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IN BRIEF

HYPERTENSION

Benefit of BP self-monitoring for hypertension

Conflicting results from trials have caused uncertainty over the benefits of self-measured blood pressure (BP) for the management of hypertension. To assess the efficacy of self-monitored BP for the titration of antihypertensive medication, 1,182 patients with hypertension were randomly assigned to three groups in which BP was controlled by self-monitoring, by self-monitoring and telemonitoring, or by clinic measurements (usual care). The primary outcome of mean systolic BP after 12 months was lower in the two self-monitoring groups with or without telemonitoring (136.0 mmHg and 137.0 mmHg of BP, respectively) compared with usual care (140.0 mmHg). Long-term follow-up of the patients will inform about the effect on mortality, but the reduction in BP in the self-monitoring groups is estimated to reduce the risk of stroke by 20% and the risk of coronary heart disease by 10%. These findings support the use of self-measured BP to guide antihypertensive therapy.

ORIGINAL ARTICLE McManus, R. J. *et al.* Efficacy of self-monitored blood pressure, with or without telemonitoring, for titration of antihypertensive medication (TASMINH4): an unmasked randomised controlled trial. *Lancet* https://doi.org/10.1016/S0140-6736(18)30309-X (2018)

HEART FAILURE

BMP9 is an inhibitor of cardiac fibrosis

Transforming growth factor-β1 (TGFβ1) is known to promote cardiac fibrosis during heart failure (HF). A study now shows that the levels of bone morphogenetic protein 9 (BMP9), another member of the TGFβ family, are increased in the blood and in heart tissues of patients with HF compared with individuals without HF. In a model of HF, ventricular function was reduced in *Bmp9*$^{-/-}$ mice compared with wild-type (WT) mice, whereas cardiac fibrosis and function were improved in mice treated with BMP9 compared with controls. These results identify BMP9 as an endogenous inhibitor of cardiac fibrosis during HF. Inhibition of endoglin, a co-receptor of TGFβ1, increased BMP9 expression and improved cardiac function in mice with HF. The study, therefore, suggests that the BMP9 pathway inhibits TGFβ1-induced fibrosis.

ORIGINAL ARTICLE Morine, K. J. *et al.* Bone morphogenetic protein 9 reduces cardiac fibrosis and improves cardiac function in heart failure. *Circulation* https://doi.org/10.1161/CIRCULATIONAHA.117.031635 (2018)

INFLAMMATION

Pericardial adipose tissue regulates granulopoiesis

Immune cells were quantified in the pericardial adipose tissue (AT) of mice before and after myocardial infarction (MI). B cells expressing granulocyte–macrophage colony-stimulating factor (GM-CSF), dendritic cells, and IL-17-producing T cells expanded in the pericardial AT after MI. B cell depletion or GM-CSF blockade reduced the number of dendritic cells and T cells in the AT of infarcted hearts. Given that IL-17 promotes granulopoiesis and neutrophil recruitment, B cell depletion also reduced the number of cardiac neutrophils after MI. These observations show that B cell expansion in pericardial AT after MI activates an inflammatory cascade that induces emergency granulopoiesis. Moreover, B cell depletion or surgical removal of AT reduced MI-induced cardiac fibrosis, which also suggests that pericardial AT influences cardiac outcomes after MI.

ORIGINAL ARTICLE Horckmans, M. *et al.* Pericardial adipose tissue regulates granulopoiesis, fibrosis, and cardiac function after myocardial infarction. *Circulation* **137**, 948–960 (2018)