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Therapeutic strategies to reduce BP variability and its controversies

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Blood pressure variability (BPV) is defined by continuous fluctuations occurring within a 24 h period, including beat-to-beat, minute-to-minute, hour-to-hour, and day-to-night changes. Also, it has been shown to occur over more prolonged periods of time, for example days, weeks, months, and even years. These fluctuations are considered as the results of complicated interactions between extrinsic environmental factors and intrinsic cardiovascular regulatory mechanisms that are not yet completely understood. Although the cardiovascular complications of hypertension are thought to depend on mean BP values, evidence from observational studies and secondary analyses of clinical trials demonstrates that these outcomes might also depend on BPV. Indeed, these studies have shown that both short-term and long-term increases in BPV are associated with the target organ damage and with an increased incidence of cardiovascular events and mortality independent of hypertension. Therefore, targeting of antihypertensive treatment towards stabilizing BPV, in addition to reducing average BP, has been suggested.

Some clinical studies have been focused on reducing morning surge and restoring the nocturnal non-dipping pattern. Thus, drugs administration-time differences have been studied in order to normalize circadian BP profile and reduce short-term BPV. It has been suggested that the administration of ≥ 1 antihypertensive drug at bedtime could be associated with a better BP control and a greater reduction in CV risk compared with conventional therapy. Recent trials have demonstrated the beneficial effects of angiotensin receptor blockers bedtime intake on nocturnal BP dipping and within-day BPV. Also, the sleep-time administration of calcium channel blockers as monotherapy or fixed-combination therapy has been also associated with decreased BPV and morning surge of BP. Despite these evidences, it should be specified that the bedtime chronotherapy may not be recommended as a general approach for all hypertensive patients. However, it may be beneficial for subjects without nocturnal BP fall, in order to reduce circadian BPV. An individualized approach may represent the optimal strategy to manage CV risk in hypertensive patients. Given their role on long-term BPV control, lifestyle modifications including weight loss and salt reduction should also be taken as an effective strategy to reduce the CV risk in hypertensive patients. Thus, an independent effect of non-

pharmacological intervention on long-term BPV control cannot be excluded, suggesting the need of further researches. In addition, poor drug adherence has been hypothesised to be one of the most common factors affecting visit-to-visit BPV. Although the association between non-adherence and visit-to-visit BPV does not fully explain the relationship between BPV and CV risk, encouraging the drug adherence can be an important target for long-term BPV reduction.

There are some controversies about the therapeutic strategies for stabilizing BPV. Because we have not yet concluded whether BPV is a substantially true risk factor or a just bystander without an active role for cardiovascular and renal outcome. Furthermore, the clinical implications of BPV can vary depending on the method of assessment and the time interval examined. There are no established threshold to discriminate pathological from physiological BPV, together with the lack of a standardized method of BPV assessment. These limitations lead to the need of further trials in order to make the BPV available and easy to use as a routine approach in clinical practice.