Pulse Pressure in Clinical Practice

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ABSTRACT

The review presents basic information about the pulse pressure. The variables related to pulse pressure are briefly explained - arterial stiffness, arterial compliance, pulse wave velocity, pulse pressure amplification and augmentation index. We present some recent trials and observational studies that show the importance of pulse pressure in clinical practice. Briefly the possibilities of influencing the pulse pressure are discussed.

BACKGROUND

Pulse pressure (PP) is defined as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP). Physiologically, both pressures increase throughout life due to the increase of stroke volume and/or peripheral vascular resistance (PVR). In the sixth decade of age, the PP increases with increasing SBP and decreasing DBP due to an increase of arterial stiffness [1]. The PP increase also leads to higher pulse wave velocity (PWV). Both PP and PWV are important independent prognostic markers of cardiovascular events, and the high prevalence of isolated systolic hypertension accompanied by a wide pulse pressure seems to be one of the most important factors [3].

Pathophysiology

Etiologic factors known to increase PP and PWV include a reduction of elastic fibres, which are replaced by collagen [4], endothelial dysfunction and an increased expression of vasoconstriction substances (angiotensin II, endotelin, tromboxan) and decrease of vasodilatation substances (NO, bradykinin). In the literature, higher PP was related to smoking, diabetes mellitus [5], dyslipidemia [6], hyperhomocysteinemia [7], obesity and power sports activity. A physiologically lower figure, lower heart rate, post menopause, in the elderly and secondary thyreotoxicosis or aortic valve insufficiency are all associated with an increase of PP.

Arterial stiffness

Arterial stiffness is a characteristic marker of arterial wall structure. Arterial stiffness index (ASI) indicates the relationship between pressure and volume in the artery at the point of measurement. The ASI significantly increases in diabetic subjects and/or hyperlipidemic subjects with hypertension in comparison to those without hypertension [8]. Physiologically, arterial stiffness is the lowest, when transmural pressure (difference between the arterial and external pressure) is almost zero. The elastic pattern of the artery dependents on the elastic properties of the tunica media and relates to an atherosclerotic progression. The arterial stiffness increases with age and is associated with increase of PP and PWV. An elevated value of ASI indicates increasing arterial stiffness and can alert the physician to the risk of atherosclerosis. In clinical practice the ASI value can be determined using for example by CardioVision device.

Arterial compliance

Compliance (C) is a ratio between arterial volume change (ΔV) and pressure change (ΔP) – an increase in blood volume occurs in a vessel with increasing pressure. The compliance value increases in proportion to arterial elasticity [9]. The compliance indicates arterial volume change against the pressure change. A large artery, which shows very large arterial volume changes against normal arterial pressure change, will result in a higher compliance value than will a smaller artery [10]. This leads to a problem with comparison of the compliance value, PP and PWV between central measurements and measurements on peripheral arteries. Reduced arterial compliance is seen in patients with hypertension and diabetes, and also in smokers.
Pulse wave velocity (PWV)

The PWV indicates the velocity of pressure waves along the artery. The physiologically value is still 12 m/s. PWV measurement is calculated from the measurements of pulse wave spreading time among carotid and femoral (or radial) artery a and from a distal point of above mentioned locations. PWV depends on arterial stiffness, ventricular ejection length and PVR intensity[11]. Aortic PWV increases with age, while peripheral PWV changes a little. Both velocities equalise in the elderly.

Pulse pressure amplification

An ejection of blood into the aorta generates a pressure wave (primary wave) that travels along the whole arterial vascular tree. A reflected wave (secondary wave) that travels backwards to the ascending aorta is principally generated in the small peripheral resistance arterioles. With increasing of arterial stiffness both the forward and the reflected waves propagate more rapidly along the vessels. Consequently, instead of reaching back the aorta during the diastole, the reflected pulse wave reaches it during the systole. The result leads to an increase of aortic pressure during systole and reduced pressure during diastole; this phenomenon is called PP amplification.

The relationship between brachial PP and height suggests that the phenomenon of peripheral pressure amplification is also affected by transmission length[12]. The increased left ventricle mass induced by the augmented afterload requires an increased oxygen supply. Therefore, a mismatch between oxygen demand and supply may occur, leading to myocardial ischemia, left ventricle diastolic and later systolic dysfunction. With decrease of DBP, the flow in coronary arteries also decreases[13]. Pressure amplification expressed by peripheral/central pulse pressure ratio was shown to be linearly related to age (r=0.7; p<0.001), with inverse linear relation to diastolic pressure in the younger group (r=0.3; p<0.001) but not in older subjects[14].

Augmentation index (AI)

AI means ratio of amplitudes difference between secondary (reflected) and primary (ejected) waves on pulse pressure. With an increase of arterial stiffness and PVR increase amplitude of reflected wave and AI too. AI is dependent on gender, heart rate and body constitution. PWV, but not central aortic AI, is associated with the extent and severity of coronary artery disease[15].

Pulse pressure and prognostic importance

The normal range of PP is not known. In a study of hypertensive subjects, those with PP 60 mmHg had higher values of left ventricle mass than those with PP 60 mmHg, despite similar mean pressures[16]. Vaccarino et al. reported that the increase of PP about 10 mmHg increases a heart failure risk about 14%, coronary artery disease about 12%, and all cause mortality about 6% in population older than 65 years[17]. The NHANES I study showed that an increase of PP about every 10 mmHg increases cardiovascular death risk about 26% in the age 25-45, and about 10% in the age 46-77[18]. As the prospective study of Franklin demonstrated the prognostic significance of systolic, diastolic and pulse pressure varies with age.

In patients < 50 years of age, brachial diastolic blood pressure was the strongest predictor of coronary heart disease risk, age 50 to 59 years was a transition period when all three blood pressure indexes were comparable predictors and from 60 years of age on, diastolic blood pressure was negatively related to coronary heart disease risk so that brachial pulse pressure became superior to systolic blood pressure[19]. A high pulse pressure represents an independent predictor for cardiovascular morbidity and mortality in those considered as having normal blood pressure[20].

Central aortic pulse pressure, which expresses the pulsatile component of left ventricle afterload exhibited strong association with LV filling pressure as the marker of diastolic dysfunction, whereas the nonpulsatile component of aortic afterload (central mean arterial pressure and central diastolic blood pressure) exhibited only weak significant association with LV relaxation[21]. It has been shown repeatedly that high central pulse pressure correlates with the extent of coronary atherosclerosis[22].

Conversely low pulse pressure below 45 mmHg in patients with advanced chronic heart failure is independent predictor of mortality[23,24].

In clinical practice there are some special situations when wide pulse pressure is an important diagnostic sign. Wide pulse pressure above 80 mmHg is an important character in severe aortic regurgitation[25]. A patent ductus arteriosus is widely considered as a difficult case when the patient shows wide pulse pressure and Corrigan’s pulse. The same applies to the aorto-pulmonary window and the proximal arterio venous fistulae[26].

Pulse pressure as therapeutic target. Antihypertensive drugs have a different effect on compliance and central blood pressure, although the effect on peripheral blood pressure is equal[27]. Optimal antihypertensive drug should decrease SBP and arterial stiffness without an increase of pulse pressure. In the Conduit Artery Functional Evaluation (CAFE) study, the combination of amlodipin and perindopril was more effective for decrease of central blood pressure than atenolol ± thiazid. This result was associated with significant decrease of cardiovascular events due to decrease of PVR and arterial stiffness and reduction of arterial wall hypertrophy. In the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) calcium channel blockers reduced risk of stroke about 27%[28]. In the same study, statin therapy did not lead to a reduction of central blood pressure or augmentation index[29]. In the Regression of Arterial Stiffness in a Controlled Double Blind Study trial, the ACEI perindopril decreased aortic systolic and pulse pressure significantly more than the beta-blocker atenolol[30].

CONCLUSION

A determination of peripheral pulse pressure is very simple and gives extra information above the systolic and diastolic blood pressure. A wide pulse pressure is due to decreased elasticity of large arteries and is a biomarker of increased cardiovascular risk. The value of peripheral PP over 55-60 mmHg should alert the clinician to likely increased arterial stiffness and risk of atherosclerosis. Although pulse pressure is not therapeutic target, the physician should consider the wide pulse pressure as the case for treatment of hypertension with ACEI or calcium blockers rather than with beta-blockers with thiazides.
REFERENCES


