

Review: History & Advancements Of Pulse Pressure Measurement

Dr. Prasanna Kumar S. C¹, Jyothsna. D³

¹ Head of Instrumentation dept., RVCE, Bangalore

³ second year MTech, BMSP&I, Dept., of Instrumentation, RVCE, Bangalore

Abstract: This paper discusses the evolution of our knowledge of the arterial pulse from ancient times to the present. The importance is discussed in three historical eras of medicine: ancient, medieval, and modern. The pursuit continues and is leading to major advancements in our knowledge of the arterial pulse and its application in diagnosis of various heart related disease [16]. The paper also discusses the parameters of the PPG waveform.

Keywords: pulse pressure, systolic blood pressure, diastolic blood pressure.

I. INTRODUCTION

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). Etiologic factors known to increase PP and PWV, include a reduction of elastic fibres, which are replaced by collagen [2], endothelial dysfunction and an increased expression of vasoconstriction substances (angiotensin II, endothelin, trombox-an) and decrease of vasodilatation substances (NO, bradykinin). In the literature, higher PP was related to smoking, diabetes mellitus, dyslipidemia, hyperhomocysteinemia, obesity and power sports activity. A physiologically lower figure, lower heart rate, post menopause, in the elderly and secondary thyrotoxicosis or aortic valve insufficiency are all associated with an increase of PP.[1]. Changes in resting heart rate over time may affect risk of death from heart disease. a low resting heart rate -- for instance, 60 beats per minute or lower -- can indicate that your heart and cardiovascular system are working more efficiently than people with a higher resting heart rate and who are less physically fit.

II. PARAMETERS

Arterial stiffness: Arterial stiffness is a characteristic marker of arterial wall structure.

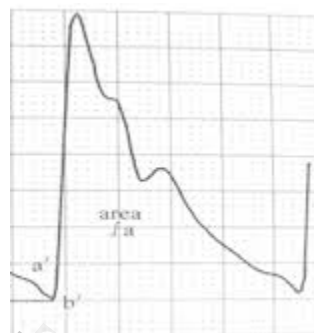


Fig1: pulse pressure waveform

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. [6]

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.

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 [7]. 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 [8].

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 [9]

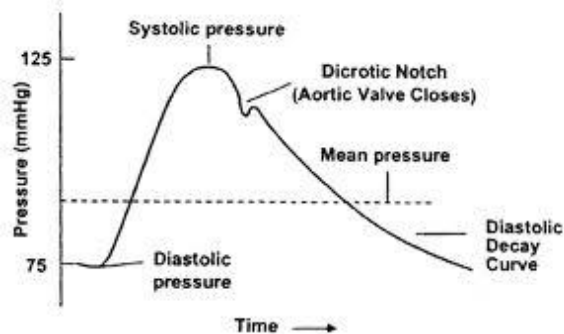


fig2: parameters of the wave.

III. HISTORY

Adolf Kussmaul (Figure 3) was the first to describe the Pulsus Paradoxus phenomenon in 1873 in a paper entitled “Ueber Schwielige Mediastino Pericarditis und den Paradoxen Puls”. This phenomenon has been described in spontaneously breathing volunteers presenting with conditions which cause right ventricular dysfunction, impaired right ventricular filling, and raised atrial pressure. Pulsus Paradoxus is induced by conditions that exaggerate the physiological mechanisms occurring during spontaneous respiration. [13]



Fig.3: Adolf Kussmaul (1822–1902).

Ancient Medicine: Ayurveda (knowledge of life) is an ancient medical science that has been originated in the Indian subcontinent and has been practiced since the time of Buddha (500 BCE). The examination of the pulse is an integral part of Ayurvedic medicine. Eight parts of a patient’s body are described for physical examination, the first one being the arterial

pulse [10]. A full description of the methodology of pulse examination has been described in three categories related to the examiner, the examined, and to the method of examination. A special method was introduced for counting pulse rate in Ayurvedic medicine. Heart rate was counted per “pal” with every 2.5pal making a minute. Moreover, different pulse rates were described for different age groups in the Ayurvedic [11].

Medieval Medicine: Arterial pulse continued to be one of the most important diagnostic and prognostic signs in medieval medicine. As an example, arterial pulse was mentioned to be valuable in prognostication of epilepsy. A medieval physician who felt the particular pulse of a patient suffering from epilepsy would project that the patient would have a seizure at some point during the natural course of the illness [12]. The above figure shows the marey’s sphygmomanometer.

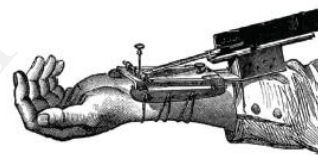


Fig 4: marey’s sphygmomanometer

Modern Medicine: From the 13th until the 16th century, when William Harvey (1578–1657 CE) discovered the greater circulation, there was no major advancement in the understanding of the physiology of the arterial pulse and circulation. Harvey fully described the circular blood flow in the body from the heart to the extremities via arteries and from extremities back to the heart via the venous system. Although the arterial pulse had been an integral guide to reach a diagnosis in equity and medieval eras, general concepts of its generation were misunderstood. Both the heart and the arteries were thought to have their own pulsation and to contract simultaneously. It was thought before Harvey’s dogma-shattering observations that the arterial pulse is the result of an active force generated in the arterial surface. It was William Harvey who for the first time attributed the generation of the arterial pulse to the contraction of the left ventricle and found the source of the heart beat in the right atrium. He contradicted his forefathers, Galen and Vesalius, in their belief of

the origin of the arterial pulse in the arterial wall and with his meticulous observations attributed the generation of the arterial pulse to a passive dilation caused by the blood inflow and compared this passive dilation of the arteries to the process of inflating a glove by blowing air into it . Furthermore, for the first time in the history of medicine, Harvey described the arteries and veins to contain nothing but blood [13-19]. The below table shows the instruments used to measure the pulse pressure over a period of time & the inventors.

Table 1: instruments used to measure pulse pressure.

| Instrument | Inventor |
|----------------|---|
| Clepsydra | Herophilus (Third century BC) |
| Pulsilogy | Santorio Sanctorius (Sixteenth-Seventeenth century) |
| Pulse watch | Sir John Floyer (Seventeenth-Eighteenth century) |
| Sphygmometer | Jules Herisson (Nineteenth century) |
| kymograph | Carl Ludwig (Nineteenth century) |
| Sphygmograph | Etienne Marey (Nineteenth century) |
| Hemotachometer | Karl Vierordt (Nineteenth century) |

| | |
|----------------|----------------------------------|
| | |
| Plethysmograph | Otto Schmitt (Twentieth century) |

IV. RECENT DEVELOPMENTS

FOR years the pulmonary artery catheter has been the gold standard for guiding fluid management in the operating room and in the critical care units. A number of studies have questioned the utilization and the safety of the PA catheter. Non-invasive techniques are being investigated to fill the void caused by the declining use of the PA catheter. The researchers, clinicians and the industry is looking for alternate and preferably non invasive monitors to fill this void. PPV has shown great potential to help optimize hemodynamic parameter using physiological data from non-invasive means. Today PPV can assist with fluid therapy and hemodynamic optimization in patients under general anesthesia receiving mechanical ventilation. With new and improved algorithms the PPV has the potential to help us guide fluid management in spontaneous breathing and non ventilated patients both in our operating room and critical care units. [13] Pulse pressure has been related to a number of surrogate end points such as LV hypertrophy, and, in addition, baseline PP has been found to be predictive of subsequent cardiovascular, particularly coronary, events. Of particular importance is the finding in several studies that PP is as good or better a predictor than other blood pressure terms. Elevated PP as Cause and Effect: While there is substantial evidence linking an elevated PP to adverse cardiovascular outcomes, there has been little study of possible mechanisms linking PP to cardiovascular pathology. Pulse pressure elevation was shown to induce endothelial dysfunction as assessed by acetylcholine reactivity (14) in small vessels, and endothelial dysfunction is a possible antecedent of atherosclerosis. As discussed, PP has also been related to LV hypertrophy. A possible additional explanation for the relationship between PP and cardiovascular end points is provided by the concept of bi-directionality—that is, an elevated PP is both a cause and a consequence of atherosclerosis. Thus, if

atheroma were widely distributed throughout the arterial system at an early, pre-symptomatic stage and if the presence of such atheroma led to increased large artery stiffness, this could result in a statistical association between baseline PP and future clinical events. Such a proposition requires evidence that atherosclerosis is indeed associated with large artery stiffness, as discussed in detail in the following text. Although such evidence cannot prove that aortic stiffness would have been increased at an earlier stage in the disease process, a recent outcome study found that aortic stiffness at baseline, as measured by PWV, was predictive of both all-cause and cardiovascular mortality, even with adjustment for other risk factors, indicating a prognostic role for aortic properties per.

V. CONCLUSION

Our intention with this historical review is to make the reader appreciate how the understanding of the arterial pulse has progressed over the centuries to the present time and give the reader an insight for future developments[16]. The paper covers the introduction to the concept of Pulse Pressure and the benefits of its measurement. The paper also talks about the advantages of pulse pressure measurement in detection of the cardiovascular diseases, it also talks a little about the history & the advances in the present day. We also see the development of the concept through different ages.

References:

1. Pulse Pressure in Clinical Practice Petr Lokaj¹, Jiri Parenical¹, Monika Pavkova Goldbergova², Katerina Helanova¹, Roman Miklik¹, Petr Kubena³, Ilona Parenicova¹, Jiri Jarkovsky⁴, Simona Littnerova⁴, Anna Vasku² and Jindrich Spinar.
2. Avolio A, Jones D, Tafazzoli-Shadpour M: Quantification of alterations in structure and function of elastin in the arterial media. *Hypertension*. 1998 Jul; 32 (1): 170-5.
3. Lehmann E, Gosling R, Sönksen P: Arterial wall compliance in diabetes. *Diabet Med*. 1992 Mar; 9 (2): 114-9.
4. Lehmann ED, Watts GF, Gosling RG: Aortic distensibility and hypercholesterolaemia. *Lancet*. 1992 Nov 7;340 (8828): 1171-2.
5. Sutton-Tyrrell K, Bostom A, Selhub J, Zeigler-Johnson C: High homocysteine levels are independently related to isolated systolic hypertension in older adults. *Circulation*. 1997 Sep 16; 96 (6): 1745-9.
6. How Your Pulse Can Predict Your Risk of Death, By Alice Park Dec. 21, 2011.
7. Sato H, Hayashi J, Harashima K, Shimazu H, Kitamoto K: A population-based study of arterial stiffness index in relation to cardiovascular risk factors. *J Atheroscler Thromb*. 2005; 12 (3): 175-80.
8. Chemla D, Hébert JL, Coirault C, Zamani K, Suard I, Colin P, Lecarpentier Y: Total arterial compliance estimated by stroke volume-to-aortic pulse pressure ratio in humans. *Am J Physiol*. 1998 Feb;274(2 Pt 2):H500-5.
9. Stergiopoulos N, Segers P, Westerhof N: Use of pulse pressure method for estimating total arterial compliance in vivo. *Am J Physiol*. 1999 Feb;276H424-8.
10. O'Rourke MF, Staessen JA, Vlachopoulos C, Duprez D, Plante GE: Clinical applications of arterial stiffness; definitions and reference values. *Am J Hypertens*. 2002 May;15(5):426-44.
11. V. Lad, *Secrets of the Pulse: the ancient Art of Ayurvedic Pulse Diagnosis*, 1996.
12. V. G. P. Upadhyay, *The Science of Pulse Examination in Ayurveda*, Sri Satguru Publication, New Delhi, India, 1997.
13. F. Wallis, "Signs and senses: diagnosis and prognosis in early medieval pulse and urine texts," *Social History of Medicine*, vol. 13, no. 2, pp. 265-278, 2000.
14. PULSE PRESSURE VARIATION: WHERE ARE WE TODAY?, Maxime Cannesson¹, Mateo Aboy², Christoph K Hofer³ and Mohamed Rehman J Clin Monit Comput 2010
15. Ryan S.M., Waack B.J., Weno B.L., Heistad D.D.; Increases in pulse pressure impair acetylcholine-induced vascular relaxation, *Am J Physiol* 268 1995 H359-H363.
16. A Brief Journey into the History of the Arterial Pulse, Nima Ghasemzadeh^{1,2} and A. Maziar Zafari^{1,2}, *Cardiology Research and Practice* Volume 2011.