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Carotid and brachial blood pressure – measurements in hypertensive subjects

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ABSTRACT

Physiologically, central pulse pressure is lower than peripheral pulse pressure for the same mean arterial pressure. This situation protects the heart from an increase in post load as a consequence of mechanisms related to wave reflections and/or arterial stiffness. Nowadays, non-invasive measurements enable to evaluate this physiological particularity. One of the methods enabling to minor measurement errors is to calculate the difference between brachial and carotid pulse pressure, so called amplification (mmHg). When this procedure is used, errors are limited to those of brachial blood pressure measurements. Amplification is reduced with age, but augmented by tachycardia, mostly in women. Amplification is poorly modified by drug treatment, except through drug-induced changes in heart rate. Longitudinal studies are needed to evaluate the role of amplification in cardiovascular epidemiology and therapeutics.

KEYWORDS

Arterial rigidity, pulse wave, hypertension.

Under antihypertensive drug therapy, the prevention of cardiovascular (CV) events has a maximal effectiveness for stroke and congestive heart failure but is less effective when coronary risk is considered[1]. The Prime study has shown that, at any given level of systolic blood pressure (SBP), coronary risk is higher in treated than in untreated hypertensive subjects, indicating a consistent residual coronary risk under treatment[2].

In hypertensive subjects under chronic antihypertensive treatment, the effectiveness of drug treatment is universally based on non invasive brachial blood pressure (BP) measurements obtained from mercury sphygmomanometer. It has been widely shown that the control of diastolic BP (≤ 90 mmHg) is easy to obtain, whereas the control of systolic BP (≤ 140 mmHg) is less frequently observed, particularly in the elderly[3]. Because DBP is quite similar in all parts of the arterial tree, and because SBP is higher in peripheral than in central arteries, it has been suggested that both brachial and central BP measurements should be important to measure and compare during chronic drug treatment[1].

Physiologically, whereas mean blood pressure (MBP) and diastolic blood pressure (DBP) are almost the same along the totality of the arterial tree, SBP and pulse pressure (PP = SBP – DBP) are significantly lower in central (thoracic aorta; carotid artery) than in peripheral (brachial artery) arteries[1]. This hemodynamic profile, called SBP and PP amplification, is the consequence of the propagation of the pressure wave along vascular conduits, with a progressive reduction of diameter and increase in wall thickness and stiffness, together with resulting changes in timing and amplitude of wave reflections. Normally, the (carotid – brachial) SBP or PP amplification approximates 11-14 mmHg, both in normotensive and hypertensive subjects. Amplification contributes to protect the heart from an increased after-load[1]. Aging is associated with a consistent reduction of SBP and PP amplification, in association with an increase of CV risk[1,4]. Increased heart rate (HR) rather contributes to enhance amplification[1]. Because non invasive central (carotid artery; thoracic aorta) BP measurements, have been developed and widely validated in the recent years, it is important to compare in normotensive and hypertensive subjects the hemodynamic profile of central and brachial BP measurements both in the presence or absence of antihypertensive drug therapy.

In a large cross-sectional population of treated and untreated hypertensive subjects, we determined carotid and brachial BP in 833 subjects including 480 men and 353 women. Both parameters

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increased with age but, at each age-range, carotid PP was lower than brachial PP[1,5-7]. The difference between brachial and carotid SBP or PP, i.e. "amplification", was reduced markedly with age. The other factors modulating amplification were also heart rate and sex, and potentially smoking, PWV and body height. From the various anti-hypertensive agents, only beta-blocking drugs reduced amplification, but only through their role on HR reduction.

In this study, we used homogeneous non-invasive determinations of brachial and carotid PP issued from a center of CV prevention in Paris City. We studied the carotid artery by transcutaneous tonometry, in order that pulse wave analysis could be performed directly, without the use of a generalized transfer function[8-14]. However, we found similar results when tonometry of the radial artery was done (data not shown) using or not a well-established transfer function. In the past, we validated both methods separately, using for each of them simultaneous determinations of intra-arterial BP measurements[9,11]. The two kinds of determinations were indeed strongly interrelated. Others and we have previously shown the high degree of reproducibility of the pulse wave analysis methodology[9-16]. In fact, the main question of such devices is that a non-invasive calibration of radial and carotid arteries BP curves requires constantly an adequate concomitant measurement of the brachial artery BP[17, 18]. When non-invasive brachial BP measurements by mercury sphygmomanometry are used, the determination of brachial SBP is commonly considered as safe. In contrast, it is not the case for DBP. Because DBP (but not SBP) is nearly the same in all parts of the arterial tree, these errors may be partly minimized. Furthermore, the calculation of SBP and PP amplification tends also to reduce the errors, so limited almost exclusively to those of brachial BP measurements. In this study, for instance, our results on SBP and PP amplifications were quite similar to those reported in the literature when invasive techniques were used[1, 13].

The principal finding of this study was that PP amplification was reduced with age while sex and smoking status played a very small additional effect. On the opposite, we observed that tachycardia as well as increased PWV tends to increase PP amplification. The former factor indicates the role of wave reflections and, possibly, of autonomic nervous system in the mechanism(s) of amplification. The latter factor suggests that hypertension and mostly diabetes mellitus might play a major role in the observed alteration[1]. Since insulin has a consistent role on the mechanism of wave reflections and since such arterial properties disappear in subjects with diabetes mellitus type 2 and insulino-resistance, the weight of evidence suggests that increased carotid PP in the elderly may be largely mediated by insulin, which is potentially associated with neurogenic mechanisms[1,19].

In conclusion, the present study has shown that central PP is implicated in the diagnosis and drug treatment of hypertension and that longitudinal studies are now needed to establish firmly such possibilities.

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CONFLICT(S) OF INTEREST/DISCLOSURE(S)

The material used in this study is original and is not submitted for publication elsewhere. There is no conflict of interest for this study.

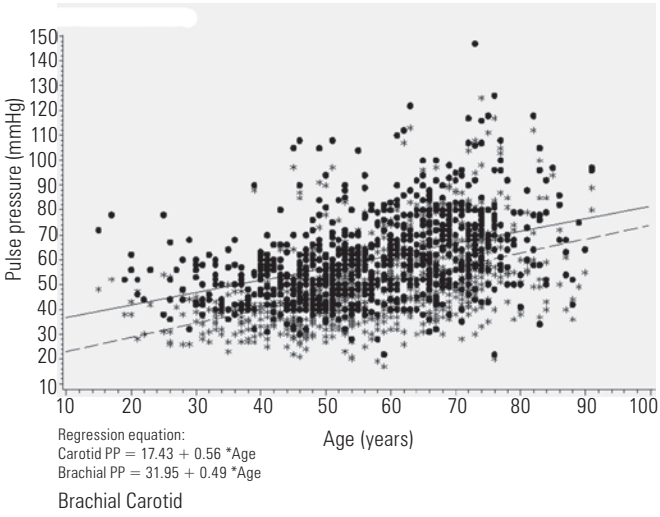

Figure 1. Relationship between brachial or carotid PP and age in the overall population.

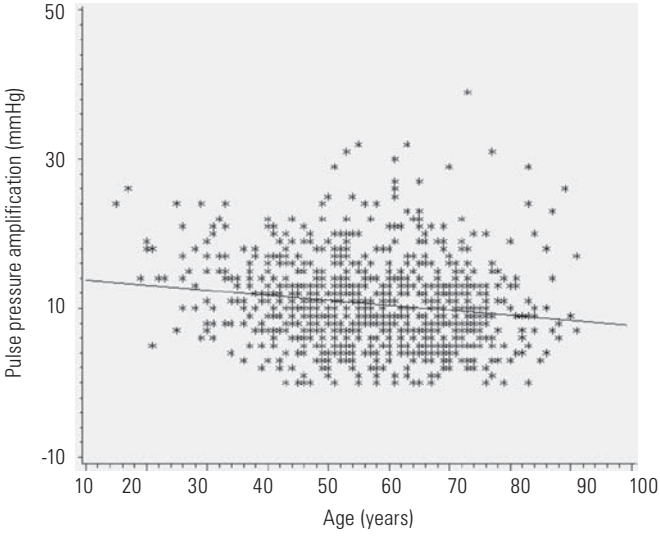

Figure 2. Relationship between brachio-carotid PP amplification and age in the overall population.

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