

Investigation of the effect of aortic stiffening and stenosis on ventriculo-arterial interaction in a porcine model of Coarctation repair

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Introduction: Systemic hypertension is commonly seen despite successful coarctation repair, regardless whether the treatment consisted of surgery or percutaneous stenting. This study aimed to investigate the effect of short- versus long-segment aortic stiffness and stenosis as sequelae of aortic coarctation repair on ventriculo-arterial interaction in an animal model

Methods : Short- and long-segment aortic stiffening was created by surgical transection/suture(SUTURE,n=6) and stent implantation(STENT,n=5) of the proximal descending aorta. Short and long aortic stenosis was realized by wrapping a prosthetic graft around the aorta to reduce the circumference with 1/3, over respectively 1 cm(SUTURESTENOSIS,n=5) and 5 cm(STENTSTENOSIS,n=6). A CONTROL-group comprised 5 animals. After 3 months, measurements consisted of aortic pressure- and flow-recordings proximally and distally from the lesion, and aortic distensibility by intravascular ultrasound. LV-performance was based on PV-loops. Baseline measurements were compared to measurements during dobutamine stimulation.

Results : Data are presented in table. An effective stenosis increases significantly the proximal aortic impedance, resulting in an increased backward-to-forward pressure-wave ratio contribution. This phenomenon is accentuated by dobutamine administration. Proximal aortic distensibility is decreased specifically in long-segment stenosis.

The increased afterload impairs the ventriculo-arterial coupling in both groups with stenosis, with blunted contractile response during dobutamine (CONTROL +140%, SUTURE +103%, SUTURE STENOSIS +77%, STENT +116%, STENT STENOSIS +57%, $p<0.001$) and increased myocardial stiffness in long aortic stenosis. Histology showed increased myocardial fibrosis in the group SUTURE STENOSIS and STENT STENOSIS.

Conclusion : This animal study, in which sequelae of aortic coarctation repair were reproduced, demonstrated that short- or long-segment aortic stiffening without stenosis had no significant effect on aortic pressure-flow characteristics. However, the negative effect of stenosis on aortic hemodynamics - especially for a longer segment - leads to rapid impairment of the ventriculo-arterial interaction, which is accentuated during inotropic stimulation. Therefore, therapeutical management needs to focus on improving aortic remodeling after coarctation repair, preferably by minimizing residual stenosis, even if it is at the cost of inducing aortic stiffness.

	CONTROL	SUTURE	SUTURE STENOSIS	STENT	STENT STENOSIS	Anova-p
Impedance Zc(mmHG/ml/s)						
Baseline	0.18(0.02)	0.15(0.14)	0.27(0.19)	0.18(0.03)	0.27(0.04)	0.009
Dobutamine	0.14(0.02)	0.14(0.02)	0.31(0.03)	0.15(0.03)	0.39(0.07)	<0.001
Forward P-wave (mmHg)						
Baseline	31.4(2.9)	32.9(2.6)	39.4(3.9)	32.6(3.3)	41.3(2.3)	0.089
Dobutamine	44.9(2.6)	44.6(4.5)	56.9(5.1)	41.4(4.9)	63.3(3.0)	0.004
Backward P-wave (mmHg)						
Baseline	13.8(1.0)	8.7(1.2)	10.7(0.6)	10.1(2.2)	17.2(1.0)	0.001
Dobutamine	14.3(2.9)	10.7(1.1)	17.3(0.7)	11.5(2.3)	31.7(2.6)	<0.001
Aortic distensibility(%)						
Baseline	27.1	24.4	12.7	17.9	8.9	<0.001
Dobutamine	32.1	26.6	14.8	21.1	10.7	<0.001
Contractility (PRSW-Mw/s)						
Baseline	47.2(1.4)	49.3(3.4)	58.5(1.7)	50.4(1.9)	46.1(5.5)	0.142
Dobutamine	113.0(3.2)	99.3(5.0)	103.3(1.9)	108.5(4.5)	71.6(8.1)	<0.001
Myocardial stiffness (β)						
Baseline	0.17(0.02)	0.18(0.02)	0.22(0.01)	0.26(0.05)	0.17(0.02)	0.120
Dobutamine	0.12(0.01)	0.13(0.02)	0.19(0.01)	0.20(0.04)	0.21(0.03)	0.118