A Quick, Easy, Unexpensive Method to Assess Pulse Wave Velocity by Echocardiography: An Early Marker of Subclinical Atherosclerosis: A Review

Pasquale Palmiero and Maria Maiello*

Cardiology Equipe, ASL BRINDISI, Brindisi District, Brindisi, Italy

Corresponding author: Pasquale Palmiero and Maria Maiello, Cardiology Equipe, ASL BRINDISI, Brindisi District, Brindisi, Italy

Abstract

High blood pressure induces pathological changes in arterial conduction and contributes to the pathogenesis of cardiovascular disease and increased cardiovascular risk. The progression of these changes involves both functional and structural components. Still, an increase in arterial stiffness of those arteries is associated with other cardiovascular risk factors, including age, sex, smoking, hypercholesterolemia, diabetes mellitus and atherosclerosis itself. Recent intervention studies have shown that tight blood pressure control by acting directly on the mechanisms that make progress the atherosclerotic disease, reduce morbidity and mortality in elderly hypertensive patients. The recent availability of non-invasive techniques in particular the determination of the pulse wave velocity for a quantitative evaluation of the stiffness, allowed the calculation of a series of indices of structure and vascular function. Our assessment of global PWVg is a more accurate parameter than PWVc to evaluate aortic stiffness, require only few minutes at the end of an echocardiographic standard examination with low time consuming, no extra equipment and no training are required. Aortic stiffness is a marker of cardiovascular disease useful to identify, at an early stage, subjects with high cardiovascular risk, so we need to implement the routine use of this method.

Keywords: Global pulse wave velocity, Hypertension

Introduction

Cardiovascular diseases remain the leading cause of mortality and morbidity in Western countries, [1-5]. Hypertension is the most common cardiovascular risk factor. The guidelines ESH/ESC 2018 for the Management of Arterial Hypertension, [6] require the assessment of individual "global cardiovascular risk", for a more appropriate diagnosis and treatment of the hypertensive patient. Risk stratification is based on several elements, including the demographic and anthropometric profile, family history, blood pressure, smoking, glycemia and lipid profile. Therefore, the guidelines recommend the careful evaluation of cardiac and vascular organ damage, encouraging early diagnosis, in hypertensive patients, especially if affected by metabolic syndrome. Organ damage affects cardiovascular prognosis, even independently of other risk factors. The organ damage must be detectedat heart level, as cardiac left ventricular mass and geometry; vascular level as intima/media thickness ratio of the carotid wall, pulse wave velocity, index ankle/arm, at eye level as retinopathy only in patients with severe hypertension and atkidney level, as albuminuria, proteinuria and creatinine clearance. The Guidelines [6] recommend the simultaneous evaluation of organ damage in all above mentioned districts: heart, blood vessels, kidney and brain, because, in spite of these alterations are often associated, their contemporary presence in many organs and districts is associated with a worse prognosis. The evaluation of organ damage is compulsory not only in patients with a new diagnosis of hypertension, in order to stratify the cardiovascular risk and treatment, but also in patients who are already on anti-hypertensive treatment, because the regression of cardiac hypertrophy and proteinuria are

possible for the cardiovascular protective effects dueto blood pressure control and pleiotropic effects of the drugs.In the assessment of organ damage are better defined some aspects such as the identification of renal damage, since it has been extended the list of renal markers of organ damage, such as the indirect calculation of creatinine clearance using the Cockcroft-Gault formula, or the glomerular filtration rate by the MDRD (variables more reliable than the value of creatinine in the evaluation of cardiovascular risk that is associated with kidney failure, even mild). Microalbuminuria is an essential parameter for evaluating organ damage, its measurement by the albumin/creatinine ratio urine spots in the morning is easy and relatively inexpensive. Finally, in the assessment of vascular damage, Guidelines since 2007 highlights the assessment of pulse wave velocity as early marker of increased stiffness of large elastic arteries, but in clinical practice this tool is yet underused, because despite its high cardiovascular predictive value, there is a low availability of necessary equipment and the assessment is expensive. The Guidelines 6 also propose the determination of the ratio between blood pressure in the upper and lower limbs, affirming that values lower than 0.9 are predictors of atherosclerotic disease.

In hypertensive patients the great arteries undergo to a continuous stress that fragments the elastic elements of the vessel wall causing dilation and stiffening of the same wall; in accordance with the law of Laplace further dilation increases the effect of stress pulsatile, causing a muscle cell hypertrophy, an increase of collagen fibers and further stiffening [7]. Arterial stiffness can therefore be considered as a potential factor of beginning and progression of atherosclerosis linked with hypertension; but it is also associated with

other cardiovascular risk factors known as elderly, male sex, lipid abnormalities, diabetes and hypertension. Therefore, the increased arterial stiffness could serve as "early marker" of initial atherosclerotic lesions and/or structural modifications caused by various risk factors above mentioned with hypertension first of all [8]. Arterial stiffness assessment means estimation of the capacity of the arteries to expand and recoil in relation to the various phases of the cardiac cycle, so increasing their diameter from diastole to systole. Artery stiffening causes a reduced power to amortize dilating blood flow ejected at each systole of the heart and result higher values of systolic pressure and lower ofdiastolic. About 600 million people worldwide have hypertension and every year some 3 million people die as a direct consequence [9]. A correct estimate of the prevalence of high blood pressure for individual variability gives us a figure that varies between 13.9% and 14.7% [10]. Hypertension increases with age: the diastolic pressure shows an increase towards 50 years age, and then decreases again, while the systolic increases steadily with advancing age. After 75 years the values of both systolic and diastolic pressure decrease [11]. The increase in pressure associated with advancing age, however, is absent in non-Western populations, characterized by lifestyles and eating habits very different from industrialized countries, underlining the importance of environmental factors in the pathogenesis of hypertension [12].

Association of Arterial Stiffness with Hypertension

In elderly we observe structural alterations of the great arteries due to aging and increased prevalence of atherosclerosis: loss of elasticity, alterations of collagen fibers, hyperplasia and hypertrophy of the smooth muscle cells that determine stiffening and decreased distensibility of great vessels wall, especially aorta [13]. The loss of the aortic compliance determines a reduced attenuation of the energy transmitted by the pulse wave so increasing the systolic blood pressure (SBP), the non-relaxation during diastole of the aortic arch causes the reduction of diastolic blood pressure (DBP). All these factors increase peripheral resistance determining the isolated systolic hypertension in the elderly. In elderly also occurs a decrease in myocardial contractility and consequently in cardiac output and a reduction in plasma volume for hypovolemia due the reduction of fluid. Often, we observe a hypertension with a reduced volume. However, the elderly may present a reduced possibility to limit excretion of sodium with the presence of hypertension at high volume [14]. There is also pressure variability due to a compromised system function of baroreceptor attendant mitigation of rapid pressure changes. The baroreceptors, stimulated by high blood pressure, induce an increase of vagal activity resulting in bradycardia and vasodilatation. A pressure decrease, on the contrary, induces a lower download vagal action resulting in prevalence of sympathetic tone and induction of tachycardia and vasoconstriction. The poor efficiency of this regulator system causes in the elderly the impairment of blood pressure control that may cause postural and/or orthostatic hypotension. Arterial hypertension is associated to hyperplasia and hypertrophy of the smooth muscle component, to an increased synthesis of collagen and to a hyper-reactivity of the wall; [13] as concerning the great arteries, aorta undergoes wall stress induced by wave of pulsation and lesions caused by atherosclerotic degradation; at level of arterioles there is a deposit of hyaline substance in the wall with loss of regular structure of the tunica media, impairment of normal endothelial permeability and passage of low molecular weight proteins. At least, the increased arterial stiffness may be independent from the influence of changes in the arterial mechanical blood pressure, for wall stress induced by the wave pulse, and acceleration of the aging process and/or atherosclerosis [15-18].

Common Assessment of Arterial Stiffness

Arterial distensibility is the capacity of great arteries to increase their diameter from systole to diastole. Among non-invasive assessment, pulse wave velocity is the most useful parameter to study arterial distensibility [19]. The transmission speed of pulse wave was considered as a sign of elasticity and stiffness of the arterial wall first by Bramwell and Hill in 1922 and then by a considerable number of authors who have used different methods: Bazett, et al. In 1922, Hickinson in 1924, Sands in 1925, Turner in 1927, Haynes in 1936. Recent developments in non-invasive methods such as ultrasonic techniques, the use of transducers and tonometer for recording of arterial flow and sphygmic waves, associated with the progress of automatic and computerized, opened new horizons to the clinical applications of registration and measurement of pulse wave velocity [19,20]. Two assessments *in vivo* are best known and used: pulse wave velocity that indirectly estimate arterial stiffness and the ultrasonic technique that allows you to calculate the changes in the diameter of the arteries. The measurement of the pulse wave velocity (PWV) is the oldest method available for the estimation of arterial stiffness but it is yet underused: the measurement itself is simple, it must be determined from the space of time between the foot of the pressure pulse waves (= means or early ejection systolic or diastolic pressure point minimum) recorded in two different sites and the distance between the registered sites. With the non-invasive procedure the delay time between the proximal and distal pulse wave can be calculated with manual measurements in which the foot of the wave is calculated, or at the point of minimum diastolic pressure or the point of intersection of tangents along the initial up-stroke systolic and the last part of the previous diastole. The interval between the two feet of the pulse waves proximal and distal is converted into time interval by making a correction for the speed of the paper. The pulse wave velocity is influenced by factors such as: wall thickness, vessel radius and density of the blood. Increased muscle tone or vascular hypertrophy, blood flow velocity and density of blood may increase the PWV. In contrast irregularities of lumen and vessel tortuosity may delay the PWV. The measurements of arterial stiffness done in a certain segment are assumed to represent the arterial stiffness along the arterial tree [21]. Arterial stiffness measured with ultrasound techniques is calculated using a series of formulas that estimate compliance, distensibility and rigidity. The degree of external pressure from the transducer as its inclination on the artery can lead to over estimation or underestimation of the diameter of the artery studied. Still, the systolic pressure is higher, and the diastolic pressure is lower in the arteries more peripheral than central, although the adoption of measures adopting oscillometric method can overcome this problem in the approximation of the measures of stiffness by ultrasonic method [22-24]. These methods and others have as objective the evaluation of arterial stiffness as marker of atherosclerosis for early prevention of cardiovascular disease. Currently available is a sophisticated but easy-to-use equipment. This system uses two transducers cutaneous pressure, applied simultaneously at the precise point of reference of the two arteries (carotid and femoral) and a computer program for registration and analysis that allows the automatic calculation and instantaneous speed of wave propagation pulse for ten consecutive systole, corresponding to a respiratory cycle. The pulse wave velocity is given by the ratio between the space path (D) and the propagation time (t) between the two seats of recording according to the formula: D = PWV (m/s) t. The propagation time (t) is determined by the delay between the foot pulse wave proximal (A) and the distal end of the wave (B). The distance (D) traveled by the pulse wave is obtained by measuring the distance between the two transducers (A and B), as shown in Figure 1. The signals obtained from the two pressure transducers are processed by a computer algorithm on the basis of the principle that the differences between the two waves are minimized once overlapped. The algorithm performs multiple comparisons between discrete sections of the profiles of the waves and uses the results to calculate automatically the delay between the

Citation: Palmiero P, Maiello M (2019) A Quick, Easy, Unexpensive Method to Assess Pulse Wave Velocity by Echocardiography: An Early Marker of Subclinical Atherosclerosis: A Review . Cardiol Cases Syst Rev. 1:003.

• Page 7 •

two waves. All the above-mentioned methods are time consuming and require expensive equipment with operator training.

Our Assessment of Arterial Stiffness

We assess aortic stiffness by global PWV measured in a partially supine position with the head of the examining table elevated by 30° after resting for at least 10 min. The examination was carried out with pulsed Doppler (3.5 MHz probe) using 2-dimensional guidance and ECG trigger. Philips Epiq 7 was used which is an echo-Doppler system equipped with a multifrequency transducer. The interval between the beginning of the QRS complex and the foot of the systolic upstroke in the Doppler spectral envelope was calculated Figure 2 and averaged over five consecutive cycles, non-simultaneously, but at the same heart rate, at the aortic valve site and at the right common femoral artery.

PWVg was calculated between the aortic valve and right common femoral artery by dividing the straight-line distance between the two by the transit time. The distance was assessed using a tape measure located at the same place as the ultrasound probe. The transit time was defined as the difference between two intervals of time using

Figure 1: Global pulse wave velocity.
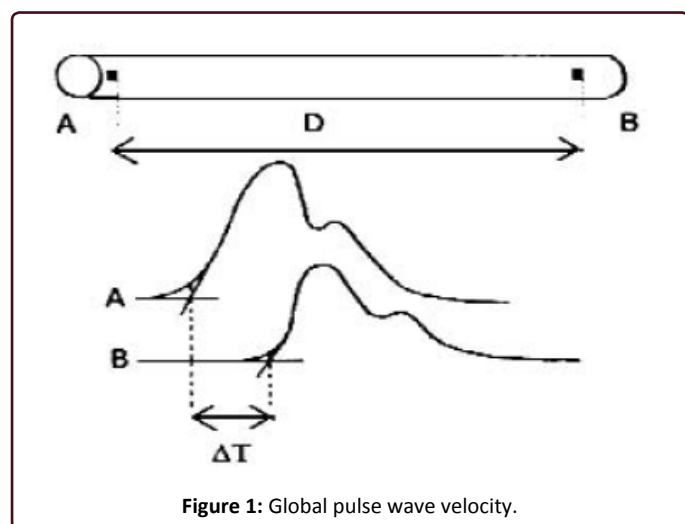
the Doppler method. The Doppler images were recorded on hard disks to be analyzed later using the calipers of the echo machine, [21].

This assessment requires only few minutes at the end of an echocardiographic standard examination with low time consuming, no extra equipment and no training are required. Moreover, the difference in value between PWVc and global PWVc is a difference in length between the territories analysed. The distance used for calculation of PWVc is less accurate since it is defined by a formula: manubrium sternum to femoral artery distance minus carotid artery to manubrium sternum distance without accurately locating the different structures. The distance used for calculation of the global Doppler PWV is measured by calculating the distances separating each site previously located by 2D ultrasound scan.

Conclusions

The study of the distensibility of medium and large caliber of arteries made it possible to obtain important information on the pathophysiology of the cardiovascular system in hypertensive patients and in metabolic diseases. To determine the distensibility of an artery are, therefore, necessary information on the thickness, vessel diameter and on the differential pressure. Compared to few years ago, there are now a number of techniques, primarily based on the use of ultrasound, used to assess, in a non-invasive method, the distensibility of the arterial wall in humans. Among these methods, our assessment of global PWVg is a more accurate parameter than PWVc to evaluate aortic stiffness, require only few minutes at the end of an echocardiographic standard examination with low time consuming, no extra equipment and no training. Aortic stiffness is a marker of cardiovascular disease useful to identify at an early stage subjects at risk, we need to implement the routine use of this method.

Conflict of Interest

None Declare.

Acknowledgement

None Declare.

Figure 2: Global pulse wave velocity, echo assessment.
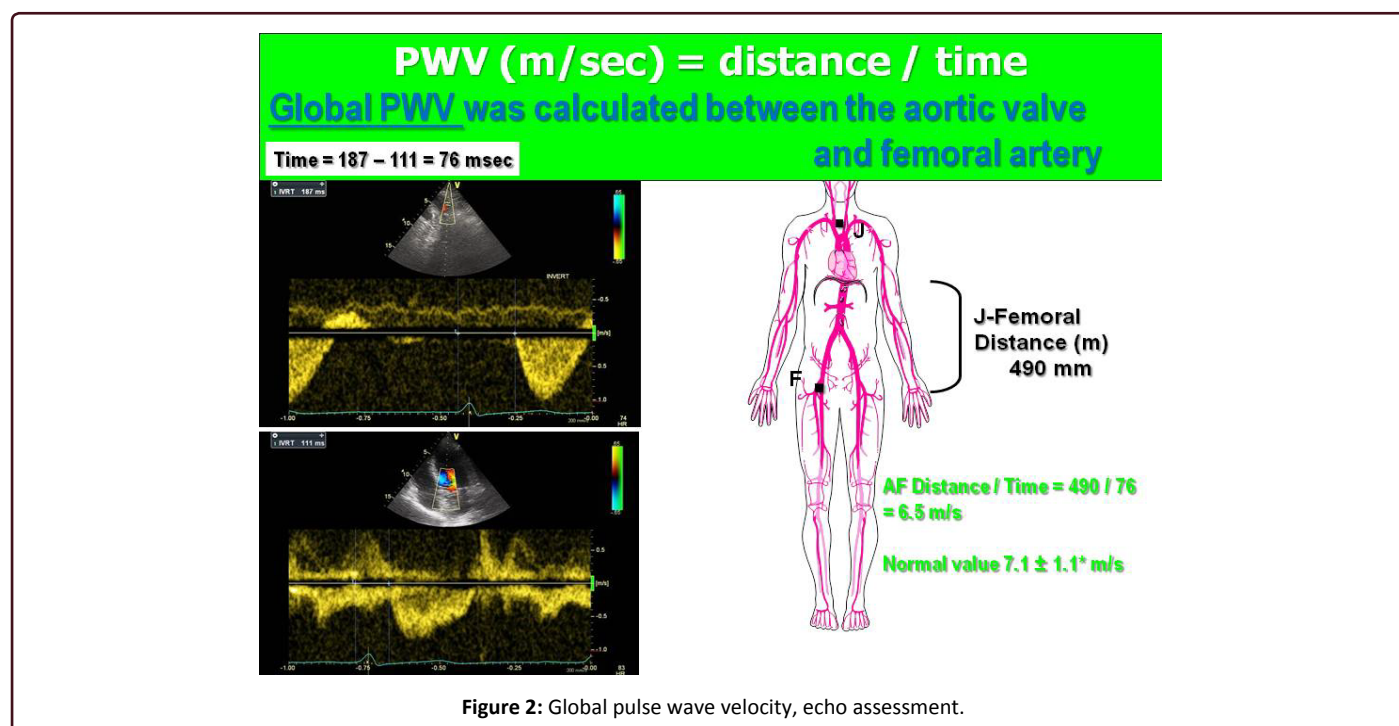
References

1. Alderman MH, Cohen H, Madhavan S (1998) Distribution and determinants of cardiovascular events during 20 years of successful anthypertensive treatment. J Hypertens 16: 761-769.

2. Black HR, Yi JY (1996) A new classification scheme for hypertension based on relative and absolute risk with implication for treatment and reimbursement. Hypertension 28: 719-724.

3. Collins R, MacMahon S (1994) Blood pressure, anthypertensive drug treatment and the risk of stroke and coronary heart disease. Br Med Bull 50: 272-298.

4. Samuelsson O, Pennert K, Andersson O (1996) Diabetes mellitus and raised serum triglyceride concentration in treated hypertension: Are they of prognostic importance? Observational study. BMJ 313: 660-663.

5. UK Prospective Diabetes Study Group (1998) Tight blood pressure control risk of macrovascular and microvascular complication in type 2 diabetes. BMJ 317: 703-713.

6. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, et al. (2018) 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 39: 3021-3104.

7. Arnett DK, Evans GW, Riley WA (1994) Arterial stiffness: a new cardiovascular risk factors? Am J Epidemiol 140: 669-682.

8. Mitchell GF (1999) Pulse pressure, arterial compliance and cardiovascular morbidity and mortality. Curr Opin Nephrol Hypertens 8: 335-342.

9. (1999) Bulletin of the World Health Organization: the International Journal of Public Health (WHO).

10. Klungel OH, de Boer A, Paes AH, Nagelkerke NJ, Seidell JC, et al. (2000) Estimating the prevalence of hypertension corrected for the effect of within-person variability in blood pressure. J Clin Epidemiol 53: 58-63.

11. Howes LG, Reid C, Bendle R, Weaving J (1998) The prevalence of isolated systolic hypertension in patients 60 years of age and over attending Australian general practitioners. Am J Hyperten 7: 139-143.

12. Staessen JA, Gasowski J, Wang JG, Thijs L, Den Hond E, et al. (2000) Risch of untreated and treated isolate systolic hypertension in the elderly: meta-analysis of outcome trials. Lancet 355: 865-872.

13. Nichols WW, Nicolini FA, Pepine CJ (1992) Determinants of isolated systolic hypertension in the elderly. J Hypertens Suppl 10: 73-77.

14. Bhattacharrya A, Das P (1999) Hypertension in the elderly: an overview. J Indian Med Assoc 97: 96-101.

15. Asmar R, Benetos A, Topouchian J, laurent P, Pannier B, et al. (1995) Assesment of arterial distensibility by automatic pulse wave velocity measurement. Validation and clinical application studies. Hypertension 26: 485-490.

16. Wilkinson IB, Fuchs SA, Jansen IM, Spratt JC, Murray GD, et al. (1998) Reproducibility of pulse wave velocity and augmentation index measured by pulse wave analysis. J Hypertens 16: 2079-2084.

17. Asmar R (1999) Arterial Stiffness and Pulse Wave Velocity. Elsevier, Paris, Itlay.

18. Mozersky DJ, Sumner DS, Hokanson DE, Strandness ED (1972) Transcutaneous measurement of the elastic properties of the human femoral artery. Circulation 46: 948-955.

19. Lehmann ED, Parker JR, Hopkins KD, Taylor MG, Gosling RG (1993) Validation and reproducibility of pressure-corrected aortic distensibility measurements using pulse-wave-velocity Doppler ultrasound. J Biomed Eng 15: 221-228.

20. Lehmann ED, Gosling RG, Parker JR, De Silva T, Taylor MG (1993) A blood pressure independent index of aortic distensibility. Br J Radiol 66: 126-131.

21. Palmiero P, Maiello M, Daly DD Jr, Ciccone MM, Nanda NC (2012) Aortic Stiffness Assessed by Global Pulse Wave Velocity in Postmenopausal Women: An Ultrasonographic Study. Echocardiography 29: 1233-1238.

22. Maiello M, Zito A, Ciccone MM, Palmiero P (2014) How aortic stiffness in postmenopausal women is related to common cardiovascular risk factors. Cardiol Res Pract 2014: 216080.

23. Palmiero P, Maiello M, Daly DD, Zito A, Ciccone MM, et al. (2014) Relationship between global pulse wave velocity and diastolic dysfunction in postmenopausal women. Int J Clin Exp Med 7: 5629-5635.

24. Blacher J, Asmar R, Djane S, London JM, Safar ME (1999) Aortic pulse pressure wave velocity as a marker of cardiovascular risk in hypertensive patients. Hypertension 33: 1111-1117.

Citation: Palmiero P, Maiello M (2019) A Quick, Easy, Unexpensive Method to Assess Pulse Wave Velocity by Echocardiography: An Early Marker of Subclinical Atherosclerosis: A Review . Cardiol Cases Syst Rev. 1:003.

• Page 9 •