THE ROLE OF MUSCLE TENSING ON THE FORCE-DEFLECTION RESPONSE OF THE THORAX AND A REASSESSMENT OF FRONTAL IMPACT THORACIC BIOFIDELITY CORRIDORS

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ABSTRACT

This paper evaluates how muscle tensing changes the structural response of the dynamically loaded thorax. Nine porcine thoraces with both ventral (supine) and dorsal (prone) loading were used to quantify the effect. Muscle tensing was assessed using repeated tests on a subject with and without forced muscle contraction. Dynamic (~1.25 m/s) and quasistatic tests were performed with a potentiometer and load cells to calculate effective thoracic stiffness. The results show that the effect of muscle tensing decreases with increasing chest deflection, which is supported by the limited human data available. The peak force increases with muscle tensing for chest deflection levels up to about 20%, after which the peak force changes negligibly when the muscles are tensed. The shape of the force-deflection curve, and therefore the work done by the deforming thorax, do, however, depend upon muscle tensing regardless of the peak deflection level attained. The findings are discussed relative to current thoracic biofidelity corridors, which include an adjustment to account for muscle tensing.

Keywords: Thorax, Biofidelity, Muscle Tensing, Dummy Validation

THORACIC DEFORMATION IN RESPONSE TO AN APPLIED ANTERIOR FORCE, often expressed as mid-sternal chest deflection, is an established indicator of injury risk (e.g., Kroell et al. 1971 and 1974, Nahum et al. 1975, Viano 1978, Kent et al. 2001a). An allowable value is specified in Federal Motor Vehicle Safety Standard 208 and the National Highway Traffic Safety Administration has published risk functions showing a clear increase in injury risk as sternal deflection increases (Eppinger et al. 1999). Rib fractures begin to occur when mid-sternal chest deflection reaches approximately 20% of the initial chest depth (Nahum et al. 1975); the rib cage loses stability at approximately 32% (Viano 1978); internal organs sustain serious crushing injuries at approximately 40% (Viano 1978); and the posterior surface of the sternum contacts the anterior surface of the thoracic spine at approximately 50% - 60% (Verriest and Chapon 1985, Kent et al. 2001a). Of particular importance for restraint design and occupant protection in a crash is the force-time history that can be applied to the chest without exceeding an injurious level of deflection. This paper investigates one potentially important factor: the presence of muscle tensing.

Researchers from the U.S. National Highway Traffic Safety Administration (NHTSA) and other groups have estimated that one-half to two-thirds of crash-involved drivers may be tensing prior to impact (Ore 1992, Petit et al. 1998). Furthermore, conscious contraction is not necessary in order to get pronounced muscle tension during an impact. Postural muscle tension is always present for joint stability, and muscles contract in response to a rapid forced extension. Muscle tensing is potentially important in several respects. First, it influences occupant kinematics and restraint loading. For example, a human can provide "propriotonic restraint" in a collision as severe as 48 km/h change in velocity (ΔV) (Crandall et al. 2003). In fact, in a series of human volunteers sled tests at speeds up to 21 km/h ΔV and 15 g peak sled acceleration, Armstrong et al. (1968) found that only 26% of the subject's kinetic energy was dissipated via work through the seatbelt while 55% was dissipated via muscle tension in the legs. Second, muscle tensing can influence the stress distribution in the tissues,

and hence the injury patterns, as external forces are applied. For example, Funk et al. (2001) found that simulated tension in the Achilles tendon produced a greater proportion of tibial pilon fractures in an axially loaded leg, while tests without tension tended to generate more calcaneus fractures. Third, muscle tensing can change the effective stiffness and mass of a body segment or region.

Consider the force-deflection response of the thorax. Lobdell et al. (in King and Mertz 1973) discussed a series of 7 tests involving male volunteers subjected to quasi-dynamic hub loading on the anterior thorax in both a "relaxed" and a "tensed" state. A 338% increase in thoracic stiffness (from 70 N/cm to 236 N/cm) was observed when the volunteers maximally tensed the muscles of their shoulders, thorax, arms, back, and neck. Stalnaker et al. (in King and Mertz 1973) corroborated this result using two human volunteers. They found a 300% increase in thoracic stiffness (from 403 N/cm to 1,140 N/cm) when the volunteers were in a "tensed" state. In both of these test series, however, loading could not be performed to potentially injurious levels of chest deflection (approximately 11% of chest depth in the Lobdell et al. study and 8% in the Stalnaker et al. study). As a result, it is not known if this dramatic increase in stiffness would remain at larger deflections when the rib cage, rather than the musculature itself, is the structure primarily responsible for the measured stiffness.

Our hypothesis is that the effect of muscle tensing on peak force will be less pronounced as chest deflection increases and that these human volunteer studies do not represent the muscle tensing effect that would be present in a dynamic test to an injurious level of chest deflection. The rationale for this hypothesis is that the human volunteer tests, by remaining in an uninjurious loading regime, engaged primarily the anterior and posterior thoracic muscles, with minimal loading of the thoracic cage or its contents. With these muscles in a tensed state, it is reasonable that there would be a pronounced increase in stiffness relative to the untensed state. In contrast, when the deflection levels are sufficient to deform the rib cage and its contents, we hypothesize that superficial muscle tensing will have a minimal influence on the peak force. Understanding this hypothesis is important because adjustments based on the above-described human volunteer results have been made to cadaver-based thoracic biofidelity corridors to account for muscle tensing in a living human (Neathery 1974). If, however, these pronounced increases in force do not remain at larger deflection levels then it will be necessary to reassess the current thoracic biofidelity corridors.

Quantifying the effect of thoracic muscle tone in a potentially injurious test is difficult because human cadavers typically cannot be obtained, screened, and prepared for testing prior to the onset of rigor mortis. Muscle tensing has been simulated mechanically for simpler systems (e.g., Funk et al. 2001), but mechanical simulation of the complex thoracic musculature is not practicable. Embalming has also been used to increase the effective stiffness of cadaveric tissue (Kroell 1995), but the degree to which this represents muscle tensing is unknown. These limitations of cadavers for studying thoracic muscle tensing led to the use of a porcine model in this study, which is an expansion of early work published by Kent et al. (2003). Electrical muscle stimulation applied within minutes of death allowed for the evaluation of full muscle tetanus on thoracic stiffness. While the pig has some limitations as a model of the human thorax, it has been used extensively in the biomechanical literature, and the requirement to test immediately after death precludes the use of a cadaver model. The intention with these tests is to expand the available data on muscle tensing by augmenting the human volunteer studies with additional non-injurious quasistatic tests and dynamic tests at potentially injurious levels of chest deflection.

BACKGROUND – MUSCLE CONTRACTION IN A POSTMORTEM SUBJECT

Since post-mortem muscle stimulation is not common in the injury biomechanics literature, the following discussion provides some background on the rationale and assumptions used to arrive at the decision to utilize this technique.

Each skeletal muscle fiber is a cylindrical cell with several oval-shaped nuclei arranged beneath a plasma membrane called the sarcolemma. Each fiber contains many parallel rod-like myofibrils that extend the length of the cell. Myofibrils consist of a chain of smaller contractile elements called sarcomeres, which contain myocin and actin molecules connected by cross bridge attachments. Shortening of the sarcomeres decreases the length of the myofibrils and causes a muscle contraction. The mechanism by which sarcomeres shorten, while still subject to some debate, is reasonably well explained by the sliding filament theory of Huxley (1957). The sliding filament action is accomplished when an electrical stimulus (action potential) propagates along the sarcolemma,

generating a transient rise in intracellular calcium ion levels. Calcium ions expose binding sites on the actin and myosin crossbridges, while reserves of adenosine triphosphate (ATP) provide energy to "cock" the myocin head. As the sequence of binding/releasing and cocking/relaxing repeats, the myosin pulls the actin toward the center of the sarcomere causing the sarcomere to shorten. This sliding of the actin filaments continues while the calcium signal and ATP are present. In the living, the action potential required to generate the calcium signal is provided by nerve stimulus. The muscles can also contract in a postmortem, pre-rigor subject through the use of an externally generated action potential as long as ATP is still present. In a vigorously exercising living human, ATP stored in the working muscles is depleted in approximately 6 seconds (Marieb 1992). ATP is then generated by the body either aerobically or anaerobically. Obviously, in a postmortem subject aerobic production of ATP is not practicable. ATP can be produced anaerobically, however, through the process of glycolisis and the formation of lactic acid. In a living human, stored ATP and anaerobic production of ATP can support strenuous muscle activity for over a minute (Marieb 1992), so it is reasonable to presume that full muscle contraction (tetanus) can be sustained for at least this long in a limited number of muscle groups using a postmortem, pre-rigor subject. This was confirmed by Jones et al. (1995), who found that muscle response to an electrical stimulus remained above 96% of the *in vivo* response even at 5 minutes post-mortem. After 5 minutes the response drops off approximately logarithmically (Figure 1) and reaches zero approximately 100 minutes after death.

METHODS

Nine post-mortem immature porcine subjects (Sus scrofa), obtained through the University of Virginia (UVa) Department of Comparative Medicine, were used to evaluate the effect of muscle tetanus on the global thoracic response. Immature subjects were chosen for their ready availability. The immature pig's highly cartilaginous rib cage would be expected to contribute less to the thoracic stiffness than the fully mature, highly ossified adult rib cage, and its muscle development would also be immature. How these factors influence the effect of muscle tensing relative to an adult human subject is unknown.

All test procedures were approved by the UVa Institutional Animal Care and Use Committee and all testing was overseen by a certified animal handler. The swine were procured at the conclusion of an independent respiratory study (the nature of the respiratory study was such that the thoracic structural characteristics were unaffected) and were euthanized immediately prior to potentially injurious biomechanical testing. Subjects had been intubated and ventilated for the respiratory study and remained so for the subsequent biomechanical testing. Thoracic anthropometry was measured with the subjects at full inhalation (approximately 1 kPa tracheal pressure and 700 mL tidal volume) (Table 1) and all tests were initiated at this point. The airway was occluded during thoracic loading, so the volume of air in the lungs remained constant.

A custom loading frame capable of generating chest deflection rates from quasistatic to 1.25 m/s was created for this testing (Figure 2). The anesthetized living subjects were positioned supine or prone in the loading apparatus and an 8.9-cm diameter rigid hub was positioned with the center of the hub on the midline midway between the xiphisternum (i.e., the caudal end of the xiphoid cartilage) and the manubrium sterni. Prior to dynamic testing, the subject was euthanized using a solution of pentobarbital, a barbiturate that affects the central nervous system and was therefore assumed to have no affect on the muscles' response to an external stimulus. Tetanus of the thoraco-abdominal musculature was achieved either percutaneously (tests 1_1 through 1_4) or intramuscularly (all others) via 12 electrodes (6 pairs) positioned as described in Table 2. A nominal 150 V pulse with duration of 0.04 μs was applied at 90 Hz to stimulate the muscles. We attempted to perform all muscle tensing tests within 5 minutes of death, but the requirement to randomize test order precluded this for some subjects. On average, the dynamic muscle tensing tests were performed 8.4 minutes after death (range 1 minute to 20 minutes). Regardless of the time after death, however, the 150 V stimulation appeared to generate muscle contraction beyond that possible consciously, again tending to exaggerate the effect of muscle tensing. Dynamic loading was applied to the chest via a dropped mass of 56 kg and quasistatic loading was achieved by simply manually pushing on the translating cross-beam. Two quasistatic and two dynamic tests were performed on each subject, one with the muscles contracted and one without (Table 3). The order of testing was randomized by location (supine/prone) and muscle state (tonic/atonic) to separate the effect of potential tissue damage in the

first test from the effect of muscle tone. The quasistatic tests were intended, in a general sense, to represent the human volunteer tests performed by Lobdell et al. and Stalnaker et al. discussed above. In the quasistatic porcine tests, a peak displacement of approximately 10% of the initial chest depth was attained in 1-2 seconds. This relatively wide range in ramp rate is due to the manual method used to apply the displacement. The dynamic tests were intended to expand on the human volunteer data by approaching injurious deflection levels (~20% of the initial chest depth) at a rate of loading similar to that experienced by a restrained occupant in a severe frontal collision. In all tests, however, we attempted to avoid rib fractures to minimize the effect of test order.

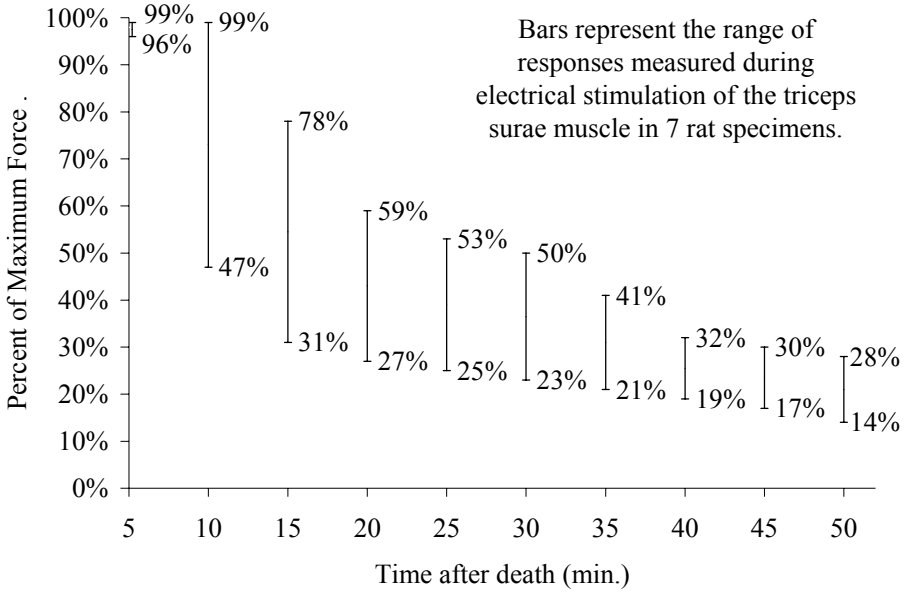


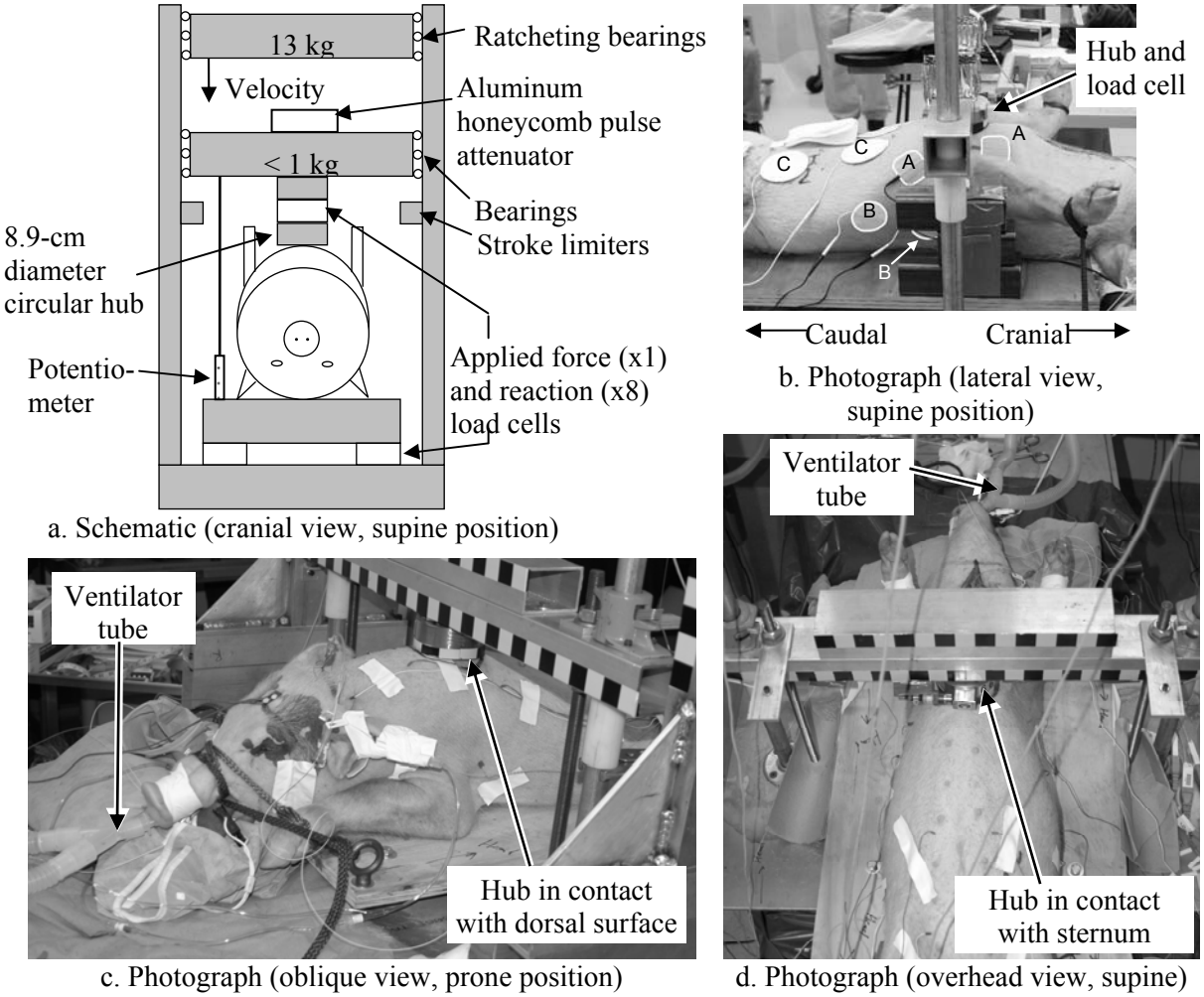
Figure 1. Decay in muscle response post-mortem (Jones et al. 1995). Note that these percentages are relative to externally stimulated muscle contraction, which is greater than conscious contraction.

Table 1 – Description of Test Subjects

Subj.	Age (days)	Weight (kg)	Gender	Proximal Tail to Distal Snout (Supine, cm)	Midsternal Chest Depth (cm)	Midsternal Chest Breadth (cm)	Midsternal Chest Circumf. (cm)
03-P-1	137	26.8	F	103	19.7	21.9	66
03-P-2	129	23.2	F	95	20.3	18.2	63
03-P-3	129	22.7	M	93	18.9	17.4	59
03-P-4	129	23.6	M	93	19.7	18.2	61
03-P-5	123	22.3	M	89	19.6	17.1	59
03-P-6	129	22.7	F	94	20.0	17.4	63
03-P-7	129	21.4	M	88	19.0	16.1	58
03-P-8	129	23.6	F	100	21.7	18.8	67
03-P-9	123	21.4	F	88	18.7	16.5	57

The applied force was measured using a piezo-resistive load transducer positioned between the hub and its support. An accelerometer mounted on the hub was used for inertial compensation. The reaction force was measured using four strain-gage load cells positioned below the subject. Transducers were inserted into the trachea to measure pulmonary pressure, through the carotid artery or the femoral artery into the aortic arch to measure arterial pressure, and through the jugular vein or the femoral vein into the inferior vena cava to measure venous pressure. Acoustic sensors were mounted on the sternum and the skull to document rib fractures. Redundant string potentiometers were used to measure hub displacement and this displacement divided by the initial chest depth at the mid-sternum was defined as the chest deflection. Quasistatic data were sampled at 1000 Hz and filtered at 8 Hz using an 8-pole Butterworth filter. Dynamic data were sampled at 10,000 Hz and

filtered at 500 Hz. To compare the various conditions, least-squares regressions were fit to the data. For the quasistatic tests, a linear regression was fit to the data from each test and the average and standard deviation of the slope was calculated and defined as the stiffness. For the dynamic tests, a fourth-order polynomial was fit to the cluster of data from all tests in each of the four test conditions (prone with tensing, prone without, supine with, supine without). A fourth-order polynomial was chosen in order to capture the nonlinearity present at the greater deflection level tested in the dynamic tests. A curve fit to the cluster of data from all tests was used because all tests were performed to different deflection levels and averaging non-linear curves fit to individual tests results in discontinuities as curves are removed from the average.



a. Schematic (cranial view, supine position)

b. Photograph (lateral view, supine position)

c. Photograph (oblique view, prone position)

d. Photograph (overhead view, supine)

Figure 2. Schematic and photographs of test setup.

Table 2. Description of Electrode Placement (see Figure 2)

Electrode Pair (Bilateral)	Primary Muscle Groups Activated
A1 – overlying pectoral muscle body A2 – midway along the costal margin	pectoralis, intercostals, ventral serratus, rectus thoracis
B1 – dorsal to the mid-coronal plane at level of the xiphisternum B2 – mid-coronal plane on the costal margin	latissimus dorsi, dorsal serratus, intercostals, thoracic trapezius
C1 – paramedian overlying epigastrium C2 – paramedian ventral abdominal wall at level of the iliac crest	external oblique abdominus, internal oblique abdominus, transverse abdominus, rectus abdominus

Table 3 – Test Matrix

Test	Subject	Test type	Position	Muscle tensing	Time since death (min.)	Cranial end of sternum to hub (cm)	Caudal end of sternum to hub (cm)
1_1	03-P-1	Quasistatic	Supine	N		8.4	2.7
1_2	03-P-1	Quasistatic	Supine	Y		8.4	2.7
1_3	03-P-1	Dynamic	Supine	Y	17	8.4	2.7
1_4	03-P-1	Dynamic	Supine	N		8.4	2.7
1_5	03-P-2	Quasistatic	Supine	Y		8.0	3.5
1_7	03-P-2	Quasistatic	Supine	N		8.0	3.5
1_8	03-P-2	Dynamic	Supine	N		8.0	3.5
1_9	03-P-2	Dynamic	Supine	Y	10	8.0	3.5
1_10	03-P-3	Quasistatic	Supine	N		4.0	4.0
1_11	03-P-3	Quasistatic	Supine	Y		4.0	4.0
1_13	03-P-3	Dynamic	Supine	Y	20	4.0	4.0
1_14	03-P-3	Dynamic	Supine	N		4.0	4.0
1_16	03-P-4	Quasistatic	Supine	Y		6.4	3.5
1_17	03-P-4	Quasistatic	Supine	N		6.4	3.5
1_18	03-P-4	Dynamic	Supine	N		6.4	3.5
1_19	03-P-4	Dynamic	Supine	Y	8	6.4	3.5
1_20	03-P-5	Quasistatic	Supine	N		4.8	4.5
1_21	03-P-5	Quasistatic	Supine	Y		4.8	4.5
1_22	03-P-5	Dynamic	Supine	Y	1	4.8	4.5
1_23	03-P-5	Dynamic	Supine	N		4.8	4.5
1_24	03-P-6	Quasistatic	Prone	Y		3.1	4.8
1_25	03-P-6	Quasistatic	Prone	N		3.1	4.8
1_26	03-P-6	Dynamic	Prone	N		3.1	4.8
1_27	03-P-6	Dynamic	Prone	Y	6	3.1	4.8
1_28	03-P-7	Quasistatic	Prone	N		3.7	3.7
1_29	03-P-7	Quasistatic	Prone	Y		3.7	3.7
1_30	03-P-7	Dynamic	Prone	Y	1	3.7	3.7
1_31	03-P-7	Dynamic	Prone	N		3.7	3.7
1_32	03-P-8	Quasistatic	Prone	Y		4.7	3.5
1_33	03-P-8	Quasistatic	Prone	N		4.7	3.5
1_34	03-P-8	Dynamic	Prone	N		4.7	3.5
1_35	03-P-8	Dynamic	Prone	Y	12	4.7	3.5
1_36	03-P-9	Quasistatic	Prone	N		4.3	3.6
1_37	03-P-9	Quasistatic	Prone	Y		4.3	3.6
1_38	03-P-9	Dynamic	Prone	Y	1	4.3	3.6
1_39	03-P-9	Dynamic	Prone	N		4.3	3.6

RESULTS

The test procedures resulted in the successful collection of reasonable data for all tests except tests 1_1 and 1_2. In those tests, the manual loading methodology generated substantially different loading rates. The methodology was refined and comparable loading rates were generated in subsequent tests. One of the dynamic test pairs (1_34 and 1_35) was excluded from analysis since multiple, bilateral rib fractures were observed during the post-test necropsy and an acoustic burst consistent with fracture was observed during test 1_34. Furthermore, the muscle tensing trend for those tests is inconsistent with the other subjects tested in this configuration, so it appears that ribs were fractured in test 1_34, which corrupted the data from 1_35. Data from 34 tests were therefore used in the analysis (Appendix A).

Quasistatic Tests

Chest deflection levels up to 10% were attained in the quasistatic tests, with peak forces of approximately 400N. Over this range, the force-deflection response was essentially linear in all tests.

The prone position generated stiffer response than the supine position, regardless of muscle tone (Figure 3, left chart). Muscle tone increased the stiffness significantly in both positions ($p<0.01$, paired student's t-test), but the effect was more pronounced with the subject prone (41.3% increase in stiffness vs. 32.8%), which is consistent with the concentration of muscle mass dorsally on the pig.

Dynamic Tests

Chest deflection up to 22% was attained in these tests, with peak forces as high as approximately 800 N (test 1_34 excluded). The force-deflection response in the supine tests was non-linear, with a pronounced increase in slope at approximately 15% deflection. Nonlinearity in the prone tests was less pronounced. Interestingly, the initial slope of the dynamic tests was comparable to the slope of the quasistatic tests, despite the difference in loading rate. Muscle tensing did not have an influence on the supine results (Figure 3, right chart), but the shape of the force-deflection curve changed when the muscles were tensed in the prone position. The instantaneous slope was greater with muscle tensing up to approximately 10% chest deflection, after which the slope was lower until the tensed and untensed force became nearly equal at approximately 19% chest deflection. As a result of this change in shape, there was a pronounced increase in work done by the deforming thorax when the muscles were tensed. To attain chest deflection levels below 10%, approximately 90% more force and energy were required when the muscles were tensed. To attain 20% chest deflection, however, the percent increase in peak force had dropped to below 5% and the increase in work performed on the deforming thorax had decreased to approximately 45% (Figure 4).

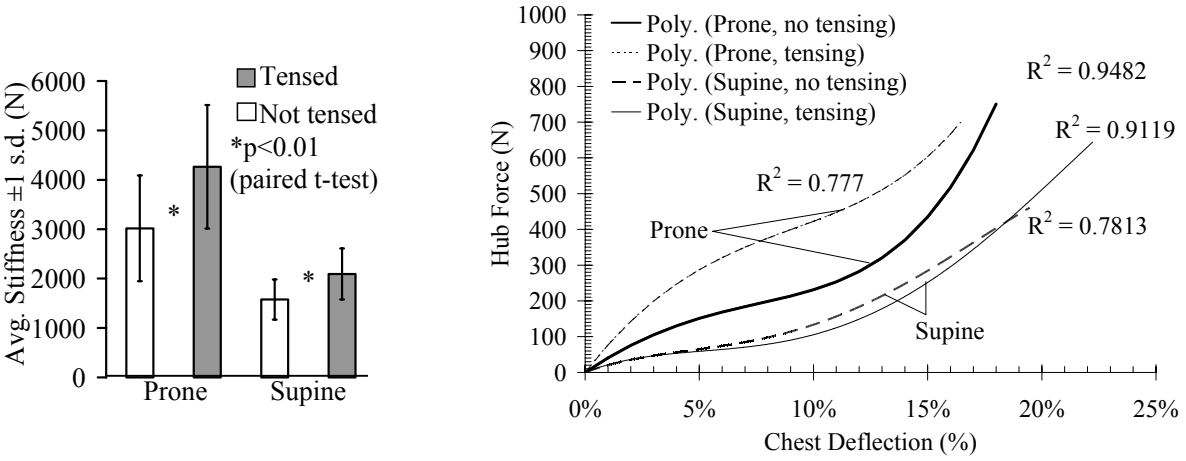


Figure 3. Average responses in the quasistatic (left) and dynamic (right) tests.

DISCUSSION

Muscle tensing is an important factor to consider in the design of human models and in the interpretation of their outputs. Occupant kinematics, stress distribution, effective segment masses, and load sharing are all influenced by the degree to which muscles are tensed during a collision. This study indicates that the effect of muscle tensing on the force generated by a deforming thorax decreases as chest deflection increases, but that the path of the force-deflection curve depends on muscle tensing. At chest deflection levels below approximately 10%, muscle tensing causes an increase in force-deflection slope, but by approximately 19% deflection, the force with and without tensing have become equal. If 20% is considered to be an injurious level of chest deflection, then the force at the point of injury is not dependent on the state of muscle tensing, but the energy required to attain injury is.

Of course, there are limitations to the use of a porcine model to study this phenomenon, so it is important to evaluate these findings relative to the available human volunteer data. As described in the Introduction, there are two series of quasistatic human volunteer tests at low levels of chest deflection (Lobdell et al. and Stalnaker et al. in King and Mertz 1973). Both of these series found a substantial increase in thoracic stiffness when the muscles were tensed. There appears to be only one instance of dynamic, potentially injurious loading being applied to a human volunteer with and without muscle tensing (Patrick 1981). This series of 8 tests involved a single subject exposed to

impacts up to 4.6 m/s with a 10-kg padded hub impactor. As part of this test series, the subject was exposed to a 3.4 m/s impact with and without muscle tension where both hub force and chest deflection are documented. With no muscle tensing, the thorax generated 1.16 kN of force at 4.57 cm of chest deflection. With muscle tensing, the force was 1.24 kN at 4.5 cm. This is a much smaller increase than would be expected based on an extrapolation of the quasistatic human volunteer results and supports both the hypothesis and findings of our study. This is illustrated graphically in Figure 5, which summarizes the results of the quasistatic and dynamic human and porcine tests. In this figure, the percent increase in peak force due to muscle tensing is plotted as a function of the chest deflection attained in each test series. Both the humans and the pigs exhibit a decreasing trend, as illustrated by a linear curve fit to the data. If linearity can be assumed, then the effect of muscle tensing on the peak force becomes negligible at 20% to 25% chest deflection. This finding is further supported by other tests in the Patrick series. Figure 5 shows only the tests in which both force and deflection were measured, but there were additional tensed and relaxed tests where chest deflection was not measured. In these tests, the difference in peak force between tensed and relaxed decreased as impact speed increased. In tests at 2.4 m/s, the tensed state generated a peak force 121% of the peak force in the relaxed state (890 N vs. 730 N), while at 3.4 m/s the tensed force was only 107% of the relaxed force (1,240 N vs. 1,160 N).

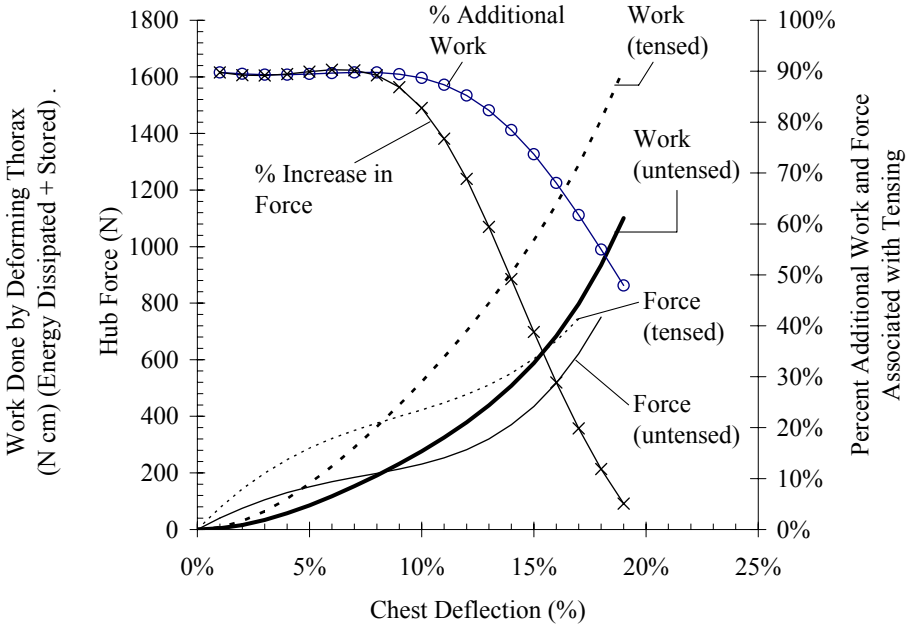


Figure 4. Effect of muscle tensing on force and work (dynamic tests on prone porcine subjects). Note that the supine tests exhibited essentially no musculature effect, presumably due to the lack of ventral muscle mass in the pig.

The most obvious application of these results is in the thoracic biofidelity corridors used for model validation in a frontal hub impact (Neathery 1974). As discussed by Lobdell et al. (in King and Mertz 1973) and later by Neathery (1974) and Kroell (1995), the dynamic response corridors for unembalmed cadavers were adjusted for muscle tensing by shifting each entire corridor vertically by 667 N. This shift was justified based on the human volunteer results of Lobdell et al. and of Stalnaker et al., but it was intended to be a suggestion only and was acknowledged to be based on "very little quantitative data" (Lobdell et al. in King and Mertz 1973). These porcine tests and the available human volunteer data suggest that a simple vertical shift in the corridors is not appropriate to capture muscle tensing effects. First, the porcine and Patrick tests indicate that the peak force should be essentially unchanged in tests having the severity of those used to develop the hub corridors. Second, a simple vertical shift does not consider tensing's effect on the work performed by the deforming thorax.

The effect of this shift is illustrated in Figure 6. The 6.7 m/s corridor has a mean peak chest deflection of approximately 30%. According to both the human and the pig tests, the peak force

should be unaffected by muscle tensing at this level of chest deflection, yet the 667 N shift represents a 19.7% increase in peak force. The shift in the 4.3 m/s corridor is more reasonable, but still too large. The 4.3 m/s corridor has a mean peak deflection of approximately 21% and the 667N shift represents a 28.6% increase in peak force. Our analysis suggests that shift in peak force in the 4.3 m/s corridor should be reduced to about 7%, or 160N, and should be eliminated in the 6.7 m/s corridor, though we have not attempted to quantify how the overall shape of the curve would be influenced by muscle tensing. In both cases, however, the shape of the corridor should be changed since a simple vertical shift does not account for muscle tension's diminishing effect as deflection increases. Determining the nature of this change is beyond the scope of this paper, but the findings of our study and of Patrick indicate that the most reasonable approach is to neglect any muscle tensing effect and simply use the "unshifted" corridors for biofidelity assessment rather than rely upon the myriad assumptions and simplifications that would be necessary in order to quantify what is undoubtedly a small change in either peak force or peak deflection.

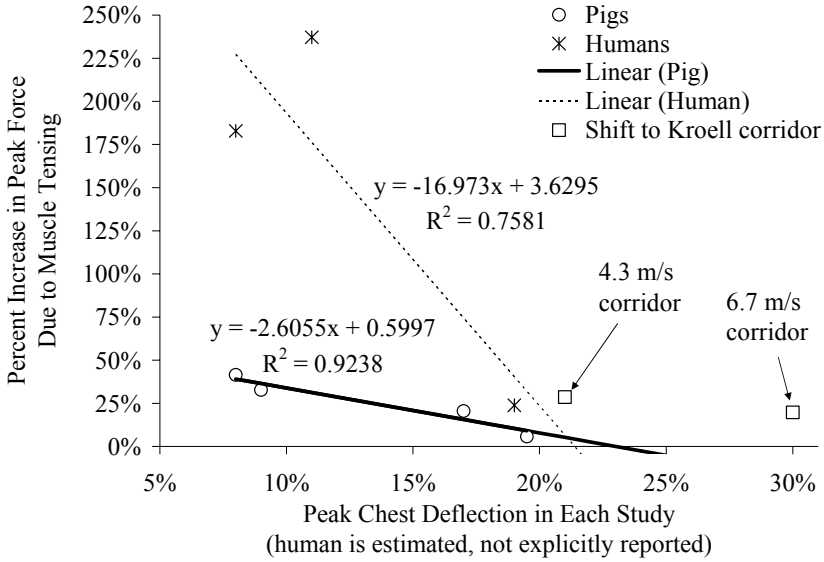


Figure 5. Muscle tensing influence on peak force in human and pig tests as a function of maximum chest deflection. Human data from Lobdell et al., Stalnaker et al., and Patrick. Pig data are from curve fits to the four conditions tested in this study. Also shown is the magnitude of the shift to the Kroell corridors used for thoracic biofidelity assessments.

Despite these important limitations, the shifted corridors have become the standard for thoracic biofidelity assessment (Neathery 1974); including validation of the Hybrid III dummy, which is used to certify vehicles worldwide, the more advanced THOR-α dummy, and thoracic finite element models. When the musculature shift is removed, however, interpretation of the models' responses is fundamentally different (Figure 6). For example, consider the THOR-α responses in the 4.3 m/s corridor. If biofidelity is defined by the shifted corridor, then the response is below the corridor for deflections below 23 mm, but inside the corridor at higher deflection levels. If the adjusted corridor is shifted down by 667 N to more accurately represent a human response, then the THOR-α response is in the corridor up to approximately 30 mm and above the corridor at greater deflections. Similarly, the response of the Hybrid-III and of the H-Thorax™ (IPS 2000) would be interpreted differently depending on whether the shifted or unshifted curve is considered to be the correct standard. Optimization of restraint systems for a human population is not possible if these validation corridors are not representative of the population in the field, so it is important to critically evaluate the pronounced and poorly justified shift in these ubiquitous biofidelity standards.

The interpretation of the porcine results is, of course, hampered by the pig's limitations as a model of the human thorax and by the limitations of our experimental design. While there are several differences in thoracic anatomy between man and pig (Huelke et al. 1986), the most critical limitations with respect to the current study are the differences in thoracic cross-sectional shape and muscle distribution. In humans, the lateral dimension of the thorax is usually larger than the anterior-

posterior dimension, while the pig has the opposite aspect ratio. Additionally, humans have more anterior thoracic musculature, with relatively large pectoralis muscles. The pig's thoracic musculature is concentrated more dorsally than ventrally. Both of these factors would seem to minimize the importance of muscle tensing in the pig model relative to the human. The geometric differences tend toward a stiffer bony structure since the moment arm between the mid-sternum and the lateral thoracic cage is shorter for the pig's aspect ratio than for the human's (i.e., an ellipse is stiffer loaded along its major axis than along its minor axis). Thus, the bony contribution might be expected to be greater in the pig than in the human, and hence the muscle contribution less, though the use of immature pigs may reduce this effect somewhat relative to the completely ossified adult human. The muscle distribution in the pig may also tend to minimize the role of muscle tensing since there is very little muscle on the ventral aspect of the pig's thorax. We addressed this issue by testing the pig both prone and supine. In the supine configuration there was essentially no muscle between the hub face and the sternum, but the prone condition resulted in large muscle groups between the hub and the bony thoracic cage. As expected, the prone configuration was more sensitive to muscle tensing both quasistatically and dynamically and may be more representative of the human sensitivity. Finally, our use of repeated tests on a single subject is a limitation of the study. For the quasistatic tests the effect of test order is negligible, but the dynamic tests, which were designed to approach injury, did cause tissue changes. We dealt with this issue in several ways. First, the order of tests was randomized with respect to muscle tensing. Second, at least four subjects were tested in each of the conditions. Finally, acoustic sensors and post-test necropsies were used to confirm that tissue damage was limited to the soft tissues and not the bony thorax.

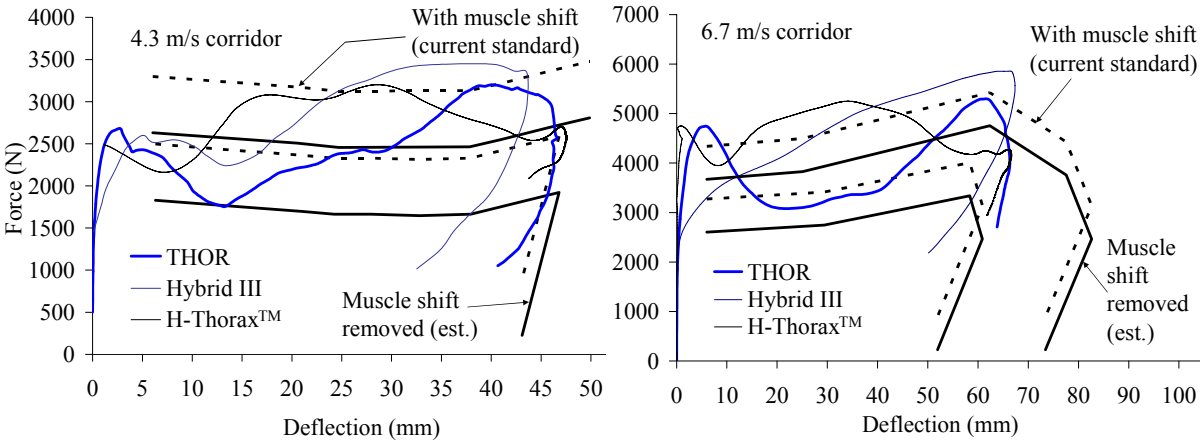


Figure 6. Comparison of THOR, Hybrid III, and H-Thorax response in blunt hub impacts relative to shifted and unshifted blunt hub impact response corridors.

CONCLUSIONS

Existing tests on human volunteers have shown that muscle tensing has a pronounced effect on thoracic stiffness in quasistatic tests at low chest deflection levels. A single human volunteer test performed in the early 1970s at higher severity dynamic loading, however, suggested that peak force is minimally affected by musculature in that environment. This study has presented both quasistatic, low-severity and dynamic, higher-severity tests of pigs with and without forced muscle contraction. These porcine tests support the human data by showing the peak force to be less sensitive to muscle tensing as chest deflection increases, with the effect becoming negligible by about 20% chest deflection. Furthermore, the pig tests have shown that the shape of the force-deflection curve, and hence the work performed by the deforming thorax, is affected by muscle tensing, even in tests where the peak force is not sensitive. These findings indicate that the current thoracic biofidelity corridors, which have been shifted in the force axis, do not accurately represent the effect of thoracic musculature at either 4.3 m/s or at 6.7 m/s. The findings of our study and of Patrick indicate that the most reasonable approach is to neglect any muscle tensing effect and simply use the "unshifted" corridors for biofidelity assessment rather than rely upon the myriad assumptions and simplifications that would be necessary in order to quantify what is undoubtedly a small change in either peak force or peak deflection.

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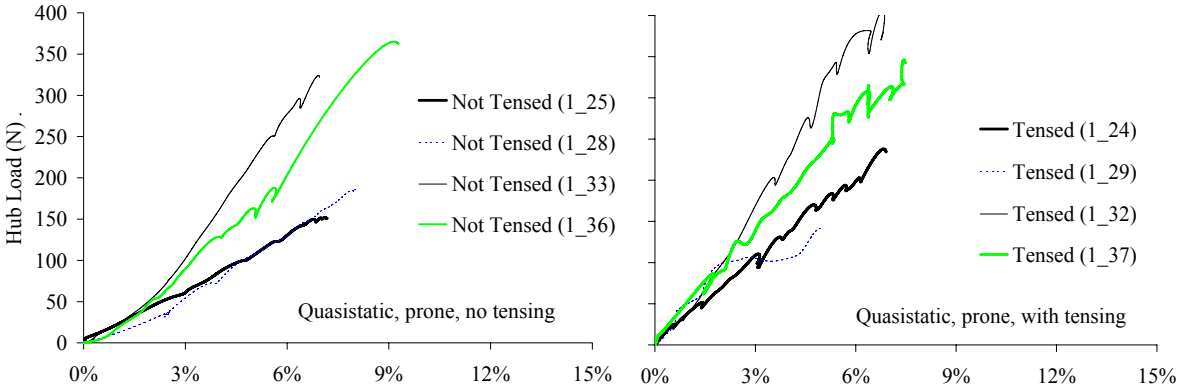
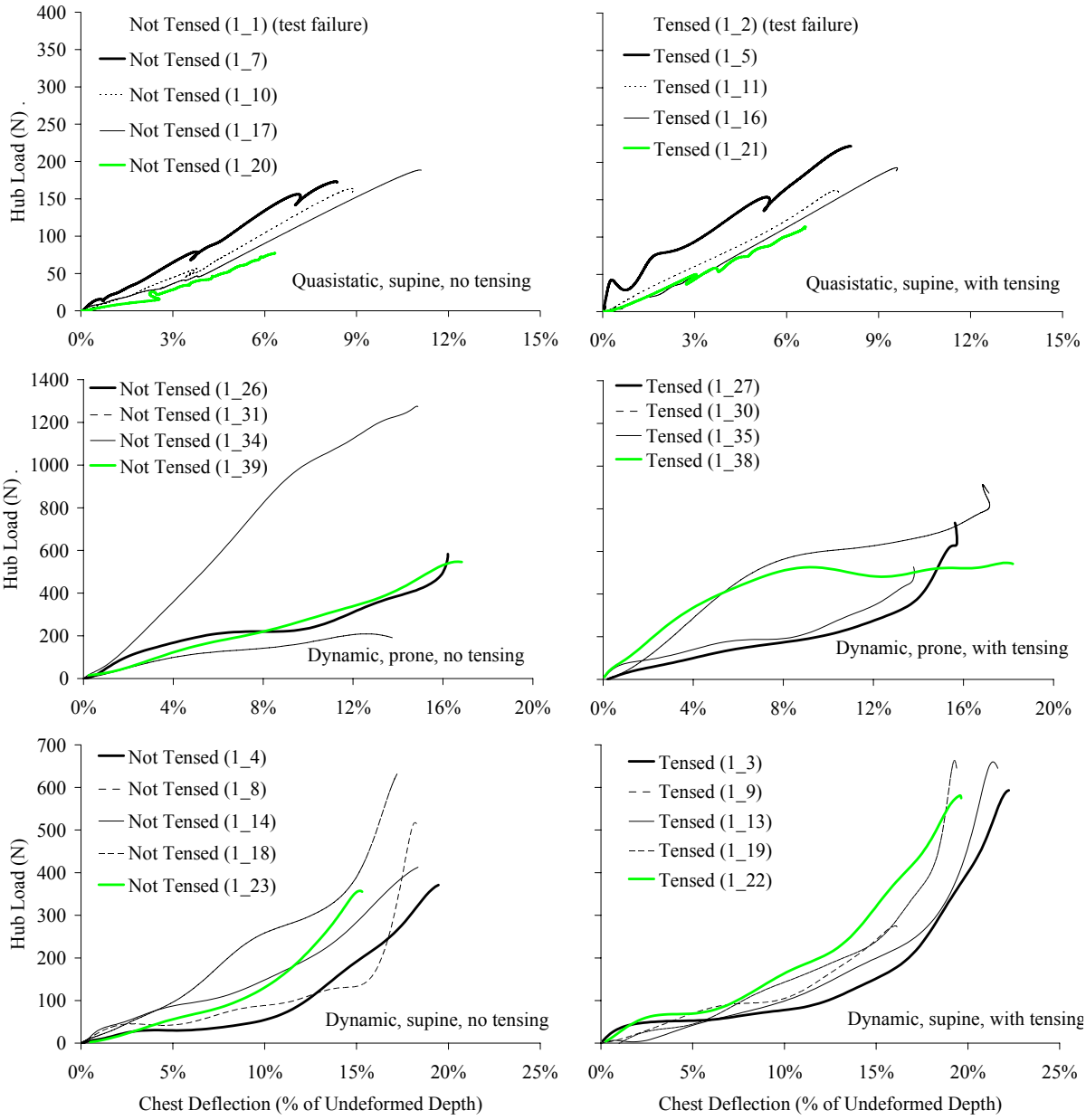
Appendix A – Force-deflection cross plots for all tests




Not Tensed (1_1) (test failure)
Not Tensed (1_7)
Not Tensed (1_10)
Not Tensed (1_17)
Not Tensed (1_20)
Quasistatic, supine, no tensing
Tensed (1_2) (test failure)
Tensed (1_5)
Tensed (1_11)
Tensed (1_16)
Tensed (1_21)
Quasistatic, supine, with tensing
Not Tensed (1_26)
Not Tensed (1_31)
Not Tensed (1_34)
Not Tensed (1_39)
Dynamic, prone, no tensing
Tensed (1_27)
Tensed (1_30)
Tensed (1_35)
Tensed (1_38)
Dynamic, prone, with tensing
Not Tensed (1_4)
Not Tensed (1_8)
Not Tensed (1_14)
Not Tensed (1_18)
Not Tensed (1_23)
Dynamic, supine, no tensing
Tensed (1_3)
Tensed (1_9)
Tensed (1_13)
Tensed (1_19)
Tensed (1_22)
Dynamic, supine, with tensing
Hub Load (N) .
Hub Load (N)
Chest Deflection (% of Undeformed Depth)