962-2 Aprotinin Prevents Fibrin Formation in Cardiac Surgery

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Aprotinin has been shown to significantly reduce bleeding following cardiac surgery, however it is unclear what is the most appropriate dosage, what risks are involved and finally what underlying physiological mechanism controls the reduction in bleeding? The effect of different concentrations of Aprotinin ($0.95 \times 10^6 - 4.45 \times 10^6$ KIU, Trasylol™, Bayer) will be assessed in 4 groups undergoing coronary artery bypass graft surgery (CABG) in relation to a control group of patients who will only receive heparin (each group, n = 40). Measurements of bleeding; platelet aggregation to collagen and ristocetin; t-PA, u-PA and PAI-1; soluble fibrin and fibrin specific degradation products (D-dimer), (AGEN Biomedical, Brisbane, Australia) and factor X11a will be obtained a) post induction of anaesthesia, b) following aprotinin, c) following heparin administration, d) during cardiopulmonary bypass, e) immediately following bypass, f) 2 hours g) 6 hours, h) day 1 and i) day 5 postoperatively. Preliminary results from 32 patients show that Aprotinin reduces circulating levels of soluble fibrin following CABG when compared to the control group (1.85 μg/ml vs 0.55 μg/ml, median) $p < 0.001$ at all doses of Aprotinin. Aprotinin modifies the increase in XDP shown in the control group at the end of bypass (290 ng/ml vs 48 ng/ml) $p < 0.05$ and reduces cross-linked degradation products on day 5 postoperatively (538 ng/ml vs 286 ng/ml) $p < 0.001$ with an apparent dose related effect. These preliminary findings suggest that the mode of action of aprotinin in reducing blood loss following CABG may be due in part to the prevention of fibrin formation with a resultant decrease in fibrin degradation products thus forming a more stable haemostatic plug with reduced bleeding following surgery. Our preliminary findings suggests higher dosages of Aprotinin have beneficial haemostatic effects which should result in a more widespread use in cardiac surgery thus reducing the amount of blood and blood products used with a decrease in attendant risks and costs. (British Heart Foundation Grant PG/94099).

962-3 Echocardiographic Predictors of Left Ventricular Outflow Tract Obstruction and Systolic Anterior Motion of the Mitral Valve After Mitral Valve Reconstruction for Myxomatous Valve Disease

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Recent studies have described left ventricular outflow tract obstruction/systolic anterior motion (LVOTO/SAM) after mitral valve reconstruction (MVR) in patients with myxomatous mitral valve disease, but have not explored predictors of SAM by transesophageal (TEE) echo prior to MVR. We therefore examined 5 consecutive patients who developed SAM after MVR (group 1) and an equal number who did not (group 2). In a standard long-axis view, the lengths of the coapted anterior (AL) and posterior (PL) leaflets (annulus to coaptation) and the distance from coaptation point to septum (C-Sept) were measured before MVR, during SAM, and when SAM resolved.

Results: Prior to MVR, group 1 had a significantly smaller AL/PL ratio ($p < 0.05$) and C-Sept distance ($p < 0.05$) compared to group 2.

	Pre MVR		LVOTO/SAM	No LVOTO/SAM	
	Group 1	Group 2	Group 1	Group 1	Group 2
AL/PL	0.92 (0.15)	1.98 (0.40)*	1.09 (0.20)	2.02 (0.38)§	3.25 (0.54)
C-Sept (cm)	2.31 (0.20)	3.16 (0.58)*	1.80 (0.35)	2.32 (0.37)§	2.99 (0.49)

*$p < 0.05$ vs group 1 before MVR; §$p < 0.05$ vs group 1 with SAM

Conclusions: These initial results suggest that a greater contribution of the posterior leaflet and a smaller C-Sept distance by TEE before MVR may indicate an increased risk of LVOTO/SAM after MVR in patients with myxomatous valves. Resolution of LVOTO/SAM was associated with shifts of the coaptation point away from the LVOT and a greater contribution of the anterior leaflet to valve closure. This is consistent with the concept that SAM relates to anterior malposition of slack leaflet portions into the LVOT, especially if the posterior leaflet is elongated.

962-4 Ventriculoarterial Coupling and the "Physiology" of Reconstructive Surgery for Left Ventricular Aneurysm

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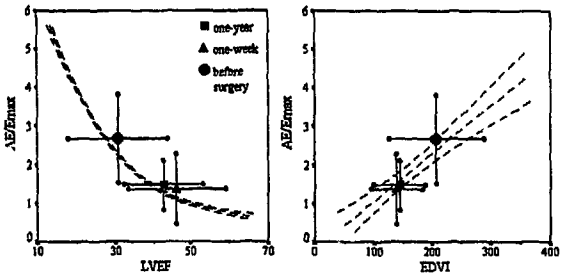
To investigate coupling between the heart and arterial system in left ventricular (LV) aneurysm and its modifications after reconstructive surgery, we studied 15 patients submitted to hemodynamic study: before the intervention (A), after 1 week (B), and after 1 year (C). The slope of arterial end-systolic pressure-stroke volume (arterial elastance – Ae) was compared to the hemodynamic data calculated by mean of frame-by-frame analysis of ventriculographies and high fidelity pressure tracings, recorded during atrial pacing (S1-S1 600 msec).

	EDVI	SVI	LVEF	Emax	Ae	Ae/Emax
A	208 ± 82	59 ± 18	31 ± 12	1.09 ± 0.73	2.35 ± 0.88	2.68 ± 1.1
B	141 ± 39*	60 ± 13	46 ± 13*	1.63 ± 0.82*	1.73 ± 0.43*	1.34 ± 0.84*
C	139 ± 44*	59 ± 16	43 ± 10*§	1.60 ± 0.92	2.07 ± 1.1*	1.48 ± 0.64*

*$p < 0.05$ vs A; §$p < 0.05$ C vs B; EDVI = end-diastolic volume index; SVI = stroke index; EF = ejection fraction; Emax = maximal pressure/volume ratio.



Ae/Emx showed an inverse hyperbolic correlation with LVEF (left graph) and a direct linear correlation with EDVI (right graph). The postoperatory increase in Emax was related to the EDVI decrease ($R = -0.58$; $p < 0.02$). *Percent changes in LVEF (A vs B) correlated to those in Ae* ($R = -0.575$; $p < 0.025$), Emax ($R = 0.53$; $p < 0.042$) and Ae/Emax ($R = -0.83$; $p < 0.0001$); *early results were confirmed after 1 year* (A vs C and B vs C).

Conclusion: ventriculoarterial coupling is the physiological link among the structural changes caused by LV aneurysmectomy, LV pump function and postoperatory cardiocirculatory equilibrium.

962-5 Intermittent Ischemia Prior to Prolonged Reversible Ischemia Protects Canine Myocyte Contractility

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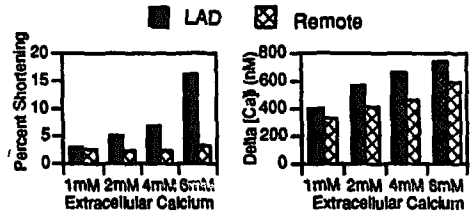
We hypothesized that contractility and calcium dynamics would be protected in myocytes from dogs (n = 6) treated with regional intermittent ischemia prior to global ischemia. After four 3 minute LAD occlusions followed by reperfusion, the heart was made ischemic with aortic cross-clamping for 20 minutes on cardiopulmonary bypass then reperfused for 60 minutes. Isolated single myocytes from LAD and remote regions were studied for differences in contractility (percent shortening, using a video edge detection system) and intracellular calcium transients (delta [Ca]i, using Fura 2 AM). LAD region myocytes showed a dose dependent increase in contractility (similar to our controls, data not shown) with increasing extracellular calcium (1–6 mM) while remote region myocytes failed to respond ($p < 0.01$, ANOVA). Both LAD and remote cells showed normal and equal increases in [Ca]i with increasing extracellular calcium concentrations ($p < 0.05$).



These data suggest that prolonged reversible ischemia decreases myofilament calcium sensitivity and that intermittent preischemia protects myofilament response to calcium.

962-6 Late Complications of Repaired Type A Aortic Dissections: Routine Follow-Up Using Transesophageal Echocardiography

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To determine the incidence of late complications of type A aortic dissection

TUESDAY POSTER