Methods We searched the electronic bibliographic databases, including Cochrane Central Register of Controlled Trials (Issue 1, 2010), PubMed (1994～2010.5). EMBASE (1994～2010.5), CNKI (1994～2010.5), VIP (1994～2010.5), Wanfang database (1994～2010.5) to assemble the randomised controlled trials (RCTs) of RVS Pacing compared with right ventricular apical (RVA) pacing. Two reviewers evaluated the quality of included studies based on the Handbook 5.0.2 and extracted data independently. Meta-analysis was performed by RevMan 5.0 software.

Results 35 RCTs involving 2054 patients were included. The results of meta-analysis showed: compared with the RVA pacing, RVS pacing could significantly reduce the QRS wave duration (MD=−0.05, 95% CI −0.07 to −0.02), significantly increase the left ventricular ejection fraction of 3 months and 18 months after operation (MD=7.10, 95% CI 3.03 to 11.17); (MD=7.44, 95% CI 5.46 to 9.42). 3 months later, there was no significant difference between the two groups with regard to pacing threshold (MD=−13.88, 95% CI −29.75 to 2.00), Compared with RVA, RVS was associated with a significant reduction in threshold perception current (MD=−0.73, 95% CI −29.75 to −0.12) and impedance (MD=−75.12, 95% CI −35.53 to −14.71).

Conclusion RVS pacing can give patients a good physiological state which is more consistent with biventricular electric conduction, and lead to haemodynamic improvement. RVS pacing might be expected to become a preferred site of ventricular pacing.

e0193 A CRITICAL ROLE OF CKIT IN CXCR4-MEDIATED PROGENITOR CELL NICHE MAINTENANCE AND MOBILISATION

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Therapeutic mobilisation of bone marrow progenitor cells (BM PCs) is a novel strategy for cardiovascular repair. Both CXCR4 antagonism and c-kit blockade can rapidly and potently mobilise BM PCs; however, the functional interaction between CXCR4 and c-kit remains unclear. We treated c-kit-deficient (c-kit $^{W/W\text{-}V}$) and wild-type (WT) mice with CXCR4 antagonist AMD3100 and evaluated PCs in the peripheral blood (PB) with a colony-forming assay. AMD3100 treatment for 2 h dramatically increased the number of PCs in the PB in WT mice but not in c-kit $^{W/W\text{-}V}$ mice (c-kit$^{W/W\text{-}V}$ vs WT: 30 vs 194 colonies/ml blood, p<0.01). To confirm that c-kit deficiency impairs BM PC mobilisation by AMD3100, we developed an in vivo BM niche clearance/occupation assay. The c-kit $^{W/W\text{-}V}$ and WT mice were firstly treated with AMD3100 to remove PCs from the niche, and 2 h later, transplanted with eGFP transgenic BM cells to competitively occupy the niche. After 3 h, BM cells were isolated and analysed by FACS. Despite the total donor-derived (eGFP+) cells were similar between WT and c-kit $^{W/W\text{-}V}$ recipients, the donor-derived CXCR4-expressing PCs, including eGFP$^+$CXCR4$^+$Lin$^-$, eGFP$^+$CXCR4$^+$Lin$^-$Sca1$^+$, and eGFP$^+$CXCR4$^+$Lin$^-$ckit$^+$ cells, were much fewer in the c-kit $^{W/W\text{-}V}$ mice (c-kit$^{W/W\text{-}V}$ vs WT: 19.7% vs 30.6%, p<0.01; 20.3% vs 29.1%, p<0.05; and 6.7% vs 17.9%, p<0.001, respectively), indicating that c-kit deficiency specifically reduced the capacity of AMD3100 to clear the CXCR4$^+$ PC niche. To better understand the mechanisms, we designed an ex vivo adhesion assay. Mouse BM mononuclear cells were isolated and applied onto plates pre-coated with BM stromal protein VCAM-1, followed by addition of AMD3100. The adhesion of BM cells to VCAM-1 resulted in marked c-kit phosphorylation. Interestingly, AMD3100 significantly attenuated the c-kit phosphorylation. In vivo, AMD3100 treatment for 15 min significantly reduced the level of phospho-c-kit in the BM as assessed by Western blotting of the BM lysates. Consistently, immunofluorescence staining of BM niche demonstrated a significantly lower ratio of phospho-ckit$^+$/total ckit$^+$ PCs in AMD3100-treated mice as compared to PBS-treated mice. We conclude that c-kit plays a critical role in CXCR4-mediated BM PC niche maintenance and mobilisation.

e0194 SRC FAMILY KINASE SFK IS ESSENTIAL FOR RECRUITMENT OF BONEMARROW PROGENITOR CELLS TO THE ISCHAEMIC MYOCARDIUM

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Background The G protein-coupled receptor CXCR4 and its ligand stromal−cell derived factor 1 (SDF-1) play an important role in directing progenitor cells (PC) homing to ischaemic tissue. The Src family protein kinases (SFK) can be activated by, and serve as effectors of, G proteins. However, whether SFK play a role in SDF-1/CXCR4-mediated PC homing is unknown.

Methods and results To investigate whether SDF-1−CXCR4 signalling activates SFK, we isolated mouse bone marrow mononuclear cells (BM MNCs) and applied onto VCAM1-coated plates, followed by addition of CXCR4 agonist SDF-1 and/or antagonist AMD3100. SDF-1 rapidly (in 2 min) and dose-dependently increased phosphorylation (activation) of Lyn, a major SFK in the BM; AMD3100 attenuated the SDF-1−induced Lyn phosphorylation. Notably, SDF-1 treatment did not increase Lyn phosphorylation in the BM MNCs isolated from Mx1-cre$^+$CXCR4$^{fl/fl}$ mice in which the CXCR4 gene had been deleted. To investigate whether SFK play a role in SDF-1/CXCR4-mediated chemotaxis, we performed Boyden chamber migration assay; SU6656, a SFK inhibitor, significantly inhibited BM-MNC migration towards SDF-1 (p<0.001, n=4). To investigate whether SFK play a role in SDF-1/CXCR4-mediated BM-MNC homing to ischaemic heart tissue, we isolated BM MNCs from CXCR4$_{BAC}$:eGFP transgenic mice and injected 1×10^6 cells into WT and SDF-1$_{BAC}$:SDF1-RFP transgenic mice (in which the expression of SDF1-RFP fusion protein is driven by the SDF-1 genomic regulatory sequence and the level of total SDF-1 protein is doubled) that had undergone surgical myocardial infarction 8 h earlier. Some recipient mice also received two i.p. injections of SU6656 (6 mg/kg) at the time of cell injection and again 4 h later. We found a significantly greater amount of eGFP+ cells (1.6-fold, p<0.01, n=5) and eGFP+c-kit+ cells (1.9-fold, p<0.01, n=5) recruited in the infarct border area of the SDF-1$_{BAC}$: SDF1-RFP recipients than in WT recipients. SU6656 treatments significantly reduced the amount of eGFP+ cells and eGFP+c-kit+ cells (p<0.01, n=5) in both WT and SDF-1$_{BAC}$: RFP recipients and abrogate the difference between the two groups.

Conclusions SFK play a critical role in SDF-1/CXCR4−mediated BM PC homing to the ischaemic cardiac tissue thus may provide a target for modulation of tissue repair.

e0195 RESEARCH ON THE EVALUATION OF OCT ON ATHEROSCLEROTIC PLAQUES OF CAROTID ARTERY IN RABBITS WITH INSULIN RESISTANCE

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Objective The purpose was to analyse the feasibility and repeatability of OCT to evaluate atherosclerotic plaque diagnosed by pathologic examination of common carotid artery in rabbits with insulin resistance.

Methods There were 26 male China White rabbits. 20 rabbits were made atherosclerotic with a high-cholesterol diet after injury of the