Review Article

DIASTOLIC BLOOD PRESSURE OR ACTUALLY IT IS BASELINE SYSTOLIC BLOOD PRESSURE?

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ABSTRACT

Blood pressure measuring represents a routine investigation in general medicine. Nikolai Korotkoff was only 31 years old when he made a short presentation to the Scientific Meeting of the Military Hospital of the Academy on 5 November 1905 concerning an easy non-invasive method of blood pressure (BP) measurement, entitled ‘Concerning the problems of the methods of blood pressure measurement’. If the pressure in the cuff is relieved, blood starts coming through the compressed arterial segment during systole and causes auscultatory sound and, the first sound, which, in Korotkoff’s opinion, is a measurement of systolic blood pressure. In the same BP measurement, when normal blood flow is fully restored, the auscultatory sounds disappear and, the last sound, which, in Korotkoff’s opinion is a measurement of diastolic blood pressure. Listening to Korotkoff sounds (K-sounds) to determine systolic and diastolic blood pressure (BP) has been the standard for noninvasive BP measurement in medical practices for nearly 100 years and it is the essential tool used for evaluation and assessment of patients with hypertension and risks of cardiovascular diseases (CVD) by physicians and nurses despite limited understanding of the nature of K-sounds. This article focuses particularly on the cardiovascular biomechanics of the first and last auscultatory sound and suggests two new terminologies; Highest systolic blood pressure and Baseline systolic blood pressure to represent the systolic pressure and diastolic pressure, respectively. Experimenting blood pressures on the basis of these two new suggested terminologies may reveal various additional undiscovered aspects of normal BP and abnormal BP.

KEY WORDS: Highest systolic blood pressure, Baseline systolic blood pressure, Korotkoff sounds.

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INTRODUCTION

Blood pressure measuring represents a routine investigation in general medicine [1]. Blood pressure means the force exerted by the blood against any unit area of the vessel wall [2]. Estimation of systemic arterial pressure by sphygmomanometry is an example of an empirically validated technique based on subjective judgments for acquisition of quantitative physiological and clinical data but despite its widespread use, our understanding of the mechanism by which the characteristic sounds are produced has not greatly improved since the technique was first reported by Korotkoff in 1905 [3]. Korotkoff was only 31 years old when he made a short presentation to the Scientific Meeting of the Military Hospital of the Academy on 5 November 1905 concerning an easy non-invasive method of blood pressure measurement, entitled ‘Concerning the problems of the methods of blood pressure measurement’ [4]. Listening to Korotkoff sounds (K-sounds) to determine systolic and diastolic blood pressure (BP) has been the standard for
noninvasive BP measurement in medical practices for nearly 100 years and it is the essential tool used for evaluation and assessment of patients with hypertension and risks of cardiovascular diseases (CVD) by physicians and nurses despite limited understanding of the nature of K-sounds [5]. If the main objective of medicine is to keep healthy people healthy and sick people well, then there is great deal of admiration for those men and women who have shaped our current medical knowledge and practice [6].

This article focuses particularly on the cardiovascular biomechanics of the first and last auscultatory signal routinely investigated and documented in the blood pressure diagnoses.

**Perspectives on Korotkoff sounds**

“Nokolai Korotkoff found that, after compressing an arm with the Riva-Rocci cuff until the distal pulse disappeared, and then gradually decreasing the pressure, a sequence of sounds could be heard under the artery distal to the compression. The origin of the sounds became the major object of discussion. Korotkoff believed that an artery cannot produce any sound when it is completely compressed, as well as uncompressed. If the pressure in the cuff is relieved, blood starts coming through the compressed segment during systole, and Korotkoff considered this to be the cause of the first sound and a measurement of systolic blood pressure. When normal blood flow is fully restored, sounds disappear, which, in Korotkoff’s opinion was a measurement of diastolic blood pressure” [7]. The Korotkoff sounds which identify systolic and diastolic blood pressure in the auscultatory method of measuring indirect blood pressure are acoustic signals which are just above the threshold of hearing when detected with a stethoscope placed distal to an occluding cuff [8].

Literature collections of a study [9] cited these mechanisms for origin of Korotkoff sounds; sudden stretching of arterial wall, turbulence, impact of blood on a stationary distal column of blood, transmission of heart sounds, flutter of the artery, cavitation, water hammer effect, amplification of perturbation by the mechanically unstable artery.

Morton E Tavel et al. supports the hypothesis that the initial Korotkoff sound is produced by rapid changes of pressure both beneath and distal to the compressing cuff, sufficient in rate to impart sonic vibrations to the vessel wall and surrounding tissues [10]. Opening tap generated as the rising intra-arterial pressure overcomes the obstructive force of the compression produced by the cuff and silences represent either laminar flow through a fully opened vessel, or absence of flow [11]. It was demonstrated that heart sounds and Korotkoff sounds are distinct and separate entities since they are separable in time and the vessel wall is not necessary to the production of Korotkoff sounds [12].

Based on these views, it is understandable that when the cuff is gradually deflated to remove its complete occlusion effect on the artery, at one point, the blood ejected during systole tends to re-enter the arterial portions distal to the cuff giving the first acoustic signal (first Korotkoff sound) with even the minimum available opening in the artery and accredited as systolic blood pressure. A combination of various individual-specific hemodynamic factors may be playing an important role in overcoming the resistance to flow through the compressed artery, in determining the point of occurrence of first, intermediate and last sounds heard through stethoscope. From the point of first acoustic signal onwards, the other acoustic signals are continuously heard, though the characteristics of sounds heard gets modified till the last acoustic signal in direct proportion to the reduction of the cuff pressure and regaining of the arterial diameter, possibly all during the systolic phase of heart. An important question arises here as to why the last acoustic signal heard just before complete elimination of occlusion effect of the cuff is also not considered occurring during systole or as systolic blood pressure but named as diastolic blood pressure since 100 years? Can human heart create Korotkoff sound at the last acoustic signal when it is in diastolic phase? An enhanced understanding of the cardiovascular biomechanics of this last acoustic signal can change our existing views about normal and abnormal blood pressures, and related medical treatments.
Diastolic blood pressure or Baseline systolic blood pressure?
The cardiac cycle consists of a period of relaxation called diastolic during which the heart fills with blood, followed by a period of contraction called systole. During the period of diastole, is there a possibility of cardiac output? If there is no scope for cardiac output during diastolic phase, then arterial pressure will be zero. Cardiac output equals arterial pressure divided by total peripheral resistance [14].

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\text{Cardiac output} = \frac{\text{Arterial pressure}}{\text{Total peripheral resistance}} \quad [14]
\]

Arterial pressure = Cardiac output \times Total peripheral resistance

Therefore, if cardiac output is zero, then arterial pressure will become zero during diastole, so we cannot call the last acoustic signal in the blood pressure test as diastolic blood pressure. There are various research standpoints that almost confirm that pressure in the artery may not be that high as we have been generally equating certain value (say, 80 mm Hg) corresponding to the last acoustic signal with diastolic blood pressure.

(i) At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left intraventricular pressures to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles, immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed [15].

(ii) Professor S. Talma of Utrecht concludes that the second sound of the heart, which is produced by the impact of a fluid column, the arterial blood, against membranous septa, the semilunar valves, originates in the vibrations of the blood, and not of the valves [16].

(iii) The negative diastolic left ventricular pressure is augmented by a decrease in end-systolic volume and by an increase in contractility and thus, left ventricular suction may play an important role in left ventricular diastolic filling [17].

(iv) The second heart sound begins significantly before aortic valve closure while forward flow is still going on, and is proportional to the magnitude of deceleration of blood flow [18].

(v) Phase I (period of filling) begins at a ventricular volume of about 45 milliliters (end-systolic volume) and a diastolic pressure near 0 mm Hg. At the end of period of ejection, the aortic valve closes and the ventricular pressure falls back to the diastolic pressure level and the ventricle returns to its starting point with about 45 milliliters of blood left in the ventricle and at an atrial pressure near 0 mm Hg [19].

(vi) The mammalian ventricle is capable of sucking blood in to its cavity during diastole [20].

(vii) Evidence for elastic recoil, minimum ventricular pressure and diastolic “suction” in the normal heart was reported by J. E. Udelson et al [21].

All these literature standpoints indicate that during diastole, there is less scope that the last auscultatory sound is created by diastolic pressure because the physiologic emphasis during diastolic phase is to refill the ventricles with blood for the subsequent systole. It is also well known that there are many silent portions too like many noise portions between the first and last auscultatory sounds noted in blood pressure measurements. Korotkoff considered the first sound created by the blood coming through the compressed segment during systole, as the cuff...
pressure is relieved, as the measurement of systolic blood pressure. It is highly possible that the sounds from the second to the last sound also belong to systolic activity and all the silent phases between first and last sound belong to diastolic activity (Figure 1 - 5).

Fig. 2: Schematic representation of occlusion effects of inflated cuff. 2a shows complete occlusion of the brachial artery caused by inflation of the cuff and 2b shows partial opening of the brachial artery caused by gradual deflation of the cuff.

Fig. 3: Schematic representation of systolic phase of heart with its blood fluid. 3a shows the streamline flow (SF) that naturally takes place. 3a also shows the fully deflated cuff with its proximal (P) and distal (D) ends. 3b shows no flow (NF) as the cuff is inflated to a level of complete occlusion of the brachial artery. 3c shows turbulent flow (TF) as the cuff is deflated and the artery begins to regain its original dimensions.

Fig. 4: Schematic representation of diastolic phase of heart with its blood fluid. 4a shows no flow (NF) as the cuff is deflated. 4b and 4c also shows no flow (NF) as the cuff is inflated and the brachial artery is in complete occlusion and partial occlusion, respectively. Regardless of the arterial diameter (fully opened or fully occluded or partly occluded), during diastolic phase, no flow (no strong flow) can be expected. But still, the blood flow caused by momentum of preceding systolic activity can be occurring during diastole also. NF here represents the lack of forward flowing strength with adequate volume of blood expected to create the arterial pressure. Diastolic suction effect can be one of the reasons for inability to create the arterial pressure during diastolic phase we routinely correlate with last acoustic signal in a blood pressure test.

Fig. 5: Schematic representation of systolic phase of heart with its blood fluid and partial occlusion of the brachial artery caused by inflated cuff. In such circumstance, the noise produced by turbulent flow (TF) can’t be listened by keeping the stethoscope (S) proximal to the cuff (5a) but by keeping the same distal to the cuff (5b). Hence, ausculatatory sounds are heard at the site of production of turbulent flow.
1) Sounds heard during auscultation are not continuous as there are multiple portions of silence, like multiple sounds between the point of appearance and disappearance of sound. During the periods of silence, the heart is in diastole or systole? If the point of the appearance of sound is considered caused by systolic pressure, why not the last sound also is considered caused by systolic pressure?

2) The scopes for minimal ventricular pressure and diastolic suction indicate the lack of possibility of blood flow during diastolic phase to create arterial pressure in the range routinely noticeable during the last sound in blood pressure measurements. So, during diastole, the heart does not pump out the blood (Cardiac output = Zero?) because the purpose of diastole is to refill the ventricles with circulating blood, though there may be some amount of blood flowing as a result of momentum created by the previous systole.

3) If all the sounds heard are considered caused by systolic activity, then the first sound can be called as 'Highest systolic blood pressure' and the last sound can be called as 'Baseline systolic blood pressure'.

**Upper and lower limits of blood pressure as noted in blood pressure tests**

Sounds are normally not heard when stethoscope alone (without blood pressure apparatus) is placed on the artery examined, because various mechanisms like turbulent flow and sonic vibrations has been believed for the production of Korotkoff sounds till complete regaining of the arterial diameter. As shown in this equation for Reynolds number, the tendency for turbulent flow increases in direct proportion to the velocity of blood flow, the diameter of the blood vessel, and the density of the blood, and is inversely proportional to the viscosity of the blood [22].

\[
Re = \frac{v \cdot D \cdot \rho}{\eta}
\]

Where \( v \) = mean velocity, \( D \) = vessel diameter, \( \rho \) = blood density, and \( \eta \) = blood viscosity

Turbulence does not begin to occur until the velocity of flow becomes high enough that the laminar flow breaks apart and therefore, as blood flow velocity increases in a blood vessel or across a heart valve, there is not a gradual increase in turbulence, instead, turbulence occurs when a critical Reynolds number (Re) is exceeded [23].

Very slight changes in diameter of a vessel can change its conductance tremendously in proportion to the fourth power of the diameter and based on Poiseuille's law, the diameter of a blood vessel plays the greatest role of all factors in determining the rate of blood flow through a vessel [24]. "Viscosity can be said to precede pressure and to be biophysically more fundamental than pressure. The relationship between blood pressure and viscosity is such that, given a constant systolic BP, if blood viscosity increases, then the total peripheral resistance (TPR) will necessarily increase, thereby reducing blood flow. Conversely, when viscosity decreases, blood flow and perfusion will increase. Because of the dependence of systemic arterial BP on cardiac output and TPR, if blood viscosity and TPR rise, systolic BP must then increase for cardiac output to be maintained" [25].

So, various biophysical factors having connections with Reynolds number and Poiseuille’s law control the upper and lower limits of blood pressure identified in blood pressure tests. Apart from these biophysical factors, posture, physical activity and clinical conditions can alter or determine the blood pressure limits. Increased arterial pressure by 30% and metabolic vasodilatation can increase muscle blood flow to a maximum of 25-fold during the most strenuous exercise [26]. Systolic and diastolic blood pressure was the highest in supine position when compared the other positions (sitting, standing, supine with crossed legs) [27]. Diastolic blood pressure (DBP) measured was significantly lower in supine position compared to standing and sitting positions [28]. “Systolic blood pressure (SBP) rise during exercise provides information about the hemodynamic response to increasing physical stress that is not available from SBP at rest. Dynamic exercise produces a large increase in SBP without much change in diastolic blood pressure” [29]. “SBP may remain elevated for a longer time if sympathetic tone does not decrease and vagal tone does not increase during the post-exercise
An exaggerated blood pressure response to heart rate during exercise is predictive of future hypertension independent of other important risk factors and lend further support to the concept that blood pressure measurement during exercise test is a valuable means of identifying normotensive individuals at high risk for developing hypertension [31]. Furthermore, hypertension accelerates the atherosclerotic process in carotid and vertebral arteries, which usually starts in the larger extracerebral arteries, particularly in the carotid bifurcation [32]. More clinical trials are needed before exercise SBP can be broadly recommended as an additional predictor for stroke [33].

“Numerous epidemiological studies have shown that the lower the blood pressure the lower the cardiovascular risk. On the other hand, intervention studies with antihypertensive agents in patients with systolic-diastolic or isolated systolic hypertension have shown that the antihypertensive treatment decreases the incidence of cardiovascular events: however it is still undefined which is the better blood pressure goal to reach during antihypertensive therapy. Observational studies and secondary analysis of large randomized trials have shown that treated hypertensive patients with diastolic values below some critical levels have a higher incidence of deaths and cardiovascular events. Studies on different populations evaluated with different protocols have given non-uniform results and many hypothesis have been suggested as causes of low diastolic pressure: 1) excessive antihypertensive treatment, 2) decreased compliance of aorta and large arteries and 3) clinical signs of a concomitant disease. Different studies suggest that diastolic blood pressure values lower than 70-80 mmHg, independently from the responsible physiopathological mechanisms, may rise the risk of cardiovascular events. Therefore, from the clinical point of view, extreme caution in the titration of the antihypertensive therapy for elevated systolic values when the range of diastolic blood pressure is below 70-80 mmHg.”[34].

CONCLUSION

Various research reports pointed out in this article clearly indicate the relationship between blood pressure, biophysical determinants of arterial pressure, diseases and the scopes for further understanding of blood pressure biomechanics. Identifying the individual-specific critical determinants of blood pressure as a result of natural and artificial vascular resistance (cuff-induced occlusion) can be deeply researched. This article indicates that all the sounds (including the last sound) heard during auscultatory BP test can actually belong to systolic activity because diastolic activity is meant for ventricular filling for the immediate next systolic activity, therefore, on this basis, the first sound can be called as ‘Highest systolic blood pressure’ instead systolic pressure and the last sound can be called as ‘Baseline systolic blood pressure’ instead diastolic pressure. Experimenting blood pressures on the basis of these two new suggested terminologies may reveal various additional undiscovered aspects of normal BP and abnormal BP. Highest systolic blood pressure can be considered as the highest hemodynamic response to overcome the artificial resistance caused by occlusion of artery for sustained circulatory functions, regulated by individual-specific biophysical and health determinants (like blood viscosity, blood volume, arterial diameter, fitness level and diseases) both at rest and during physical activity. Baseline systolic blood pressure can be considered as the hemodynamic response to overcome the natural resistance for sustained circulatory functions (in the absence of artificial resistance like cuff-induced occlusion), regulated by individual-specific biophysical and health determinants (like blood viscosity, blood volume, arterial diameter, fitness level and diseases) both at rest and during physical activity.

Conflicts of interest: None

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