It can therefore be hypothesised that aldosterone plays a role in the pathophysiology of atrial fibrillation. Taking into account the aforementioned deleterious effects one can speculate that aldosterone antagonists such as spironolactone may ameliorate atrial remodeling. Undoubtedly, the favourable effects of aldosterone antagonism on ventricular remodeling observed in heart failure do not imply similar actions on atrial myocardium. In conclusion, we believe that the role of aldosterone in atrial fibrillation deserves further study since it may contribute to the development of effective strategies against atrial remodeling.

References

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Atrial remodelling in persistent atrial fibrillation: the potential role of aldosterone: reply

The letter by Dr. Korantzopoulos and co-workers made several good points that are of general interest to the issue of aldosterone in atrial fibrillation (AF). Without a doubt, our findings provide mechanistic support for clinical observations pointing to efficacy against AF-induced structural remodelling of angiotensin converting enzyme (ACE) inhibitors after cardioversion of persistent AF. Increased understanding of pathophysiology of AF has shown that activation of the renin-angiotensin–aldosterone (RAA) system has been implicated in the progression of atrial structural remodelling in this setting. Goette et al. has shown that atrial expression of ACE is increased in interstitial tissue of fibrillating human atria. In addition to direct effects of angiotensin II on myocardial tissue, angiotensin II causes the release of aldosterone from the adrenal gland and extra-adrenal tissues. Recently, it has been shown that aldosterone is also produced in the failing human heart. Aldosterone has been shown to stimulate cardiac collagen synthesis and fibroblast proliferation via activation of local mineralocorticoid receptors, or indirectly, interfering with angiotensin II type 1 receptors and enhancing local ACE expression.

Surprisingly, to date, clinical data directly linking aldosterone with AF are scarce. Systemic aldosterone levels are increased in patients with persistent AF. Thus, it is likely that elevated systemic levels of aldosterone, perhaps as well as increased local synthesis of aldosterone acting in a paracrine/autocrine fashion, during AF may contribute to atrial remodelling.

The deleterious effect of an activated RAA system has been clearly demonstrated in patients with heart failure. A recent clinical trial has shown a marked benefit in patients with chronic heart failure from co-administration of the aldosterone receptor antagonist and ACE-