

BLOOD PRESSURE PHYSIOLOGY

Systolic and Diastolic Blood Pressure: Do We add or subtract to estimate the Blood Pressure Burden?

Tiny Nair

ABSTRACT

The parameters that are taken into account during diagnosis or treatment of hypertension are the systolic blood pressure (SBP) and the diastolic blood pressure (DBP). Also, the importance of pulse pressure (PP), which is the difference between SBP and DBP, has been highlighted in determining the outcome of hypertension. A radically new concept of blood pressure burden (BPB), which is the sum of SBP and DBP (SBP+DBP) is proposed.

Keywords: Blood pressure burden, Diastolic blood pressure, Hypertension, J curve, Physiology, Systolic blood pressure.

How to cite this article: Nair T. Systolic and Diastolic Blood Pressure: Do We add or subtract to estimate the Blood Pressure Burden? *Hypertens J* 2016;2(4):221-224.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

The global burden of hypertension is huge and rising. It is estimated that by 2025, around 1.5 billion people worldwide would suffer from hypertension.¹ Hypertension is one of the most important risk factors for the development of premature cardiovascular (CV) disease. A meta-analysis 68 BP lowering trials involving 245,885 individuals show that a reduction of 10 mm Hg systolic and 5 mm Hg diastolic reduction translates into a 36% stroke reduction, 38% heart failure reduction, coronary event reduction by 20%, and CV death reduction by 16%.² The elevated risk of CV disease is mostly driven by systolic blood pressure (SBP) in the elderly population (above the age of 60 years) and by diastolic blood pressure (DBP) in those who are younger.³ Pulse pressure (PP), the difference between SBP and DBP, which is a measure of large artery stiffness also predicts CV risk.⁴ Thus SBP, DBP, and their subtracted value of PP remain the standard parameters that are measured at the bedside.

The phasic cardiac contraction produces a phasic “pulsatile” flow into the vascular system. A series of

vascular channels of different resistance and conductive characters make sure that this “pulsatile” flow is translated to a near continuous flow to the organs for proper perfusion. Despite significant advances in invasive and noninvasive hemodynamic monitoring, many areas of vascular physiology remain obscure and difficult to explain. Emergence of widespread use of central aortic blood pressure has given us an insight into the circulation, demonstrating that the arterial waveform in central aorta is different in contour and pattern compared to the peripheral arteries. As the pulse wave travels through the vascular system, it transforms into shape and contour. Also with aging as the central aorta becomes less compliant, the pulse wave contour changes.

THREE-COMPARTMENT CONCEPT

To understand and explain the pulse wave patterns, the vascular system may be divided functionally into three distinct compartments.

Compartment 1: The heart

Compartment 2: The proximal aorta and branches. It contains large volume reservoir with lots of elastic tissue. It has two basic functions:

1. Reservoir (capacitance) function in systole and
2. Recoil function in diastole.

Both the functions depend on elastic tissue content of the arteries.

Compartment 3: The peripheral resistance arteries, with muscular arterioles. These create the resistance to the flow and are dynamic, being under delicate neurohormonal control.⁵

During ventricular systole, blood flow out of the left ventricle (LV) into the proximal capacitance vessels (compartment 1 to compartment 2). These large vessels dilate partly attenuating the rise of pressure while transmitting the rest of it to the periphery. The rate of rise of pressure (in the central aorta) depends on the force of ventricular ejection, the compliance of the reservoir (compartment 2), and the resistance of the peripheral resistance vessels (compartment 3) (Fig. 1).

During ventricular diastole, the aortic valve is closed so that there is no active forward flow across the aortic valve. The “reservoir” large vessels (compartment 2) recoil and passively push out blood to the periphery. Here the rate of decay of the diastolic pressure depends on the

Head

Department of Cardiology, PRS Hospital, Thiruvananthapuram Kerala, India

Corresponding Author: Tiny Nair, Head, Department of Cardiology, PRS Hospital, Thiruvananthapuram, Kerala, India
 Phone: +919847061455, e-mail: tiny_nair@gmail.com

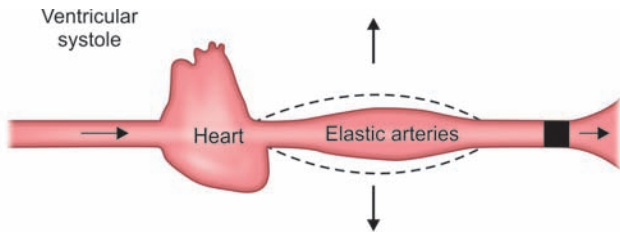


Fig. 1: Blood flow during ventricular systole

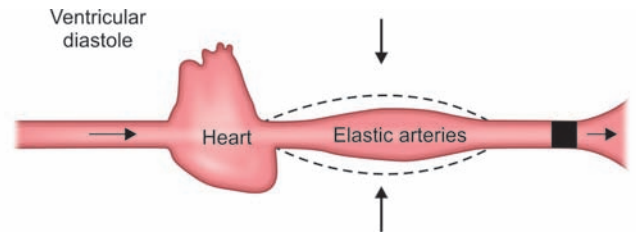


Fig. 2: Blood flow during ventricular diastole

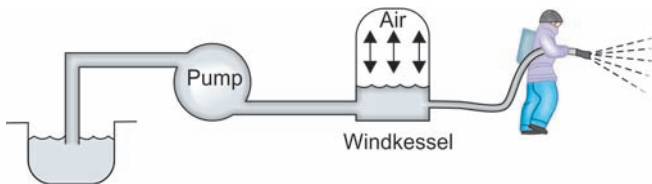


Fig. 3: Phasic pump converted to continuous flow in a fire-fighting equipment (Windkessel effect)⁶

recoil of the large vessels (compartment 2), competence of the aortic valve, and the resistance of the peripheral vessels (compartment 3) (Fig. 2).

This effect is called Windkessel effect (used by fire fighters to convert phasic pump flow into a continuous flow (Fig. 3).

Graph 1 shows the LV pressure curve and the aortic pressure curve. The calculated “Windkessel” pressure is superimposed.

Despite all the efforts of the vascular system to flatten out the pressure wave in the aorta in an effort to convert it from a phasic flow to a continuous flow, we can see that it is not very efficient in such a conversion.

If we calculate the flow rates and convert them into force (Graph 2), we can clearly see that the major driving energy is the contracting ventricle [forward compression wave (FCW)]. But subsequently, there is a smaller albeit

clear propagative force [forward expansion wave (FEW)] generated by the recoil of the reservoir vessels that drive blood in diastole in an effort to make the flow as continuous as possible.

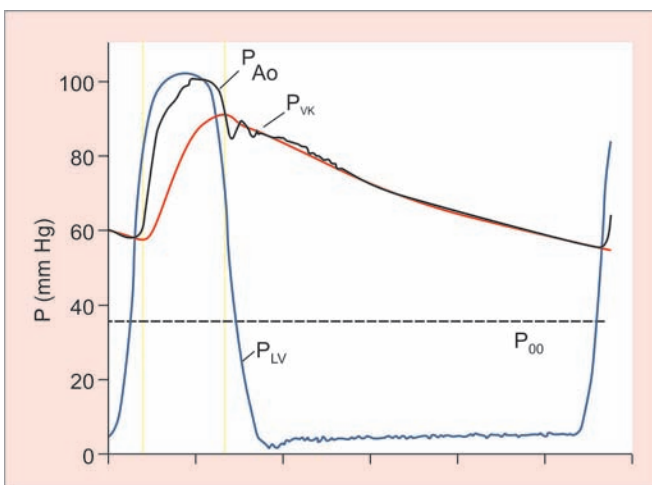
If we look at the volume flow, the majority flow is in systole. The flow in diastole in contrast, is very low in quantity as shown in Graph 3.

The arterial impedance is a ratio of pressure divided by the flow at any point of time. Most often it is the input impedance (the impedance at the entry of the circuit), is considered important in physiology. During systole there is high pressure (e.g., 120 mm Hg) and high flow with acceptable impedance in the healthy normotensive individual (Graph 4) (shaded area in systole).

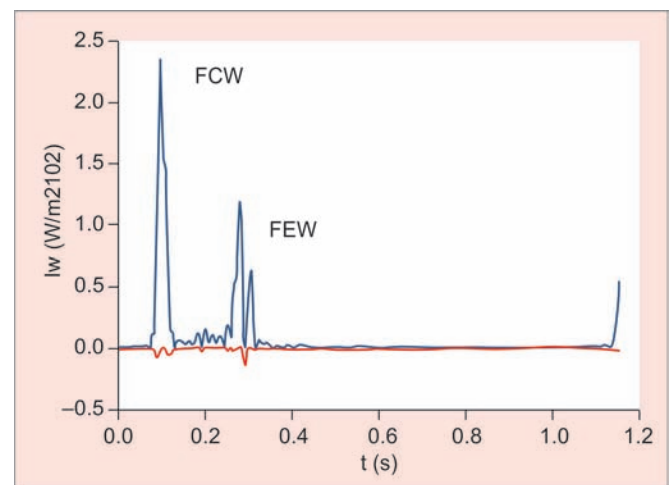
During diastole the pressure is lower (e.g., 80 mm Hg) but the flow is very low (driven only by compartment 2 recoil) (Graph 4). This means there is significant impedance even during ventricular diastole when apparently the flow is minimal (low pressure and very low flow).

Shaded Area in Diastole

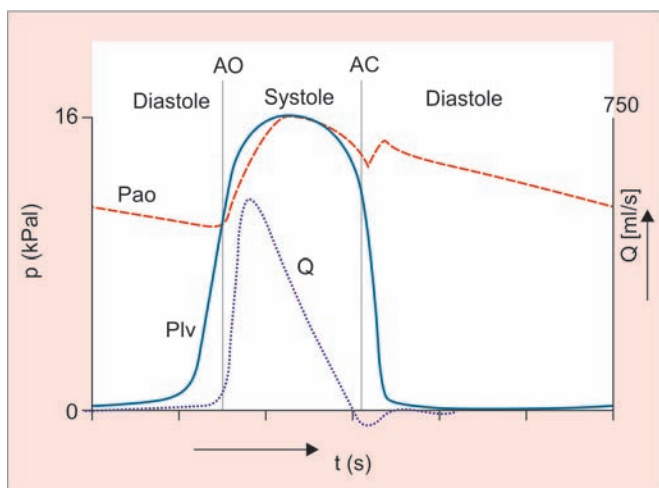
So when we consider the total impedance (burden of BP to the circulatory system), we should think beyond systole



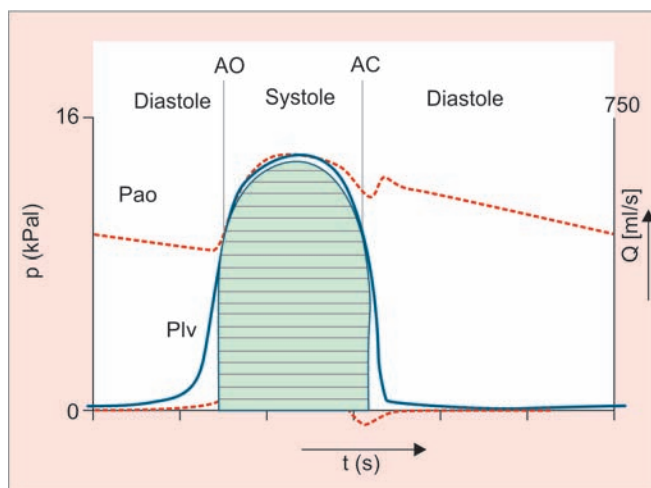
Graph 1: Left ventricular pressure curve and the aortic pressure; P_{Ao} : Aortic pressure; P_{LV} : LV pressure; P_{Wk} : Windkessel pressure (calculated)⁶



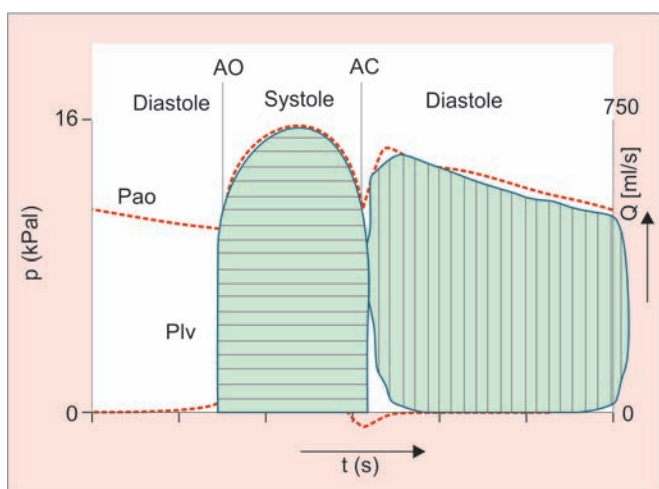
Graph 2: 0.5 rates⁶; FCW: Forward compression wave; FEW: Forward expansion wave



Graph 3: Flow in diastole⁶; Y-axis left: Pressure (P); Y-axis right: Flow (Q)

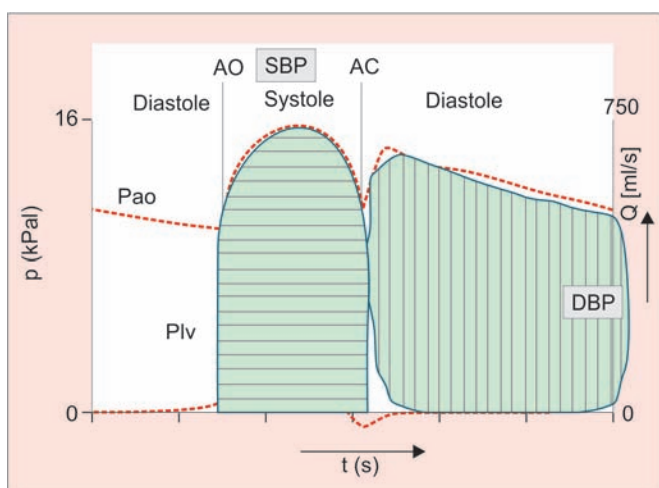


Graph 4: Shaded area in systole indicates systolic impedance (conceptual)



Graph 5: Horizontal shaded area (systolic); vertical shaded area diastolic impedance (conceptual)

[total shaded area (horizontal and vertical, Graph 5)]. Note that the area under the diastolic curve is actually larger than systole.



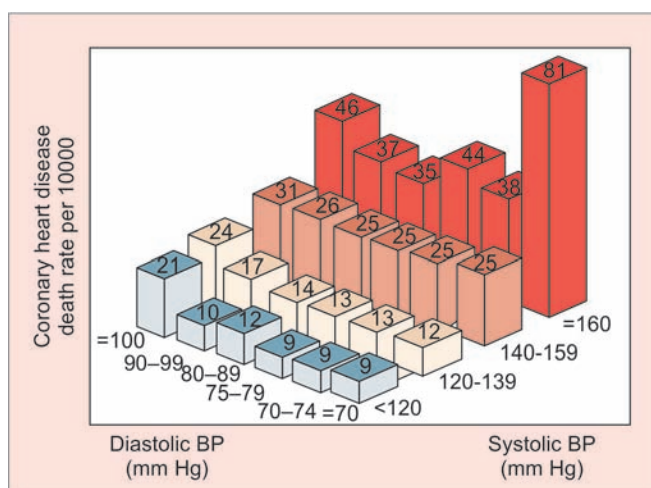
Graph 6: Area under the systolic and diastolic pressure shown. Addition of SBP (horizontal lines) with DBP (vertical lines) would predict BP burden better

CONCEPT OF BLOOD PRESSURE BURDEN OR BLOOD PRESSURE LOAD

The BP burden (BPB) can be considered as a load taken by the vascular system in total which would mainly be the impedance to flow, both systolic and diastolic (Graph 6). Since measurement of impedance is not available to the clinician at the bedside, the nearest figure would be to *add* up SBP number to the DBP number instead of subtracting it.

Initially, such a concept might look counterintuitive, but a little thinking makes it clear that it solves many problems of clinical management of hypertension.

The data of Graph 7³ shows that at any given level of DBP, increase in SBP increases CV risk. Similarly, addition of incremental SBP on any given DBP increase coronary heart disease event rate albeit to a lesser extent. This proves that a parameter which takes into account both the SBP and the DBP would be a better risk predictor compared with SBP or DBP alone.



Graph 7: Value of SBP and DBP in predicting CV death

Levels

A normal BPB should be $120 + 80 = 200$ mm Hg.

A target of therapy and goal being $140 + 90 = 230$ mm Hg.

Elder population age more than 65 BPB goal being $150 + 90 = 240$.

Advantage of BPB over SBP and DBP

- We know SBP and DBP independently predict vascular event risk, the predictability of SBP being more than DBP. A BPB (SBP + DBP) of >230 would include isolated systolic hypertension, systolo-diastolic hypertension, and isolated diastolic hypertension. The clinician needs to remember just one cutoff number. The moment this number is exceeded, the alarm bell should ring.
- High SBP with low DBP as seen in hyperkinetic circulatory state like thyroid disease and anemia or even aortic incompetence (e.g., 160/40) will no longer be unnecessarily classified as hypertension, since the BPB would still be $160 + 40 = 200$. This would avoid unnecessary drug therapy.
- Both SBP and DBP demonstrate a J curve, with event rates increasing as the SBP or DBP goes down below a specified level (110 systolic, 70 diastolic). A common problem is treating isolated systolic hypertension with high SBP and normal DBP (e.g., 160/70). Such BP on treatment might go down to 140/60. In an endeavor to achieve goal SBP, we push down the DBP to the tail of the J curve, and thus increasing event rate. A cutoff level of BPB of 200 would ensure that the DBP would not go down below a prespecified level. A J curve, systolic or diastolic would be quickly found out.

A patient of isolated systolic hypertension with a BP of 180/70 being treated by a drug, develops a BP of 140/50. The BPB would drop from 250 to 190. A cutoff of lowest attained BPB of 200 would warn the physician to a J curve problem.

- Many physicians consider mildly elevated systolic and diastolic hypertension as “borderline risk” and would not start treatment. Adding SBP to DBP would bring out a higher absolute number, which might enable a better mindset to start treatment.
- SBP and DBP are two different but independent risk factor. Adding the two would be justified as we risk stratify our patients like adding up diabetes, dyslipidemia, obesity, and smoking giving weightage to each individually.

CONCLUSION

Addition of SBP number to DBP number would give us a better predictable value of BPB. A single number BPB may be a better predictor of CV events compared with SBP, DBP, and PP. A BPB would also avoid pushing the patient to a low DBP (diastolic J curve) while treating high SBP. A meta-analysis of applying BPB to the available hypertension database would indicate whether it is worthwhile to use this value as a goal for diagnosis and a target for treatment endpoint.

REFERENCES

1. Ibrahim MM, Damasceno A. Hypertension in developing countries. *Lancet* 2012 Aug;380(9841):611-619.
2. Thomopoulos C, Parati G, Zanchetti A. Effects of blood pressure lowering on outcome incidence in hypertension. 1. Overview, meta-analyses, and meta-regression analyses of randomized trials. *J Hypertens* 2014 Dec;32(12):2285-2295.
3. Izzo JL Jr, Levy D, Black HR. Clinical Advisory Statement. Importance of systolic blood pressure in older Americans. *Hypertension* 2000 May;35(5):1021-1024.
4. Vasan RS. Pathogenesis of elevated peripheral pulse pressure: some reflections and thinking forward. *J Hypertens* 2008 Jan;51(1):33-36.
5. London GM, Pannier B. Arterial functions: how to interpret the complex physiology. *Nephrol Dial Transplant* 2010 Dec;25(12):3815-3823.
6. Wang JJ, O'Brien AB, Shrive NG, Parker KH, Tyberg JV. Time-domain representation of ventricular-arterial coupling as a Windkessel and wave system. *Am J Physiol Heart Circ Physiol* 2003 Apr; 284(4):H1385-H1368.