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The Use of a Postmortem Porcine Model to Study the Effect of Muscle Tetanus on Thoracic Force-Deflection Response

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ABSTRACT

This paper evaluates how muscle tensing changes the structural response of the dynamically loaded thorax. Structural models of two porcine thoraces were used to quantify the effect. A quasilinear viscoelastic formulation was used to model the elastic and viscous response, with ramp-hold tests used to determine the model coefficients. The effect of thoracic musculature was assessed using repeated tests on a subject with and without forced muscle contraction. Even maximally contracted thoracic musculature is shown to have a minimal effect on the response, with similar elastic and viscous characteristics exhibited by each subject regardless of muscle tone.

INTRODUCTION

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, Kroell et al. 1974, Nahum et al. 1975, Viano 1978, Kent et al. 2001a). A maximum 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 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), and it is

unknown how this may affect thoracic response. Quantifying the effect of muscle tone is difficult because, while it may be possible to stimulate muscle contraction electrically in a post-mortem subject, 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. (2001a) presented a series of lower extremity tests in which achilles tension was simulated using a clamp-and-cable system), but mechanical simulation of the complex thoracic musculature is not practicable. As a result, attempts have been made to use non-injurious human volunteer tests to estimate the effect of muscle tensing (Lobdell et al. and Stalnaker et al. in King and Mertz 1973), but these tests have not been performed to potentially injurious levels of chest deflection. Porcine tests presented here are intended to augment the human volunteer studies by approaching injurious levels.

METHODS

Two post-mortem porcine subjects (Sus scrofa), obtained through the UVa Department of Comparative Medicine, were used to evaluate the effect of muscle tetanus on the global thoracic response. All test procedures were approved by the UVa Institutional Animal Care and Use Committee. 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 biomechanical testing. Subjects had been intubated and ventilated for the respiratory study, and remained so for the subsequent thoracic testing. Thoracic anthropometry was measured with the subjects at full inhalation (approximately 1 kPa tracheal pressure) (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.

Table 1. Description of Porcine Subjects

Identification number	1	2
Age (months)/gender	4/M	4/M
Weight (kg)	20.5	25.0
Proximal tail to distal snout (cm)	85	91
Supine chest depth (mid-sternum/bottom of sternum) (cm)	17.5/17.9	20.0/20.0
Supine chest breadth (mid-sternum/bottom of sternum) (cm)	17.2/19.0	20.0/22.3
Supine chest circumference (mid-sternum/bottom of sternum) (cm)	56.5/56.5	63.8/63.8

Note: All measurements taken with ventilated subject at maximum inhalation

A custom loading frame was designed for thoracic characterization of the porcine subjects. The apparatus is capable of generating a 5-cm ramp in approximately 40 ms (1.25 m/s) and then holding that displacement indefinitely via a ratcheting mechanism (Figure 1). The anesthetized living subjects were positioned supine 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 (Figure 1). The subjects were then 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. Immediately following death, tetanus of the thoraco-abdominal musculature was achieved via 12 electrodes (6 pairs) positioned as described in Table 2. Immediately following

contraction, the ramp displacement was applied to the chest and held for 60 seconds. Tetanus was maintained throughout the hold portion of the test. Two tests were performed on each subject, one with the muscles contracted and one without (Table 3). The order of testing was reversed from the first subject (tonic then atonic) to the second subject (atonic then tonic) to separate the effect of potential tissue damage in the first test from the effect of muscle tone.

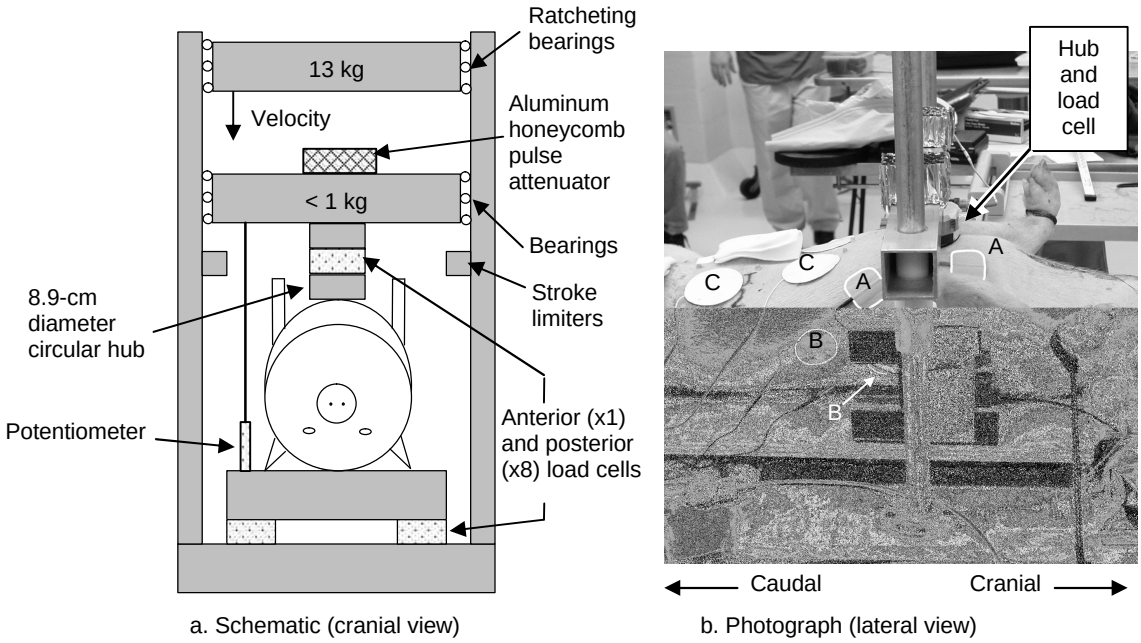

Figure 1: Porcine test methodology and electrode placement.

Table 2. Description of Electrode Placement (see Figure 1)

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

The force applied anteriorly 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 posterior reaction force was measured using 8 piezo-electric load crystals positioned below the posterior loading platform. 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. Data were sampled at 1 kHz.

Data Analysis and Model Development

The posteriorly measured force and the mid-sternal chest deflection were used in the development of quasilinear viscoelastic (QLV) structural models of the porcine thoraces. The posterior force was used in the QLV model development because the formulation chosen does not include consideration of the accelerated sternal mass (i.e., there is no inertial term in the model). template

Development of QLV Structural Formulation. Depending on the characteristics of a material, the form of a viscoelastic model used to describe it can include multiple spring and dashpot elements arranged singly, in series, or in parallel. The elastic and viscous characteristics of biological solids have often been described using a model consisting of an elastic element in parallel with one or more Maxwell elements. This model form will be used in the current study, but the model will be used to describe the relationship between the applied force, F, and the resulting mid-sternal chest deflection, c, rather than a stress-strain description of a material.

The force-deflection response of this model can be described using a differential equation for the force in terms of the time derivatives of the deflection. Experimental determination of the model coefficients, however, is facilitated by a hereditary integral description of the model's response to an applied step in deflection. The hereditary integral formulation results from the rule of linear superposition (see Flugge 1967). For the thorax, consider the case of several displacements of different magnitudes applied successively. The force response is a function of the magnitude of each applied displacement and of all displacements prior to it. If the limiting case of a series of infinitesimal displacements is considered, the result is the hereditary integral description of the structural response:

$$F(t) = \int_{-\infty}^{t} F_R(t-\tau)\frac{\partial c(\tau)}{\partial \tau}\,d\tau \qquad [1]$$

where FR(t-τ) is the relaxation function of the material, c(t) is the mid-sternal chest deflection, and F(t) is the force response to the applied displacement. Fung's theory of QLV assumes that the force relaxation function can be divided into a spatial component (i.e., a strain-dependent or, in this case, a chest deflection-dependent function) and a temporal component (i.e., a time-dependent function) (Fung 1981):

$$F_R(c,t) = G(t)\cdot F^{elastic}(c) \qquad [2]$$

where $F^{elastic}(c)$ is the instantaneous elastic function (i.e., the force response to an instantaneously applied step deflection) and G(t) is a normalized, or reduced, relaxation function, which is a monotonically decreasing function of time. The elastic function may assume a nonlinear form and, for biological materials subjected to finite strains, a nonlinear form of the elastic response is usually required. Despite this nonlinearity in the force response, the principle of superposition remains valid due to the assumption of time linearity, and the resulting hereditary integral describes the response of a QLV structure:

$$F(t) = \int_{-\infty}^{t} G(t-\tau)\frac{\partial F^{elastic}[c(\tau)]}{\partial c}\frac{\partial c(\tau)}{\partial \tau}\,d\tau \qquad [3].$$

In this study, an exponential form was assigned for the instantaneous elastic function:

$$F^{elastic}(c) = A \cdot [Exp(B \cdot c) - 1] \quad [4]$$

where A and B are loading condition-specific model coefficients determined from the experimental data. Four exponential terms were used in the reduced relaxation function, G(t), which was assigned the form of a sum of exponentials:

$$G(t) = \sum_{i=1}^{4} G_i \cdot Exp(-\beta_i t) + G_\infty \quad [5]$$

where G_i, G_8, and β_i are model coefficients. The coefficients G_i and G_8 are normalized so that their sum is unity.

RESULTS

Due to limitations of the laboratory setup for the porcine testing, preconditioning tests were not possible. As a result, the ramp-hold model development tests were influenced by the order of testing. As noted in Table 3, a clear change in the thoracic depth was observed after the first ramp-hold test on each subject. Following the initial test, the thorax depth decreased 0.7 cm and 1.2 cm. This deformation is not believed to be due to thoracic hard tissue damage for multiple reasons. First, palpation before and after all tests indicated no fractures or instability of the chest wall. Second, these subjects were 4 months of age (approximately 2 months before sexual maturity and approximately 14 months before maximum size) and pediatric ribs typically exhibit large strains prior to fracture. Finally, there was no dramatic change in the thoracic force-deflection response once the offset due to the change in chest depth was considered (Figure 2). We hypothesize that, rather than hard-tissue damage, the change in chest depth is due to damage induced in the superficial musculature and viscera and in the internal organs and soft tissues as well as a long-time viscous response. The result of this change in chest depth is that the initial condition was different between tests. This change is quantified in Figure 2, which shows the posteriorly measured force as a function of the position of the string potentiometer. The force-displacement curves for the first and second tests are clearly offset. These figures also show, however, that the slope of the force–position curve is essentially unchanged from the atonic to the tonic condition for both subjects, regardless of the order of testing. By reversing the order of the tonic and atonic tests, it is possible to evaluate the effect of muscle tone independently of the effect of test order, and it is clear that the effect of muscle tone is small. This insensitivity to muscle tension is also apparent in the QLV model coefficients calculated for each test (Figure 3 and Table 4). While differences in both the elastic function and the reduced relaxation function can be seen with muscle tension, the differences are small and are likely within the range of repeatability for the tests.

DISCUSSION

The influence of thoracic musculature has been studied in the past using human volunteers. 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 linear elastic 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

Table 3. Porcine Test Description

Test number	Subj.	Muscle State	Step Magnitude	Time after death	Notes
3_1	1	Tensed	5.07 cm	53 sec.	No hard tissue injury found via palpation after test 3_1, but thorax depth decreased 1.2 cm due to test 3_1 loading. Subject ventilated for approximately 5 minutes between test 3_1 and test 3_2, but original chest depth could not be obtained.
3_2	1	Relaxed	3.87 cm	< 600 sec.	
3_3	2	Relaxed	3.78 cm	94 sec.	Decrease in thoracic depth after test 3_3 present, but less pronounced (less than 1 cm) than that exhibited by subject 1 after test 3_1.
3_4	2	Tensed	3.87 cm	520 sec.	

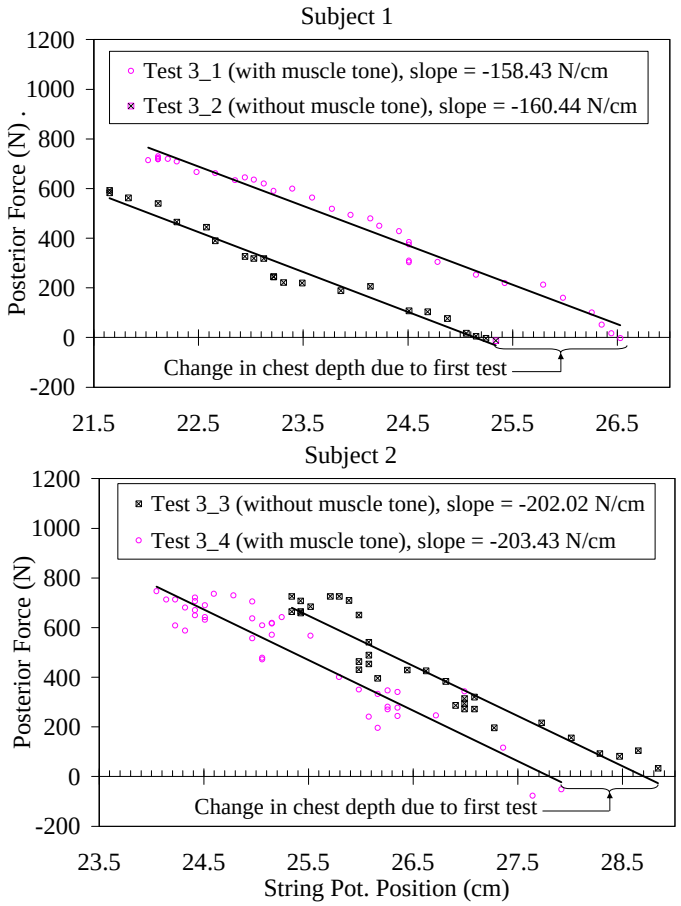

Figure 2: Porcine force-deflection response with linear regression. Note the shift due to test order and also that the slope is essentially unchanged between tests for both subjects, indicating a negligible effect from muscle tetanus.

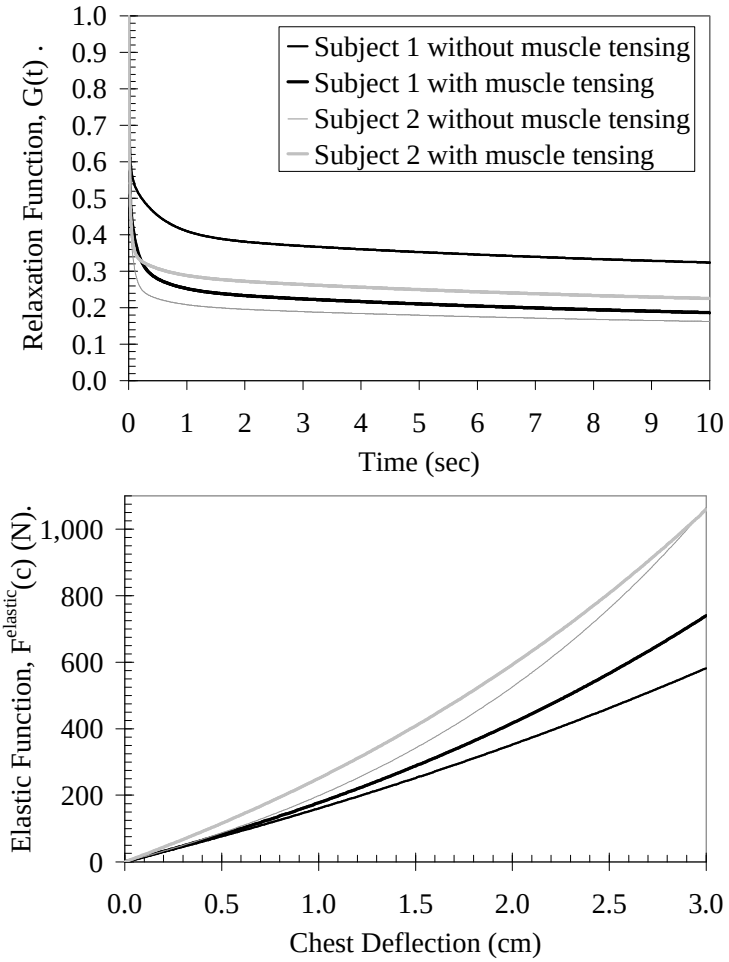

Figure 3: Reduced relaxation functions and elastic functions for porcine tests with and without muscle tone showing inter-specimen difference in response but a small musculature effect.

Table 4. QLV Coefficients for Porcine Tests

Test	A (N)	B (cm^{-1})	G_1	G_2	G_3	G_4	G_8	β_1 (s^{-1})	β_2 (s^{-1})	β_3 (s^{-1})	β_4 (s^{-1})
3_1	507	0.30	0.10	0.09	0.19	0.47	0.15	0.10	2	10	100
3_2	813	0.18	0.12	0.16	0.09	0.35	0.28	0.10	2	25	100
3_3	306	0.50	0.06	0.06	0.25	0.49	0.14	0.10	2	15	100
3_4	690	0.31	0.10	0.06	0.22	0.43	0.19	0.10	2	25	100

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 was 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. The current study addressed this issue by performing tests to potentially injurious levels on postmortem swine with and without muscle tetanus. These tests were performed to deflection levels of approximately 15% (subject 2) and 25% (subject 1) of the initial chest depth. At this level of

deflection, the rib cage appears to be primarily responsible for the elastic response of the thorax, particularly in the pig with its less pronounced anterior musculature. It is logical that the response at low levels of chest deflection would be dominated by the effect of musculature, but that this effect would become less pronounced as the deflection level increases and the rib cage becomes loaded more heavily.

It has been established that the Hybrid III thorax is stiffer than a human's (e.g., Cesari and Bouquet 1991, 1994) and the current study provides a partial explanation for this. Based on the human volunteer-based studies of muscle tensing discussed above, the cadaver-based force-deflection corridors used in the design of the Hybrid III dummy and the THOR dummy (and often used as finite element model validation corridors) were adjusted by approximately 650 N (Kroell in Backaitis 1994). In other words, due to the observed increase in stiffness when the muscles were tensed in those low-deflection tests, the force values for the corridor were increased to simulate a tensed driver or passenger in a collision. The porcine tests presented here cast some doubt on the validity of this adjustment. The obvious result of this adjustment is that the chest deflection measured by a Hybrid III in a sled test is not the same deflection that would be experienced by a human. As a result, injury risk functions used in the interpretation of Hybrid III chest deflection measures must be developed explicitly for that dummy (e.g. Kent et al. 2001b) and the determination of Hybrid III deflection thresholds based on cadaver thresholds is not straightforward.

The use of a porcine model is a limitation of this study. The swine's thoracic structure differs from the human's in several important ways. Most importantly, due to its body position during locomotion, the swine's musculature is more concentrated on the dorsal aspect of the thorax than the human's. The pig thorax also has a different shape than the human, being narrower with a greater depth, and the pig's ribs are less sloped than the human's. These characteristics may result in stiffer response for a porcine chest compared to its human counterpart (Viano and Warner 1976) and may decrease the effect of muscle tensing. The pig is used often as a reasonable surrogate for a human, however, and its thoracic structural response has been used as a model for the human's in the past (e.g., Viano and Warner 1976, Viano et al. 1977). Future research should evaluate the effect of muscle tensing when the porcine thorax is loaded from directions other than frontal. For example, loading the porcine thorax posteriorly, where the musculature is concentrated, may better simulate anterior loading on a human.

Another limitation of this study is that it was not possible with the current test setup to quantify the degree of muscle tensing attained in the various muscle groups. While the muscle tension could be verified both visually and palpably, it is difficult to define the degree of muscle tone and it can be described only in quantitative terms. Upon initiation of the action potential, the subject visibly tensed including adduction of the extremities due to contraction of the pectoralis muscles and the abdominal muscles. If the degree of adduction of these extremities is used as a marker of the degree of muscle tone, then the muscle tensing could not be observed to change during the hold portion of the test. Following the test, strong muscle contractions could still be obtained by activating the stimulus and the muscle could be felt to flex strongly up to several minutes after the completion of testing. It is possible, however, that the diaphragm was not tensed using the external electrodes. Tensing of this muscular structure may play a role in stiffening the thoracic response since a tensed diaphragm may be less prone to displace inferiorly when the pressurized lungs are loaded by the hub. Future research should include attempts to quantify the degree of muscle tensing obtained and to ensure that the diaphragm is being stimulated.

CONCLUSIONS

The results of this study indicate that full tetanus of the thoracic muscle groups may not generate an appreciable increase in the effective structural stiffness of the anteriorly loaded thorax. The validity of adjustments to cadaver-based thoracic force-deflection corridors, intended to account for muscle tensing, should be re-evaluated.

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DISCUSSION

PAPER: **The Use of a Postmortem Porcine Model to Study the Effect of Muscle Tetanus on Thoracic Force-Deflection Response**

PRESENTER: ***Richard Kent, University of Virginia***

QUESTION: *Guy Nusholtz, Daimler/Chrysler*
If I read your graph correctly, it looked like it was less than a 100 newtons was the difference between–

ANSWER: This?

Q: No. I was looking at the force deflection. You passed it. Go back. Keep going. There. The dark and the light red and the dark and light blue looks like you're less than 100 newtons difference between the force. So, you'd add a 100 newtons to the cadaver responses as opposed to the 667.

A: Oh, no. These are two different subjects where the dark line is with muscular and the light line is without. So, this is Subject 1 and Subject 2. So, the affect of musculature here is about 5 newtons maybe and the difference between subjects at this point is maybe 50 newtons.

Q: That's down at the bottom. But if you go in the middle of the range, it might be a little bit more. Right here.

A: Yeah. Yeah.

Q: It's less than a 100 though.

A: That's correct.

Q: So, it's an order of magnitude different than what we–at least, in order of magnitude.

A: Something like that up here.

Q: Did you check to see any–You just chose one depth?

A: Yes. We've only tested two subjects so far.

Q: Okay. Are you planning to look at additional depths to see if there's any difference?

A: Yes. We would like to be able to answer the question: because you see a pronounced musculature effect at low deflection in the human, do you still see that in a pig? In other words, if you repeated those old human volunteer tests using the pigs, would you see the same dramatic stiffening they saw? I think the answer will be no because the musculature on the pig is different than on a human. A pig doesn't have big, thick pectoralis muscles in the middle of the chest. They're more lateral. As you know, a pig walks around on its legs. And so, we're actually loading almost directly on the pig's sternum. From the standpoint of answering the question: How much does the rib cage response change when there's musculature–the pig's a reasonable model. But in terms of interpreting low deflections in the human, it's not quite as good a model because there is this effect of the anteriorly located pectoralis muscles being flexed, which we don't mimic with the pig. So, yes, we should look at different levels, but at some point you lose the applicability of the model, I think, in terms of representing a human.

Q: And, do you have any speculation as to what is causing the hysteresis difference?

A: Not right now. I did those curves on the plane on the way here, so.

Q: Okay.

A: I haven't thought about them that much. I thought it was an interesting finding to share but at this point I can't explain it.

Q: *John Melvin, Tandelta*
Back in the early 80's when we were doing the advanced ATD project at Michigan, I was looking around at spec'ing chest response, and there's a pretty amazing series of tests that Larry Patrick subjected to himself and was published in the 25th Stapp, which he hit–He basically did the 208 calibration test on himself, and everybody was amazed that he had done this years ago, but he just published it then. But, there–He has tensed and relaxed living human–himself–and I analyzed those relative to the Kroell data and concluded exactly as you say: You shouldn't increase the stiffness because of muscle tension. It doesn't have any effect.

A: I'm glad to hear you say that.

Q: *Barry Myers, Duke*
This doesn't matter for the conclusions in this study, but it might as you sort of push along. We did some work, years ago now, stimulating the nerve to excite the muscle, and when we went to publish were criticized because by saturating the nerve, we were over driving the muscle. And, you're sort of saturating the chest. So, just as–If you get situations where muscle starts to show up as matter and you may need some alternate strategy to lighten up on your loading.

A: Yeah. This was kind of done as an ultimate test, you know: How–What's the largest effect we could see. Because if it didn't show up, we thought anything less would be–

Q: Yeah, well, in that regard, it would really buttresses your conclusion.