DOI: 10.1111/jch.13708

COMMENTARY

WILEY

Evening blood pressure rise, from myth to reality

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Blood pressure (BP) lowering by antihypertensive drugs is a relatively safe option without serious adverse effects. And it can be easily adopted once BP elevation is associated with cardiovascular events. Moreover, regarding the sudden nature of vascular events or attacks, the closer the temporal link between these events and BP changes, the stronger the association. In this case, BP within the specific time window related to the attack could be targeted by BP-lowering therapy.

There have been many studies reporting the circadian patterns of myocardial infarction and stroke.[1-5] Most reported a single peak in events in the morning, but some controversy remains.[1] Symptoms upon awakening could reflect accumulated events during sleep periods such that events counted in the morning could be overestimated.[2] Some studies reported an additional minor peak of acute myocardial infarction and stroke in the evening, especially in Asian populations.[3,4,6,7]

The biological plausibility of a morning peak is largely based on the intrinsic circadian rhythms of the autonomic nervous system and hormone and/or coagulation systems, which are superimposed through additional psychosocial activation in the morning.[8,9] However, regarding the minor peak in the evening, there is no neuronal evidence supporting intrinsic ultradian components in the biological clock [10] even though many studies favored a multiple-component single cosinor model.[8] Several extrinsic factors such as stress, physical activity, smoking, and alcohol drinking are temporally intermingled with potential ultradian rhythms such as appetite, blood circulation, hormone, and more. Moreover, the presence of a circasemidian rhythm with 12-hour cycles could be interpreted as the simple first-order harmonics of a circadian biological clock or rhythm generator.

Murakami et al[11] reported a minor rise in BP in the evening clearly demonstrated by 7-day consecutive ambulatory BP monitoring (ref). This study has meaningful clinical implications in several aspects. First, the time when rhythmicity started was set as the time when the patient awoke instead of clock time. Second, they adopted an ultradian rhythm with a 6-hour cycle, on which the reference time interval from which the 3-hour evening BP rose was set to 6 to 9 hours after awake.[12] Third, unusual 7-day ambulatory BP monitoring could effectively prove the real-world presence of an ultradian or circasemidian peak of BP in the evening regardless of the origin of the rhythm from the harmonics of circadian rhythm or from extrinsic factors.

Regarding an interpretation scheme on this rhythmicity, there are two apparently competing ideas. One is the blunting of the circadian rhythm represented by non-dipper is harmful for the cardiovascular system with which the blunting of a few ultradian rhythms were also observed.[13] The other is the exaggeration of an ultradian rhythm with 4- to 6-hour cycles represented by a morning surge or evening rise could be harmful for the cardiovascular system. Conceptually, the former is focusing on cumulative damage by blunting the rhythm, whereas the latter is focusing on direct triggering of a cardiovascular attack.[14] Triggering can be assumed by a tight temporal link between the rhythm and the event. As expected by the design of this study, extrinsic factors with an effect on BP with several-hour cycles, such as alcohol and smoking, were associated with a pre-awake morning surge. As shown in a systematic review, temporal changes in blood pressure depend on the time lapsed after alcohol ingestion.[15] Blood pressure changes during sleep in smokers could be a manifestation of nicotine withdrawal for the several-hour period interfering with sleep more and more as the morning comes.[16] Hence, these two factors could set separate BP change cycles spanning several hours.

With regard to evening rise, sustained smoking cessation for a several-hour period and alcohol drinking in the daytime are unlikely causes. In this study, as expected, there seemed to be complex interplay between vascular stiffness and the half-life of antihypertensive drugs excluding smoking and alcohol drinking as related factors. In general, the causality between eGFR and increased diastolic BP or higher baPWV is unclear because eGFR within this range does not necessarily mean volume retention, but rather could mean vascular aging. As in the case of morning surge, vascular stiffness could exaggerate BP changes in response to extrinsic factors in the evening.

For more active clinical application or investigation of evening BP rise, reconsideration of the optimal measurement window for evening BP during home BP monitoring, and further studies to

find out when to measure evening BP (before sleep, in the evening, or 12 hours after awakening) will be of value. The feasibility issue related to multi-day ambulatory BP monitoring for rhythmology could be addressed by adopting recent cuff-less technologies, which should be complemented by standard ambulatory BP monitoring.[17,18]

In summary, based on 7-day ambulatory BP-monitoring data, an evening rise in BP could provide a very strong explanation for the minor epidemiologic peak in cardiovascular events in the evening. Evening BP rise could be affected by insufficient antihypertensive medication and vascular stiffness, which is contrasted with the pre-awake morning surge, which is mainly affected by lifestyle factors. This calls for further investigation of antihypertensive drug administration to manage evening BP and to prevent vascular aging or stiffness.

CONFLICT OF INTEREST

The author has no conflict of interest to declare.

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