, C6-C7 as you progress left to right). Empty Cells – Specimen not available for testing.

All data is plotted on a 20 mm by 3000 N grid for comparison purposes. T02P and T16P are arranged at the end of the data set and not in the order of age. The cervical segments tested for T02P and T16P were not consistent with the remainder of the testing sample (T02P – O-C6; T16P – C3-C4 and C5-C6).
PMHS ID: T07PO2
Segment: O-C2
Age: 0 months (perinatal)
Initial Failure: -
Ultimate Failure: 168.1 N

PMHS ID: T09P45
Segment: C4-C5
Age: 0 months (perinatal)
Initial Failure: 178.2 N
Ultimate Failure: 180.4 N

PMHS ID: T13PO2
Segment: O-C2
Age: 0 months (perinatal)
Initial Failure: -
Ultimate Failure: 274.8 N

PMHS ID: T13P45
Segment: C4-C5
Age: 0 months (perinatal)
Initial Failure: 334.3 N
Ultimate Failure: 360.5 N

PMHS ID: T07PO2
Segment: O-C2
Age: 0 months (perinatal)
Initial Failure: -
Ultimate Failure: 196.9 N

PMHS ID: T09P67
Segment: C6-C7
Age: 0 months (perinatal)
Initial Failure: 124.8 N
Ultimate Failure: 154.1 N

PMHS ID: T13P45
Segment: C4-C5
Age: 0 months (perinatal)
Initial Failure: 334.3 N
Ultimate Failure: 360.5 N

PMHS ID: T13P67
Segment: C6-C7
Age: 0 months (perinatal)
Initial Failure: 194.9 N
Ultimate Failure: 204.1 N
PMHS ID: T05P45  
Segment: C6-C7  
Age: 0.03 months  
Initial Failure: 172.6 N  
Ultimate Failure: 181.3 N

PMHS ID: T05P45  
Segment: C4-C5  
Age: 0.03 months  
Initial Failure: 188.4 N  
Ultimate Failure: 207.5 N

PMHS ID: T05PO2  
Segment: O-C2  
Age: 0.03 months (perinatal)  
Initial Failure: 188.3 N  
Ultimate Failure: 208.9 N

PMHS ID: T05PO2  
Segment: O-C2  
Age: 0.03 months (perinatal)  
Initial Failure: -  
Ultimate Failure: 241.9 N

PMHS ID: T08P45  
Segment: C4-C5  
Age: 0.03 months (perinatal)  
Initial Failure: 188.4 N  
Ultimate Failure: 207.5 N

PMHS ID: T08P45  
Segment: C6-C7  
Age: 0.03 months (perinatal)  
Initial Failure: 177.8 N  
Ultimate Failure: 209.7 N

PMHS ID: T10P67  
Segment: C6-C7  
Age: 0.03 months (perinatal)  
Initial Failure: 177.8 N  
Ultimate Failure: 209.7 N

PMHS ID: T10P67  
Segment: C4-C5  
Age: 0.03 months (perinatal)  
Initial Failure: 188.4 N  
Ultimate Failure: 207.5 N

PMHS ID: T10P67  
Segment: C4-C5  
Age: 0.03 months (perinatal)  
Initial Failure: 188.4 N  
Ultimate Failure: 207.5 N
PMHS ID: T11P67
Segment: C6-C7
Age: 0.53 months
Initial Failure: 182.9 N
Ultimate Failure: 191.4 N

PMHS ID: T11P45
Segment: C4-C5
Age: 0.53 months
Initial Failure: -
Ultimate Failure: 167.3 N

PMHS ID: T06P67
Segment: C6-C7
Age: 0.37 months
Initial Failure: 140.2 N
Ultimate Failure: 151.9 N

PMHS ID: T06P45
Segment: C4-C5
Age: 0.37 months
Initial Failure: 173.8 N
Ultimate Failure: 176.0 N

PMHS ID: T03P67
Segment: C6-C7
Age: 0.1 months
Initial Failure: 139.3 N
Ultimate Failure: 142 N

PMHS ID: T03P45
Segment: C4-C5
Age: 0.1 months
Initial Failure: 165.8 N
Ultimate Failure: 182.7 N

PMHS ID: T03PO2
Segment: O-C2
Age: 0.37 months
Initial Failure: -
Ultimate Failure: 257.6 N

PMHS ID: T03P45
Segment: C4-C5
Age: 0.37 months
Initial Failure: 173.8 N
Ultimate Failure: 176.0 N

PMHS ID: T06P67
Segment: C6-C7
Age: 0.37 months
Initial Failure: 140.2 N
Ultimate Failure: 151.9 N

PMHS ID: T06P45
Segment: C4-C5
Age: 0.37 months
Initial Failure: 173.8 N
Ultimate Failure: 176.0 N

PMHS ID: T03P67
Segment: C6-C7
Age: 0.1 months
Initial Failure: 139.3 N
Ultimate Failure: 142 N

PMHS ID: T03P45
Segment: C4-C5
Age: 0.1 months
Initial Failure: 165.8 N
Ultimate Failure: 182.7 N

PMHS ID: T03PO2
Segment: O-C2
Age: 0.37 months
Initial Failure: -
Ultimate Failure: 257.6 N
PMHS ID: T14PO2
Segment: O-C2
Age: 9 months
Initial Failure: 260 N
Ultimate Failure: 262.3 N

PMHS ID: T14P45
Segment: C4-C5
Age: 9 months
Initial Failure: 394.2 N
Ultimate Failure: 400 N

PMHS ID: T14P67
Segment: C6-C7
Age: 9 months
Initial Failure: 673.2 N
Ultimate Failure: 840.1 N

PMHS ID: T12PO2
Segment: O-C2
Age: 5 months
Initial Failure: 382.3 N
Ultimate Failure: 461.7 N

PMHS ID: T12P45
Segment: C4-C5
Age: 5 months
Initial Failure: 382.3 N
Ultimate Failure: 461.7 N

PMHS ID: T12P67
Segment: C6-C7
Age: 5 months
Initial Failure: 241.6 N
Ultimate Failure: 292.9 N

PMHS ID: T04PO2
Segment: O-C2
Age: 0.8 months
Initial Failure: 250.4 N
Ultimate Failure: 262.2 N

PMHS ID: T04P45
Segment: C4-C5
Age: 0.8 months
Initial Failure: 237.2 N
Ultimate Failure: 240.8 N

PMHS ID: T04P67
Segment: C6-C7
Age: 0.8 months
Initial Failure: 250.4 N
Ultimate Failure: 262.2 N

PMHS ID: T04PO2
Segment: O-C2
Age: 0.8 months
Initial Failure: 260 N
Ultimate Failure: 262.3 N

PMHS ID: T04P45
Segment: C4-C5
Age: 0.8 months
Initial Failure: 237.2 N
Ultimate Failure: 240.8 N

PMHS ID: T04P67
Segment: C6-C7
Age: 0.8 months
Initial Failure: 250.4 N
Ultimate Failure: 262.2 N
PMHS ID: T15P02  
Segment: O-C2  
Age: 11 months  
Initial Failure: 152.5 N  
Ultimate Failure: 1019 N

PMHS ID: T15P45  
Segment: C4-C5  
Age: 11 months  
Initial Failure: 406.1 N  
Ultimate Failure: 631.2 N

PMHS ID: T15P67  
Segment: C6-C7  
Age: 11 months  
Initial Failure: 489.1 N  
Ultimate Failure: 492 N

PMHS ID: T17P02  
Segment: O-C2  
Age: 22 months  
Initial Failure: 1097 N  
Ultimate Failure: 1231 N

PMHS ID: T17P45  
Segment: C4-C5  
Age: 22 months  
Initial Failure: 466.8 N  
Ultimate Failure: 844.8 N

PMHS ID: T17P67  
Segment: C6-C7  
Age: 22 months  
Initial Failure: 646.3 N  
Ultimate Failure: 806.5 N

PMHS ID: T18P02  
Segment: O-C2  
Age: 9 years  
Initial Failure: 1460 N  
Ultimate Failure: 1925 N

PMHS ID: T18P45  
Segment: C4-C5  
Age: 9 years  
Initial Failure: 1400 N  
Ultimate Failure: 1757 N

PMHS ID: T18P67  
Segment: C6-C7  
Age: 9 years  
Initial Failure: 1400 N  
Ultimate Failure: 1757 N
PMHS ID: T16P34
Segment: C3-C4
Age: 18 months
Initial Failure: 855.8 N
Ultimate Failure: 915.7 N

PMHS ID: T16P56
Segment: C5-C6
Age: 18 months
Initial Failure: 515 N
Ultimate Failure: 732.8 N
Appendix C

Supplemental Digital Content 1, Injury Descriptions

Injury descriptions from all twenty-four PMHS segmental load-to-failure tests are presented in this supplemental content.
<table>
<thead>
<tr>
<th>PHMS ID</th>
<th>Age</th>
<th>Sex</th>
<th>Segment</th>
<th>Injury Descriptions</th>
</tr>
</thead>
<tbody>
<tr>
<td>01P</td>
<td>14 YR [168 MN]</td>
<td>F</td>
<td>O-C2</td>
<td>Occipito-atlantal dislocation with C1 fracture in right anterior arch; right occipital condyle fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td></td>
</tr>
<tr>
<td>02P</td>
<td>20 WKG [0 MN]</td>
<td>F</td>
<td>WCS (OC-C6)</td>
<td>Physeal failure (superior physis endplate failure of C6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>O-C2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td></td>
</tr>
<tr>
<td>03P</td>
<td>3 DY [0.1 MN]</td>
<td>M</td>
<td>O-C2</td>
<td>C2-C3 dislocation with associated fractures (bilateral fractures in C2 lamina proximal to posterior synchondroses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (superior physis endplate failure of C4) with bilateral C4 neurocentral synchondroses fractures; bilateral C5 lamina fractures</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure at C6) with unilateral right C6 neurocentral synchondrosis fracture</td>
</tr>
<tr>
<td>04P</td>
<td>24 DY [0.8 MN]</td>
<td>F</td>
<td>O-C2</td>
<td>C2-C3 dislocation (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [inferior])</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (superior physis endplate failure of C4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure of C6)</td>
</tr>
<tr>
<td>05P</td>
<td>1 DY [0.03 MN]</td>
<td>F</td>
<td>O-C2</td>
<td>Physeal failure (inferior physis endplate of C2) - C3 fractures (through right neurocentral and posterior synchondroses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (inferior physis endplate of C4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (inferior physis endplate of C7)</td>
</tr>
<tr>
<td>06P</td>
<td>11 DY [0.37 MN]</td>
<td>F</td>
<td>O-C2</td>
<td>Physeal failure (inferior physis endplate of C2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (inferior physis endplate of C4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (inferior physis endplate of C6)</td>
</tr>
<tr>
<td>07P</td>
<td>33 WKG [0 MN]</td>
<td>M</td>
<td>O-C2</td>
<td>Atlanto-axial dislocation with Type III dens fracture (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior])</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (superior physis endplate failure of C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (inferior physis endplate failure of C6)</td>
</tr>
<tr>
<td>08P</td>
<td>37.5 WKG [0 MN]</td>
<td>M</td>
<td>O-C2</td>
<td>C2-C3 dislocation with associated C2 fractures (fx through subdental synchondrosis and bilaterally through neurocentral synchondroses [inferior]; fx through right lateral mass, inferior-superior oblique &amp; line exiting at superior articulating process; bilateral &amp; through C2 lamina proximal to posterior synchondrosis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td></td>
</tr>
<tr>
<td>09P</td>
<td>33 WKG [0 MN]</td>
<td>M</td>
<td>O-C2</td>
<td>Physeal failure (inferior physis endplate failure of C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure of C7)</td>
</tr>
<tr>
<td>10P</td>
<td>35 WKG [0 MN]</td>
<td>M</td>
<td>O-C2</td>
<td>Physeal failure (inferior physis endplate failure of C4); C4 right neurocentral synchondrosis to posterior synchondrosis fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure at C7)</td>
</tr>
<tr>
<td>11P</td>
<td>16 DY [0.53 MN]</td>
<td>F</td>
<td>O-C2</td>
<td>Physeal failure (superior physis endplate failure of C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure of C7)</td>
</tr>
<tr>
<td>12P</td>
<td>5 MN</td>
<td>M</td>
<td>O-C2</td>
<td>Atlanto-axial dislocation with Type III dens fracture through fixation; bilateral occipito-atlantal facet capsule disruption on lateral half of capsules</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C4-C5</td>
<td>Physeal failure (inferior physis endplate failure at C5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C6-C7</td>
<td>Physeal failure (superior physis endplate failure of C7)</td>
</tr>
</tbody>
</table>
**Supplemental Digital Content 1 (cont.). Perinatal, Neonatal, Pediatric and Young Adult Injury Descriptions Associated with Failure Tests**

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>Age</th>
<th>Sex</th>
<th>Injuries Described</th>
</tr>
</thead>
<tbody>
<tr>
<td>13P</td>
<td>29 WKG (5 WPB)</td>
<td>F</td>
<td>O-C2: Atlanto-axial dislocation with Type III dens fracture through fixation (fix through subdental synchondrosis and bilaterally through neurocentral synchondroses [superior]); C4-C5: Physseal failure (superior physis endplate failure at C5); C6-C7: Physseal failure (superior physis endplate failure at C7)</td>
</tr>
<tr>
<td>14P</td>
<td>9 MN</td>
<td>M</td>
<td>O-C2: Atlanto-axial dislocation with C2-C3 dislocation (Type II dens fracture); С4-C5: Physseal failure (superior physis endplate failure at C4); C6-C7: Physseal failure (superior physis endplate failure at C7)</td>
</tr>
<tr>
<td>15P</td>
<td>11 MN</td>
<td>F</td>
<td>O-C2: Atlanto-axial dislocation with ossiculum terminale fracture of dens; fracture in left C2 superior facet surface; C4-C5: Physseal failure (superior physis endplate failure at C4); C6-C7: Physseal failure (superior physis endplate failure at C6)</td>
</tr>
<tr>
<td>16P</td>
<td>18 MN</td>
<td>M</td>
<td>C3-C4: Physseal failure (inferior physis endplate failure at C4); C5-C6: Physseal failure (superior physis endplate failure at C5)</td>
</tr>
<tr>
<td>17P</td>
<td>22 MN</td>
<td>F</td>
<td>O-C2: Atlanto-axial dislocation (dens fracture Type II/III); C4-C5: Physseal failure (superior physis endplate failure at C4); C6-C7: Physseal failure (superior physis endplate failure at C6)</td>
</tr>
<tr>
<td>18P</td>
<td>9 YR [108 MN]</td>
<td>M</td>
<td>O-C2: Atlanto-axial dislocation with C2-C3 dislocation (Type II dens fracture); C4-C5: Physseal failure (superior physis endplate failure at C5); C6-C7: Physseal failure (superior physis endplate failure at C6)</td>
</tr>
<tr>
<td>19P</td>
<td>7 YR [84 MN]</td>
<td>F</td>
<td>O-C2: Type III dens fracture secondary to C2 inferior vertebral body fracture through the fixation</td>
</tr>
<tr>
<td>20P</td>
<td>12 YR [144 MN]</td>
<td>M</td>
<td>O-C2: Occipito-atlantal and parietal atlantal axial dislocation with occipito-atlantal fracture of left condyle and avulsion of right alar (Complete AOD; posterior ligamentous disruption AAD); C4-C5: Complete ligamentous and intervertebral disk disruption; C6-C7: Complete ligamentous and intervertebral disk disruption</td>
</tr>
<tr>
<td>21P</td>
<td>16 YR [192 MN]</td>
<td>F</td>
<td>O-C2: C2 endplate/vertebral body and bilateral spinous process fractures through fixation (occipito-atlantal-axial junction intact with physiologic mobility retained and lack of crepitus); C4-C5: Complete ligamentous and intervertebral disk disruption (right anterior quadrant C4 vertebral body fracture); Physseal failure (superior physis endplate failure of C4); C6-C7: Complete ligamentous and intervertebral disk disruption (anterior C6 vertebral body fracture), Physseal failure (superior physis endplate failure of C6)</td>
</tr>
<tr>
<td>22P</td>
<td>17 YR [204 MN]</td>
<td>M</td>
<td>O-C2: Occipito-atlantal dislocation; C4-C5: Physseal failure (inferior physis endplate failure at C4); C6-C7: Physseal failure (inferior physis endplate failure at C6)</td>
</tr>
<tr>
<td>23P</td>
<td>18 YR [216 MN]</td>
<td>F</td>
<td>O-C2: Atlanto-axial dislocation with C1 anterior tubercle fracture along midline propagating in an inferior to superior direction (fracture was not complete); C4-C5: Complete ligamentous and intervertebral disk disruption; C6-C7: Complete ligamentous and intervertebral disk disruption</td>
</tr>
<tr>
<td>24P</td>
<td>6 YR [72 MN]</td>
<td>F</td>
<td>O-C2: Basilar skull fracture (occipito-atlantal-axial junction intact with physiologic mobility retained and lack of crepitus); O-C2: Atlanto-axial dislocation with Type II dens fracture through fixation; C4-C5: Physseal failure (superior physis endplate failure at C5); C6 vertebral body fracture</td>
</tr>
</tbody>
</table>

C3-C4 - C3 to C4 segment; C4-C5 - C4 to C5 segment; C5-C6 - C5 to C6 segment; C6-C7 - C6 to C7 segment

YR - years; MN - months; DY - days; WPB - weeks post birth; WKG - weeks gestation

1 - Initial failure test per protocol; 2 - Second failure test administered from fixated occiput through Occ-C1-C2 junction

- Injuries identified subsequent to second failure test with possibility of occurrence during either failure test

Spinal segment not tested; ALL - anterior longitudinal ligament; IVD - intervertebral disc; O-C2 - Occiput to C2 segment

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>Age</th>
<th>Sex</th>
<th>Injuries Described</th>
</tr>
</thead>
<tbody>
<tr>
<td>23P</td>
<td>18 YR [216 MN]</td>
<td>F</td>
<td>O-C2: Complete ligamentous and intervertebral disk disruption; C4-C5: Complete ligamentous and intervertebral disk disruption</td>
</tr>
<tr>
<td>24P</td>
<td>6 YR [72 MN]</td>
<td>F</td>
<td>O-C2: Basilar skull fracture (occipito-atlantal-axial junction intact with physiologic mobility retained and lack of crepitus); O-C2: Atlanto-axial dislocation with Type II dens fracture through fixation; C4-C5: Physseal failure (superior physis endplate failure at C5); C6 vertebral body fracture</td>
</tr>
</tbody>
</table>

C3-C4 - C3 to C4 segment; C4-C5 - C4 to C5 segment; C5-C6 - C5 to C6 segment; C6-C7 - C6 to C7 segment

YR - years; MN - months; DY - days; WPB - weeks post birth; WKG - weeks gestation

1 - Initial failure test per protocol; 2 - Second failure test administered from fixated occiput through Occ-C1-C2 junction

- Injuries identified subsequent to second failure test with possibility of occurrence during either failure test

Spinal segment not tested; ALL - anterior longitudinal ligament; IVD - intervertebral disc; O-C2 - Occiput to C2 segment
Appendix D

Comparison of QLV Model Response to Experimental Results

WCS and Myers Model

Pediatric whole cervical spine experimental, quasi-linear viscoelastic (Myers approach) and nonlinear elastic model responses: force-time and force-displacement responses. (a) 0.01 Hz, (b) 0.05 Hz, (c) 0.1 Hz, (d) 0.2 Hz, (e) 0.5 Hz, (f) 1 Hz, (g) 5 Hz, (h) 10 Hz.
**O-C2 and Myers Model**

Pediatric O-C2 experimental, quasi-linear viscoelastic (Myers approach) and nonlinear elastic model responses: force-time and force-displacement responses. (a) 0.01 Hz, (b) 0.05 Hz, (c) 0.1 Hz, (d) 0.2 Hz, (e) 0.5 Hz, (f) 1 Hz, (g) 5 Hz, (h) 10 Hz.
**C4-C5 and FSHSE Model**

Pediatric C4-C5 experimental, quasi-linear viscoelastic (FSHSE approach) and nonlinear elastic model responses: force-time and force-displacement responses. (a) 0.01 Hz, (b) 0.05 Hz, (c) 0.1 Hz, (d) 0.2 Hz, (e) 0.5 Hz, (f) 1 Hz, (g) 5 Hz, (h) 10 Hz.
C6-C7 and Best Model

Pediatric C6-C7 experimental, quasi-linear viscoelastic (Best approach) and nonlinear elastic model responses: force-time and force-displacement responses. (a) 0.01 Hz, (b) 0.05 Hz, (c) 0.1 Hz, (d) 0.2 Hz, (e) 0.5 Hz, (f) 1 Hz, (g) 5 Hz, (h) 10 Hz.
References


Mann, D.C., and J.A. Dodds. 1993. Spinal injuries in 57 patients 17 years or younger. *Orthopedics* 16, no. 2:159-64.


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Biography

Name: Jason Frederick Luck
Born: San Diego, CA, November 12, 1975
Spouse: Vera M.R. Luck
Children: Genevee D. Luck; Emelia Jean G. Luck
Parents: Charles W. and Kathy W. Luck

Educational Background

Duke University, Durham, North Carolina. Advisor: Dr. Barry S. Myers


Publications

Book Chapters


Peer Reviewed Journal Articles


**Conference Proceedings and Abstracts**


specimens (PMHS) osteoligamentous cervical spines. Abstract presented at the 6th World Congress of Biomechanics (WCB), August 1-6, Singapore.


Awards

2008 Stapp Student Award, Third Place, paper presented at the 52nd Stapp Car Crash Conference