with adenosine alone and 158 combined stress testing. Ischemic electrocardiography (ECG) changes, typical angina symptoms as well as cardiac wall stress quantified by levels of brain natriuretic peptide (BNP) measured before and 1 minute after stress testing were used to compare the 3 testing modalities. Myocardial ischemia was defined as a summed difference score (SDS) of ≥3 detected by SPECT.

Results: Median BNP-increases were significantly higher in the bicycle stress group as compared to the adenosine only group (22 vs. -3 pg/ml; p < 0.001). In the bicycle group, patients with evidence of myocardial ischemia on imaging more often had ischemic ECG changes (33% vs. 12%, p < 0.001) and a greater increase in BNP levels (28 vs. 16 pg/ml, p < 0.001) compared to those without ischemia (figure). This was not observed in the adenosine group and in the combined group. Patients in the combined group (28% vs. 15%, p = 0.047) and bicycle group (25% vs. 9%, p = 0.001) more often had angina symptoms if showing reversible perfusion defects compared to those without. This was not observed in the adenosine only group.

Bicycle stress: But not adenosine stress results in an increase of cardiac wall stress. The lack of increase in cardiac wall stress and the absence of more ischemic ECG changes in patients with indolent perfusion defects, suggests that stress testing using adenosine alone does not necessarily induce myocardial ischemia.

Conclusion: Bicycle stress but not adenosine stress results in an increase of wall stress. The lack of increase in cardiac wall stress and the absence of more ischemic ECG changes in patients with indolent perfusion defects, suggests that stress testing using adenosine alone does not necessarily induce myocardial ischemia.

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Microvascular obstruction in the right ventricle in reperfused anterior myocardial infarction: macroscopic and pathologic evidence in a swine model

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Methods: Left ventricular adult human cardiomyocytes (HCM) were subjected to normoxia or simulated ischaemia for 4 h or I/R (3 h normoxia followed by 1 h reperfusion) all in the absence or presence of IMD. Cell viability was assessed using Trypan blue dye exclusion and oxidative damage by commercial Oxylight assay.

Aim: To identify if IMD and its receptor components are expressed in human ventricular cardiomyocytes; to investigate whether IMD protects these cells against simulated I/R injury and if so, determine receptor subtype involvement.

Results: IMD, AM and their receptor component mRNAs were expressed in HCM; IMD was detected at 30 min, and remained increased at 4 h of I/R.

Conclusion: IMD exerts protective effects against I/R injury. IMD administration during cardiac reperfusion protects cardiomyocytes in vitro.

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Intermedin protects against ischaemia and reperfusion injury in human microvascular cardiomyocytes: receptor subtype involvement

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Purpose: Early reperfusion following myocardial infarction is essential but results in ischaemia/reperfusion (IR) injury, whereby myocardial cells die largely due to oxidative stress. Intermedin (IMD; adrenomedullin-2), a novel peptide related to adrenomedullin (AM), is up-regulated in cardiovascular disease and is protective against I/R injury in rodent hearts and human coronary microvascular endothelial cells and cardiac non-mycocytes. Protection is dependent on calcitonin receptor-like receptor (CRLR) signaling in association with one of three receptor activity-modifying proteins (RAMPs). IMD has yet to be investigated in human cardiomyocytes for a cardioprotective role.

Aims: To identify if IMD and its receptor components are expressed in human ventricular cardiomyocytes; to investigate whether IMD protects these cells against simulated I/R injury and if so, determine receptor subtype involvement.

Methods: Left ventricular adult human cardiomyocytes (HCM) were subjected to normoxia or simulated ischaemia for 4 h or I/R (3 h normoxia followed by 1 h reperfusion) all in the absence or presence of IMD. Cell viability was assessed using Trypan blue dye exclusion and oxidative damage by commercial Oxylight assay.

Aims: Exogenous IMD is protective against I/R in HCM, attributed to AM1 receptor subtype involvement. IMD administration during cardiac reperfusion could improve levels of morbidity and mortality in patients following myocardial infarction.

Conclusion: Exogenous IMD is protective against I/R in HCM, attributed to AM1 receptor subtype involvement. IMD administration during cardiac reperfusion could improve levels of morbidity and mortality in patients following myocardial infarction.

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Multi center, prospective, randomized, single blind, consecutive enrollment evaluation a novolimus-eluting CSS with bioabsorbable polymer compared to a zotarolimus-eluting coronary stent

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Aims: To evaluate the long term safety and efficacy of the Elixir DESyneTM BD Novolimus Eluting Coronary Stent System (CSS) with a bioabsorbable polymer compared to the Endeavor Zotarolimus Eluting Coronary Stent System.

Methods and results: 149 patients were randomized 3:1, either to the Elixir DESyne BD Novolimus Eluting CSS loaded with 5mg per cm stent length of Novolimus, a sirolimus metabolite, eluted via a bioabsorbable polylactide-based polymer (ElixirDES) or the Endeavor Zotarolimus-eluting CSS loaded with 10mcg per cm stent length of Zotarolimus eluted via a durable polycholene polymer. All patients were analyzed for the primary endpoint of in-stent late loss (assessed by quantitative coronary angiography (QCA) at 6 months). Moreover, all patients underwent evaluation for the secondary endpoints including the Device-oriented Composite Endpoint (DoCE) defined as: cardiac death, MI not clearly attributable to a non-intervention vessel, and clinically-indicated target lesion revascularization; clinically-indicated Target Vessel Revascularization (TVR), and stent thrombosis at 1, 6, 9, and 12 months and annually through 5 years. Lesions were also evaluated for angiographic endpoints at 6 months including: in-segment LLL, percent diameter stenosis, minimal lumen diameter post-procedure, and angiographic binary restenosis (ABR) (≥50%). A subset of patients underwent intravascular ultrasound (IVUS) evaluation including percent reference vessel area and neointimal area at 6 months demonstrating both non-inferiority and superiority of the DESyne BD compared to the control (0.12±0.15 vs 0.67±0.47, p < 0.001), additionally, in-stent ABR was significantly lower for DESyne BD (8% vs 12%, p < 0.001). Excellent clinical results at 6 months were demonstrated for both devices (DoCE 2.7% vs. 3.2%, p= 1.00). Sustained low event rates were observed at 12 months (DoCE 2.7% vs. 3.2% p=1.0). Clinical results through 24 months and complete angiographic and IVUS results will be presented.

Conclusion: First Report of Clinical results through long term (24 months) and complete angiographic and IVUS results will be presented.