Blood pressure (BP), not a constant number in any individual, varies from beat to beat. Variation occurs due to several factors emanating from person, environment, atmosphere, time of day, and season. BP variability (BPV) has been noted in both normotensive and hypertensive subjects, especially in the latter. Frattola et al., for the first time, reported the prognostic importance of BPV.¹

All parameters of BP are associated with sub-clinical organ damage culminating in end-organ dysfunction, either acutely or chronically. Most prognostic studies utilized absolute and mean BP values as indicative of target organ damage (TOD), with BPV not given due importance, probably due to the non-availability of a standardized method for its assessment. Lately, many observational studies and analysis of clinical trial data (post-hoc) have shown prognostic importance.²

BPV can be ‘physiological’, enabling an individual to cope with the stresses of daily life, or ‘pathological’, being a harbinger of diseases. No matter what time interval is taken for BP measurements, readings differ, at times, substantially. Different devices can record this variation. Initially, BPV was assessed intra-arterially, not applicable in routine practice. Non-invasively, beat-to-beat variability is measured by finger plethysmograph – an oscillometric method. Ambulatory blood pressure monitor (ABPM) and time-triggered home blood pressure monitor are used for 24-hour BP monitoring, whereas digital, mercurial, and aneroid BP monitors are utilized for day-to-day, i.e., home and visit-to-visit BP monitoring.

Depending on the duration of successive measurements, BPV is of five types: very short-term (beat-to-beat), short-term (within a day), mid-term (within a week), long-term (visit-to-visit), and very long-term (visit-to-visit > five years).³ A difference in the prognostic impact of these BPVs has been noted.⁴

In a systematic review of 33 studies (over one million subjects), Stevens SL et al. examined the association of three types of BPVs (short-, mid- and long-term) with various endpoints. They concluded: “Long-term variability in SBP was associated with risk of all-cause mortality (HR 1.15, 95% CI 1.09 to 1.22), CVD mortality (HR1.18, 95%C I 1.09 to 1.28), CVD events (HR 1.18, 95% CI 1.07 to 1.30), CHD (HR 1.10, 95% CI 1.04 to 1.16), and stroke (HR 1.15, 95% CI 1.04 to 1.27). Mid-term and short-term variability in daytime SBP were also associated with all-cause mortality (HR 1.15, 95% CI 1.06 to 1.26 and HR 1.10, 95% CI 1.04 to 1.16, respectively)”.⁵ The hazard ratio for BP variability with respect to cardiovascular disease (CVD) mortality has been found to be 1.18.

In a similar meta-analysis (41 cohorts), Diaz KM et al found modest association of visit-to-visit BPV with various endpoints, “for each 5 mmHg higher SD of SBP, the pooled hazard ratios for stroke across seven cohorts was 1.17 (95% CI:1.07–1.28), for CHD across
for four cohorts was 1.27 (95% CI:1.07–1.51), for CVD across five cohorts was 1.12 (95% CI:0.98–1.28), for CVD mortality across five cohorts was 1.22 (95% CI:1.09–1.35), and for all-cause mortality across four cohorts was 1.20 (95% CI:1.05–1.36).6

The prognostic impact of BPV has been noted in normotensives also. Liu M et al. studied 7065 patients with optimal systolic blood pressure (SBP) for MACE (major adverse cardiac events), which was higher by 21% in subjects with the highest SBP variability (quartile 4) as compared to those with the lowest (quartile 1) (HR 1.21; 95% CI, 1.09–1.35).7

For the assessment of BPV without any loss of prognostic information, at least 48 readings are needed.8

Various statistical parameters assess the variability of BP, the foremost being ‘standard deviation (SD)’, a measure of the dispersion of BP readings around a central mean value. The number of readings obtained during day and nighttime are sometimes different, and ‘dipping’ in nighttime BP is also noted. Many researchers have used another measure of ‘weighted SD’ to remove bias created by these two factors. To obviate the total dependency of BPV on the mean value, the ‘coefficient of variation (CV)’, calculated by dividing SD with the mean value, was advocated by some researchers, but this also did not totally remove the effect of mean BP. ‘Variation independent of mean (VIM)’ and ‘average real variability (ARV)’ obviate the effect of mean maximally. In VIM, SD is divided by mean BP raised to the power of x, which nullifies the correlation between these two. In ARV, the sum of differences between successive BP measurements is divided by the number of total readings minus one and has been shown to provide the best estimate of BPV.9

The therapeutic impact of BPV is yet to be established. However, certain studies have shown that Calcium channel blockers decrease, whereas beta-blockers and diuretics are inferior in this regards.10

Awareness of BPV amongst physicians could be better, as shown by Setia S et al. in a survey of 60 Singaporean physicians. Approximately 82% of physicians had no training for BPV.11

Although the literature is loaded with evidence for BPV, basic knowledge (definition, types, acquisition methods, advantages, and drawbacks along with prognostic information – total or differential), has not yet been detailed for a busy clinician. To remove this paucity, it is entirely rational to narrate a text where the subject has been explained in an understandable manner. Due to the very low application of this important phenotype of BP, the main objective of this review is to apprise the treating physician of the applicability of BPV in routine clinical practice, making the management of BP in the long term more stringent.

CRITICAL OVERVIEW OF LITERATURE

The appreciable spurt of clinical evidence has been noted for BPV. A PubMed search for this term fetched only three articles in 1948. Since then, the number of studies has constantly risen, reflected by 2754 studies last year and 1537 this year till this manuscript was written.

1. TYPES OF BLOOD PRESSURE VARIABILITY (PHENOTYPES)

Variability in BP is time-constrained and has been divided into five classes, as shown in Figures 1 (Ultra short-term and mid-term) and 2 (mid-term and long-term) with intervals for measurements mentioned in Table 1. Devices used for measurement of different types of variability and the advantages/disadvantages of each are shown in Table 1.

Figure 1: A- Ultra short-term BP variability. The red line is for systolic BP, and the blue line is for diastolic BP,
with dots representing BP values. B- Short-term variability recorded by 24-hour ABPM.

Figure 2: Upper panel: Mid-term variability recorded by HBPM. Lower panel: Long-term BP variability recorded by data on visit-to-visit basis, at least 3-10 records are needed for meaningful analysis. If record is available for more than five years in the same fashion, very long-term variability can be assessed.

Table 1: Variability types, devices used for recording, advantages, and disadvantages

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Type of variability</th>
<th>Time interval and duration</th>
<th>Device used</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Short-term</td>
<td>BP is recorded every 15-30 minutes for 24-48 hours.</td>
<td>ABPM/Time-triggered HBPM</td>
<td>Provides many phenotypes of BP. Low cost. Plethora of clinical studies available.</td>
<td>Repeated use is not possible. Disturbance during sleep may not depict night readings perfectly.</td>
</tr>
<tr>
<td>3</td>
<td>Mid-term</td>
<td>BP is recorded in the morning and evening for 7 days.</td>
<td>HBPM</td>
<td>Low cost and well accepted.</td>
<td>Provides BP reading during awaking and not during work/stress. Requires training and medical supervision.</td>
</tr>
</tbody>
</table>
2. PARAMETERS TO MEASURE BLOOD PRESSURE VARIABILITY

Initially, the standard deviation (SD) of BP records was taken as the criterion of variability. It is a measure of the dispersion of BP measurements around a central mean value, Figure 3A. Many drawbacks have been noted for SD as a measure of variability. Since SD and mean BP are correlated, both cannot be used in multivariate analysis simultaneously. SD gives one average value of the whole dataset, disregarding the sequence of change in variability.

In a 24-hour study of BP records, more measurements are obtained during daytime compared to nighttime. This preponderance of daytime readings has a biased effect on the 24-hour mean value (and any derived measurement). Nocturnal dipping of BP also creates bias. To obviate these confounders, another method has been introduced in which the measurements of day and nighttime are weighted according to their numbers, and the SD derived is known as weighted SD (wSD), Figure 3B.12

Another method to remove the dependency on the mean is the coefficient of variation, in which SD is divided by mean BP and expressed as a percentage, Figure 3C. Being a proportion, data recorded in different units can be compared by this parameter.

During routine chores, BP sometimes varies so abruptly that transient changes go unnoticed, which causes more target organ damage than steady state higher BP values. To catch these transient changes, another parameter came into vogue, ‘time rate of BP variation (TRBP)’ by Zakopoulos.13 In this, the mean of absolute ratios in the difference of successive BP readings and time between them are noted.

Two measures of assessment of BPV known as ‘variation independent of the mean (VIM)’ and ‘average real variability (ARV)’ totally disregard the mean BP, hence providing a better estimate of variability. VIM is derived by dividing SD with the mean, which has been raised to the power of x (derived from a fitting curve obtained by plotting mean SD against mean BP), rendering the correlation between the two almost zero, Figure 3D.

The ARV provides a better estimate of BP dispersion and therapeutic measures. It has shown greater predictive power for subclinical organ damage, mortality, and non-fatal events. ARV is the preferred index for very short-term and short-term variability assessment. Absolute differences between successive BP readings are summed up and divided by the total number of BP readings minus one, Figure 3E.

Although derived from the same data, the prognostic information obtained by these parameters differs. This has been explicitly shown by Mena LJ et al. in their remarkable meta-analysis of 19 studies comparing the predictive powers of these indexes.14

3. FACTORS AFFECTING BLOOD PRESSURE VARIABILITY

Many intrinsic and extrinsic factors affect BPV, varying with type. However, most specifically affects short-term variability, as shown in Figure 4 (blue ovals). Behavioral and environmental factors affect both types of variability (white margins), and drugs or inappropriate estimation of blood pressure (yellow margins) affect long-term variability more often.
Eating causes a slight rise in BP, which usually falls 1 hour later due to vasodilation in viscera and is seen especially after carbohydrate intake in elderly and hypertensive subjects.

Sodium causes an elevation in BP more at nighttime, especially in salt-sensitive patients, as excretion of this mineral also shows diurnal variation. Physiology of other minerals like potassium, magnesium, and calcium is exactly the opposite as they lower BP.

Neurohormonal factors in the form of sympathetic activation and baro-receptor reflexes play an important role in very short-term and short-term BPV. G Mancia et al. have elaborated on it in their study of 82 patients.

Hormones also have a role in the modulation of BPV. Catecholamines, Insulin, and Angiotensin II have been studied clinically. Vasodilators like bradykinin, nitric oxide, and endothelins cause an effect on BPV. These agents mostly affect very short-term and short-term BPV.

Seasonal alteration by sympathetic modulation mainly affects both short-term and long-term variability as higher blood pressures are recorded in winter, especially in the elderly.

Each 1-degree centigrade fall in temperature results in a 1% increase in mortality due to alteration in BP. A seasonal variation in SBP of 2.9/3.4 mmHg and 1.4/0.9 mmHg in the Northern/Southern hemispheres has been reported, respectively, from data across three continents and 24 studies (cross-sectional).

A working group of the European Society of Hypertension issued a consensus statement on seasonal variation in BP, mentioning the evidence and recommendations for clinical practice.

Behavioral factors, especially mental stress, physical activity, and postural changes, by producing changes in sympathetic activity, volume status, and cardiac output, affect both short- and long-term BPV.

Adherence to medical treatment and restriction of social habits (smoking and alcohol consumption) ensure effective control of BPV.

4. BLOOD PRESSURE VARIABILITY AS A PROGNOSTIC MARKER

The prognostic significance of BPV has been shown in a large number of studies. This has varied from individual target organs to cumulative evidence of damage, as demonstrated by all phenotypes. Intra-phenotypic comparison has also been done for various endpoints, showing mixed results. BPV has demonstrated clear superiority over other BP parameters like SBP, DBP, and mean BP.

- **Target organ damage:** Clinical evidence for target organ damage (TOD) has been noted in earlier studies. Gianfranco H et al. studied 108 hypertensive patients by intra-arterial 24-hour BP measurement. Parameters for LVH (ECG), cardiac volume (CXR), ocular fundi (Keith-Wegner classification), heart failure, cerebral and peripheral vascular insufficiency (clinical measures), and renal dysfunction (BUN, creatinine, and proteinuria) were scored for TOD. Short-term and long-term variabilities were assessed within half-hour SD and among half-hour SD of mean BP. High BPV, both short- and long-term (p < 0.05 and < 0.01), respectively, were found to have significant prognostic values for TOD.

The association of increased incidence of stroke due to arterial stiffness and carotid intima malformation with short- and mid-term BPV has been studied. Zhou TL et al., in 1671 patients, demonstrated that an increase of 1-SD in BPV (derived from a composite of short-, mid-and long-term BP record) increases carotid-femoral pulse wave velocity [0.10 m/s (95% CI, 0.01–0.20)], carotid circumferential wall tension (0.84 dyne/cm [0.51–1.17]), circumferential wall stress [0.79 kPa (0.31–1.27)], and intima-media thickness [8.6 µm (1.0–16.3)], but not carotid distensibility [−0.033×10⁻³/kPa (−0.255 to 0.190)]. This shows carotid maladaptation, which can result in stroke.

An increase in left ventricular mass is one of the targets of high blood pressure. Mustafa et al. demonstrated a significant association of short-term variability (by ABPM record) of nocturnal diastolic BPV (SD) with increased LV mass and hypertrophy. Similar associations were noted.
for 24-hour ARV and nocturnal diastolic ARV. An increase in LV mass is a harbinger of many cardiovascular complications.

#### Major adverse cardiovascular events

In a retrospective study of 42482 patients, Ebinger JE et al. searched for an association of BPV with multiple endpoints (MI, stroke, heart failure, and death), individually and cumulatively, over a period of six years. Data was obtained from digital health records corresponding to the real-life methods of BP recording and not the stringent confines of a robust clinical trial. The investigators obtained a significant correlation of VIM, SBP (HR, 1.22; 95% CI 1.17–1.28), and DBP (HR, 1.24: 95% CI 1.19–1.30) with endpoints. VIM was chosen as a reference parameter as other measures depend highly on mean BP. In this study, the association of BPV with clinical endpoints was demonstrated, and the utility of routinely obtained BP measurements was emphasized.

#### Mortality

Sierra ADL, in a Spanish study of 249 elderly in-hospital patients (>80 years of age), studied the relation of various parameters on 24-hour ABPM like arm and central BP (aortic), BP and heart rate variabilities, ratio of systolic and diastolic BP variabilities and aortic pulse wave velocity with 1-year mortality. Considering only BP variabilities, they found that a 1% increase in SBP variability raised the mortality rate by 38% (HR: 1.38: 95% CI: 1.06–1.80). Brachial BP was also associated with mortality significantly (HR: 1.31; 1.06–1.62). The researchers recommended a cautious approach in the administration of short-acting antihypertensive medicines to these hospitalized patients as variation in BP would be further enhanced.

#### Ischemic heart disease

Groove JS et al. studied 1433 men of the Honolulu heart program for a 10-year period, recording their blood pressures at four different times approximately three years apart. They studied the variation of SBP as a cause of coronary heart disease for a subsequent 11.3 years and found a significant correlation (p<0.001). This group demonstrated the variability of blood pressure with cardiac events. Soh MS et al. studied 343 post-PCI patients for long-term BPV for a median period of 76 months. Systolic BPV was 13.2 ± 7.6 mmHg and diastolic BPV was 8.9 ± 4.4 mmHg. Subjects were divided into two groups (high BPV >12.3 mmHg and low BPV < 12.3 mmHg) based on systolic BPV. They found a significant association only in the high BPV group for MACE, whereas no difference was noted for other study endpoints (recurrent MI, target vessel revascularization, and all-cause mortality), and neither an association was noted for diastolic BPV with any of the endpoints.

Weasel CL et al. studied 471 post-angioplasty patients (10 BP measurements in a span of 3-60 months), correlating visit-to-visit BPV (SD and largest change) with MACE (myocardial infarction, cerebrovascular accident, death, and all-cause hospitalization). Systolic BPV was significantly higher in patients who died or were re-admitted by both parameters, whereas diastolic BPV was significantly higher in subjects showing large change only.

#### Stroke

BPV affects the prognosis of patients after neurological insult markedly, especially in the early phase of acute ischemia/infarction. Higher BPV is associated with more significant residual disability. Control of mean BP and variability at this stage will lead to better neurological outcomes.

Naito H et al. studied short-term BP (by 24-hour ABPM) variability parameters (SD and CV of SBP, DBP, and morning surge) with functional status in acute ischemic stroke patients. Total cohort comprised of 626 subjects with 497 subjects analyzed at three months by modified Rankin score (mRS) for disability status. SD and CV of 24-hour SBP and DBP, along with morning surge and non-dipping pattern, were significantly associated with a worse mRS.

Han X et al., in a study of 137 patients with acute ischemic pontine infarct, found worse outcomes (assessed by modified Rankin score) in subjects with high BPV (assessed by CV of systolic and diastolic pressure on 24-hour ABPM record), paramedian location of infarct and higher National Institute of Health Stroke Score (NIHSS).

Chang JY et al. studied 90 patients who had undergone successful endovascular thrombectomy but had poor collateral circulation and found that BPV (assessed by SD, CV, and VIM of SBP and mean PR) during the first 24 hours after recanalization had a greater impact on functional outcome (assessed by early neurological recovery on day one and mRS at three months) in patients with poor collateral
circulation. This necessitates adequate control of BPV for effective functional recovery.

Neurological and cardiac insults in their earlier stages demand stricter control of BPV as these patients show fluctuation in BP levels more often in the first seven days. Control at this time makes recovery smooth and better for long-term functional recovery.

- **Cognitive function and dementia:** Chiu TJ et al., in a systemic review of 20 cohort studies (7 924 168 persons), found SBP variation assessed by CV and SD was associated with a higher risk of dementia of all causes, especially during shorter follow-ups in elderly subjects (> 65 years). Haverkamp RA et al., in a study of 279 elderly patients, assessed the risk of mortality with cognitive decline and BP variability by 7-day HBPM record. No correlation was found for day-to-day variability irrespective of the mean (VIM) for mortality and cognitive decline, but a significant association was noted for morning systolic BP VIM and mortality (adjusted HR: 1.09, 95%-CI 1.01–1.18, p = 0.033).

Dementia and cognitive decline are associated with small cerebrovascular disease (sCVD). This entity is identified on MRI by white matter hyperintensities, microbleeds, lacunes, and perivascular spaces’ enlargement. In a study of 82 middle-aged hypertensive patients, an association of BPV with sCVD was evaluated by de Heus RA et al. by HBPM. Significant association of systolic BPV and evening SBP was noted with sCVD. Association for DBP, mean BP, or morning BP was not significant.

In 3511 Chinese patients comprised of two groups [45-59 years (mid to old life) and 60 years or more (old to old life)], Xu et al. found a significant negative association between SBP variability and various parameters of cognitive function like orientation, language, recall, and total (assessed by Mini-mental state exam-MMSE). For every one-unit increase in SBP variability, the MMSE score decreased by 9.5 points. No such correlation was noted for DBP and pulse pressure variability. A significant association was stated only in the older group, emphasizing better control of SBP in this group to reduce cognitive dysfunction.

- **Heart failure:** Admission BP and variability in cases of heart failure have shown prognostic value with regard to mortality. A higher admission blood pressure is associated with a better prognosis, but a higher BPV in heart failure showed an adverse prognosis. Wei FF et al., in 1006 patients with acute decompensated heart failure (preserved ejection fraction), assessed mean admission BP, SD, and CV of BP during multiple hospitalizations for a median follow-up of 1.54 years. For every 1% increment in SD and CV of SBP, the risk of all-cause mortality increased by 10% and 11%, respectively (SD: HR, 1.10, 95%-CI, 1.01-1.21, P=0.029, CV: HR, 1.11, 95%-CI, 1.02-1.21, P=0.017), which further increased to 18% and 19% after adjusting confounders.

Similarly, patients with heart failure with reduced ejection fraction (HFrEF) also showed poorer prognosis regarding mortality and heart failure re-hospitalizations. Rossignol P et al. studied 3834 patients of HFrEF for 6.8 years divided into two groups receiving low (50 mgs) and high (150 mgs) doses of Losartan and assessed by visit-to-visit variability of blood pressure by SD, CV, and ARV. All parameters showed increased hazard ratios (HR: 1.023, 95% CI 1.013–1.034, P < 0.0001) for outcome irrespective of dose of Losartan.

- **Endocrinology:** Bisogni V et al., in their study of 23 patients of Pheochromocytoma and paraganglioma (PPGL), assessed short-term BPV parameters. Removal of PPGL showed "a significant decrease in 24-h systolic BP ARV (8.8 ± 1.6 vs. 7.6 ± 1.3 mmHg, p < 0.001), in 24-h diastolic BP ARV (7.5 ± 1.6 vs. 6.9 ± 1.4 mmHg, p = 0.031), and wSD of 24-h diastolic BP (9.7 ± 2.0 vs 8.8 ± 2.1 mmHg, p = 0.050) compared to baseline measurements." In a large Swedish study of 9855 diabetic patients followed up for a median of 4 years, the correlation of BPV (by SD, CV, and VIM) with cardiovascular and all-cause mortality was studied with no change in hypertension treatment. No significant association was noted for the entire group, but in the subset of patients not taking any antihypertensive medicine (n=2949), an association was noted for all-cause mortality with only a small increase in discrimination when this variable was added to other measures. This study doesn't support the use of BPV in this group of patients as a marker of mortality.

- **Chronic kidney disease:** The role of BPV in assessing CKD patients is taken with care as

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volume and pressure status vary significantly due to interventions (dialysis, etc.). However, in certain areas, BPV plays a significant prognostic role.

Hsieh MY et al., in their study of 1011 patients on regular dialysis, found BPV was associated with an increased risk of access thrombosis [HR= 1.27, 95% CI, 1.18–1.44, /SD increase in BPV]. The risk in patients was 2.45 times in the highest BPV quartile.

In the HEMO trial comprising 1844 patients on regular hemodialysis, Chang TI et al. studied the association of BPV (by CV and ARV) with all-cause and cardiovascular mortality. They found an 18% higher risk of death from any cause with every 10% rise in BPV by CV, and ARV also showed a similar association. "Black race, a history of heart failure and diabetes mellitus, catheter use, and having more frequent intradialytic hypotension are associated with higher visit-to-visit blood pressure variability."

Chia YC et al., in their long (15 years) retrospective study of 874 patients, studied the relation of BPV with a decline in eGFR. A significant negative correlation was noted (SD: r=0.16, p<0.001; CV: r=0.14, p<0.001). They found that an SD of 13.5 mmHg and CV of 9.74% were associated with the onset of chronic kidney disease.

Wang G et al., in their study of 245 non-dialyzed patients of renal failure (grade 1-4), found a correlation of high BPV (assessed by SD, CV, and VIM) with the progression of renal disease (seen only in unadjusted model and not in fully adjusted model) and cardiovascular disease but not with total mortality. They found hyperkalemia, LV end-diastolic diameter, hypertension, and BMI as markers of high BPV.

Kumanan T et al., in their one-year study of long-term blood pressure variability of 406 patients, found that female sex (p=0.023), DM (p=0.013), CKD (p=0.007), and the tendency for developing OSA (p=0.004) were associated with significant variability. A median value of 11.69 differentiated low and high variability groups. In multivariate analysis, only DM and CKD were significantly associated with high variability.

- **Obstructive sleep apnea:** Obstructive sleep apnea (OSA) is associated with hypertension. Steinhorst has studied the association of BPV and OSA, AP et al. in 107 patients.

Damage to targets of high blood pressure is accrued by every component of it (SBP, DBP, and mean BP), but the narration mentioned above shows clinical evidence of a higher damaging role of blood pressure variability, emphasizing its recognition and effective control.

5. **TARGETING BP VARIABILITY THERAPEUTICALLY**

Although the position of BPV as a prognostic marker has been clearly established, there are clinical lacunae in its implementation as a therapeutic target. First, there is no clear demarcation for normality at present; secondly, pharmacological trials especially directed against BPV, need to be improved. However, evidence is accumulating from earlier large-scale trials (ALLHAT trial) that calcium channel blockers reduce BPV significantly more than ACE inhibitors (lisinopril). In a retrospective analysis of MRC trial and ASCOT-BPLA trials, beta-blockers proved inferior to diuretics (Chlorthalidone) and calcium channel blockers (Amlodipine). Combinations of antihypertensive agents, including calcium channel blockers, are better at reducing BPV than combinations lacking this agent (COPE trial). The superiority of calcium channel blockers for modulation of BPV extends to all types of it and combination with ACEI/ARB, outperforming other combinations, especially those involving beta blockers. However, more dedicated research is still needed in this regard.

6. **DIFFERENTIAL PROGNOSIS OF BLOOD PRESSURE VARIABILITY PHENOTYPES**

The prognostic impact of the different phenotypes of BPV differs, as demonstrated by comparative clinical studies.

Tahir ZA studied 152 hypertensive patients retrospectively, comparing short-term (ABPM record) and long-term (home BP record) BPVs for a period of 10 years. The endpoints of the study were “acute coronary syndrome (ACS), chronic ischemic heart disease (IHD), heart failure (HF), or stroke”. Systolic BPV of day (OR=1.94; 95% CI=1.09–3.45; p=0.025) and nighttime (OR=1.23; 95% CI=1.00–1.51; p=0.048) showed significant association with IHD, whereas SD of visit-to-visit BPV was significantly associated with ACS (OR=1.10; 95% CI=1.01–1.21; p=0.04).
In another comparative study (n = 508) of long-term (visit-to-visit BPV) vs. short-term (24-hour BPV) for mortality in treated hypertensive patients (age > 65 years), Chowdhury EK et al. also showed differential results. SD of daytime SBP and SDw of 24-hour SBP were more significantly associated with mortality, whereas SD of visit-to-visit SBP was not. The low association of VV BPV with endpoint was attributed to a small number of patients by the researchers, as other studies have shown an association in this regard.

CONCLUSION

Blood pressure variability, a parameter of great prognostic importance, is yet to gain widespread clinical acceptance in daily practice. Long-term variability can be gauged easily by serial BP measurements obtained from patients' medical records. The application needs the knowledge of the treating physician in this regard, which is the primary purpose of this manuscript. Short-term variability assessment needs some sophisticated measures (home BP measurement or ABPM), which are now becoming more readily available with advancements in medical care. The medical community must refrain from showing inertia in using these gadgets and apply all the available parameters to ensure better health care. Reasonable control of BPV in acute phases of vascular events ensures better survival and limited damage in the long term.

Similarly, encouraging results can be obtained if this parameter is closely observed in cognitive and renal dysfunction areas. No specific therapeutic agent is available at present for BPV normalization, but there are clues from large-scale studies showing the efficacy of certain agents (calcium channel blockers). This area needs special attention for better clinical outcomes in the future. BPV phenotypes differ regarding prognostic capability in different clinical conditions, and variability of various blood pressure parameters also show prognostic differences for clinical endpoints. A thorough knowledge of these subtleties of blood pressure management will ensure high-quality care for hypertensive patients

AUTHORS’ CONTRIBUTION

IH: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. IH: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

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REFERENCES


Address for Correspondence:
Dr. Imran Hameed, Anklesaria Nursing Home, Sir Aga Khan III Road, Karachi, Pakistan.
Email: imranhameed61@hotmail.com

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