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### abstract

#### *Effect of antihypertensive treatment with RAS inhibitors or calcium channel blockers on vascular stiffness and central pressure.*

Aortic stiffness, as represented by carotid-femoral pulse wave velocity, has a significant prognostic value in cardiovascular disease, in particular in hypertension and in chronic kidney disease. Central aortic pressure and peripheral blood pressure are significantly different in younger individuals, with systolic central aortic pressure being significantly lower than brachial artery systolic pressure. As humans become older, vascular stiffness and reflected waves result in augmentation of central systolic pressure, and decreases in the difference between central aortic systolic pressure and systolic peripheral (brachial) blood pressure.

Antihypertensive agents may exert differential effects on vascular stiffness and on central pressure. Some of these actions may depend on the heart rate effects of the antihypertensive medications used as well as direct effects on the vascular wall. RAS blockers and mineralocorticoid receptor blockers as well as calcium channel blockers reduce collagen and fibronectin deposition, which may contribute to decrease the stiffness of the blood vessel wall. These agents and calcium channel blockers may improve endothelial function, which may also be accompanied by reduced vascular wall stiffness resulting from the action of greater release or bioavailability of nitric oxide.

The CAFE study has demonstrated that an amlodipine and angiotensin converting enzyme inhibitor based study resulted in lower central pressures than a beta blocker and diuretic based treatment although brachial blood pressure was similar in both groups. Other studies using calcium channel blockers or renin-angiotensin (RAS) inhibitors have demonstrated similar results. Mineralocorticoid receptor blockers have also been shown to beneficially affect aortic stiffness in rodent models. It is likely that as in the ASCOT study, of which CAFÉ was a substudy, lower achieved central pressures may explain better outcomes with some antihypertensive agents than with others.