Compressive Mechanical Properties of the Perinatal, Neonatal and Pediatric Cervical Spine

Jason F. Luck, Roger W. Nightingale, Cameron R. "Dale" Bass

Abstract Quantifying the biomechanical response of the pediatric cervical spine is a fundamental step in improving our understanding of the genesis of pediatric neck injuries and determining the influence of neck response on head trauma. Pediatric cervical spine biomechanics research has historically been limited due to the lack of pediatric cadaveric human donors. This investigation characterizes mechanical properties of the developing human cervical spine, providing structural response characterization from birth to maturity under compressive loading. A cohort of whole osteoligamentous cervical spines (WCS) and sub-components (UCS: O-C2; LCS: C4-C5, C6-C7) from 20 weeks gestation to 18 years (n = 23) were tested in compression. Segments were compressed under load control at a rate designed to maintain a constant stress rate throughout the age (size) range of the cohort. The segments were compressed to 5% of the expected failure load. Compressive structural stiffness was calculated by regressing the force-displacement response from 50-100% of peak load. The WCS and UCS were more compliant than LCS for all ages and the stiffness for all segments increased with age. These data may further our understanding of compression related pediatric neck injury and the potential association of these injuries with head contact and a muscularly active neck in a dynamic loading environment.

Keywords cervical spine, compression, neonatal, pediatric, stiffness

I. INTRODUCTION

Quantifying the biomechanical response of the pediatric cervical spine is a critical step in improving our understanding of pediatric neck injuries and elucidating the influence of neck response on head injuries. Pediatric spinal trauma accounts for up to 12% of all spinal injuries and these injuries are often associated with high mortality rates [1-9]. While the pediatric neck may be vulnerable to catastrophic injuries, of equal importance is how the response of the neck governs head kinematics and the potential for head and brain trauma.

Pediatric cervical spine biomechanics research has historically been limited due to the lack of pediatric cadaveric human donors [10-14]. In lieu of human data, juvenile animal studies have served an important role in approximating the biomechanical response of the developing human spine [15-24]. Recent biomechanical data on the tensile and bending behaviour of the human osteoligamentous cervical spine from birth to young adulthood [25-26] have provided structural response and tolerance data to assist in developing mechanical surrogates (ATDs) and computational models, while also offering the ability to assess previous animal models. One benefit of improving the biofidelity of pediatric osteoligamentous response in computational modelling involves understanding the role of active neck musculature in head-neck response. Not surprisingly, recent modelling work utilizing a validated head-neck model has suggested that a stabilized head-neck complex incorporating the osteoligamentous and active musculature places the osteoligamentous spine into compression [27]. The implications of this compressive pre-load may be of importance in better understanding neck tolerance under compressive loading, for example, in head-impacts during motor vehicle crashes. A number of studies have investigated the compressive loading response of the adult cervical spine [28-30], but no studies have investigated the response of the human osteoligamentous cervical spine from birth to young adulthood under compressive loading. Thus, increasing our understanding of the compressive structural response of the cervical spine over the entire developmental range, from birth to young adulthood, is an

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important component in furthering our understanding of neck injuries, providing additional insight between current juvenile animal models and human response, in addition to providing additional guidance for development of ATD and computational models of the pediatric neck.

Table 1: Perinatal, Neonatal, Pediatric and Young Adult Post-Mortem Human Subject (PMHS) Anthropometry

PHMS ID	Age (MN)	Sex	COD	Whole Body Mass (kg)	Whole Body Height (cm)	Head Mass (kg)	Head Breadth (cm)	Head Length (cm)	Spine Cervical Length (cm)	Spine T1 Endplate Width (mm)	Spine T1 Endplate Depth (mm)	Scale Factor	Scaled Loading Rate (N/s)
Perinatal and Neonatal PMHS													
02P	0	F	Heart Failure	-	-	0.094	5.9	7.1	2.46	9.35	4.85	0.115	5.8
13P	0	F	-	-	-	0.492	7.1	10.2	3.40	13.40	6.50	0.222	11.1
09P	0	M	Fetal Demise	2.04	43.2	SC	9.1	-	3.61	11.05	7.00	0.197	9.9
07P	0	M	-	-	-	0.434	8.2	10.1	3.68	13.65	6.20	0.215	10.8
10P	0	M	Fetal Demise	-	-	SC	7.9	10.0	4.12	15.60	8.15	0.323	16.2
08P	0	M	P-Hyp	-	50.8	0.780	9.4	11.4	4.21	15.00	8.00	0.305	15.3
05P	0.03	F	Diaphragmatic Hernia	2.75	-	0.665	9.1	11.7	4.14	13.00	6.00	0.198	9.9
03P	0.1	M	HIE; cerebral infarction	-	-	0.492	8.5	10.3	3.61	12.25	6.75	0.210	10.5
06P	0.37	F	NIHF; ICH	2.02	44.5	0.702	10.4	11.2	3.83	13.40	6.60	0.225	11.3
11P	0.53	F	Anencephaly	2.27	-	SC	6.3	6.1	3.66	15.35	6.55	0.256	12.8
04P	0.8	F	DWS	2.72	45.7	1.152	10.5	17.5	4.84	15.35	8.70	0.340	17.0
Pediatric PMHS > One Month [Older Cohort]													
12P	5	M	Respiratory Failure	-	-	1.071	12.3	13.2	5.22	20.45	9.00	0.468	23.4
14P	9	M	COPD	7.00	-	1.950	11.5	15.0	5.36	20.00	8.25	0.420	21.0
15P	11	F	SIDS	8.16	71.1	1.570	11.9	14.8	6.12	21.70	10.85	0.599	29.9
16P	18	M	Drowning	11.80	81.3	SC	13.5	15.2	7.17	21.90	12.20	0.680	34.0
17P	22	F	NHL	-	-	-	12.5	16.4	6.73	21.30	11.50	0.623	31.2
24P	72	F	GCT	27.22	137.2	-	14.2	15.8	8.98	25.40	13.75	0.888	44.4
19P	84	F	Renal Failure; HIE	-	-	-	14.9	15.5	7.37	26.15	12.20	0.812	40.6
18P	108	M	ESRD; ↑K+	-	-	2.440	13.1	16.3	8.66	23.60	15.10	0.906	45.3
20P	144	M	Brain Cancer	56.70	-	SC	13.4	16.6	11.03	27.55	15.45	1.08	54.0
21P	192	F	Seizure Disorder	90.72	149.9	-	14.5	19.2	10.68	27.35	14.50	1.01	50.5
22P	204	M	Gunshot	66.68	160.0	SC	-	-	11.95	28.45	18.85	1.36	68.0
23P	216	F	Overdose	-	-	SC	-	-	12.46	25.45	15.90	1.03	51.5

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; HIE - Hypoxic Ischemic Encephalopathy; DWS - Dandy-Walker Syndrome

Equation for area of an ellipse $\frac{1}{4}\cdot(\pi\cdot W\cdot D)$ used to approximate area of T1 endplate for scaled loading rate

II. METHODS

Twenty-four whole osteoligamentous cervical spines from 20 weeks gestation to 18 years were tested in multiple modes of loading as part of a comprehensive test battery focused on characterizing the biomechanics of the cervical spine from birth to young adulthood [12, 25-26]. The whole cervical spine (WCS) was initially tested in tension, and a sub-sample of this cohort was also tested in compression, followed by sectioning of the spines into three segments that included two lower cervical spine segments (LCS: C4-C5 and C6-C7) and one upper cervical spine segment (UCS: O-C2). Similar to the WCS, a sub-sample of the motion segments were tested in compression along with the tension battery. A total of twenty-three spines were tested in compression. These included eleven perinatal and neonatal (younger cohort: 20 wk gestation to 24 d) and twelve infant to young adult specimens (older cohort: 5 mo to 18 yr) (Table 1). Segments were compressed under load control at a rate designed to maintain a constant stress rate throughout the age (size) range of the sample. The segments were compressed to 5% of the expected failure load, where expected failure was approximated based on tensile failure data from previous work [15-16, 23, 32]. Compliance of the fixation techniques and testing apparatus were removed for the frame-cup compliance (WCS and O-C2) and cup-cup compliance (C4-C5 and C6-C7) [26]. Compressive structural stiffness was represented by the slope of a line calculated by regressing the force-displacement response from 50-100% of peak load. Perinatal and neonatal donors were grouped to assess differences in compressive stiffness by level. All perinatal donors were designated zero months of age, while all neonatal donor ages were converted to a fraction of a month based on 30 days/month. The WCS response of 02P and 07P were not included in the average response as neither sample represented a WCS from the head to T1. A single-factor ANOVA (α = 0.05) and *post hoc* Tukey-Kramer HSD multiple-comparison test was used to evaluate differences in stiffness in the younger cohort.

Table 2: Compressive Stiffness for Perinatal, Neonatal and Pediatric PMHS (N/mm)

PMHS ID	Age (months)	Pediatric PMHS < 1 Month Compressive Stiffness (50-100%)			
		WCS	O-C2	C4-C5	C6-C7
02P¥	0	3.1	—	—	—
07P	0	1.6	4.0	27.4	21.7
08P	0	1.8	1.5	—	—
09P	0	—	—	18.6	24.0
10P	0	—	—	27.5	13.6
13P	0	4.1	1.5	33.1	22.7
05P	0.03	1.0	2.2	25.7	36.4
03P	0.1	2.5	1.8	40.3	10.0
06P	0.37	2.0	4.1	20.4	30.1
11P	0.53	—	—	19.2	39.0
04P	0.8	1.2	0.9	25.3	38.3
Average		2.1	2.3	26.4	26.2
SD		1.1	1.3	7.0	10.5
PMHS ID	**Age (months)**	**Pediatric PMHS > 1 Month Compressive Stiffness (50-100%)**			
		WCS	O-C2	C4-C5	C6-C7
12P	5	6.9	9.9	32.8	22.0
14P	9	16.2	20.7	100.9	61.9
15P	11	6.5	36.5	109.8	91.7
16P†,‡	18	—	—	138.6	125.2
17P	22	—	41.0	93.0	86.0
24P	72	—	110.6	227.7	213.0
19P	84	—	—	179.2	—
18P	108	—	71.7	—	192.6
20P	144	—	—	180.5	—
21P	192	—	—	202.9	189.6
22P Δ	204	—	—	318.0	301.1
23P Γ	216	—	—	235.9	282.5

¥ - 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

— - Specimen not available

III. RESULTS

The child cervical spine exhibited a non-linear stiffening response under compressive loading (Figure 1). This response was similar to previously observed force-displacement behaviour observed under tensile loading in the child osteoligamentous spine [12, 25]. Compressive stiffness for the WCS and motion segments are reported for the younger and older cohorts (Table 2).

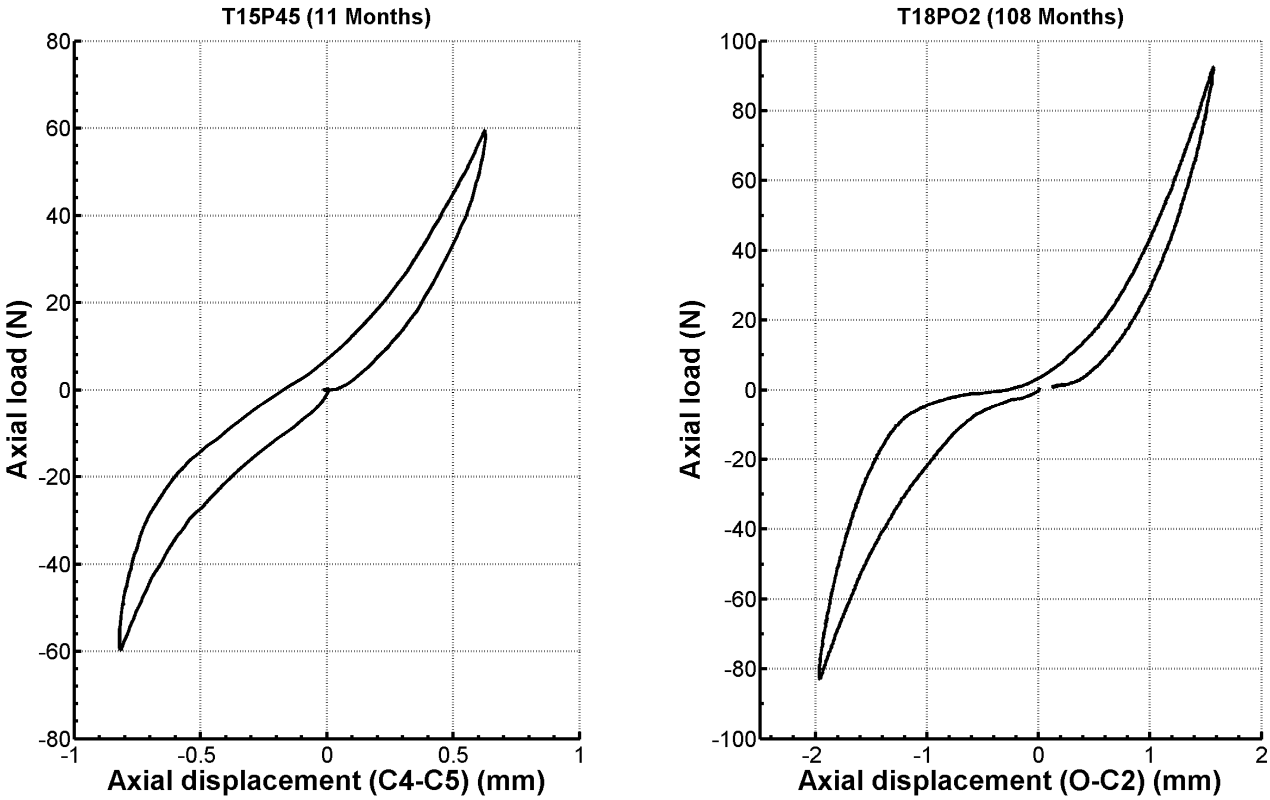

Fig. 1. Non-linear stiffening responses were generally observed in both the compressive and tension phases in both the upper (O2: O-C2) and lower (45: C4-C5) cervical spine and at younger (T15P-9 mo) and older ages (T18P–108 mo). Hysteresis was also typically observed in both phases of loading. Positive load and displacement represents tensile loading, while negative load and displacement represents compressive loading.

Compressive stiffness increased with age for the WCS and motion segments (Figure 2). Both lower motion segments exhibited increases in stiffness from birth to 18 years, while the O-C2 segment increased in compressive stiffness over the age range tested (birth to 9 years). The WCS was tested over a smaller age range, birth to 11 months, but still showed an increasing trend. The WCS stiffness increased approximately three fold from birth to one year of age. Larger increases in compressive stiffness over the first year of life were observed in the motion segments. The UCS stiffness increased 15 fold during the initial year of life, while more modest gains of roughly four fold were observed in the LCS. From birth to 9 years of age the UCS increased in stiffness by approximately 30 fold. The LCS increased in stiffness approximately 10 fold from birth to 18 years, with much of this increase occurring by roughly 6 to 9 years of age.

The younger cohort included perinatal cervical spines up to one month of age and were grouped to assess differences in compressive stiffness by level (Figure 3). The stiffness of the WCS and UCS were significantly lower than the LCS stiffnesses ($p < 0.001$, ANOVA). The LCS were approximately 11 times stiffer than the WCS and UCS in the younger cohort. No significant differences were observed between the two LCS or between the WCS and UCS.

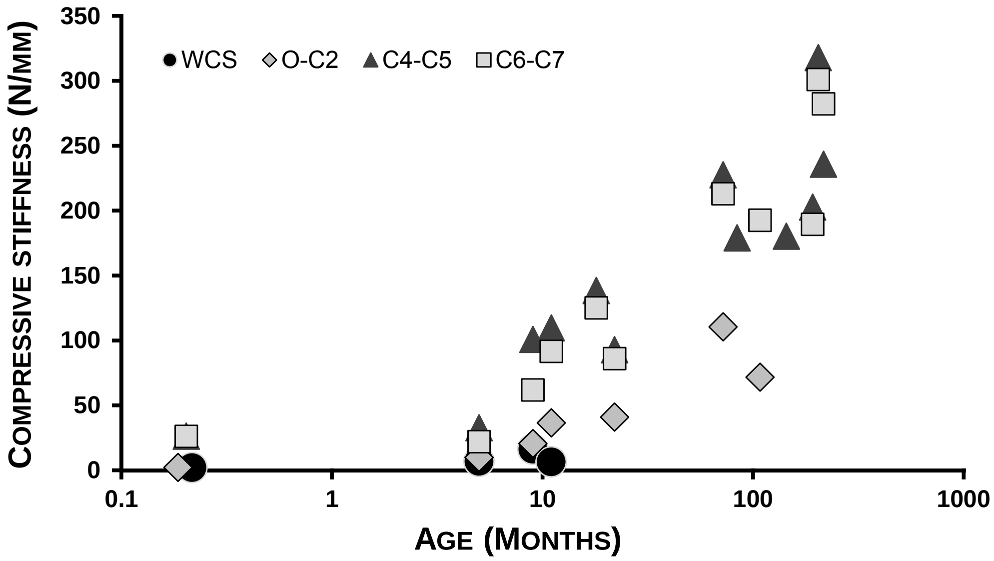

Fig. 2. Compressive stiffness of the perinatal, neonatal and pediatric osteoligamentous cervical spine. The WCS and all motion segments exhibit an increase in stiffness with age (over that range of ages tested).

IV. DISCUSSION

This work provides compressive stiffness data from birth to young adulthood for the LCS, in addition to UCS and WCS compressive response from birth to 9 yrs and 11 mos, respectively. These data along with previously reported data on the tensile response of the child spine increase our understanding of the biomechanical response of the developing cervical spine [12, 25-26] and are especially applicable in the development of age-dependent cervical spinal computational models [27, 31].

The results of the current study should be considered in the context of a number of existing studies that have investigated various aspects of the compressive loading response of human age equivalent juvenile and young adult animal models [17, 19-20]. Reference [17] observed that compressive stiffness increased in juvenile and young adult baboons corresponding to the human equivalent age range of 1 to 26 years. The authors also observed that the compressive stiffness of the O-C2 segment was statistically more compliant than the stiffness of the LCS in the animal model which was similar to the observations of the current study. The compressive stiffness in the O-C2 joint increased threefold in the animal model compared to a twofold increase in the human from 1 to 9 years of age. A similar twofold increase in the stiffness of the human LCS from 1 to 9 years of age was observed, while the animal model saw roughly a 2.5 fold increase over the same human equivalent age.

Recent modelling efforts of the pediatric head and neck may indicate that the compressive tolerance of the cervical spine is affected by the active musculature of the neck during pre-loading [27]. Using active musculature and the stiffness of the osteoligamentous spine the head is stabilized against gravity. The effect of this stabilization is to place the osteoligamentous spine into compression. In a loading environment associated with a motor vehicle crash the potential exists where the occupant may experience head contact that may exacerbate the compressive state of the spine. In this event, the amount of compressive loading required to reach the tolerance of the neck will be less than traditional estimates of compressive tolerance. The current study provides compressive response from birth to young adulthood in the LCS and can provide assistance in estimating compressive tolerance at all ages of development, which is of fundamental importance in continuing to progress our understanding of the interplay between active muscle, osteoligamentous spine and head response in dynamic loading environments.

A number of limitations existed in the current study. The limited number of samples, overall and at any one age, was the most significant limitation. Approximately half of the spines were one month or less in age and provided increased confidence in the response at younger ages. A similar total number of spines occupied the age range of the older cohort, but unlike the younger cohort, these spines extended from one month up to 18 years. The compression protocol was one portion of a larger testing battery that focused on tensile and bending response and this limited the extent of some testing. There were instances based on potential loading instability that tests in the UCS and WCS were not conducted in compression and as a result limited the age range tested for these cervical regions. The maximum level of compressive loading was also a limitation, especially at the youngest ages, as the intent of the experimental protocol was to remain in a sub-injurious region of loading [12, 32].

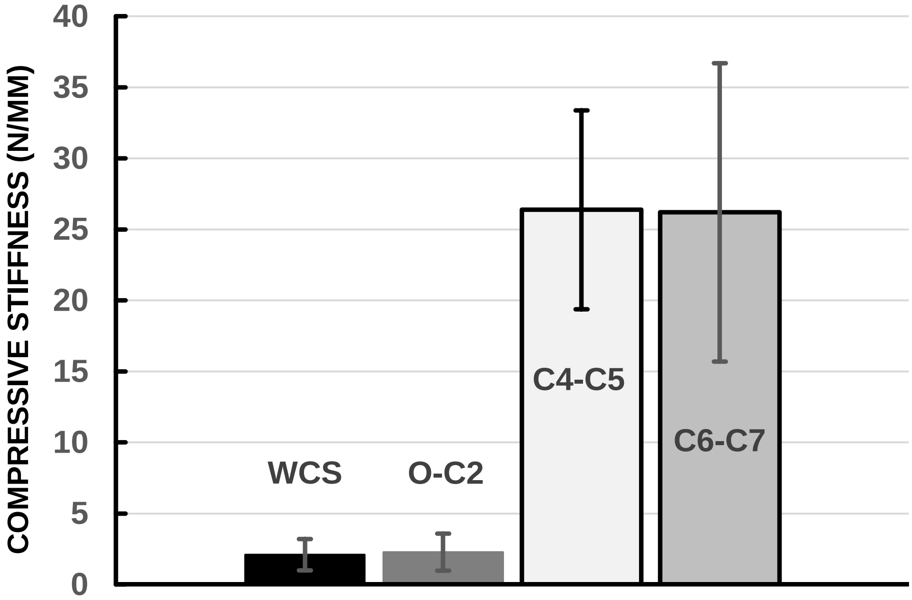

Fig. 3. Whole cervical (WCS; n=7) and upper cervical spine (UCS; n=7) compressive stiffness in the younger cohort (perinatal and neonatal ages) was significantly less stiff than the lower cervical spine (C4-C5; n=9 & C6-C7; n=9). The lower cervical spine was approximately 11 fold stiffer than the upper and whole spine.

V. CONCLUSIONS

This study provides compressive structural response in the developing cervical spine that will be critical for furthering our overall understanding of pediatric neck biomechanics and progressing physical and computational models of the pediatric neck. The presented data encompasses ages from around birth (perinatal) to young adulthood (18 years) and includes WCS, UCS and LCS response. The WCS (2.0 ± 1.0 N/mm) and UCS (2.3 ± 1.3 N/mm) of the younger cohort was statistically less stiff than both LCS (C4-C5: 26.4 ± 7.0 N/mm & C6-C7: 26.2 ± 10.5 N/mm) segments and no differences were observed between LCS or the WCS and UCS segments. The LCS in the younger cohort was approximately 11 fold stiffer than the WCS and UCS. LCS compressive stiffness from birth to 18 years increased by tenfold, while UCS stiffness increased by 18 and 31 fold from birth to 2 and 9 years, respectively. These data accompanied with previously published pediatric data are important building blocks for defining the response of the core component of the pediatric cervical spine – the osteoligamentous neck. Improved biofidelity of osteoligamentous neck models establishes a foundation for investigating the role of musculature in head and neck response and the implications of these responses on the evolution and mechanisms of head and neck injury. Moreover, through the realization of validated child-like physical and computational models the ability to effectively assess countermeasures against head and neck injury from motor vehicle related crashes, falls and sports-related traumatic events is possible.

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