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Introduction. Inflammation is thought to be one of the major factors in the progression of arrhythmogenic atrial remodeling that promote atrial fibrillation (AF). The aim of this study was to investigate the effects of inflammatory state before and immediately after catheter ablation on clinical outcomes after catheter ablation of persistent AF. Methods. We investigated 176 patients with long-standing persistent AF (sustained AF duration; 1 to 20 years, with a mean of 3.4 ± 3.8 years) undergoing catheter ablation. The high-sensitivity C-reactive protein (hs-CRP) level was measured as an inflammatory marker before and immediately after the catheter ablation. Patients were divided into two groups according to the hs-CRP level in the baseline: high hs-CRP group ($n = 84$, >0.075 mg/dl) and low hs-CRP group ($n = 92$, <0.075 mg/dl). Results. Catheter ablation was successfully performed in all patients. After 12-month follow-up, 53.4% of the patients had AF recurrence. The hs-CRP level before catheter ablation was significantly associated with AF recurrence ($p = 0.024$), however, neither the hs-CRP level immediately after catheter ablation nor the increment of hs-CRP after catheter ablation was not associated with AF recurrence. Multivariate Cox regression analysis revealed that longer duration of AF ($p < 0.001$), larger left atrial diameter ($p = 0.049$), and higher hs-CRP level ($p = 0.033$) were significantly associated with AF recurrence. In Kaplan–Meier AF free curves, there is a significant difference in AF free rates between low hs-CRP group (57% at 1 year) and high hs-CRP group (35% at 1 year) ($p = 0.007$). Conclusions. The increased hs-CRP level reflecting an inflammatory state before catheter ablation may is one of the important predictors of recurrence of AF after catheter ablation in patients with long-standing persistent AF.

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Pericardial resistance artery contractile responses to endothelins

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Because the parietal pericardium is opened during cardio-thoracic surgeries, the tissue might be used for translational research. We investigated whether i) contractile resistance-sized arteries can be isolated from the parietal pericardium and ii) whether their arterial smooth muscle is responsive to endothelin (ET). Tissue was harvested from pigs i) in a local slaughterhouse ($N = 8$) or ii) at the end of experimental surgery under a cocktail of anaesthetics and analgesics ($N = 7$) and stored overnight. Arterial segments were microdissected from the tissue and studied in wire myographs. Arterial lumen diameter at a distending pressure of 100 mm Hg was 251 ± 24 μm. Contraction in response to 32 mM K^+ averaged 3.9 ± 0.9 N/m. Responses to ET-receptor stimulation were investigated after desensitization of sensorimotor nerves and irreversible blockade of α-adrenergic receptors and in the continuous presence of inhibitors of NO synthases, cyclooxygenases and small- and intermediate-conductance Ca^{2+}-activated K^+ channels. These pharmacological tools were applied to concentrate on arterial smooth muscle responses. Sarafotoxin 6c (ETB-agonist) caused potent and strong contractions (EC50 42 pM, Emax 4.1 ± 0.3 N/m). Thereafter and in the presence of 30 nM A-192621 (ETB-antagonist) the sensitivity and maximal contractile response to ET-1 averaged 1.3 nM and 8.5 ± 2.4 N/m, respectively. 100 nM PD-156707 (ETA-antagonist) reduced the sensitivity and maximal response to ET-1 in these conditions. Differences between arteries from both sources were not statistically significant. We conclude that resistance arteries from the parietal pericardium of patients undergoing cardio-thoracic surgery may be useful for translational studies of arterial smooth muscle responses to ETA- and ETB-receptor stimulation and blockade.

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Regional differences in the effect of hypoxia on endothelin-1-induced contraction in rat arteries

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Acute arterial occlusion due to an embolus or a thrombus causes hypoxia in the vascular bed, usually resulting in critical injury. Hypoxia affects more or less vascular function, but the response to low oxygen differs in individual vascular beds. The present study examined the influence of hypoxia on endothelin-1 (ET-1)-induced contraction in isolated rat carotid and mesenteric arteries. Although the addition of ET-1 (10^{-10} to 10^{-8} M) produced a dose-dependent contraction either in carotid or mesenteric arteries, the response to ET-1 was significantly attenuated by hypoxia in carotid, but not in mesenteric, arteries. The impaired contraction to ET-1 in carotid arteries was also observed in endothelium-denudated preparations or in the presence of an endothelin type B (ETB) receptor antagonist (BQ-788, 10^{-6} M). Meanwhile, ET-1-induced contraction of carotid arteries in the presence of an endothelin type A (ETA) receptor antagonist (BQ-123, 10^{-6} M) was not affected by hypoxia. Incidentally, ET-1-induced contraction was largely inhibited by antagonism of ETA receptors either in carotid or mesenteric arteries. In addition, IRL-1620 (<10^{-7} M), a selective ETB receptor agonist, did not cause any contraction in both arteries. Although a crucial feature of the response to hypoxia is to produce reactive oxygen species like superoxide, the treatment with superoxide dismutase (200 U/mL) did not affect the influence of hypoxia on ET-1-induced contraction in both arteries. These findings suggest that although ET-1 induces contraction through ETA receptors either in carotid or mesenteric arteries, hypoxia impairs this pathway only in carotid arteries. Furthermore, extracellular superoxide seems not to be a causal factor responsible for this regional difference.

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Urinary ET-1 excretion after exposure to radio-contrast media in diabetic patient and patients with preexisting impaired renal function

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Preclinical studies indicate that the renal endothelin system is involved in the pathogenesis of acute renal failure. Contrast media