AORTIC ARCH ANGULATION INCREASES BLOOD PRESSURE IN AN EX-VIVO PORCINE MODEL

Ariel F. Pascaner (1), Tim Mandigers (2)(3), Martina Schembri (1), Sonja Jelic (1), Maurizio Domanin (2)(4), Ferdinando Auricchio (1), Santi Trimarchi (2)(4), Michele Conti (1)

1. Civil Engineering and Architecture Department, Università degli Studi di Pavia, Pavia, Italy; 2. Department

of Vascular Surgery, Fondazione IRCCS Cà Granda Ospedale Maggiore Policlinico, Milan, Italy;

3. Department of Vascular Surgery, University Medical Centre Utrecht, Utrecht, The Netherlands;

4. Clinical and Community Sciences Department, Università degli Studi di Milano, Milan, Italy.

Background

It is well established that increased central blood pressure has major effects on cardiovascular risk and disease manifestation. However, the relationship between aortic geometry and hemodynamics has not been completely elucidated yet. This study explored the impact of severe aortic arch angulation on blood pressure in an *ex-vivo* porcine model.

Materials and methods

We harvested the aortas of 20 pigs (commercial hybrid, near 12 months old, 160 to 180 Kg), and prepared them by removing the surrounding tissue and clamping all ramifications between the aortic root and the celiac artery. The animals were raised and sacrificed for commercial use only at a local abattoir and not for the purpose of this study. Therefore, authorization of a local ethics committee was not requested.

Two cases of aortic arch angulation were studied; namely Type I and Type III arch [1]. To emulate these arch geometries the aortas were placed into custommade guides, which were virtually designed and subsequently 3D-printed (Figure 1). The aortas were connected into a mock circulatory loop endowed with a pulsatile pump [2]. For each aorta, the experimental workflow was the following. We first connected the aorta using the Type I arch guide and configured the circuit parameters to achieve: 4.5-5.5 L/min of cardiac output at 75 beats per minute and 70-80 mmHg – 115-125 mmHg of diastolic and systolic pressure, respectively. Pressure and flow waveforms were registered at 1 KHz for at least 20 seconds (Honeywell



Figure 1: (A) and (B): CAD models of the Type I and Type III guides, respectively. (C) and (D): Example of aorta placed under the Type I and Type III arch guides, respectively.

40pc015g series, Morristown, NJ, U.S.A. and Em·tec, Finning, Germany, respectively). After the acquisition, we placed the aorta into the Type III arch guide and reconnected it to the circuit. To emulate the compensatory mechanisms of the heart, after changing the geometry, the stroke of the pump was adjusted to reach the same cardiac output as under the Type I geometry. Similarly, pressure and flow were registered using the Type III arch guide.

For each experiment flow and pressure data were automatically separated into individual heartbeats for post-processing. For each heartbeat, we extracted: mean flow (Q), diastolic (Pd), systolic (Ps) and mean (Pm) pressures and then averaged among the available cardiac cycles. Data were compared with paired t-tests after normality confirmation using Shapiro-Wilk test.

Results

Table 1 shows the obtained values of mean flow and pressures for the Type I and Type III configurations. Mean flow was similar for both types of aortic arch consistently with the experimental workflow. All pressures resulted higher in a Type III arch with respect to the Type I arch (p < 0.001).

Parameter	Type I	Type III	р
Q [L/min]	4.73 ± 0.41	4.74 ± 0.39	0.14
Pd [mmHg]	73.6 ± 1.6	82.5 ± 5.7	< 0.001
Ps [mmHg]	122.0 ± 2.2	134.1 ± 11.1	< 0.001
Pm [mmHg]	89.7 ± 1.5	98.8 ± 6.5	< 0.001
Table 1. Results of mean flow (O) diastolic (Pd)			

Table 1: Results of mean flow (Q), diastolic (Pd), systolic (Ps) and mean (Pm) pressures for Type I and Type III arch geometries.

Conclusion

Our study showed that an increased angulation of the aortic arch results in higher blood pressure levels, which is a main factor for the progression of cardiovascular disease. Further studies are needed to translate these observations to *in-vivo* human arteries and to assess its impact on cardiovascular risk.

References

- 1. S. Madhwal et al, J Invasive Cardiol, 20(5), 2008.
- 2. H. de Beaufort et al, Ann Vasc Surg, 43:302-308, 2017.

