Cardiomyocyte proliferation occurs over approximately two days alongside budding and bridging of cardiomyocytes from the wound margins. Macrophages migrate to the necrotic wound border, peaking in numbers at 12 hours post-injury and remain at the site in elevated numbers for up 48 hours after injury.

Conclusions During heart regeneration individual macrophages undergo a phenotypic switch at the site of injury with upregulation of TNF-α, suggesting that wound-associated macropahages transition to an inflammatory activation state. This zebrafish injury model appears to replicate the secondary apoptosis of peri-infarct cardiomyocytes observed in human MI, offering a simple system in which to investigate the impact of interventions on macrophage function following cardiac injury.

90 ABSTRACT WITHDRAWN

91 PROPHYLACTIC USE OF CARVEDILOL TO PREVENT VENTRICULAR DYSFUNCTION IN PATIENTS WITH CANCER TREATED WITH DOXORUBICIN
1Abdulhalim Kinsara, 2Ahmed abuosa, 3Ayman Elsheikh, 2Kahekashan Qureshi, 2Mohammed Abrar, 2Mona Kholeif, 2Abdulwahab Andejani, 2John Cleland.
1King Saud bin Abdulaziz University for Health Sciences, COMJ. King Abdul Aziz Medical City -WR. King, Jeddah, Saudi Arabia; 2King Saud bin Abdulaziz University for Health Sciences, COMJ. King Abdul Aziz Medical City -WR. King; 3National Heart and Lung Institute, Royal Brompton and Harefield Hospitals, Imperial College, London
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Aims Deterioration in ventricular function is often observed in patients treated with anthracyclines for cancer. There is a paucity of evidence on interventions that might provide cardio-protection. We investigated whether carvedilol can prevent doxorubicin-induced cardiotoxicity and whether any observed effect is dose related.

Methods and results A prospective, randomised, double-blind study in patients treated with doxorubicin, comparing placebo (n=38) with different doses of carvedilol [6.25 mg/day (n=41), 12.5 mg/day (n=38) or 25 mg/day (n=37)]. The primary endpoint was the measured change in left ventricular ejection fraction (LVEF) from baseline to 6 months. LVEF decreased from 62%±5% at baseline to 58%±7% at 6 months (p=0.002) in patients assigned to placebo but no statistically significant changes were observed in any of the 3 carvedilol groups. At 6 months, only one of 116 patients (1%) assigned to carvedilol had an LVEF <50% compared to four of the 38 assigned to placebo (11%), (p=0.013). No significant differences were noted between carvedilol and placebo in terms of the development of diastolic dysfunction, clinically overt heart failure or death.

Conclusions Carvedilol might prevent deterioration in LVEF in cancer patients treated with doxorubicin. This effect may not be dose related within the studied range.

92 HIGH HEART RATE PREDICTS CARDIAC FIBROSIS IN PATIENTS WITH ATRIAL FIBRILLATION AND PRESERVED LEFT VENTRICULAR FUNCTION
1Farhan Shahid*, 2Gregory Lip, 3Eduard Shantsila.
1University of Birmingham, Birmingham, University of Birmingham, Institute of Cardiovascular Sciences, Birmingham, UK; 2University of Birmingham, Birmingham
10.1136/heartjnl-2018-BCS.91

Introduction Abnormal cardiac fibrosis predisposes to diastolic function, heart failure and adverse outcomes in patients with cardiovascular disease. Patients with atrial fibrillation (AF) are known to have excessive left ventricular (LV) fibrosis but contributing factors are poorly understood.

Purpose To investigate whether high heart rate predisposes to cardiac fibrosis in AF.

Methods We studied 215 patients with permanent AF (median (IQR) age 73 (51–88) years, 78% males) over a 2 year period from general practice and outpatient clinics. Exclusion criteria included age <50 years, ejection fraction <55%, recent CABG, severe airways disease, BP >160/90 mmHg, advanced renal dysfunction (table 1).

Integrated calibrated backscatter (cIB) was assessed using echocardiographic acoustic densitometry. LV cIB was calculated