Does Acute Catastrophic Psychological Stress Disrupt Diurnal Cardiovascular Variability?

To the Editor:

Prof Parati et al \(^1\) reported an interesting case in which ambulatory blood pressure (BP) monitoring was performed at the time of an earthquake (4.7 on Richter scale), which demonstrated than acute catastrophic psychological stress can raise BP and heart rate in an ambulatory setting.

In a recent study, \(^2\) we investigated earthquake-induced BP elevation in elderly hypertensive outpatients (mean age, 69 years) living near the epicenter of the 1995 Hanshin-Awaji earthquake (7.2 on Richter scale). In this population, in which BP had been measured before the earthquake, clinic systolic and diastolic BP were 14 mm Hg and 6 mm Hg higher during the first 2 weeks after the earthquake while major aftershocks persisted, but returned to baseline by 3 to 5 weeks.

There is thus a discrepancy between Dr Parati’s case and our hypertensive patients. In Dr Parati’s case, although pronounced BP variability persisted throughout the following 6 hours, the earthquake-induced BP elevation lasted only 1 hour. In our study, \(^2\) the BP elevation varied according to whether the patients had microalbuminuria: in those who did not, BP returned completely to the baseline value 4 weeks after the earthquake, but in those who had microalbuminuria, the BP increase was prolonged for at least 2 months. Microalbuminuria is closely related to endothelial cell dysfunction, and the different time courses in BP recovery after the earthquakes between Parati’s case and our report might be owing to differences in endothelial cell function and to the persistence of aftershocks. Parati’s patient was a young (34-year-old) normotensive (BP 130/85 mm Hg) woman without any target organ damage.

Even in the absence of target organ damage, acute stress might affect nocturnal BP. Parati’s case seems to show a nondipping pattern from inspection of his figure, and the absence of a nocturnal reduction in heart rate suggests persistent sympathetic activation during the night and poor sleep quality. Our recent studies have suggested that in addition to poor sleep quality, \(^3\) psychological factors such as subclinical depression may contribute to nondipping of nocturnal BP.\(^4\)

This earthquake-induced nondipping status might also be related to the nighttime onset of cardiovascular events. In our previous study of the 6 districts in the Awaji Island near the epicenter, coronary heart disease (myocardial infarction and sudden death within 24 hours) and stroke were increased 1.5-fold and 1.9-fold, respectively, during the 3-month period after the earthquake compared with the same period of the previous year.\(^5\)\(^6\) This stress-induced increase in cardiovascular events was observed only in the period from midnight to early in the morning, and there was no increase of cardiovascular deaths during the active daytime period, from noon to midnight. And a previous paper\(^7\) reporting on the time of onset of acute myocardial infarctions found that 53% of depressed patients compared with 20% of nondepressed patients reported an onset of symp-toms between 10:00 PM to 6:00 AM.

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References


Response

The observations made by Drs Kario, Shimada, and Pickering in their letter\(^1\) on the possibility that blood pressure changes might persist not only for hours but also for weeks or months after a catastrophic seismic episode, thus leading to an increased rate of cardiovascular events, represent an important complement to our case report.\(^2\)

It is not surprising that a threatening event such as an earthquake, which is often characterized by shocks of variable intensity that repeatedly occur over several days, might have both short-lasting and more sustained effects on cardiovascular vari-ables. These effects may differ in different subjects and at the time of different behaviors, and the evidence collected by Kario et al.\(^5\)\(^6\) that the earthquakes that hit Japan in recent years had an impact of different magnitude and duration on the cardiovascular system in hypertensive subjects as a function of their degree of target organ damage may indeed reflect one of the mechanisms underlying the different patterns of cardiovascular “adaptation to stress”.

These observations deserve a few comments. First, the occurrence of a cardiovascular sympathetic hyperre sponsiveness after an earthquake is also supported by our report, which offers unique information on the time course of the acute effects of a major seismic episode on ambulatory blood pressure mean values and variability in real life conditions. As shown in our paper, beyond the peak blood pressure and heart rate increases observed at the time of the strongest strike (increases that were “sensitized” by the preceding milder earth shocks),\(^2\) our patient also displayed a more pronounced blood pressure variability over the following hours. An increased blood pressure variability has been shown to reflect a higher level of sympathetic cardiovascular modulation.\(^6\)\(^7\) which in our patient might have resulted in a lower threshold of blood pressure reactivity to milder aftershocks. Because of the dynamic nature of our observations, we have been able to document the time course of these acute changes in blood pressure hour by hour after the
acute seismic event, showing a return toward baseline ambulatory blood pressure levels about 6 hours after the strongest quake. The pattern of these acute blood pressure and heart rate changes obviously does not exclude the occurrence of persistently elevated office blood pressure readings weeks after the seismic episode.8 This might be owing to a persistently enhanced “white coat effect,” as a result of a catastrophe-induced sensitization to stress (possibly further enhanced by the recurring milder quakes that followed the main seismic event), leading to a more pronounced sympathetic responsiveness to even mild stressful events (as in the case of consultation in the physician’s office),9,10 even weeks or months after the acute seismic episode. Second, an acute stressful event, such as a strong earthquake, might have an important impact on nocturnal blood pressure levels. Kario et al11,12 suggest that the resulting nondipping status may in itself be responsible for the reported increased rate of cardiovascular events after a seismic event. Indeed, a persistent state of enhanced anxiety and depression might contribute to a persistently altered nocturnal behavior of blood pressure, which might in turn favor a higher rate of cardiovascular complications. Our data do not allow us to draw any definitive conclusion in this regard. We do believe, however, that some caution is needed before concluding for the systematic occurrence of a nondipping status after an earthquake. In our case report5 it seems indeed much more likely that the patient, rather than being a nondipper, was more simply a nonsleeper, as probably anyone would have been in the night immediately after a major earthquake that had seriously damaged the house in which he or she was trying to fall asleep. In fact, the degree of nocturnal blood pressure fall is known to depend on several mechanisms, among which the depth and duration of sleep play a major role.13,14 Because our case report was based on an isolated ambulatory blood pressure recording, we are unable to conclude either in favor for or against the occurrence of a reproducible and persisting nondipping pattern of blood pressure during sleep after an earthquake. Our data, however, also on the background of patient’s diary, strongly suggest that the lack of nocturnal blood pressure fall reflected the effects of an acutely disturbed sleep pattern, induced by the recent occurrence of a strong seismic episode.

Finally, the methodological considerations we made in our paper on the usefulness of ambulatory blood pressure monitoring in documenting the effects of stressful events on daily life blood pressure levels and variations2–15 may be relevant to the data reported by Kario et al. In fact, our data on the increase in blood pressure variability after a major seismic episode and the available evidence on the role of a higher degree of blood pressure variability in determining a higher rate of cardiovascular complications16–21 are in line with the finding of Kario et al of more pronounced increases in blood pressure levels after an earthquake at the time of a physician’s visit in those hypertensive patients who display signs of target organ damage. An enhanced degree of sympathetic drive to the cardiovascular system, as our data suggest to take place after a strong seismic event, may actually lead to important increases in blood pressure variability, as we were able to show some years ago, by performing beat-to-beat 24-hour ambulatory blood pressure monitoring in patients with a phaeochromocytoma22 and in patients facing major stressful conditions in their daily life.23 This further underlines the importance of ambulatory blood pressure monitoring as a tool able to provide us with a unique insight into the features of neural cardiovascular regulation during daytime and nighttime activities.24

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