ular shape. However, the largest source of error in the compliance estimation was the accurate measurement of the change in vessel diameter. For a cross sectional image taken perpendicularly to the vessel direction, a change in vessel area over the cardiac cycle would be properly displayed in the cycle reconstruction. An acquisition made in the longitudinal axis of the vessel would only show an increase in it's height over the cardiac cycle therefore underestimating the real area change. The use of a quadratic fitting procedure on the velocity profile was also investigated but results were not conclusive.

4.5. Interpretation of flow pulsatility modelling with regards to ex vivo results

The observed differential change between smaller and larger vessels when comparing ATX and WT mice could be simulated using three scenarios: decreasing smaller vessel compliance (Fig. 7C)), increasing larger vessel compliance (Fig. 7D) or increasing compliance of both smaller and larger vessels (Fig. 7E).

Application of the $\hat{C}$ estimator to vessels $< 80\,\mu$m displays a trend towards an increase in compliance for the ATX mice compared to the WT mice leading to a reduced likelihood for the first scenario. Both subsequent scenarios involve an increase in the compliance of larger arteries. This hypothesis is corroborated by recent measurements performed on isolated resistance arteries [14]. Resistance arteries extracted from the base of the brain (which are directly connected to the circle of Willis) were subjected to pressure myography. These results indicated an increase compliance in the ATX model compared to WT. Resistance arteries have internal diameter in the 100-300 μm range at physiological internal pressure, thus the dimension of these vessels is comparable to that of the larger vessel group in the present study therefore suggesting a similar behaviour with regards to changes in compliance. In view of these results, a likely scenario is that compliance in both smaller and larger vessels increase but that our estimator $\hat{C}$ did not have high enough SNR to measure a statistical difference in larger vessels.

5. Conclusion

In this work we have demonstrated a new ECG gated reconstruction technique for evaluating flow pulsatility in CMV and vessel compliance. Comparison between an ATX and a WT group showed differences in flow pulsatility on different arterial segments. As it has been largely recognized, atherosclerotic lesions cause a hardening of the carotid arteries leading to a loss of compliance and higher blood pressure variations at the input to the circle of Willis. Recent evidence suggests that the resistance arteries at the base of the brain compensate this effect by increasing compliance. Our results showed increased flow pulsatility in small arterioles. This increase was modeled, amongst different scenarios, by an increase in compliance in both small and larger vessels corroborating ex vivo results. This supports that OCT can be used to study vascular function although modelling may be required to interpret its results.

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