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A REVIEW ON CENTRAL AORTIC PRESSURE AS AN INDEPENDENT PREDICTOR OF CARDIOVASCULAR RISK

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ABSTRACT

Central aortic pressure predicts future cardiovascular disease (CVD) events in the general population with hypertension. Central blood pressure induces direct mechanical stress on the left ventricle and vital organ vasculature. Central aortic pressure was also demonstrated to be more valuable than other BP variables in predicting cardiovascular mortality. Multiple factors influence central aortic function and hemodynamics. Use of antihypertensive medications and allow a greater chance of preventing cardiovascular events. One among the many indexes of measuring central aortic pressure is the BP amplification Index which (difference between central SBP and brachial SBP) has been established as strong indicator of cardiovascular risk. Central BP and arterial stiffness have been found to be more relevant pathophysiologically than peripheral pressures in the pathogenesis of CVD. One of the most convenient forms of measuring central aortic blood pressure is the applanation tonometry which records the radial artery pressure waveforms. Though there is no suggestion that brachial blood pressure measurement should

be fully abandoned but the adoption of central aortic pressure into clinical practise could gain an opportunity to change the phase of medicine.

KEYWORDS: Central aortic pressure, amplification index, cardiovascular risk, Antihypertensive medications.

INTRODUCTION

The provocative proposition of "hypertension is a myth" has generated many a debate and speculations.[1] Traditionally, the diagnosis and management of hypertension has been done based on brachial blood pressure (BBP), also considered as the surrogate marker for predicting the cardiovascular risks. However, emerging data shows that central aortic pressure (CAP) rather than BBP is a more sensitive marker of cardiovascular events such as stroke and myocardial infarction, and a better predictor of progression of hypertension, target-organ damage, and long-term cardiovascular outcomes and could also be used to optimize better treatment strategies.[2]

Central aortic pressure is a net effect of left ventricular contraction and peripheral vascular resistance and undergoes augmentation and peripheral amplification due to changes in the diameter and elasticity of arterial tree. Augmentation increases the absolute aortic systolic pressure and is attributed to variation in cardiac ejection pattern, alteration in the various reflecting sites, arterial reservoir pressure, and an increased stiffness in aorta as well as in large arteries resulting from age or disease processes.[3-4]

It is evident from recent literature that measurement of CAP (rather than measurement of BBP alone) helps in evaluation of the actual pressure load imposed on the left ventricles. Besides, the BP amplification (difference between central SBP and brachial SBP) has been established as strong indicator of cardiovascular events.[5] In this review we discuss our current understanding about central aortic pressure and the current evidence required to bring blood pressure measurement, and cardiovascular risk assessment into the modern era.

Central BP and arterial stiffness have been found to be more relevant pathophysiologically than peripheral pressures in the pathogenesis of CVD. With every cardiac contraction, a pressure waveform is transmitted to the peripheral circulation via large arteries that become progressively smaller in diameter, as well as stiffer in wall tension, with distance from the heart. Owing to these changed vascular properties and the nature of arterial pressure wave

travel within a relatively ‘closed-end’ vascular system, systolic blood pressure (SBP) in the distal vasculature (that is, brachial/radial arteries) is generally higher than in the central arteries (that is, aorta/carotid artery).

The various pressure indices studied were systolic BP (SBP) and diastolic BP (DBP) by direct measurements and PP (SBP − DBP), mean arterial pressure (MAP = DBP + PP/3), pulsatility (PP/MAP), pulsatility index (PP/DBP), absolute BP amplification (peripheral PP − central PP), relative BP amplification (peripheral PP/central PP), and PP amplification(PPA = [peripheral PP − central PP/central PP] × 100) by indirect derived calculations. Pulsatility and pulsatility index were calculated in the central aorta only as they are relevant only in the central arteries.[6-8]

Central Aortic and Its Significance

Normally, aortic or carotid (central) systolic and pulse pressures are lower than peripheral (brachial) systolic and pulse pressures, while mean and diastolic pressures vary from the aorta to the brachial artery due to the absence of indicative resistance at the level of the large conduit arteries.[9] One of the most convenient forms of measuring central aortic blood pressure is the applanation tonometry which records the radial artery pressure waveforms. Though there are various criticisms based on the accuracy of the devices but independent researchers have shown to work out the math where the efficiency of the device is concerned.

These amelioration pave the way to integrate central BP into clinical practise for effective hypertension management. The central aortic pressure reflects the pressure in the large conduit arteries, and is representative of the real hemodynamic stress imposed on the heart, the coronary circulation, the cerebral vessels, and the renal microcirculation. Several studies have addressed this point, and have found a relationship between central aortic systolic or pulse pressure and left ventricular (LV) hypertrophy, concentric geometry of the left ventricle, extent of coronary atherosclerosis, and intima-media thickness. Very recently, the closer relationship of central BP with retinal abnormalities (wall-to-lumen ratio or retinal arterioles) was reported.[10]

For the analysis of the arterial (carotid, radial, or brachial) waveform, three different approaches have been proposed: 1) the use of a generalized transfer function; 2) the derivation of the inflection point on the descending slope of the systolic blood pressure wave, called the secondary systolic wave (SBP2) method; and 3) the N-point moving average

(NPMA) method. The oscillometric method with a generalized transfer function, and the NPMA method allow for 24-hour central systolic pressure monitoring.[11]

Another interesting aspect that has been only partially addressed is the relationship between 24 hour ambulatory central versus brachial BP and target organ damage. Ambulatory peripheral blood pressure is highly related to the development and progression of target organ damage. At the same time, it has been shown that central BP may exert a stronger influence on LV mass increase, on the impairment of LV systolic and diastolic dysfunction, myocardial ischemia, but also carotid intima-media thickening and plaque,66 or retinal arterioles' wall-to-lumen ratio.[12-13] It will be necessary to evaluate the prognostic value of 24-hour ambulatory central aortic pressure reasonably, it can be hypothesized that 24-hour central BP is superior, in this regard, to brachial 24-hour BP.[14] Anti-hypertensive medications have shown to show major response in codifying blood pressure. The individual differences were more prominent in central aortic blood pressure the brachial blood pressure.

Risk Assessment Using Central Aortic Pressure

Central aortic pressure predicts future cardiovascular disease (CVD) events in the general population with hypertension. Central BP induces direct mechanical stress on the left ventricle and vital organ vasculature. Central aortic pressure was also demonstrated to be more valuable than other BP variables in predicting cardiovascular mortality. Multiple factors influence central aortic function and hemodynamics. Age, gender, height ethnicity, exercise, smoking, and heart rate affect various parameters of central aortic pressure. Use of anti hypertensive medications and allow a greater chance of preventing cardiovascular events.

Central Aortic Blood Pressure and Aortic Stiffness in Coronary Heart Disease

Associations between coronary heart disease (CHD) and increased aortic stiffness. In most cases of CHD, brachial blood pressure was measured and because brachial blood pressure is physiologically higher than aortic blood pressure for the same mean arterial pressure (MAP). Aortic blood pressure is expected to be more relevant to the investigation of cardiovascular risk than brachial blood pressure, it is closer to the heart, coronary arteries and carotid arteries, which are the most important sites of cardiovascular risk. An independent association between the level of invasive aortic blood pressure and the extent of CHD in a population who have the same angioplasty procedure. Finally the central wave reflections was shown to be an independent predictors of CHD.

Relationship Between Age and Arterial Stiffness

Age is the important factor of large artery stiffness. Central arteries stiffen increased with age. The stiffening of aorta and other central arteries is a potential risk factor for cardiovascular morbidity and mortality. Aortic stiffness decreases sharply with age in the first stage of life. Age-dependent changes is pathologically understood by fracture and fragmentation of the elastin fibers after repetitive stress cycles, with consequent dilation and stiffening have been observed. A more dynamic and cellular ionic basis for age-related changes in arterial compliance have also been described. Free magnesium levels decrease and cytosolic free calcium levels increase and with age in platelets, skeletal muscle, circulating red cells and brain.[15]

The clinical studies analyzed the effects of age on aortic stiffness, mainly with the method of pulse wave velocity (PWV). The central artery stiffness increase with age, occurs gradually and continuously, similarly for men and women. The Large artery stiffness increases with age independently of the presence of cardiovascular risk factors or other associated conditions. The extent of this conditions increase may depend on several environmental or genetic factors. The PWV increased less with age in populations with low salt diet than in those with high salt consumption. Role of other environmental factors is less documented. Relationship between age and aortic PWV was influenced by the angiotensin II type 1 (AT1) genotypes.

That indicating the genetic variants may influence the development of arterial stiffness with age.

The vascular stiffening and increase in systolic and diastolic blood pressure have been considered as a part of normal aging. Because no treatment for these alterations has been proposed. Although arterial stiffening is a common situation, now it has been confirmed that older patients with increased arterial stiffness and increased systolic and diastolic pressure have higher cardiovascular morbidity and mortality. Therefore, large artery stiffening can be considered as a arterial age marker, and should be considered as a major risk factor for cardiovascular events.

Relationship Between Arterial Stiffness and Hypertension

In patients with hypertension, the main structural modification of the vessel wall is hypertrophy of the medial layer. In adult hypertensive patients, the alterations of the mechanical properties result mainly from the elevated blood pressure, as reduced carotid

compliance and distensibility disappear in isobaric conditions.[16] In some other conditons such as the femoral artery or thoracic aorta, intrinsic changes in stiffness (ie, increased stiffness in isobaric conditions) may be observed.

The elderly hypertensive patients with medial hypertrophy is associated with a considerable development of the extracellular matrix of the media. In patients with hypertension, active mechanisms within the arterial wall are certainly involved because, at the site of peripheral muscular arteries diameter is unchanged despite the elevated blood pressure, but in the central arteries, the diameter is increased in proportion with the level of blood pressure.[17] These changes are observed in the site of central arteries but not peripheral arteries.

A major role is involved in the changes of nitric oxide (NO) production. The NO production is of particular importance for peripheral arteries in which NO and vasoconstrictive compounds in relation with vascular smooth muscle cells that may in constant interaction.[18] The renin-angiotensin-aldosterone system (RAAS) activity may regulate an important role in the regulation of arterial stiffness in hypertensive patients. But hypertensive patients not in normotensives. The angiotensin II and 1 receptor and aldosterone synthase gene variants are significant determinants of arterial stiffness. These results suggest the RAAS may be have synergistic effects with mechanical factors on large arterial stiffness.

Dyslipidemia and Arterial Stiffness

The presence of atherosclerotic plaques in the coronary artery was assessed by the ultrasonographic images of common, internal, and bifurcation sites of the coronary artery for the presence of atherosclerotic lesions. On the basis of clinical and experimental studies, it has been reported that elevated cholesterol level substantially alter the endothelial function. This leads to a decreased relaxation of the arterial vessels.[19] These defect has been mainly described in atherosclerotic patients but also in asymptomatic patients with hypercholesterolemia. But some studies reported increased rigidity of the aortic wall in groups of hypercholesterolemic patients.

This abnormality associated with an increased stiffness of the arterial wall in patients. Subsequently decreased as atheroma progressed in the later stages of the disease.[20] In adult patients with heterozygous familial hypercholesterolemia, sex- and age-matched normocholesterolemics. That patients with familial hypercholesterolemia had significantly more distensible aorta. The significant positive correlations between cholesterol (CH), low-

density lipoprotein cholesterol (LDL), compliance and duration of disease. But a negative correlation between high-density lipoprotein cholesterol (HDL) and aortic compliance. The adults with familial hypercholesterolemia had significantly less distensible aortas than normo-cholesterolemics, with an inverse correlation between LDL-cholesterol and aortic distensibility.

Age and LDL-cholesterol is the best future predictors of aortic stiffness. In the long-term treatment of hypercholesterolemicpatients with a statin was able to increase arterial stiffness or compliance. In some population studies, the different fractions of lipoproteins to be not evaluated. So it described as a negative relationship between arterial stiffness and LDL-cholesterol levels.

Correlation Between Heart Failure and Arterial Stifness

In the congestive heart failure (CHF), use of invasive methods, to assess the presence of abnormalities of large conduit vessels, and more specifically that physical properties of the aortic wall are significantly altered.[21] The conduit artery stiffness affects the pulsatile component of afterload and contributes to altered left ventricular function. This may be partly reflected by the reduction of blood pressure with heart failure, since PWV remains high. Some studies shows using non-invasive procedure or methods have been conducted to evaluate the role of large arteries in heart failure, and to determine the comparable abnormalities to those seen at the ascending aorta are present in other parts of the arterial tree.

The arterial compliance and distensibility evaluated at the brachial, aortic, iliac, or carotid artery levels are impaired in different populations of patients with CHF.[22] The sublingual glyceryl trinitrate induced similar effects in these patients. The role of the endothelium on the arterial properties in CHF and endothelium relaxing factor mediated increasesed distensibility are impaired in CHF patients. The radial artery level, baseline is altered in patients with severe CHF but not in mild CHF. The post-ischemic increased compliance in both mild and severe CHF. That arterial compliance and modulation are impaired in CHF.

The impaired arterial compliance occurring in CHF was increased by the treatment regimen with angiotensin converting enzyme (ACE) inhibitors. The ACE inhibitor could counteract the adverse effects of reduced compliance in patients with CHF such as increased oxygen consumption, cardiac work, decreased coronary perfusion, and altered baroreflex. Because the increased arterial stiffness in patients with heart failure.

Effect of Smoking on Arterial Stiffness

The smoking is alter to the arterial wall, the endothelial function, and to accelerate atheromatosisin several arterial territories. The influence of smoking on arterial stiffness. Some studies show that in smokers without any cardiovascular disease, the acute smoking habits decreased distensibility in both medium and large arteries. It already demonstrated that smoking increased PWV in hypertensive subjects. Few studies showed the acute smoking apparently decreased aortic and brachial distensibility, and elevated blood pressure. Because no basal differences related to long-term effects of smoking was found as compared to non-smokers.

CONCLUSION

As technology keeps pace with time, the evidence based justification for the technology is of greater importance as far as clinical practise is concerned. With this comes quality and accuracy issues. Suitable datas are also required to compare the validity of the commercial device with state of art invasive catheters. Furthermore, risk mortality assessment for cardiovascular diseases in future is also possible by this method and the extent and severity of CVD risks can also be predicted. Though there is no suggestion that brachial blood pressure measurement should be fully abandoned but the adoption of central aortic pressure into clinical practise could gain an opportunity to change the face of medicine.

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