	Males ($n = 13$)	Females ($n = 7$)
Noradrenaline	0.73 ± 0.25 μM	0.23 ± 0.40 μM
Phenylephrine	3.36 ± 0.29 μM	0.36 ± 0.22 μM*
U46619	11.29 ± 0.12 nM	2.32 ± 0.41 nM*
Serotonin	4.08 ± 0.27 μM	0.16 μM ± 0.10 μM*
Endothelin	9.36 ± 1.05 nM	7.65 ± 1.49 nM

Conclusion: The internal mammary arteries of women are more sensitive to constriction by thromboxane, phenylephrine and serotonin, compared with men. Ongoing studies are evaluating the molecular mechanisms responsible for these differences.

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Heme Oxygenase-1 is Necessary for Ischaemia-mediated Neovascularisation

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Left Atrial Specific Endothelial Dysfunction and Inflammation in Atrial Fibrillation

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Introduction: Atrial fibrillation (AF) is associated with a prothrombotic state, with thrombus formation frequently occurring within the left atria (LA). We have previously shown that platelet reactivity is increased within the left atria in patients with AF. However little is known about endothelial function and inflammation within the LA.

Methods: Fifteen AF patients and 16 left sided accessory pathway (REF) undergoing radiofrequency ablation were studied. Following transseptal puncture and prior to the administration of heparin a blood sample (20 mL) was collected from the LA. Markers of endothelial dysfunction (von Willebrand factor [vWF]), and inflammation (intracellular adhesion molecule [ICAM-1] and vascular adhesion molecule [VCAM-1]) were measured using enzyme linked immunosorbent assay.

Results: AF patients had significantly higher levels of vWF within the LA compared to the LA of REF patients, indicating endothelial dysfunction. ICAM-1 and VCAM-1 were raised in the LA of AF patients when compared to REF (see table).

Conclusion: AF is associated with left atrial specific endothelial dysfunction and inflammation. This data suggests that LA endothelial dysfunction and inflammation are important contributing factors in the prothrombotic risk associated with AF.

	REF left atria	AF left atria	*P* value
vWF (mU/mL)	571 ± 132	1669 ± 223	<0.0001
ICAM-1 (ng/mL)	121 ± 76	200 ± 98	0.05
VACM-1 (ng/mL)	386 ± 190	499 ± 246	0.21

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Lipid-free Apolipoprotein A-I Enhances Endothelium generation from Human Blood Monocytes

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We have recently reported that human blood monocytes are capable of rapidly transforming into endothelial-like cells after adhering to preexisting endothelium. This indicates that blood monocytes are a potential source for the repair/maintenance of intact endothelial layers. In this study, we investigated the effect of lipid-free apolipoprotein (apo) A-I, the main apolipoprotein in HDL, on endothelium generation from endothelial-adherent blood monocytes. Peripheral blood mononuclear cells (PBMCs) obtained from healthy HLA-A2+ donors were co-cultured with HLA-A2− human umbilical vein endothelial cells (HUVECs), thus allowing the PBMC-derived cells to be tracked by their HLA-A2 expression. The co-cultures were exposed to lipid-free apoA-I at final concentrations of 0.5 and 2.0 mg/ml. The cell layers were analysed by immunofluorescence and dual-color flow cytometry at serial time points. After 24 hours of co-culture, the proportion of monocyte-derived endothelial-like cells (M-ELCs) in the controls (no apoA-I) was 17 ± 0.9% while the proportion of M-ELC in co-cultures containing 0.5 mg/ml and 2.0 mg/ml of apoA-I were increased to 33 ± 1.4 and 35.8 ± 1.7% (both $P < 0.01$ compared to control). At an earlier two hour time point, at which potential M-ELC proliferation is minimal, a similar two-fold increase in the proportion of M-ELC was also observed. Endothelial transformation of the adherent blood monocytes was characterised by the acquisition of CD105, CD144, and eNOS expression. This was not affected by exposure to apoA-I. In conclusion, a physiologically relevant concentration of apoA-I enhances the generation of endothelium from endothelial-adherent blood monocyte by increasing monocyte recruitment.

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