with patients randomized to placebo (3.8 years (95% CI: 3.3–4.2) for BQ-123 versus 2.8 years (2.1–3.4) for placebo, p = 0.032, Figure 1). Conclusion: Short-term administration of BQ-123 in patients undergoing primary PCI for STE-ACS leads to a longer cardiovascular event-free survival.

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Clinical features between heart failure and sleep disordered breathing

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Introduction: Little has been known about clinical background of the patients with heart failure (HF) and sleep disordered breathing (SDB). The aim of this study was to elucidate the relationship between HF and SDB. Methods: 1121 patients admitted to our institute with the diagnosis of HF between 2006 and 2012 was enrolled. SDB was defined by apnea-hypopnea index (AHI). Obstructive sleep apnea (OSA) group and central sleep apnea (CSA) group were defined based on the data of type III sleep monitor (Morpheus). Results: Among 1121 patients 328 (29%) underwent screening of type III sleep monitor. In the 328 patients, 275 (84%) patients showed SDB. Among these 275 SDB patients, 135 (41%) were OSA, and 140 (43%) were CSA. AHI was significantly higher (OSA: 22.5 ± 16.2, CSA: 29.8 ± 14.9, P < 0.05) and ejection fraction (EF) was significantly lower (OSA: 40.1 ± 17.1%, CSA: 33.5 ± 14.1%, P < 0.05) in CSA group between two groups. Among 140 CSA patients, 80 (57%) patients have heart failure with reduced ejection fraction (HFREF) and among 135 OSA patients, 60 (44%) patients have HFREF. Conclusions: SDB was highly associated with HF and the clinical features between OSA and CSA with HF were different. CSA patients were associated with lower EF and higher AHI than OSA patients. This study suggested that SDB was one of an important target of treatment HF and to treat HF according to these clinical subsets of SDB was clinically required in the future.

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Selective deletion of endothelin B receptors from vascular smooth muscle does not inhibit neointimal lesion formation

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Pharmacological inhibition and genetic deletion (Murakoshi et al., 2002; Kirksy et al., 2012) suggest that endothelin (ET) A-selective antagonists are preferable to mixed ETA/B antagonists for prevention of neointimal lesion formation. ETB receptors expressed in smooth muscle cells may, however, contribute to lesion development. It was proposed that ETB deletion from smooth muscle (SM) would reduce lesion formation following arterial injury. Methods: Mice bearing a floxed ETB gene or expressing cre-recombinase under the SM22 promoter were crossed to produce SM-selective ETB deletion. SMETB knockout mice were identified by genotyping and backcrossed to C57Bl/6J (4–6 generations). Functional confirmation of ET deletion was determined by exposing trachea, and mesenteric artery and vein, to sarafotoxin 6c in a myograph. Femoral injury was performed in adult, male SMETB knockout mice and littermate controls and arteries were harvested 33 days later for structural analysis. Results: SMETB knockout reduced (~55%), but did not abolish, ETB-mediated contraction in trachea. In contrast, S6c-mediated contraction in mesenteric veins (130 ± 46% KPSS, n = 4), and in mesenteric arteries cultured for 24 h (72 ± 24% KPSS, n = 4), was abolished by SMETB deletion (5.1 ± 3.4% KPSS and 0% KPSS, respectively). Femoral artery injury produced large, neointimal lesions (47.4 ± 10.6%; n = 7) but SMETB knockout did not alter lesion size (42.2 ± 4.5%; n = 9; P = 0.64). Conclusions: Stimulation of ETB receptors in SM does not influence neointimal lesion formation. This supports the suggestion that ETA-selective antagonists are preferable to non-selective antagonists for prevention of neointimal proliferation. The study was funded by the BHF (project grant and CoRE). Murakoshi et al. (2002) Circulation 106:15; Kirksy et al. (2012) Cardiovasc Res, 95, 19.

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Neointimal lesion formation does not induce endothelin (ET) B-mediated contraction in murine femoral arteries

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Incubation of arteries ex vivo induces ETB-mediated contraction (Adner et al., 1998), possibly via transcriptional mechanisms (Skovsted et al., 2012). ETB receptors are also expressed in neointimal lesions (Azuma et al., 1994). It was proposed that ETB-mediated contraction would be induced by neointimal lesion formation. Methods: Femoral arteries from adult, male C57Bl/6J mice (n = 6) were harvested 36 +/− 2 days after ligation. Isolated mesenteric and femoral veins and arteries from uninjured mice were cultured (DMEM; 37 °C; 5% CO2; 3% humidity; 5 days) before analysis in a myograph. Contractile function was assessed using endothelin-1 (10−9−3 × 10−5 M), endothelin-1 (10−9−3 × 10−5 M) and sarafotoxin 6c (10−11−10−8 M). Relaxant function was assessed using endothelium-dependent (acetylcholine; 10−7M) and independent (sodium nitroprusside; 10−9−3 × 10−5 M) agents after contraction with phenylephrine. Results: Freshly isolated mesenteric veins contracted in response to S6c whereas mesenteric arteries and femoral veins did not. Some (4/10) femoral arteries produced small S6c-induced contractions (21.86 +/− 3.72% KPSS,