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hypertension in the present model: atrial hypertrophy, increased atrial fibrosis, conduction slowing, and prolonged duration of induced AF. Blockade of the renin-angiotensin system can prevent structural remodelling associated with AF in dogs with pacing-induced heart failure. In the LIFE study, regression of cardiac hypertrophy—assessed as left ventricular hypertrophy—was associated with prevention of new-onset AF, irrespective of classical risk factors for AF or treatment allocation. The sequence of events suggested by new-onset AF, irrespective of classical risk factors for AF or study, regression of cardiac hypertrophy—assessed as left AF in dogs with pacing-induced heart failure. In the LIFE system can prevent structural remodelling associated with the data of Kistler et al. and by these studies—hypertension activates the renin-angiotensin system, induces (atrial) hypertrophy, and thereby causes structural remodelling of the atria—could explain why inhibition of the renin-angiotensin system is so effective in the prevention of AF. Further studies are warranted to assess the activity of the renin-angiotensin system and the effects of its pharmacological blockade in this model. In addition, the effect of lowering blood pressure without interfering with the renin-angiotensin system on the structural remodelling process and the substrate for AF might be of interest.

As in all models, there are limitations. The authors did not search for spontaneous AF. The role of atrial stretch as a trigger for atrial or pulmonary vein automaticity has not been explored. Furthermore, although pre-natal steroid treatment is accepted as a model for hypertension, increased blood pressure is a consequence of a foetal intervention, causing altered steroid-regulated gene expression. Cardiac hypertrophy could be a consequence of this prenatal intervention rather than a response to hypertension. Last but not least, other signalling cascades than the renin-angiotensin system can provoke atrial hypertrophy, fibrosis, and cell death; these should be studied. The paper by Kistler et al. establishes a link between hypertension and AF and thereby introduces atrial hypertrophy as a potential new target for rhythm control treatment in patients with AF.

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