THE MAGNITUDE, PHASING, AND ATTENUATION OF PRESSURE WAVES IN THE AORTA DURING ABDOMINAL BELT LOADING.

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ABSTRACT

Rupture of the aorta is a leading cause of death after motor vehicle crashes, but the exact mechanisms of this injury remain unidentified. It is well documented that transient pressure waves develop within the aorta during impact loading and this increase in blood pressure has been postulated as a primary or contributory mechanism of aortic rupture. This paper investigates the *in situ* intra-aortic pressure generated during dynamic belt loading of the abdomen.
Ten juvenile swine (average weight 21.4±1.4 kg) were subjected to dynamic belt loading on the abdomen using a customized pneumatic device. Instrumentation included ultra-miniature vascular pressure transducers inserted into the distal (abdominal), thoracic, and proximal (arch) aorta to track the magnitude and propagation of the blood pressure wave generated by the loading. Belt penetration depth and rate, muscle tension, and belt location (upper and lower abdomen) were considered.
Belt penetration ranged from 35% to 68% of the unloaded abdominal depth at a peak rate of 3.5 m/s to 7.0 m/s. The average peak pressure measured in the abdominal aorta during belt loading was 847±277 mm Hg. The peak pressure measured in the thoracic aorta was on average the 69% of that in the abdominal aorta, and the peak in the aortic arch was on average 53% of the abdominal aorta. No macroscopic aortic trauma was observed in any subject.

Keywords: Abdomen, animals, pressure, safety belts, thorax.

TRAUMATIC AORTIC RUPTURES (TAR) are the cause of death in 5% to 24% of motor vehicle fatalities (Dischinger et al. 1988, Shkrum et al. 1999, Richens et al. 2003). They represent a serious threat to life, with a survival in scene of 9%, and a overall mortality of 98%. Motor vehicle crashes are the largest cause of laceration or rupture of the aortic wall (Fitzharris et al 2004, Richens et al 2003). Several studies have found that the survival time after this injury is less than 8 hours in over 95% of cases (e.g., Skrum et al. 1999). Since the majority of deaths occur prior to arrival at the emergency room, it is reasonable to assert that there is far more potential benefit in preventing TAR than in better triage and treatment of them. The first step in preventing them is a detailed and specific understanding of how they happen.

Early studies hypothesized several individual mechanisms of blunt TAR (Oppenheim 1918, Klotz 1932, Roberts et al. 1966). These mechanisms include 1) a transient rise in arterial blood pressure 2) inertial loading as the chest is accelerated or to 3) chest deformation as the sternum is displaced posteriorly relative to the spine. As independent mechanisms, however, these hypotheses fall short of explaining what is known about blunt TAR. For instance, Bass et al. (2001) performed a series of in-vitro and in-situ pressurization tests of human and porcine aortas, finding that a pressure magnitude of 102 kPa corresponded to a 50% risk of an aortic tear. However, in the tested aortas, the tear was longitudinal 50% of the time, while ruptures in the field are predominantly transverse. This indicates a fundamental difference in the stress distribution and effectively eliminates pressure as a sole mechanism in most field cases, though could be a cofactor in many cases.

Thoracic acceleration has likewise been eliminated as a sole mechanism of aortic rupture in most field cases. In an analysis of over 260 recordings of chassis acceleration during Indy race car crashes in the U.S., Melvin et al. (1998) estimated that maximum thoracic acceleration levels of approximately 90g (frontal), 180g (side), and 125g (rear) with a duration as long as 20 ms had been tolerated by the drivers with no cases of blunt TAR. This tolerance level has recently been confirmed in a more controlled laboratory setting (Forman 2008), nine high-acceleration cadaver sled tests were performed resulting in rearward x-axis accelerations up to 98 g, mid-spine accelerations up to 113 g, and chest deflections less than 10% of the undeformed chest depth. These tests resulted in no significant injuries to the aorta, just in one test an intimal iatrogenic laceration to the aorta was found.

Finally, chest deformation in the absence of aortic fluid pressure seems insufficient as a sole mechanism of blunt TAR in the field. Several studies have imposed a posterior sternal deflection with relatively low levels of thoracic acceleration and found no aortic injuries. For example, research at General Motors in the 1970s (summarized in Backaitis 1994) involved blunt impacts to cadavers of sufficient severity to cause the posterior sternum to contact the anterior spine, yet no blunt TAR were noted. Recent studies at UVA using computed tomography to study the deforming thorax have illustrated that even large lateral displacements of the heart in a static loading environment do not necessarily generate aortic injury (Kent et al. 2001). In contrast, Hardy et al. (2008) demonstrated that TAR could occur in cadavers subjected to blunt-hub impact from various directions and abdominal belt loading when the subjects' aortas were initially perfused to nominal *in vivo* fluid pressures.

Numerous studies have hypothesized that TAR is caused by a combination of fluid pressure, acceleration loading, and distraction of the heart from compression of the chest (Ben-Manachem 1981, Moar 1985, Bass et al. 2001, Richens et al. 2002). Using a finite element model of the aorta, Lee and Kent (2007) suggested that strains in the aortic wall caused by acceleration, fluid pressure, and distraction of the heart may combine in a non-linear manner when all of these loading mechanisms are applied to the heart and aorta.

Some studies have suggested that TAR may be caused by loading of the abdomen. Such loading may cause distraction of the heart through the motion of a portion of the abdominal contents into the thoracic cavity, and may cause the generation of a fluid pressure wave in the abdomen that may travel superiorly to the thoracic aorta. Voigt and Wilfert (1969) suggested that cranially-directed impact to the inferior portion of the sternum and ribcage may cause a "shovelling effect" that may cause TAR. Hardy et al. (2008) performed one test subjecting a cadaver to belt loading to the abdomen, and found that that test resulted in a partial tear of the aorta through the media layer. This paper seeks to further investigate the *in situ* intra-aortic pressure generated during isolated dynamic belt loading of the abdomen, and to observe the magnitude and propagation of the intraluminal fluid pressure wave as it travels superiorly to the thoracic aorta.

The porcine model was chosen for this study rather than a human cadaver primarily due to the rapid availability following death. Human cadavers typically cannot be collected, screened, prepped, and tested before some soft tissue degradation has occurred. In contrast, the experiments presented here were performed within minutes of death. There are, however, anatomical differences between the human and pig that limit the applicability of the study findings. Various anatomical structures of the human and porcine aorta compare very favorably, including circumference of the aortic valves (Sim et al. 2003) and overall dimensions (Kent et al. 2006). In fact, porcine aortic values are frequently transplanted into humans. Anatomical differences include the central point of coaptation, which is skewed toward the left coronary cusp in humans and toward the non-coronary cusp in swine (Sim et al. 2003). These differences probably affect blood flow in the aorta (e.g., Grande et al. 1993) and thus the biomechanics of the fluid-structure interaction. This may alter slightly the transient properties of the pressure wave generated during abdominal loading. With those limitations in mind, we believe that the overall morphologic (Dellman et al. 1991), structural, and material (cf. Bass et al. 2001) similarities between human and porcine aortas make the results of this study reasonably representative of the pressure wave propagation characteristics of human children exposed to abdominal loading.

METHODS

Ten juvenile swine (average weight 21.4±1.4Kg) were subjected to dynamic belt loading on the abdomen using a customized pneumatic device. The load was applied on two locations: lower and upper abdomen due to the different responses to loading observed (upper abdomen is composed of solid organs whereas lower abdomen is composed of hollow organs) (Rouhana 2002). All tests were performed with a loading surface initially in contact with the abdomen. Two different shapes of the displacement wave were used: ramp-hold and ramp-release. The muscle tensing was considered and intramuscular needle electrodes were inserted in order to obtain the muscle stimulation. The tests were performed at a nominal mean abdominal deflection rate of 3 m/s or 6 m/s, to reproduce the abdominal deflection rate experienced by a belted occupant in a severe crash (3 m/s) and to evaluate the influence of the deflection rate (6 m/s), because the abdomen was found to be rate sensitive in structural response (Hardy et al. 2001) and its injury criteria at a given penetration magnitude (Viano and Lau, 1989), see Table 1.

Table 1. Test configuration and independent variables for the statistic analysis.

Test	Abdominal Position	Muscle Tensing	Waveform	C max [%]	V max [m/s]	Max force applied [N]
PAC1.01	Lower	Yes	RH	45,0%	5,7	1690
PAC1.02	Lower	Yes	RH	54,0%	4,3	3444
PAC1.03	Lower	No	RH	48,0%	3,5	3143
PAC1.04	Lower	No	RH	49,0%	5,3	3423
PAC1.05	Upper	No	RH	50,0%	7,0	3972
PAC1.24	Upper	No	RR	39,0%	6,4	1947
PAC1.26	Upper	No	RR	44,0%	3,9	1945
PAC1.44	Lower	No	RR	61,0%	6,6	3285
PAC1.45	Upper	No	RR	68,0%	5,7	3349
PAC1.47	Lower	No	RH	56,0%	5,5	4245
Average				51,4%	5,4	3044
s.d.				8,6%	1,2	883

The porcine subjects were housed at the vivarium at the UVA (University of Virgina) Health System until a pig reached a target whole-body mass of 21.4 Kg. (This size of pig was chosen to approximate the abdominal dimensions and organ masses of a 6-year-old human; the procedure to identify this model is explained in Kent *et al.* 2006). The test was performed the following day. The pigs were anesthetized, weighed, endo-tracheally intubated and anthropometric measurements were then taken on the subject. To track the magnitude and propagation of the blood pressure wave from the site of abdominal loading into the thoracic cavity and to the heart three pressure transducers were inserted into the abdominal aorta, the thoracic aorta, and the aortic arch. The pressure transducers were surgically placed while the subject was under general anesthesia: the aortic arch transducer was inserted via the right common carotid artery; the abdominal and thoracic aorta pressure transducers were inserted via the left and right femoral arteries. All pressure transducer implantation was performed under the supervision of an attending physician from the department of Emergency Medicine and Pediatrics. The length of each pressure transducer that was introduced into the surgical incision was measured to approximate the final position of the transducer and the incisions were closed. (Figure 1)

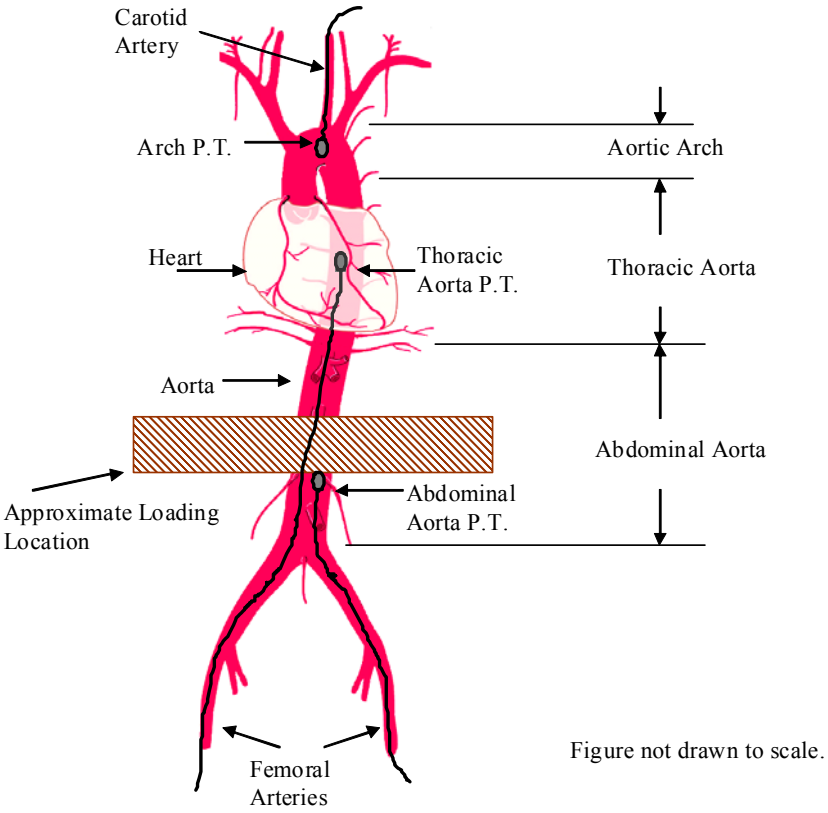
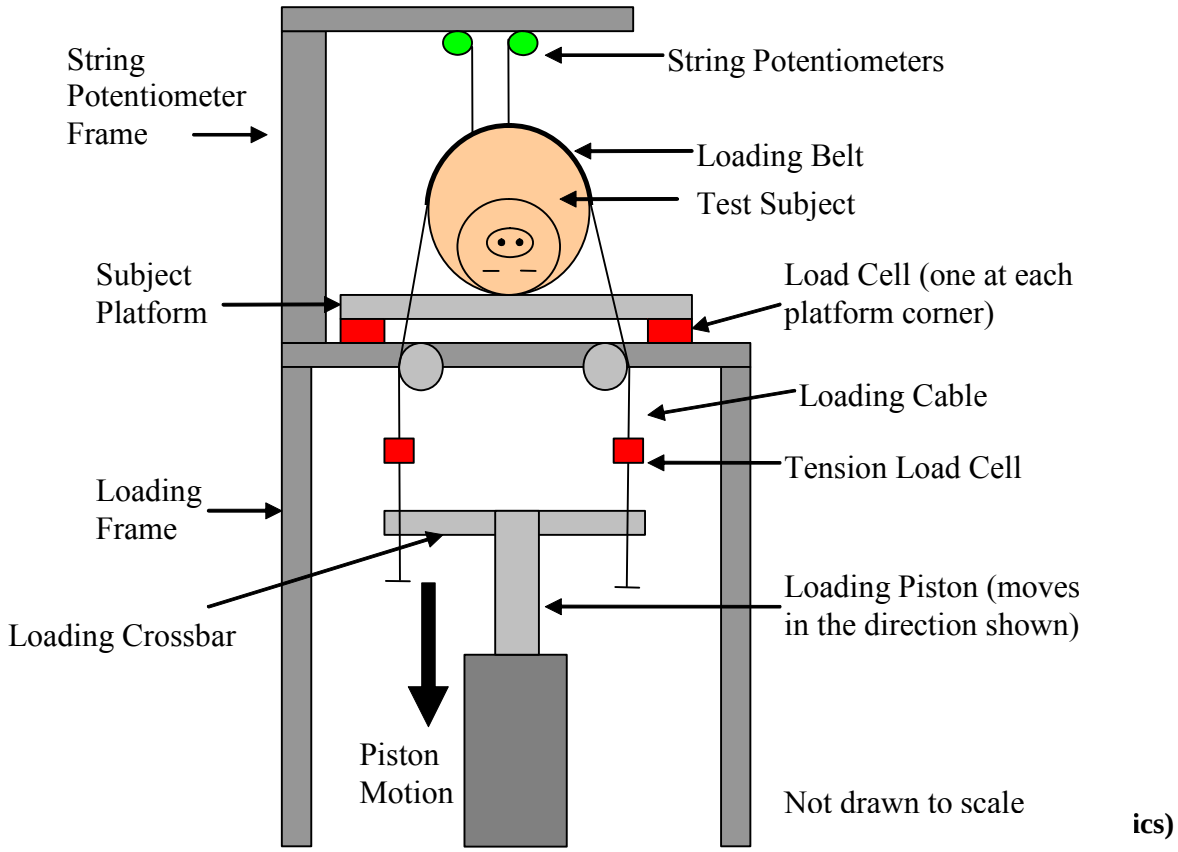


Figure 1. Pressure transducers location (Source: Center for Applied Biomechanics)

The swine were placed supine on a table frame, and a 2 ply, 5 cm wide, 30 cm long belt of an inelastic high-strength cloth was used laid across the anterior aspect of the abdomen, positioned according to the test matrix (Figure 2). A pneumatic cylinder and valve system applied belt tension at dynamic velocity. For ramp-release tests, the quick release mechanism on the pneumatic cylinder was configured to release the belt tension at the pre-set point of maximum abdominal penetration. For the ramp-hold tests, the quick-release mechanism was disabled, so the piston held the maximum abdominal deflection for 120 seconds. The abdominal depth of the subject was measured at the belt location, and this measurement was used for all subsequent calculations. The belt was preloaded to a nominal value of 10 N to ensure the belt was initially in contact with the anterior surface of the abdomen at the beginning of the test to impose a "non-impact" initial condition. Four intramuscular needle electrodes were inserted for tests involving active muscle stimulation (only lower abdominal tests): 2 on the right and 2 on the left, each 2 cm above or below the belt at the mid-nipple line. Immediately prior to the start of the test, the subject was euthanized, the lungs were filled to maximum physiologic capacity, and the endotracheal tube was clamped off. The tests were performed immediately after respiratory and cardiac activity had ceased. The test apparatus and methodology is discussed in greater depth in Kent *et al* 2006).



ics)

Instrumentation included miniature vascular pressure transducers (Millar® Instruments, Model SPR-524) to measure the *in situ* fluid pressure. A Phantom® high speed digital imager recording at 3,000 frames per second was used to capture a detailed, lateral view of each test. The video was used to track the position of the belt in time, and provided a displacement/time history for the midline belt motion. To provide a redundant measure of abdominal displacement, a string potentiometer (SpaceAge Controls Model L-01) was placed at the belt mid-line. The tension in the loading belt is measured with two tension load cells (Sensotec Model 31) installed in series with the belt loading cables.

Electronic signals from sensors mounted to the test fixture and to the subject were recorded at 10,000 Hz and digitized by a 16-channel Dewetron, model DEWE-2010, data acquisition system. The post-test processing and plotting of the digital data was performed using DADisp 2002 (DSP Development, Newton, MA) and Excel (Microsoft Corp., Redmond, Wash.). Each of the displacement, acceleration, and force signals was debiased using pre-trigger data and digitally filtered through a low-pass 8th-order Butterworth filter with a 200-Hz cutoff frequency.

The belt midline was tracked frame by frame during the loading phase for each test using the Phantom point tracking software (version 6.07). This data was then plotted against the data obtained with the string potentiometer and both signals were synchronized temporally by aligning the first positive deflection which indicated the onset on belt movement. Once “time zero” was established for the video displacement data, it was filtered to CFC 180 (BWfilter software package). The displacement determined from the video tracking was considered to be the most accurate measure of the actual belt penetration into the abdomen.

STATISTICAL ANALYSES: A correlation matrix was performed involving all test variables: the abdominal load position, the existence of muscle tension, the type of applied waveform, the test

velocity and the abdominal deformation. The measured pressures in the abdominal aorta, thoracic aorta, the aortic arch were also included, as was the ratio between the pressure measured in the aortic arch and the pressure measured in the abdominal aorta. The correlation matrix indicates the strength and direction of a linear relationship between two pair of variables; p values were also calculated to determine the statistical significance.

After the correlation matrix, several linear multivariate regression models were performed for the different dependent variables (different pressure measurements depending on the experiment test matrix), to determine the influence from the 6 independent variables (load position, waveform, muscle tension, high or low speed, high or low compression, and high or low applied force) variables.

Due to the small number of tested specimens, the continuous variables maximum compression, test velocity and maximum force applied were transformed in dichotomous variables, to increase the model robustness. The thresholds were respectively 50% compression, 5 m/s test velocity and 2 kN applied force (Table 1).

Multivariate linear regression models were developed for each of the 3 dependent variables (abdominal, thoracic, and aortic arch pressures). In each model we evaluated the role of all 6 dependent variables (i.e., experimental parameters as listed in Table 1). Should there have been any truncated values in the experiment, sensitivity analyses were performed to evaluate to role of such cases in the models.

RESULTS

ABDOMINAL COMPRESSION TESTS: Tests were completed successfully on all 10 pigs. The pressure (mmHg) curves in the abdominal aorta, thoracic aorta, and aortic arch were the primary data of interest.

The test was considered started after the belt load reached 100N, belt penetration ranged from 39% to 68% of the unloaded abdominal depth at a peak rate of 3.5 m/s to 7.0 m/s, and the belt force applied in the test ranged from 4245 N to 1690 N. The average peak pressure measured in the abdominal aorta during belt loading was 848±330 mm Hg. The abdominal aortic pressure exceeded 1000 mmHg in three cases (and three more truncated signals). The peak pressure measured in the thoracic aorta was the 586±128 mm Hg , The peak pressure measured in the aortic arch was the 448±122 mm Hg. The peak pressure measured in the thoracic aorta was on average the 69% of that in the abdominal aorta, and the peak in the aortic arch was on average 53% of the abdominal aorta. (Table 2)

The abdominal pressure time history is presented in the Figure 3, the pressure increased approximately linearly from 0.005s to 0.02s, before exhibiting a concave-down characteristic that was smooth in most cases. However this characteristic was not homogeneous, some pressures exhibiting a more gradual pressure drop. The peak pressure varied substantially from 1427 to 541 mmHg according to the test configuration, obtaining its peak between 0.018s and 0.026s

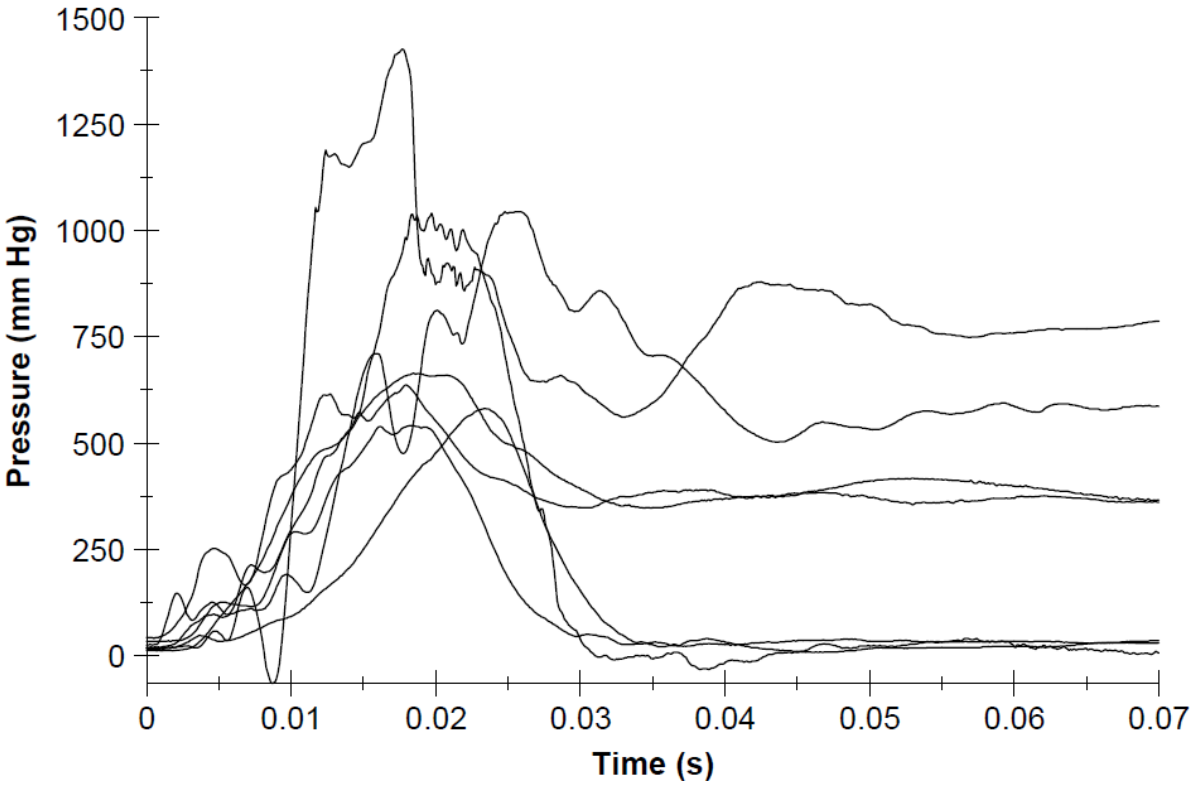


Figure 3. Abdominal aorta pressure.

The thoracic pressure time history is presented in the Figure 4, the pressure increased approximately linearly from 0.015s to 0.023s, before exhibiting a concave-down behaviour, more pronounced than the abdominal pressure. This characteristic was quite consistent, but only five tests were recorded properly. The peak pressure varied substantially from 710 to 393 mmHg, obtaining its peak between 0.018s and 0.026s

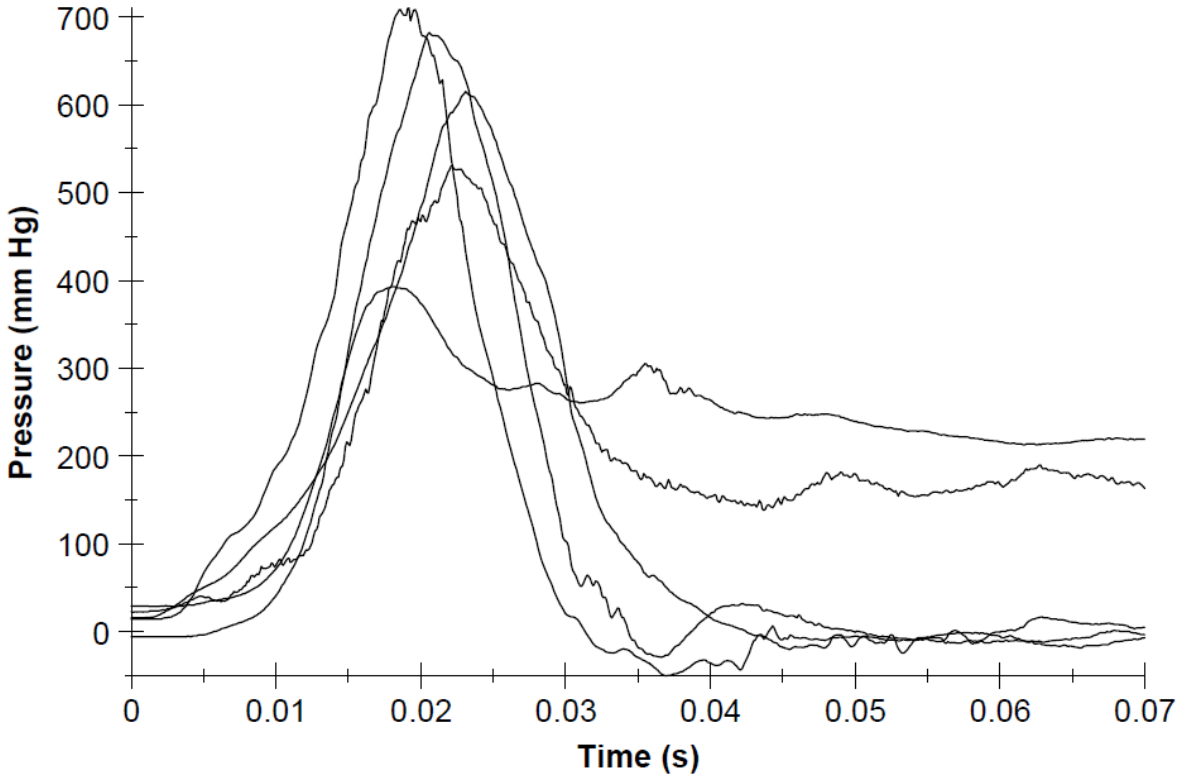


Figure 4. Thoracic aorta pressure.

The pressure recorded in the aortic arch is presented in the Figure 5 in front of time, this pressure increased approximately linearly from 0.01s to 0.033s, before dropping. However this characteristic was not consistent across tests; some pressures exhibited a more gradual pressure drop. The peak pressure varied substantially from 641 to 293 mmHg according to the test configuration, obtaining its peak between 0.018s and 0.037s

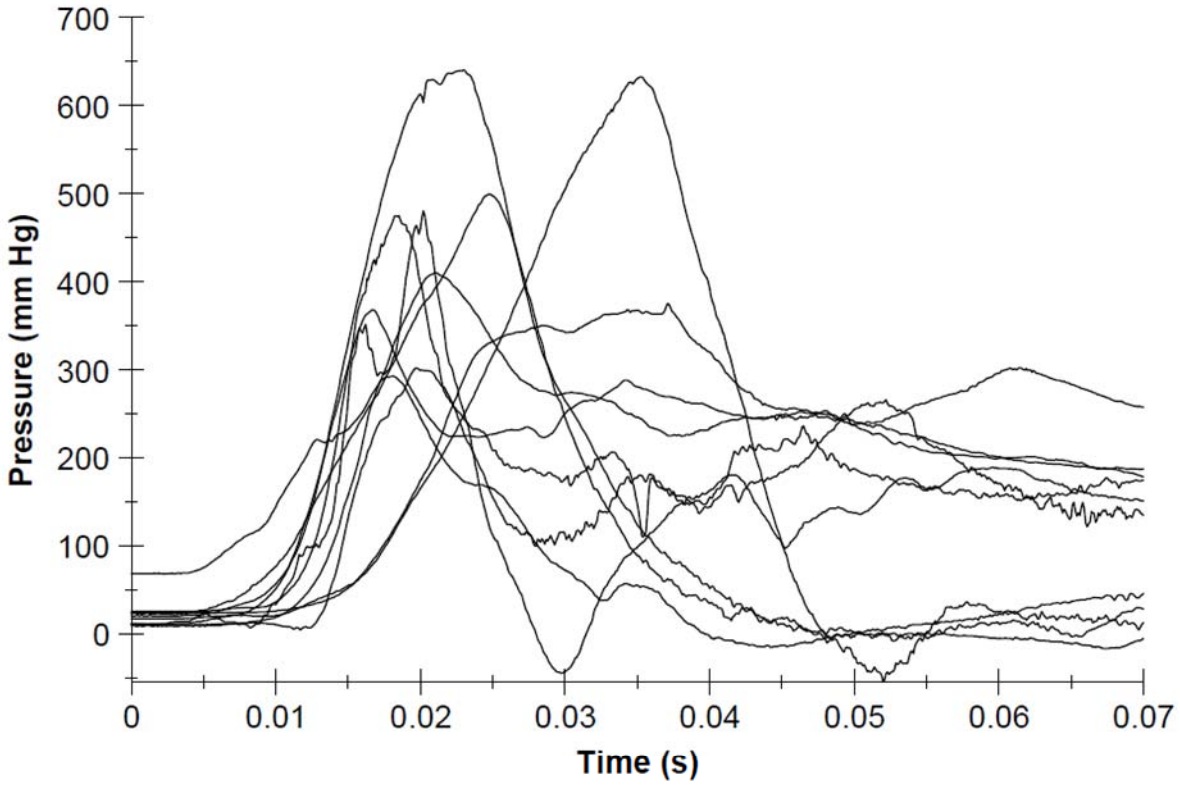


Figure 5. Aortic arch pressure.

After the tests a detailed necropsy was performed immediately following each test to identify and document injuries according to the AIS scale (1990 version, 1998 Update). No macroscopic aortic trauma was observed in any subject.

STATISTICAL ANALYSIS

Following the described experimental methodology, the measured pressure peaks obtained in the different referred locations of the pigs' aortas are presented in the table 2. An indirect value is also included, the ratio of the peak aortic arch pressure to the peak abdominal aortic pressure. Due to experimental problems just 5 test lead to satisfactory data in the thoracic aorta, thus this variable was dropped from the analysis. Three pressure signals from the abdominal aorta were truncated exceeding the sensor capacity.

Table 2. Experimental pressure maximum values, dependent variables for the correlation matrix.

Test	Abd Aorta P max [mmHg]	Thor Aorta P max [mmHg]	Arch P max [mmHg]	Arch / Abdominal [mmHg]
PAC1.01	636	393	368	57,9%
PAC1.02	999*	-	376	37,6%*
PAC1.03	1044	-	410	39,2%
PAC1.04	1427	-	302	21,2%
PAC1.05	663	-	481	72,5%
PAC1.24	541	710	293	54,2%
PAC1.26	581	615	500	86,0%
PAC1.44	1040	682	641	61,6%
PAC1.45	1002*	-	633	63,2%*
PAC1.47	2000*	530	475	23,8%*
Average	848	586	448	55,2%
s.d.	330	128	122	23,1%

* pressure signal clipped

\- pressure not aquired

CORRELATION ANALYSIS OF EXPERIMENTAL DATA: According to the correlation matrix, presented in the Table 3, the abdominal load position was positively correlated ($R^2 \geq 0.5$, see bold fonts) with the maximum pressure in the abdominal aorta, and inversely with the ratio pressure aortic arch/ abdominal aorta. This was the only variable significantly related to abdominal aortic pressure. The muscle tensing during the test did not show any influence on the rest of variables, just a small correlation with the pressure in the thoracic aorta, but as only 5 values were obtained in this aorta region, the statistical power may not be sufficient. The high compression was strongly positively correlated ($R^2=0.777$) with the maximum pressure measured in the aortic arch, and this result was statistically significant ($p=0.008$). The test speed velocity was not correlated with the rest of variables, and the maximum force applied by the seat belt was positively correlated ($R^2=0.706$) with the maximum pressure measured in the abdominal aorta; this result was also statistically significant ($p=0.022$).

Table 3. Correlation matrix for the experimental results (bold indicates $R^2>0.5$ or $p<0.05$).

	C max [%]	V max [m/s]	Max force Applied [N]	Abd Aorta P Max [mmHg]	Thor Aorta P max [mmHg]	Arch P Max [mmHg]	% P arch/ Abd. aorta
C max	1.000						
V max	0.169 0.638	1.000					
Max force applied	0.612 0.059	0.157 0.663	1.000				
Abd Aorta P max	0.444 0.198	-0.096 0.790	**0.706** **0.022**	1.000			
Thor Aorta P max	0.032 0.958	0.241 0.695	0.089 0.886	-0.159 0.797	1.000		
Arch P max	**0.777** **0.008**	0.196 0.586	0.308 0.386	0.079 0.828	0.216 0.727	1.000	
% P arch/ abd. aorta	-0.072 0.842	0.186 0.606	-0.454 0.187	**-0.777** **0.008**	0.228 0.711	0.468 0.171	1.000

A multivaried regression using the maximum abdominal aorta pressure as the outcome variable and the 6 independent variables as regressors shows that only muscle tension (p=0.046), the wave form (p=0.078), and maximum abdominal compression (p=0.058) exhibit a statistically significant (although sometimes only weakly so) relationship. These effects can be observed in the Table 4.

Table 4. Linear regression for Abdominal aorta maximum pressure in front of the independent variables (bold indicates p<0.05) R^2=0.91.

Abdominal aorta P max	Coefficient	P> I t I	95% Conf. Interval	
Abdominal Position	335,0	0,199	-314,9	985,0
Muscle Tensing	-1009,5	**0,046**	-1986,8	-32,2
Wave form	792,3	0,078	-162,8	1747,4
C max [%]	-833,8	0,058	-1719,1	51,4
V max [m/s]	29,5	0,869	-495,3	554,0
Max force applied [N]	517,6	0,212	-523,3	1558,6

Removal of experiments with the truncated values resulted in less of this significant relationship, possibly due to sample size problem.

The multivariate regression focused on the maximum thoracic aorta pressure assessing the effect of the 6 independent variables was not possible to be done, due to the lack of statistical power of the 5 values. No statistically significant relationship was found for the aortic arch maximum pressure or the ratio between the aortic arch and the abdominal aorta.

DISCUSSION

As intent to isolate the multiple physical events that occur simultaneously stressing the aorta during a car accident, an experiment loading only the abdomen was performed, and the experimental pressure curves are presented.

Abdominal seat belt loading generates an intra-aortic pressure wave that travels through the aorta and is not completely attenuated before reaching the arch. In this study, the pressure observed in the aorta exceeded the pressure limit reported in some recent studies performed *in vitro*, defined as 900 mmHg for 50% risk of failure for subjects below 68 years, or 1200 mmHg as ultimate pressure, extracted from the Weibull injury risk proposed curve (Bass 2001), but no damage was observed when 1421 mmHg were measured. Previous studies have documented in-phase extra-aortic pressure waves, which mitigate the total change in pressure across the in situ aorta when the mechanism of pressure generation is external loading (Forman et al. 2008).

The peak pressure measured in the thoracic aorta was on average the 69% of that in the abdominal aorta, and the peak in the aortic arch was on average of 53% of that in the abdominal aorta. Thus, the peak value of the pressure was attenuated approximately 50% from the abdominal aorta to the aortic arch. The correlation matrix shows a large influence of the belt position (p=0.021) for this pressure ratio, if the belt is located upper in the abdomen the abdominal aorta reduces its pressure, the pressure in the aortic arch it is increased, and subsequently the ratio arch aortic pressure/abdominal pressure is increased.

The high compression was strongly positively correlated R^2=0.777 with the maximum pressure measured in the aortic arch, and this result was statistically significant p=0.008. The test speed velocity was not correlated with the rest of variables, and the maximum force applied by the seat belt

was positively correlated R^2=0.706 with the maximum pressure measured in the abdominal aorta; this result was also statistically significant p=0.022.

These results suggest that abdominal loading by a seatbelt can cause considerable fluid pressures in the thoracic aorta that may contribute to the occurrence of TAR in motor vehicle collisions. The extent to which this pressure contributes to TAR is not currently understood, but this study suggests that aortic pressures from abdominal loading should at least be considered in future investigations of TAR in simulated vehicle collisions.

There are several important limitations to this study. The limited statistical power available using 10 subjects may have prevented the detection of other relationships, to minimize this limitation the independent variables were treated as dichotomic in the statistical analysis. Furthermore, while most of the abdominal aorta and aortic arch pressures are considered reliable, several of the measured thoracic pressure tranducers failed during the test. Further, placing the pressure tranduscers at consistent positions *in vivo* was difficult, since the insertion of the transducers was essentially done blindly. Lastly, although the porcine model has been the model of choice for several previous studies of the biomechanics of TAR (e.g., Bass et al. 2001) the anatomical differences between pigs and humans are also a limitation.

CONCLUSIONS

The effect in the aorta due to an isolated abdominal load has been presented, then animal have been performed obtaining the different time pressure curves for the abdominal aorta, thoracic aorta and the aortic arch, the pressure exceeds without damage the maximum pressure reported in the literature.

The peak value of the pressure was attenuated approximately 50% from the abdominal aorta to the aortic arch, this effect can't be negligible. The statistical analysis shows a large influence of the belt position for this pressure ratio, and the high compression was strongly positively correlated with the maximum pressure measured in the aortic arch.

More research have to be performed to understand completely the pressure load in the aorta during a motor vehicle collision event.

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