

Blunt Traumatic Aortic Rupture and the Aortic Response to High Speed Impact

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Introduction

You always hear about the pitfalls of driving. Mentions of fender-benders, DUIs, and road rage saturate the airwaves. All this talk of danger and even the possibility of death, and we never stop to ask ourselves: what actually kills us? Statistics will tell you that blunt force trauma to the head is the leading cause of death. This is understandable, but the second leading cause of death is something of a mystery to most: Blunt Traumatic Aortic Rupture (BTAR) [1].

A condition classified as the rupture of the aorta (the main artery that acts as the exit for blood from your heart) due to blunt force trauma, BTAR causes 19% of all fatalities in auto accidents [1] [2]. Yet, oddly enough, a large number of these induced deaths happen after the patient has been taken to hospital due to the difficulty of diagnosis. Even with all the medical technology we have available to us, 30% of people who survive to reach hospital die within 6hrs, and 50% die within 24hrs [3]. To make matters worse, we are not exactly sure why BTAR occurs. While the general cause for BTAR is understood to be high-speed impact, the physiological causes are scarcely understood due to the clinical and ethical constraints of human testing for such a condition [4]. As such, we have decided to develop a method for analyzing the response of blood (pressure, flow rate, etc...) in the human aorta during high speed collision. Working under the hypothesis that high speed impact will cause a large increase in pressure due to the resulting forces and possibly cause rupture, we hope that this system, dubbed OSCAR (Ongoing Search for Counteracting Aortic Rupture), will allow us to gain insight into one of the possible causes of BTAR, as well aid in the possibility of preventing it.



Figure 1: Synthetic Aorta



Figure 2: 3D Printed Rib Cage

Materials and methods

The OSCAR system consists of a cart carrying an experimental synthetic aorta with an attached heart valve (mechanical or porcine). The synthetic aorta is anatomically correct, and is surrounded by a 3D printed ribcage to allow for a more realistic recreation of real life conditions. Water is pumped through the system continuously (in place of blood). The internal conditions of the aorta are initially governed to produce an approximate flow rate and pressure waveform consistent with the average adult (5 L/min and 120/80 mmHg respectively). The cart is pulled along a track using a motor and is brought up to a speed of approximately 8 km/h. After reaching a pre-selected point along the track, a sensor (photodiode) cuts power to the motor, and the cart enters a deceleration phase up until the moment of impact with our "wall" (a shock absorber).

An electronic setup using a pressure sensor, flow meter, speed sensor, and high speed camera, allows us to continuously acquire data throughout the experiment that can then be analyzed using LabView (a data acquisition and analysis software). This data allows us to determine the effects of high speed impact on the fluid dynamics of the human aorta.

Results

Our results showed a significant increase in pressure during impact compared to at rest. On average, the peak pressure was found to be 250 mmHg, or a bit more than double the normal "high pressure" (systolic pressure) of the heart. The results shown were acquired while using a porcine valve, similar results were seen with a mechanical valve.

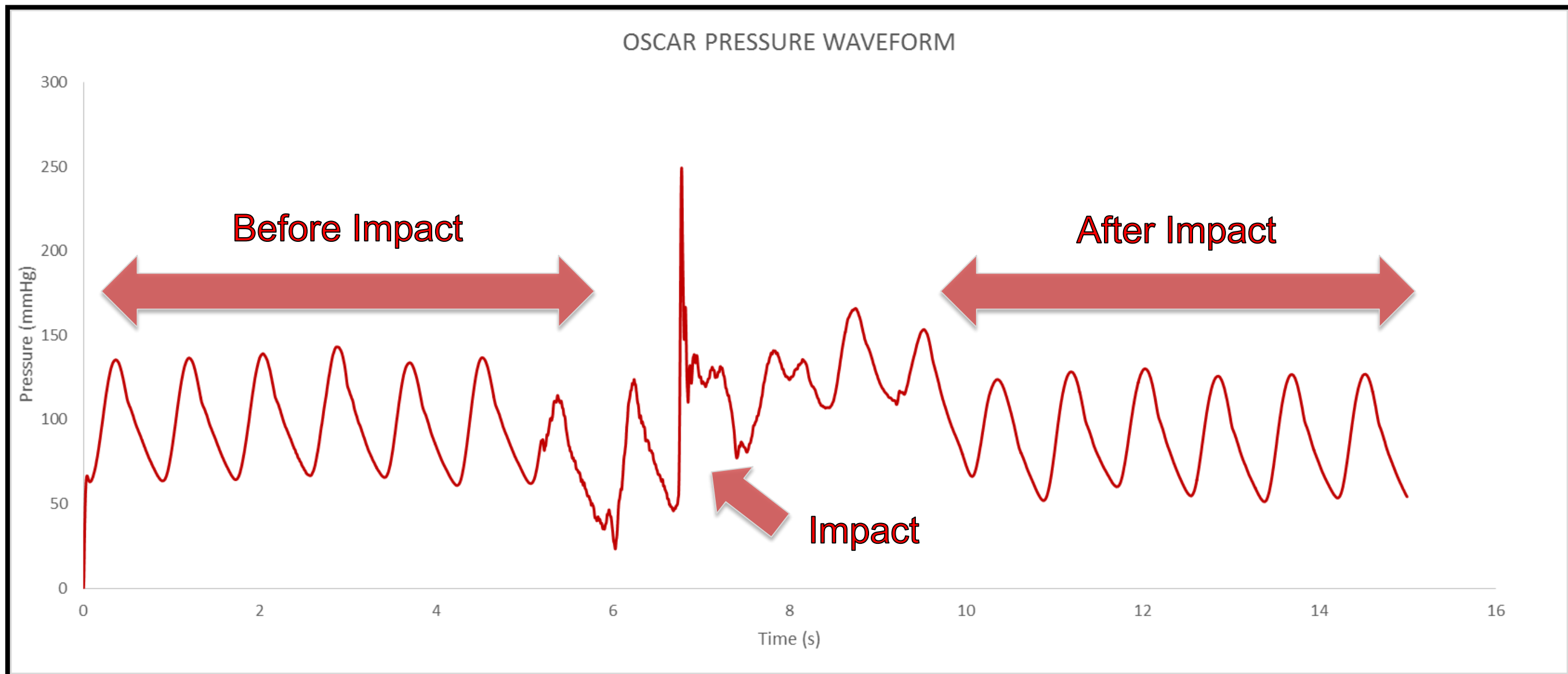


Figure 3: Oscar Results

There is a noticeable dip in the waveform (figure 3) at the 5 second mark. This corresponds to the moment that the cart begins accelerating. As we approach 7 seconds, the cart impacts with the shock absorber and a massive spike is recorded. After approximately 10 seconds (4 seconds after impact), we can clearly see that the pressure waveform returns to normal.

These results prove that high speed impact does have an effect on the regular pressure waveform of the heart. This significant spike in pressure can have a multitude of effects on the heart's mechanics, such as increased stress in the aortic wall or increased stress on the aortic valve (bicuspid valve).

To observe the cause of this pressure spike, we analyzed captures (Figures 4 and 5) from a high-speed camera operating at 2000 fps (frames/second). The massive amount of deformation in the aorta is the clear cause of this huge increase.

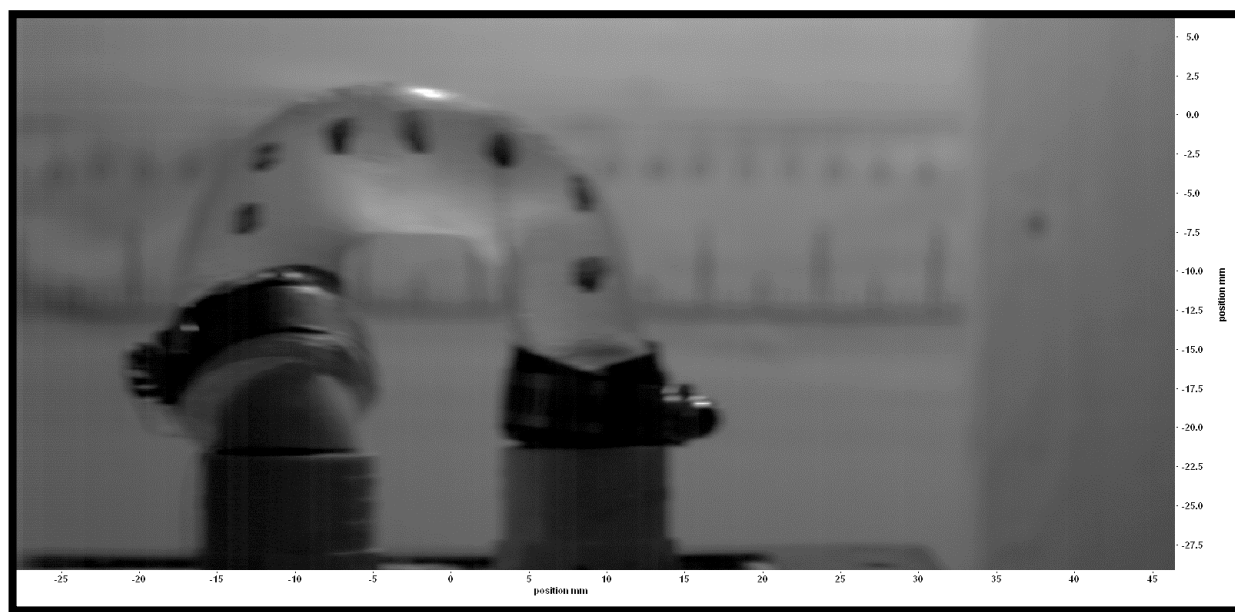


Figure 4: Aorta Pre-Impact

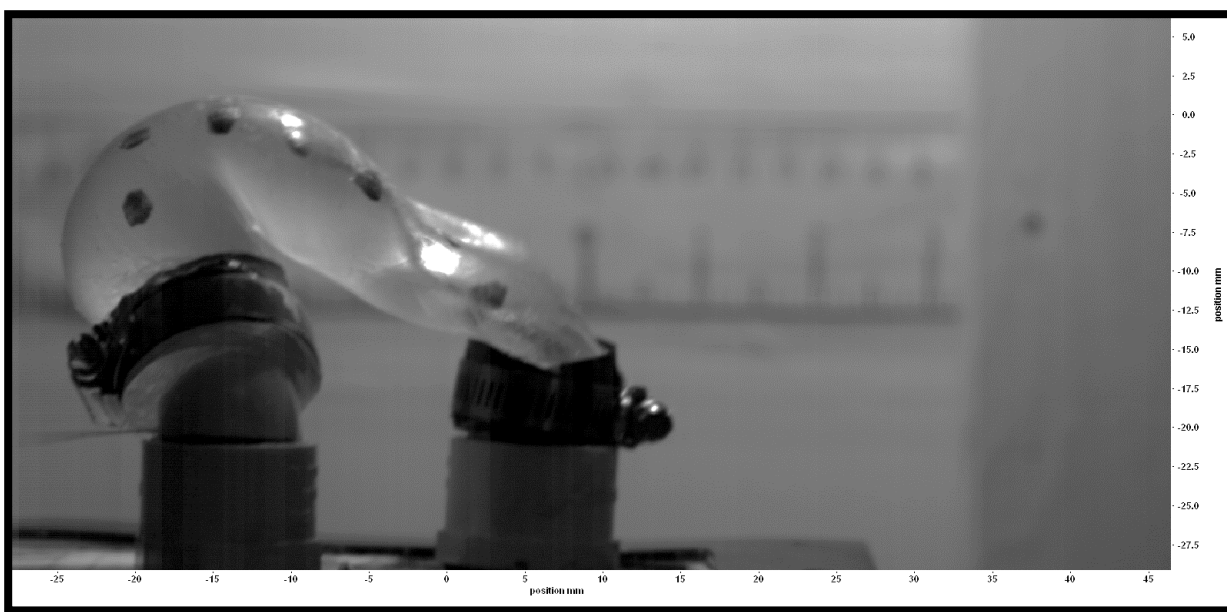


Figure 5: Aorta at Impact

Conclusions

Though our hypothesis was verified, and a significant pressure spike was recorded (120 mmHg to 250 mmHg), we are unable to say with any certainty that the pressure response is the sole reason for BTAR.

According to the literature, the threshold for rupture of aortic tissue is at a minimum 1.58 MPa (approximately 11 800 mmHg) [5]. Though these results are a product of biaxial stress testing, a different mechanism of rupture than ours, they are still a useful guideline for the strength of human aortic tissue. Furthermore, these results are orders of magnitude higher than our value of 250 mmHg, and this allows us to deduce that though our pressure response does have an effect, it should not be considered a standalone mode of failure.

Our next step is to improve the system in such a way as to gain even more accurate results. This will be done by:

- Adding an accelerometer to the system.
- Using Particle Image Velocimetry (PIV) to analyze fluid flow at impact.
- Undergoing tests with different synthetic aortae to verify results.
- Modification of the system to allow for different aorta configurations (frontal vs side impact)

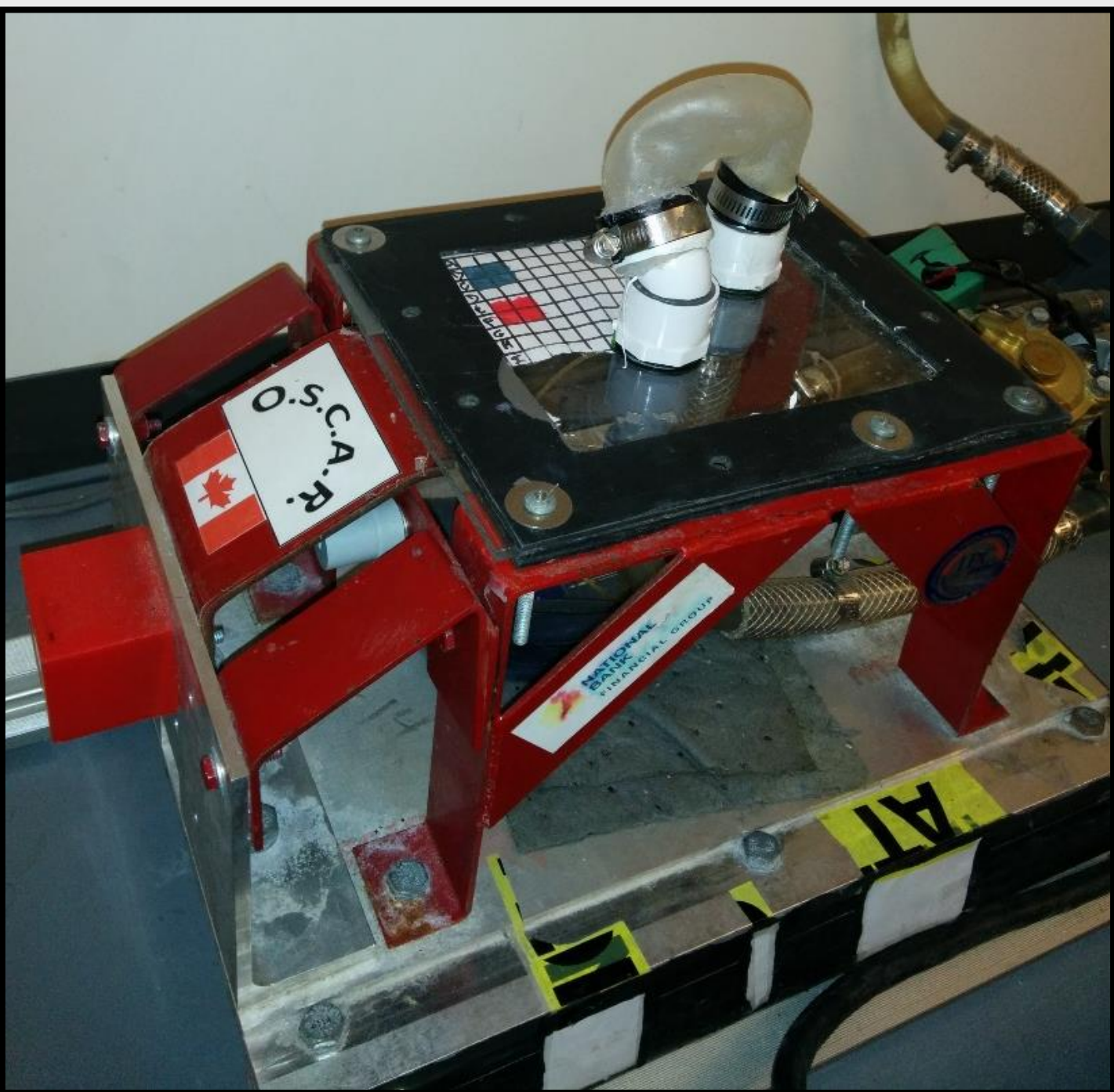


Figure 6: The OSCAR System

Acknowledgments

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