## [2003] [P1574] Effects of aortic coarctation on left-ventricular arterial coupling and mechanical efficiency are not baroreflex-mediated

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**Objectives:** To compare LV response to afterload augmentation when the baroreflex was intact or inhibited by hexamethonium infusion.

**Methods:** Six pigs, instrumented for aortic pressure and flow, LV pressure and volume measurements were studied under pentobarbital-sufentanil anaesthesia. Vascular properties [input (R1) and peripheral (R2) resistances, compliance (C), arterial elastance (Ea)] were estimated with a windkessel model. LV function was assessed by the slope (Ees) of end-systolic pressure-volume relationship (ESPVR) and stroke work (SW). Ventriculo-arterial coupling was defined as Ees/Ea, and mechanical efficiency as SW/pressure-volume area (PVA). After baseline recordings, LV afterload was increased with an aortic coarctation. Haemodynamic measures were obtained after 30 minutes. The coarctation was lifted, and 30 minutes later, autonomous nervous system (ANS) was inhibited by infusion of hexamethonium. The coarctation was reinstalled, and haemodynamic measurements recorded after 30 minutes.

**Results:** While aortic coarctation increased R1 (from  $0.132 \pm 0.010$  to  $0.352 \pm 0.007$  mmHg.sec/ml; p<0.001) and decreased C (from  $0.57 \pm 0.04$  to  $0.41 \pm 0.05$  ml/mmHg; p<0.005) independently of hexamethonium infusion, R2 and heart rate increased (from  $1.50 \pm 0.11$  to  $1.70 \pm 0.06$  mmHg.sec/ml and from  $115 \pm 5$  to  $125 \pm 2$  beats/min, respectively; p<0.05) only when the ANS was intact. Independently of hexamethonium infusion, Ees increased from  $2.81 \pm 0.18$  to  $3.69 \pm 0.20$  mmHg/ml, while dead volume Vd decreased from  $-3.6 \pm 0.2$  to  $-6.8 \pm 0.3$  ml (p<0.01). Ees/Ea remained unchanged in both conditions. At matched end-diastolic volumes and independently of baroreflex integrity, SW and PVA increased (from  $2012 \pm 168$  to  $2912 \pm 114$  mmHg.ml and from  $2874 \pm 352$  to  $4520 \pm 224$  mmHg.ml, respectively; p<0.005) and SW/PVA decreased (from  $0.70 \pm 0.12$  to  $0.64 \pm 0.10$ ; p<0.05).

**Conclusions:** Our results demonstrate that afterload augmentation has a composite effect on LV function. Ventricular performance is increased (ESPVR leftward shift, increased Ees and SW), but mechanical efficiency is reduced. These changes, observed independently of baroreflex integrity, are of paramount importance in heart transplant patients, which although lacking cardiac innervation, can adapt LV performance without simultaneous changes in HR.

## Session Info: Poster display III

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