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Why should we measure central blood pressure?: Implications from the Anglo-Cardiff Collaborative Trial

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Blood pressure varies throughout the arterial tree due to the phenomenon of pressure amplification. This means that blood pressure in the ascending aorta is not necessarily the same as that in the brachial artery. A number of factors influence the amount of pressure amplification within meaning that in most individuals, central pressure cannot simply be estimated by measuring the brachial pressure with a cuff. The disparity between peripheral and central pressure holds a number of important implications for the diagnosis and categorization of hypertension, risk stratification and the use of therapies aimed at reducing blood pressure and the associated risk of cardiovascular events. Across the age span, there is marked inter-individual variability in pulse pressure amplification. When categorizing individuals on the basis of brachial blood pressure, this variability leads to considerable overlap in aortic pressures. Indeed, ~70% of individuals with high-normal blood pressure have similar aortic systolic pressures to those individuals with Stage 1 hypertension. This holds important implications for the categorization and treatment of essential hypertension, since a large proportion of individuals who are classified as normotensive based on current guidelines might actually be at increased risk according to their central BP. It also seems likely that that the assessment of central pressure may improve the identification and management of patients with elevated cardiovascular risk. Traditionally, the lowering of blood pressure with pharmacological intervention was considered to be more important for risk reduction than the specific anti-hypertensive agent used. However, recent evidence suggests that beta-blocking drugs are less effective at improving outcome than other drugs. This has stimulated intense interest in the influence of beta-blockers on the central pressure waveform. Beta-blockers appear to be less good at reducing aortic systolic pressure than other drugs, which is most likely due to beta-blockers increasing wave reflection within the arterial tree. However, further studies are necessary to elucidate the precise mechanisms responsible for the increase in wave reflections with beta-blockers and also to determine the impact of beta-blockers and other anti-hypertensive drug classes on central pressure.