Haemodynamic Disturbances Along the Aorta and the Resulting Deformations of the Aortic Arch Following Deceleration

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I. INTRODUCTION

Blunt traumatic aortic ruptures (BTAR) are the second cause of fatalities related to road accidents [1]. Reference [2] showed that 94% of BTAR occurs in the peri-isthmic region, which is the weakest part of the thoracic aorta [3][4]. These ruptures are characterised by a transverse tearing of the aortic wall [5] and mostly occur above the arterial ligament that connects the aorta to the pulmonary artery. The mechanisms leading to ruptures have been discussed for many years [3]. These ruptures can occur when the body undergoes an abrupt change in speed due to inertial loading [5]. Owing to the experimental complexity, and to injury often not being reproduced experimentally, none of the previous studies have been able to conclude on the actual causes that lead to aortic injuries [6]. Among the various mechanisms, the present work focuses on the effect of a blood pressure wave on the aortic wall. According to [6], a critical overpressure at the level of the aortic isthmus can be generated by two physical phenomena involving either a mass displacement of blood or a pressure wave propagation. These haemodynamic phenomena may be triggered by an abrupt change in body velocity during an impact. This study focuses on the influence of such abrupt decelerations on the haemodynamic within the aortic arch and the potential development of local overpressure and local high strain in the aortic wall.

II. METHODS

An experimental setup designed to reproduce a cardiac pulsatile blood flow and to induce high-energy loadings was developed. The objective was to characterise the aortic behaviour by applying a significant transient loading, representative of a deceleration pulse [7], and accounting for fluid–structure coupling. To achieve this, porcine cardiovascular systems (CS) (heart, lungs and vessels) to preserve a complex anatomical structure were mounted on a support designed to maintain a physiological configuration: the carotids and distal end of the aortas were fixed on the support, and the heart was positioned to preserve the natural curvature of the aorta.

The aortas were instrumented with five pressure sensors placed every 5 cm from the near first carotid to the descending aorta to record the transient pressure wave (@10kHz). A fine and irregular speckle pattern was applied to the aortic arch for digital image stereo-correlation using three high-speed cameras (@1kHz) to measure the wall displacements. The principal Green Lagrange strains were then computed (VIC3D software).

A human ascending aorta pulsatile flow with a blood-mimicking fluid (60% water-40% glycerol) was introduced through the left ventricle. Repeated pulsations were applied, then, a sudden deceleration of the support was imposed on the base of the support. The deceleration was applied at two different instants corresponding to the diastolic and systolic physiological pressures. Three different deceleration magnitudes were tested for each instant (100, 200 and 300 Gs). They were chosen to induce a significant hemodynamic response to disturb the fluid pressure and the deformation at the aortic arch. The displacement of the aortic wall and the evolution of the pressure along the aorta were measured during five cycles before the impact and three cycles after. Each impact condition was repeated on five different cardiovascular systems (13 CS in total).

III. INITIAL FINDINGS

Applying a deceleration to the thoracic aorta generated a transient pressure wave that propagates backwards to the aortic root. This transient pressure wave (P_{impact}) was superposed to the physiological pressure wave. The GL strain wave, E_1, in the radial direction (1st principal direction) was found to be greater than the GL strain E_2 in the longitudinal direction (2nd principal direction). This pressure wave induces a deformation wave that occurs with a delay relative to the pressure wave. Figure 1 represents an example of a test with a deceleration pulse of 300 Gs applied at the instant of the systolic pressure. Similar results were found for the other test conditions.

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Figure 2 shows the mean (over the tested aortas) variation of the maximum of the transient pressure wave P_{impact} compared to the maximum of the physiological pressure ($P_{systolic}$) at different positions of the pressure sensors along the aorta for the different loading conditions. The pressure wave propagated backwards along the aorta, with a decrease at the exit of the aortic arch (with respect to the direction of the flow, pressure sensor 2) followed by a further amplification close to the aortic root (sensor 1). The amplitudes of the transient pressure wave increased with the level of deceleration. The instant when the deceleration was applied also had an importance, 2 shows that the pressure peak was higher at the systole compared to the diastole. This difference was less pronounced for higher levels of deceleration. Moreover, the peak of the transient wave was found lower than the physiological systolic pressure depending on the sensor position and the instant of the deceleration.

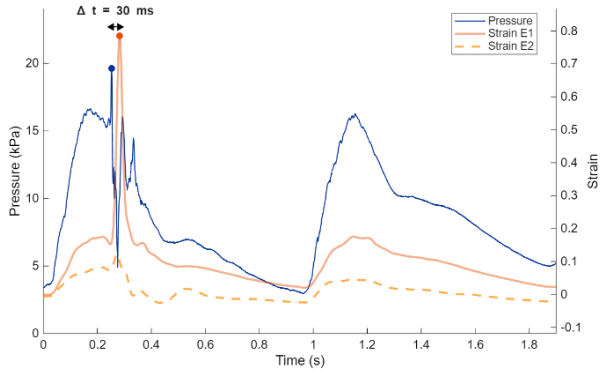

Figure 1: Temporal evolution of pressure and principal Green Lagrange strain at the aortic arch submitted to a deceleration pulse with a magnitude of 300Gs applied at the instant of the systolic pressure.

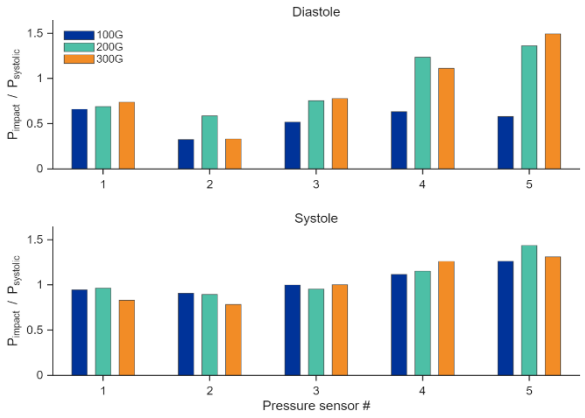

Figure 2: Maximum normalised pressure ($P_{impact}/P_{systolic}$) measured at five locations along the aorta from the aortic root (sensor 1) to the end of the descending aorta (sensor 5) for the three deceleration levels (100G, 200G, 300G), under diastolic and systolic conditions.

IV. DISCUSSION

These preliminary results show that the application of a deceleration to the thoracic aorta generates a transient pressure wave propagating along the aorta. Higher pressure peaks are observed when deceleration occurs during systole compared with diastole. However, analysing only the peak of the transient pressure wave does not fully describe the haemodynamic behaviour following the impact. In particular because the transient wave is quickly damped and dissipated. A more in-depth analysis of the spatiotemporal evolution of this transient wave is necessary and is currently ongoing to better understand the effect of this pressure wave, particularly on wall deformation at the level of the aortic arch, which is the area of structural weakness and the primary site of blunt traumatic ruptures [2]. These elements show that the timing of deceleration within the cardiac cycle has an important role in the resulting hemodynamic response.

In addition to the transient pressure wave, a transient strain wave is observed at the time of deceleration; the maximum peak strain appears after a time delay, highlighting that the structural response of the aortic wall is not instantaneous. This shows that there is a coupling between the fluid dynamics that induce the wall loading and wall deformation, likely governed by the viscoelastic response of the aortic wall. The anisotropic properties of the aortic wall are also highlighted by the differences in amplitude between the two principal strains (E_1 and E_2).

V. REFERENCES

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