Estimation of Central Systolic Blood Pressure from Peripheral Pressure Waves in Rabbits with and without Atherosclerosis Using a New Second Systolic Peak Pressure-Based Method

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Abstract—We attempted to estimate central systolic blood pressure from peripheral pressure waves using a new method without the use of generalized transfer function in young normal and Kurosawa and Kusanagi-hypercholesterolemic rabbits under pentobarbital anesthesia. Central and peripheral pressure waves in response to intravenous infusion of angiotensin II and sodium nitroprusside were simultaneously recorded under regular cardiac pacing. We found that the average of the first and second systolic peak pressures of peripheral pressure waves approximated central systolic pressure irrespective of presence of atherosclerotic lesion and blood pressure level.

I. INTRODUCTION

The ASCOT CAFE study [1] elucidated that depressor effects of vasodilating drugs were more potent on central systolic blood pressure (cSBP) than peripheral systolic blood pressure (pSBP). Although the second systolic blood pressure (pSBP2) of peripheral pressure waves has been shown to approximate cSBP in elderly hypertensive patients [2], there was considerable dissociation between pSBP2 and cSBP in younger normotensive subjects [3]. We attempted to estimate cSBP from peripheral pressure waves using a new method without the use of generalized pressure transfer function in normal and Kurosawa and Kusanagi-hypercholesterolemic (KHC) rabbits aged 12 months.

II. METHODS

Two catheters (2Fr) with a micromanometer at the tip were advanced to the ascending aorta (AA) and distal region of the right brachial artery (Br) via the right common carotid and radial arteries, respectively, under pentobarbital anesthesia (30 mg/kg, i.v.). Changes in pressure waves in response to intravenous infusion of angiotensin II and sodium nitroprusside were simultaneously recorded in AA and Br under regular cardiac pacing.

III. RESULTS

pSBP, pSBP2 and an average of pSBP and pSBP2 (pSBPm) were strongly correlated with cSBP in the two rabbit strains. In Bland & Altman plot, the differences between pSBP and cSBP, and between pSBP2 and cSBP decreased and increased significantly with increasing their average in the two strains, respectively. When the difference between pSBPm and cSBP was plotted against their average, instead of plotting the difference between pSBP2 and cSBP against their average, the difference between pSBPm and cSBP was almost zero irrespective of their average values in the two strains. Systolic amplification (pSBP/cSBP×100) (%) was negatively correlated with mean arterial pressure (MAP), whereas amplification of pSBP2 to cSBP (pSBP2/cSBP×100) (%) was positively correlated with MAP in the two strains. There was no significant correlation between amplification of pSBPm to cSBP (pSBPm/cSBP×100) (%) and MAP in the two strains because the negative and positive correlations with MAP were balanced out each other. Similar findings have been found in humans [4].

IV. CONCLUSION

We conclude that pSBPm in the brachial artery could be a precise estimate of cSBP despite presence of atherosclerotic lesion and blood pressure level.

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REFERENCES