

Management of Cardiovascular Risk in Disaster: Jichi Medical School (JMS) Proposal 2004

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Abstract

There is growing evidence that psychological stress contributes to cardiovascular disease. In proportion to the damage of the disaster, the number of cardiovascular events increases in a high-risk older population. Typical increase was found during nighttime, suggesting that poor sleep quality might affect disaster-induced cardiovascular events. Acute stress can trigger cardiovascular events predominantly through sympathetic nervous activation and potentiation of acute risk factors (blood pressure increase, endothelial cell dysfunction, increased blood viscosity, and platelet and hemostatic activation). Chronic stress resulting from environmental change contributes to the atherosclerotic process through the neuroendocrine and immune systems (sympathetic nervous system and hypothalamus-pituitary adrenal axis) and related chronic risk factors (metabolic syndrome, hypertension, diabetes, and hyperlipidemia). In this Jichi Medical School (JMS) Proposal 2004, we propose the practical management of disaster-induced risk factor and stress, and hope immediate management could achieve effective primary and secondary prevention for cardiovascular disease in disaster.

Key words Disaster, Stroke, Coronary artery disease, Sudden death, Hypertension, Sleep quality

Introduction

In recent years, both animal and human studies have demonstrated that psychological stress can influence chronic disease processes such as hypertension and atherosclerosis and trigger cardiovascular disease (CVD) events.^{1–7} In humans, there are substantial individual variations in the perception of stress and in the subsequent physiologic responses, which mean that the consequences are not uniform across all individuals. However, unanticipated catastrophic natural disasters like the major earthquake and its sequellae are among the strongest acute and subacute psychological forms of stress. Several reports have shown that the incidence of fatal and non-fatal CVD such as stroke, and coronary heart disease (CHD) including unexplained sudden death increased at the time of the

Hanshin-Awaji earthquake.^{8–12} Further, in the more recent terrorist disaster of September 11, 2001 cardiovascular consequences were also observed to have occurred more frequently.¹³

In this paper, we discuss the effects of psychological stress on cardiovascular risk factors and how it can affect the CVD, based mainly on the findings of the Hanshin-Awaji and other earthquakes.

Hanshin-Awaji Earthquake

At 5:46 am on January 17, 1995, the southern part of Hyogo Prefecture, Japan, was struck by a major earthquake measuring 7.2 on the Richter scale. This was a typical earthquake in that it was most strongly felt directly above the epicenter. It caused 5,488 deaths and tens of thousands of casualties. The epicenter, the Awaji-Hokudan district of the Awaji Island, was one of the most

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heavily damaged districts. About one-third of the houses in the district were completely destroyed, and about one-third of the residents temporarily moved to shelters. In the Awaji-Hokudan district, which has a resident population of about 11,500, there were 10 CVD deaths during the 6 weeks immediately following the earthquake, as compared to 3 during the same period in the previous year.

Disaster-induced CVD

It is well known that acute CVD events (acute coronary syndrome and stroke) can be triggered by abrupt emotional or physical stressors such as intense anger or physical exercise.³ An increase in the CHD deaths following a major earthquake has been reported on several occasions.^{14–16}

The Jichi Medical School Cohort Study (JMS Cohort Study) is a longitudinal study of cardiovascular risk factors that started in 1991 involving

Table 1 Characteristics of disaster-induced cardiovascular disease

- The number of cardiovascular events (stroke, coronary artery disease, cardiac sudden death, and pulmonary embolism) increase.
- The increase persisted for 2–3 months.
- Cardiovascular events were more common in high-risk elderly subjects.
- Cardiovascular events occurred in proportion to degree of disaster damage.
- Increases occurred during the night and in the morning.

people living in the Awaji-Hokudan district, which is near the epicenter of the Hanshin-Awaji earthquake.¹⁷ In the 6 districts in the Awaji Island near the epicenter, we investigated earthquake-induced CVD, and summarize the characteristics in Table 1. Both stroke events and CHD events (myocardial infarction and sudden death within 24 hours after the onset) increased 1.9-fold and 1.5-fold respectively during the 3-month period after the earthquake, when compared with the same period in the previous year.^{11,12} Pulmonary embolism may occur during this period. This increase in earthquake-induced CVD was predominantly found in a high-risk elderly population, and the frequency of earthquake-induced CVD death in each district was positively correlated with the earthquake-induced damage to that district (Fig. 1).

The number of CVD deaths, specifically, the number of subjects with acute myocardial infarction during the first 4 weeks after the Hanshin-Awaji earthquake was 3.5 times higher in the region.^{9,10} The increase was significantly greater in women than men, and the mean post-traumatic stress disorder reaction index score was also significantly higher in women.

Persistent Stress and Disaster-induced CVD

The estimated duration of the influence of the Hanshin-Awaji earthquake on CVD events differs from the findings of previous studies. Thus, in the Athens earthquake,¹⁴ the Newcastle earth-

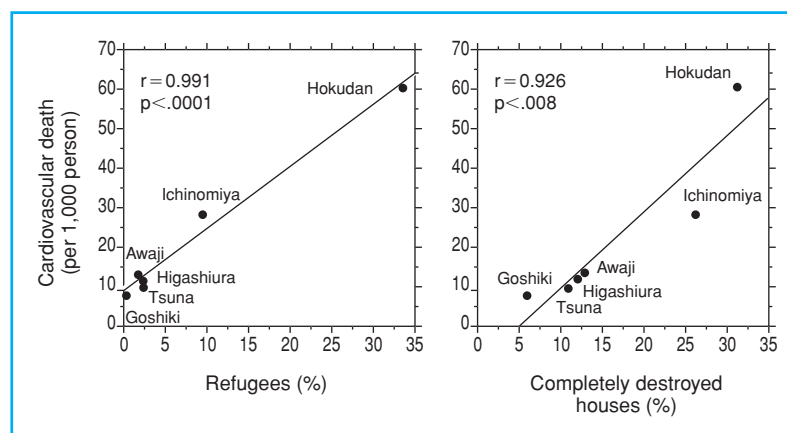


Fig. 1 Correlation between damage of the Hanshin-Awaji earthquake and cardiovascular death around the epicenter

quake of Australia,¹⁵ and the Northridge earthquake of Los Angeles,¹⁶ the increase in CVD deaths was limited to a few days. In the Northridge earthquake, after a few days of increased CVD deaths following the earthquake, the subsequent death rate was lower than the baseline rate.¹⁸ However, in our study of the Hanshin-Awaji earthquake, the increase in CVD deaths persisted for around a month.¹⁹ This difference may be due to the characteristics of the study population, and to the duration of the stressor. In the study of the Newcastle earthquake, CHD death was investigated only in younger adults aged 70 years or less. However, in our study, more than 30% of the subjects were over the age of 60 years, and more than 90% of subjects who died of cardiovascular events after the earthquake were 70 years or older. Furthermore, our study was only concerned with the most heavily damaged area.

Persistent stress resulting from extensive damage to the environment could explain the persistence of increased CVD deaths after the earthquake. Following the World Trade Center attack on September 11, a study of the frequency of ventricular arrhythmias among patients fitted with an implantable cardioverter defibrillator (ICD) at the time of the destruction, showed that ventricular arrhythmias increased more than 2-fold among ICD patients.²⁰ The first arrhythmic event did not occur for three days following 9/11, with events accumulating in a progressive non-clustered pattern. The delay in onset and the non-clustered pattern of these events differ sharply from effects following other disasters, suggesting that subacute stress may have served to promote this arrhythmogenesis.

These results indicate the impact of chronic stress by environmental change on CVD and the importance of the immediate reduction of chronic stress to achieve effective prevention of disaster-induced CVD.

Diurnal Variation in CVD Onset

There is a marked diurnal variation in the onset of CVD events, with peak incidence of myocardial infarction, sudden cardiac death and ischemic and hemorrhagic stroke occurring in the morning (between 6 am and noon).¹⁻³

This normal diurnal variation of CVD onset may be modified in disaster situation. Figure 2

shows the distribution over 24 hours of the onset of the earthquake-induced CVD deaths in comparison with the rate in the year preceding the earthquake.^{11,12} In the year before the earthquake, both CHD events and strokes occurred more frequently in the period from early morning to noon than at other times of day. After the earthquake, CVD deaths during this period were further increased. However, the most prominent increase was observed in the period from midnight to early in the morning. On the other hand, there was no increase of CVD deaths during the active daytime period, from noon to midnight.

In this context, it is of interest to note that a previous study of the timing of the onset of acute myocardial infarctions found that 53% of depressed patients, as compared with 20% of non-depressed patients, reported an onset of symptoms between 10:00 pm and 6 am.²¹ In addition, in patients with sleep apnea syndrome, cardiac sudden death markedly increases during the sleep period.²² Thus, depression or sleep impairment caused by the earthquake, which are closely associated with each other, might contribute to these nighttime onset cardiovascular deaths. In a

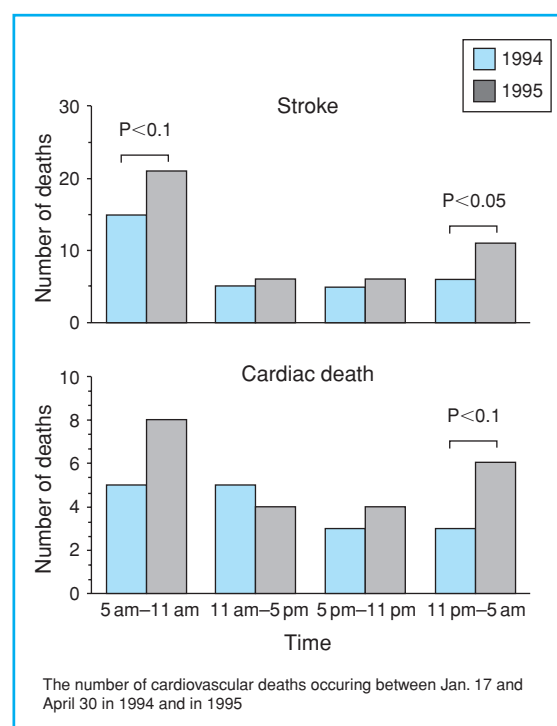


Fig. 2 Onset of cardiovascular death after Hanshin-Awaji earthquake of Jan. 17, 1995

general population, approximately 15% of CVD events occur during the night, which is a lower rate than at other time periods.

Stress-induced Potentiation of Acute Risk Factors

Recent advances in the field of neuroscience have greatly improved our understanding of how the brain perceives and responds to stress, and how it can affect various target organs such as the brain itself, the cardiovascular system, and the immune system.^{1,2} Table 2 and Fig. 3 show the

characteristics and mechanism of disaster-induced potentiation of acute risk factors, respectively. The neuroendocrine system, autonomic nervous system, and immune system are mediators of adaptation to challenges of disaster stress. The first physiological mediators such as noradrenalin from sympathetic activation, glucocorticoids from hypothalamus-pituitary adrenal (HPA) axis activation, and cytokines from cells of the immune system act upon receptors in various tissues and organs.

Risk factors associated with sympathetic nervous activation could be considered as acute risk

Table 2 Disaster-associated potentiation of cardiovascular risk factors

| | |
|---------------------|---|
| Blood pressure | <ul style="list-style-type: none"> • transient increase (decrease 2–4 weeks after disaster) • by 15 mmHg of systolic pressure • correlated with white-coat effect • in smaller degree in diabetics and hypertensives treated with antihypertensives inhibiting sympathetic activity • for longer time in those with microalbuminuria |
| Thrombotic tendency | <ul style="list-style-type: none"> • hypercoagulable and hyperfibrinolytic state • increased blood viscosity (hematocrit and fibrinogen) • endothelial cell dysfunction • poorly controlled in those treated with anticoagulation (warfarin) |
| Inflammation | <ul style="list-style-type: none"> • increased inflammatory reaction |
| Lipid profile | <ul style="list-style-type: none"> • unchanged in relatively shorter term (may worsen in longer term) |
| Blood sugar | <ul style="list-style-type: none"> • increase in plasma glucose and new-onset diabetes • poorly controlled in treated diabetics |

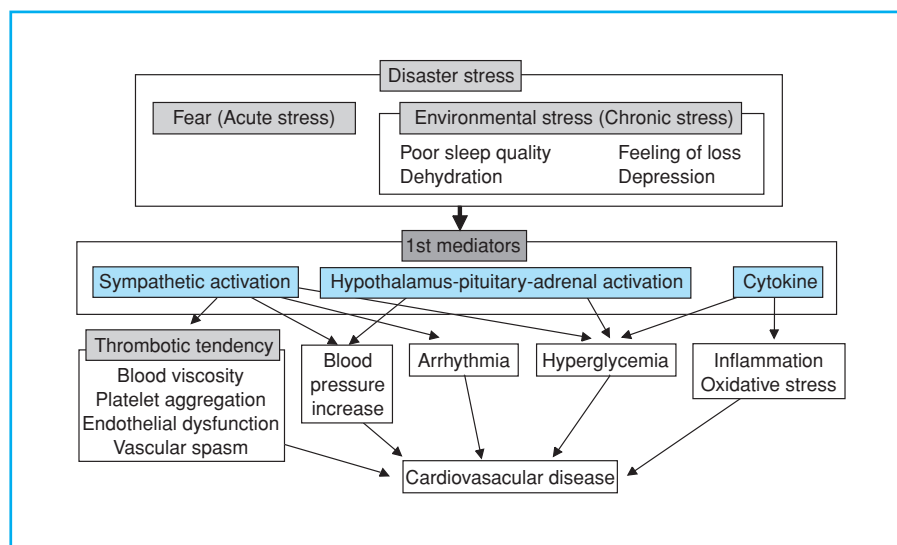


Fig. 3 Mechanism of disaster-induced cardiovascular disease

factors, which trigger CVD events.²³ In contrast to chronic risk factors which advance the atherosclerotic process like hypertension, diabetes, dyslipidemia, and smoking these acute risk factors include: 1) transient blood pressure (BP) increase, 2) endothelial cell dysfunction, 3) increased blood viscosity, 4) platelet activation, and 5) imbalance between coagulation and fibrinolysis (augmented procoagulant activity and impaired fibrinolytic activity).

Early morning activation of the sympathetic nervous system and HPA axis potentiates several sympathetically mediated risk factors such as the vulnerability to arrhythmias, and various clinical and subclinical cardiovascular events, described below.²³

1. Disaster-induced BP increase

There are several reports that BP and heart rate

are increased at the time of a disaster. In our study of well-controlled hypertensive patients, increases of approximately 18 mmHg in systolic BP and 8 mmHg in diastolic BP were found during the second week following the earthquake, when compared with BP levels before the earthquake²⁴ (Table 3). In addition, further immediate BP increase might have occurred as a result of felt after-shocks that triggered CVD events. As shown in Fig. 4, marked BP elevation occurs in some patients' higher BP response to acute stress.

In most patients, this increase was transient, and returned to the pre-earthquake baseline levels within 4 weeks. A similar time course of BP changes was also observed by Saito et al.,²⁵ and by Minami et al. using home BP monitoring.²⁶ This characteristic of disaster-induced BP increase is important because persistent intense antihypertensive treatment for subjects with high BP

Table 3 Earthquake-induced change in cardiovascular risk factors

| | Hanshin-Awaji earthquake | |
|---------------------------|--------------------------|-------------------|
| | Before | 7–14 days after |
| Systolic BP (mmHg) | 152 (142–164) | 170 (160–178)** |
| Diastolic BP (mmHg) | 83 (79–88) | 91 (84–96) |
| Heart rate (bpm) | 72 (67–86) | 80 (69–87) |
| Hematocrit (%) | 38.1 (35.9–40.7) | 39.7 (38.3–42.9)* |
| Total cholesterol (mg/dl) | 201 (185–226) | 198 (179–213) |
| HD-cholesterol (mg/dl) | 42 (38–49) | 41 (35–48) |
| Lipoprotein (a) (mg/dl) | 14 (7.0–23) | 15 (7.8–21) |

** P<0.001, * P<0.0001

(Kario et al. J Am Coll Cardiol. 1997;29:5:926–933.)

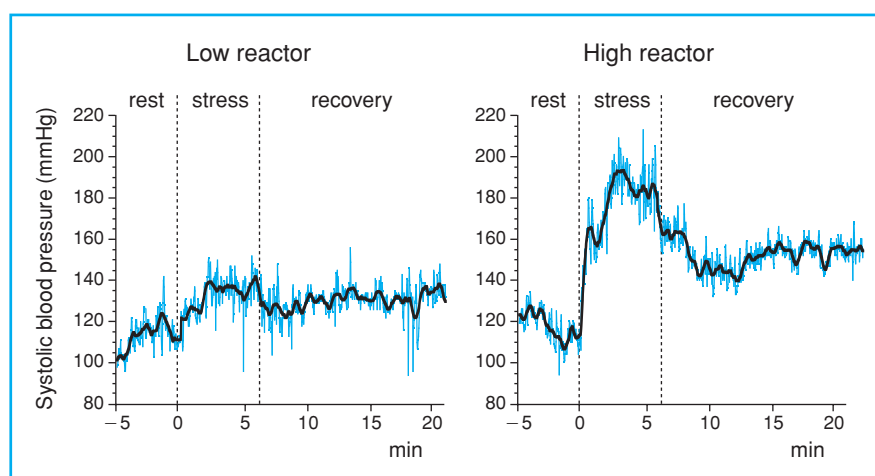


Fig. 4 Blood pressure change during acute mental stress

at the time of disaster could result in excessive reductions of BP, as we observed in a patient who had been started on treatment with antihypertensive agents just after the earthquake. She was referred to our clinic because she developed dizziness 3 months later. We discontinued her antihypertensive medication and monitored her ambulatory BP level, which was normal, and her symptoms disappeared.²⁷

This transient, disaster-induced BP increase may persist for longer periods in patients with chronic kidney disease. In hypertensives having microalbuminuria, which is a risk for CVD in elderly Japanese,²⁸ recovery from the transient BP increase was impaired and BP elevation per-

sisted for at least several months (Fig. 5).²⁹

White-coat hypertension, where ambulatory BP is normal but BP measured by health professionals in conventional clinical settings is high, is diagnosed in approximately 20–25% of the hypertensive subjects,³⁰ and may have slight cardiovascular risk.³¹ We observed some patients with white-coat hypertension which shifted to a pattern of sustained hypertension (both conventional and ambulatory BP measurement high) after the earthquake. In these patients, the BP increase was still present two months after the earthquake, and antihypertensive medication was then needed to control BP.³² After one year of treatment, the 24-hour ambulatory BP

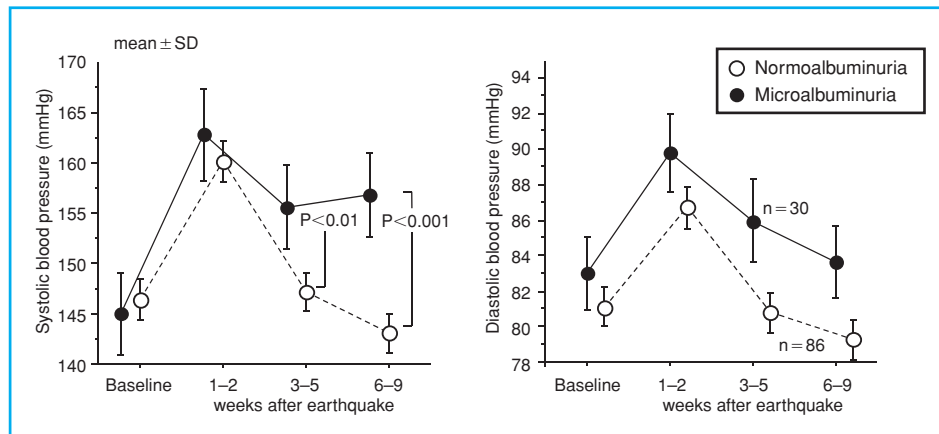


Fig. 5 Earthquake-induced blood pressure change in hypertensives with and without microalbuminuria

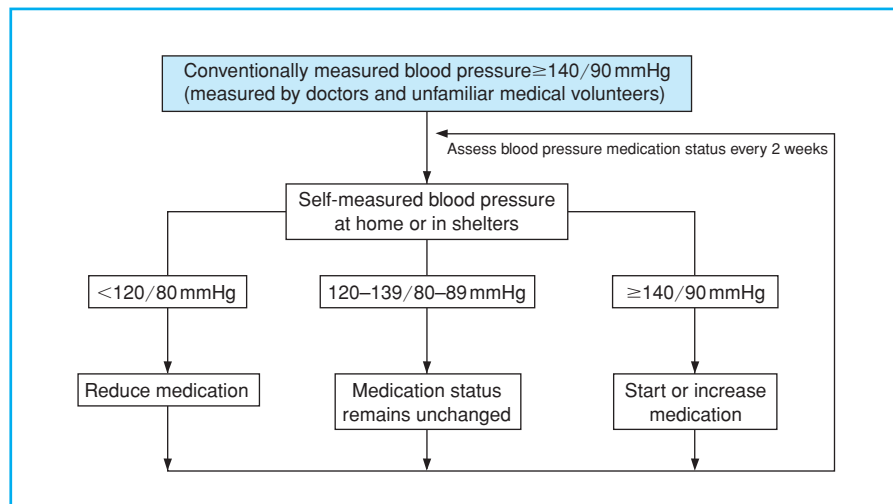


Fig. 6 Management of blood pressure in disaster

decreased to the level before the earthquake. However, the conventionally measured BP remained high, indicating that the white-coat effect can persist in treated patients.³³

Based on these findings, we propose the management of BP in disaster situations as shown in Fig. 6. To exclude the white-coat effect on BP, self-measured, BP-based BP management is recommended,^{34–37} and antihypertensive medication status should be evaluated repeatedly every 2 weeks.

2. Thrombosis and hemostasis

The formation of a thrombus following the rupture of a coronary atherosclerotic plaque is one of the major mechanisms of acute CHD events such as myocardial infarction and unstable angina. Plaque rupture may be triggered by increased shear stress from a sudden increase of blood pressure and by coronary vasospasm resulting from endothelial cell dysfunction. Although the pathogenesis of stroke is not necessarily the same as for acute CHD events, it is likely that the same processes contribute to ischemic stroke, and that transient BP increases may trigger hemorrhagic strokes. Increased platelet activity and an imbalance between coagulation and fibrinolysis (hypercoagulability and hypofibrinolysis) are associated with the progression of silent cerebral infarction (a predisposing condition for clinical cerebral infarction)³⁸ and the onset of ischemic

stroke. Increased blood viscosity, platelet activation, and abnormalities in blood coagulation and fibrinolysis may further facilitate thrombus formation.

As shown in Table 3 and Fig. 7 respectively, hematocrit and fibrinogen, both of which are major determinants of blood viscosity, increased after the earthquake. Increases in two endothelial cell-derived factors (vWF and tPA antigens), markers of endothelial cell injury, after the earthquake were also observed in the high-stress hypertensive group (Fig. 7). Figure 8 shows that

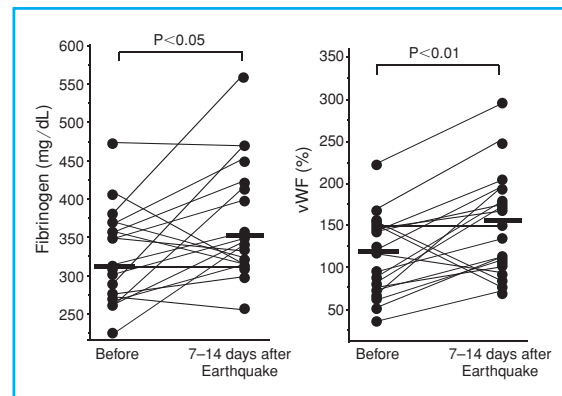


Fig. 7 Earthquake-induced change in fibrinogen and von Willebrand factor in high-stress hypertensives*

* whose housing was completely destroyed or whose family members experienced hospitalization due to earthquake-related injury

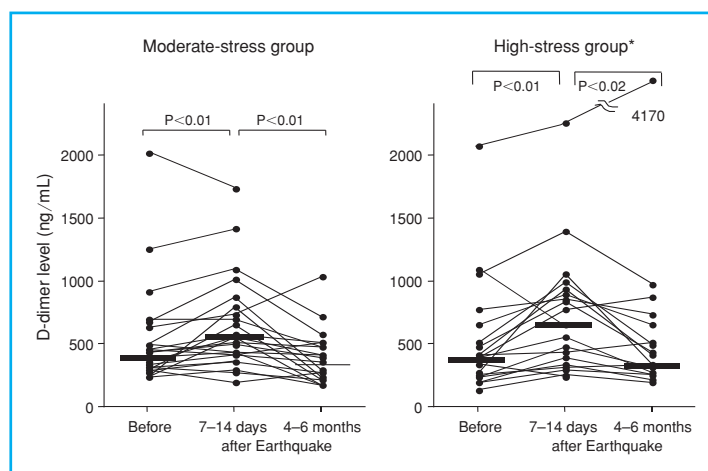


Fig. 8 Earthquake-induced change in D-dimer in high-stress hypertensives*

* whose housing was completely destroyed or whose family members experienced hospitalization due to earthquake-related injury

the most prominent increase was found in plasma levels in D-dimer, an activation marker of both coagulation and subsequent fibrinolysis, one to two weeks after the earthquake, and this increase was higher in the high-stress group (whose housing was completely destroyed or whose family members experienced hospitalization due to earthquake-related injury), than in the moderate-stress group. In the high-stress group, plasma PIC and tPA antigen levels were also higher. Four to six months after the earthquake, when the incidence of cardiovascular events returned to the level of the previous year, these indicators of a hypercoagulable state and subsequent fibrinolysis had all returned to the baseline level. There were positive correlations between the levels of these two factors and D-dimer levels after the earthquake,²⁴ suggesting that the endothelial cell dysfunction caused by the earthquake was associated with an increase in fibrin turnover. All of these changes could have contributed to the increase of cardiovascular events following the earthquake.

The lipid profile was not changed in the relatively shorter term (one to two weeks) after the earthquake. However, a high-calorie lipid-rich diet with reduced physical activity may worsen the lipid profile over the longer period.

3. Oxidative stress and inflammation

Oxidative stress, a cellular or physiological condition of elevated concentrations of reactive oxygen species that cause molecular damage to vital structures and functions, might be one of the mechanisms that explains the association between earthquake-induced increase in cardiovascular events. Several environmental factors influence the susceptibility to oxidative stress by affecting the antioxidant status or free oxygen radical generation.³⁹ Various factors have effects on the development of oxidative stress.³⁹ Regular exercise and carbohydrate-rich diets seem to increase the resistance against oxidative stress. Alcohol in lower doses may act as an antioxidant on low density lipoproteins and thereby have an anti-atherosclerotic property. Cigarette smoke and psychological stress increase oxidative stress.³⁹ Chronic stress can induce oxidative stress as assessed by increased plasma superoxide anions and malondialdehyde.⁴⁰ In addition, oxidative stress is linked to activation of the coagulation system in atherothrombotic disorders. The

overall oxidation state of plasma proteins is associated with changes of circulating pro- and anti-coagulant markers in healthy subjects.⁴¹ Vitamin E treatment *in vivo* restores in part the equilibrium between pro- and anticoagulant pathways.⁴¹

Acute and chronic psychological stress induces a significant increase in plasma levels of inflammatory cytokines such as interleukin-6 as a possible mechanism for how psychological stress might contribute to cardiovascular disease.⁴²

4. Mechanism of activation

The potentiation of the acute risk factors that we observed could be attributed to sympathetic activation resulting predominantly from earthquake-induced stressors. In support of this, is the observation that earthquake-induced BP increase was less pronounced in patients taking alpha- and beta-adrenergic blockers than those taking other kinds of antihypertensive drugs.^{25,29}

There are some reports that psychological stress and its related sympathetic activation cause platelet hyperactivity, and increase in two of the determinants of blood viscosity (hematocrit and fibrinogen).⁴³⁻⁴⁵ This platelet activation has been observed in both healthy subjects and those with advanced atherosclerotic disease.⁴⁴ In addition, increases of BP induced by psychological stress could augment shear stress-induced platelet activation in patients with atherosclerotic stenoses.⁴⁶

There are no experimental data showing that hormonal mediators of the sympathetic or HPA axis directly trigger a hypercoagulable state. In a study of healthy subjects, an infusion of stress hormones (epinephrine, cortisol, glucagon, angiotensin II, and vasopressin) for 24 hours did not affect procoagulant and fibrinolytic factors.⁴⁷ *In vivo*, the hypercoagulable state is determined by changes in both the coagulation system (leading to thrombin generation) and platelet hyperactivity (leading to microthrombus formation). In this context it is of interest to note that the reduction of acute myocardial infarction by aspirin has been found to be stronger in the morning, particularly during the 3-hour interval immediately after waking, a period characterized by sympathetic activation and a risk of infarction twice that of any other comparable time interval during the day or night.⁴⁸ Beta-adrenergic blockade suppressed the early morning rise in PAI-1 and tPA in patients with chronic coronary artery

disease.⁴⁹ In monkeys, psychosocial stress (72-hour exposure of male monkeys to a social stranger) caused a significant increase in the number of injured endothelial cells, and this endothelial cell injury was significantly inhibited by beta-adrenergic blockers.⁵⁰

Diurnal Variation of Acute Risk Factors

Figure 9 shows the chronobiological mechanism of the diurnal variation of stress-induced CVD events.

As the ambulatory BP level also increases during the period from night to early morning, and moreover, in the early morning, other cardiovascular risks such as thrombophilic tendencies and endothelial dysfunction are potentiated, the

effect of high BP on cardiovascular risk is greater in the morning than during other periods of the day. Theoretically, there are two types of morning hypertension (Fig. 10).^{51,52} The non-dipper/riser (nocturnal hypertension) type, with persistent high BP from nighttime to morning, is well known to be associated with risk for damage to all target organs (brain, heart, and kidneys) and CVD events.⁵³⁻⁵⁸ The other type, the morning BP surge type, is associated in part with the extreme-dipping status of nocturnal BP, which we have previously reported to be associated with a stroke risk.^{55,56} In our previous result in Jichi Medical School ABPM Study, wave 1, the early morning rise in BP, is a risk factor for CVD in elderly hypertensives.⁵⁹ It is predominantly determined by alpha-adrenergic activity,⁶⁰ and

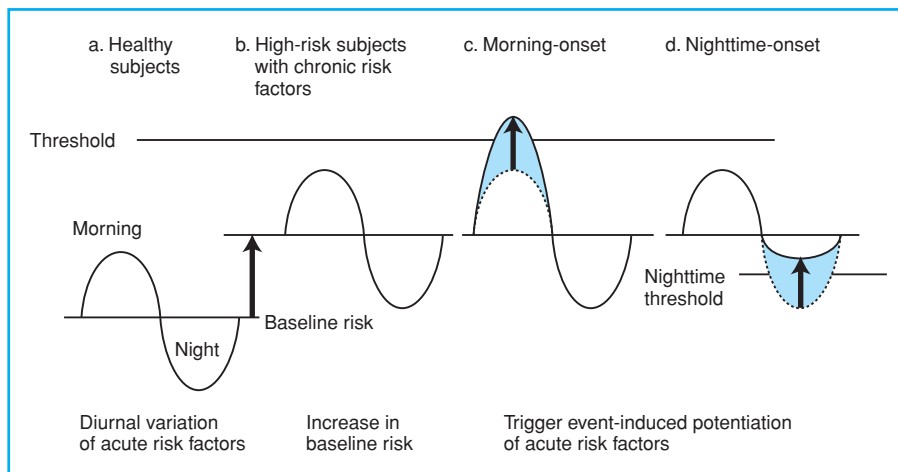


Fig. 9 Diurnal variation of disaster-induced risk factor and onset of cardiovascular disease

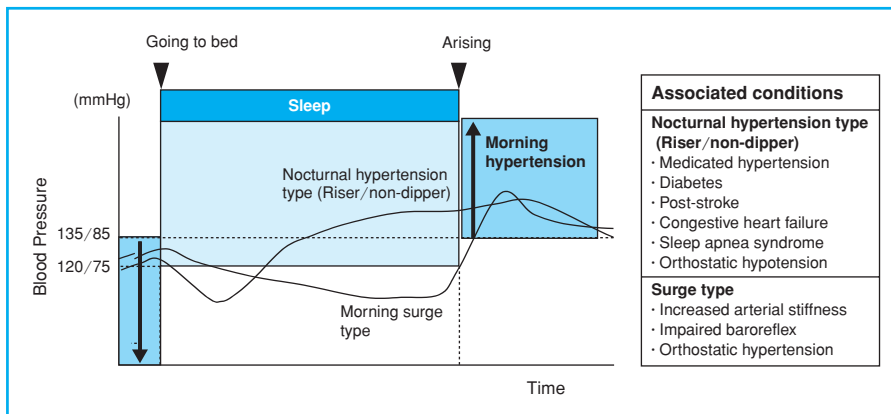


Fig. 10 Morning hypertension and diurnal blood pressure variation

is selectively attenuated by alpha-adrenergic blockers.^{61,62} Morning BP surge attenuated by alpha-blocker is associated with hypertensive silent cerebral disease.⁶³ It has been related to increased left ventricular mass.^{64–68} Morning surge and variability in BP are also associated with carotid atherosclerosis.^{69,70} Morning BP surge is a part of various forms of ambulatory BP variations, and recent animal and human studies indicate that increased BP variation accelerates atherosclerosis to become a potential risk for CVD.^{71–76}

Several risk factors for thrombotic events are potentiated early in the morning. These include endothelial cell dysfunction and vasospasm, plasma levels of blood viscosity determinants (hematocrit and fibrinogen), beta-thromboglobulin, and platelet factor-4. Aspirin selectively prevents the morning peak of myocardial infarctions.⁴⁸ A recent study of the diurnal variation of activation markers of coagulation showed that plasma levels of both activated factor VII and F1+2 were higher in the morning than in the afternoon.⁷⁷ In addition, plasma levels of plasmin-alpha₂/plasmin inhibitor complex were lower, and accompanied by an increase in PAI-1 level in the morning.⁷⁷ This result indicates that fibrinolytic activity is suppressed in the morning. Taken together, these diurnal changes indicate a morning prethrombotic state.

Aggravation of acute risk factors, found early in the morning, would be associated with hemorrhagic and ischemic cardiovascular events, while

imbalance between hemostatic factors and BP level may be involved in the triggering of hemorrhagic cardiovascular events during the period from afternoon to evening,⁷⁸ as shown in Figs. 9b and 9c.¹

In healthy subjects and hypertensive patients, sympathetic nervous activity is suppressed and parasympathetic nervous activity increases in proportion to the depth of sleep, leading to falls of nocturnal BP.⁷⁹ During rapid-eye-movement (REM) sleep, bursts of sympathetic activation result in marked BP variations and increase myocardial susceptibility to arrhythmias. Various genetic and environmental factors including psychological and physiological factors that influence abnormal autonomic nervous activity and sleep quality such as sleep apnea syndrome contribute to the non-dipping status.^{80–89} Although a precise assessment of sleep requires polysomnography, sleep quality can be indirectly assessed using actigraphy, because physical activity increases due to microarousals during sleep.⁹⁰ We have found a positive association between physical activity during sleep assessed by actigraphy and nocturnal BP fall in healthy adults, and increased sleep physical activity in non-dippers.⁹¹ Sleep disturbance is an important dimension of post-traumatic stress disorder, as shown by a recent report which demonstrated that subjects affected by Hurricane Andrew showed an increased number of arousals and entries into stage 1 sleep.⁹² These arousals are associated with sympathetic nervous activation,

Table 4 Management of disaster-associated cardiovascular risk

| | |
|---------------------|--|
| Sleep quality | <ul style="list-style-type: none"> • Turn off lights in shelters at night • Ensure privacy in shelters |
| Blood pressure | <ul style="list-style-type: none"> • measure morning blood pressure levels at home or in shelter • frequent BP assessment (every 2 weeks) and antihypertensive medication • reduce salt intake and increase high potassium-containing diet (green vegetables, fruits, and seaweeds) |
| Thrombotic tendency | <ul style="list-style-type: none"> • increase water intake (ensure access to temporary restroom facilities) • encourage physical activity (regular walking) • frequent assessment of anticoagulation activity in patients treated with warfarin |
| Infection | <ul style="list-style-type: none"> • distribute gauze masks • ensure a hygienic environment |
| Lipid profile | <ul style="list-style-type: none"> • reduce lipid-rich diet |
| Blood sugar | <ul style="list-style-type: none"> • reduced intake of sugar and carbohydrate • frequent assessment of glucose in diabetics and those with glucose intolerance |

and transient increases of BP.⁷⁹ Nocturnal behaviors like nocturia sometimes trigger falls, syncope, or CVD events in elderly subjects, and the sympathetic activation and BP variations associated with these activities are considered to be triggering mechanisms.⁹³ In addition, the ischemic threshold may be decreased during the night⁹⁴ (Fig. 9d). Based on these considerations, a possible mechanism for the increased incidence of nighttime-onset CVD events after the earthquake could be an impaired sleep pattern, and subsequent increases in nocturnal activity and microarousals.

Improvement of sleep quality in an adequate sleep environment seems to be vitally important in reducing disaster-induced potentiation of acute risk factors and subsequent CVD events during nighttime and in the morning.

Practical Management of Cardiovascular Risk

Table 4 shows the practical management of cardiovascular risk. The importance of improving the sleep environment in order to improve sleep quality is stressed. Measures can include turning lights off in disaster shelters during the night and ensuring the privacy of those who survived.

In disaster situations BP management guided by self-measured BP, as shown in Fig. 6, is recommended. Particularly important for the effective prevention of CVD is the frequent assessment of self-measured, morning BP levels (every 2 weeks). Once-daily antihypertensives are now widely used,^{95–98} however, conventionally measured and home BP control is still poor.^{99,100} In medicated hypertensives, even those whose clinic BP is well-controlled, the morning BP level before taking medicine is frequently high.^{101,102} Therefore, morning hypertension is a blind spot

in the current clinical practice for hypertension. More specific management targeting morning hypertension will achieve a more beneficial cardiovascular outcome in hypertensive patients also in disaster situation.

To reduce thrombotic tendency, water intake should be increased, even though nocturia may also increase. Accordingly, adequate provision of temporary restrooms should be arranged. Ensuring physical activity, such as regular walking, is particularly important for the prevention of deep vein thrombosis and subsequent pulmonary embolism. Anticoagulant activity should be carefully monitored in patients treated with warfarin.

To prevent the spread of infection, it is important to distribute gauze masks and to maintain a hygienic environment. After the acute stress of the disaster, during the subsequent chronic stress period, a high-calorie and lipid-rich diet should be avoided, and restrictions imposed on sugar intake. Additionally reducing salt intake and encouraging a high potassium diet (green vegetables, fruits, and seaweeds) is also recommended.

Conclusions

Acute and chronic stress in disasters trigger CVD through hemodynamic changes such as BP increase and thrombogenic factors, with effects being greatest during the nighttime hours. Disaster studies indicate the importance of the role of acute and chronic psychological stress, which also feature in daily life, on CVD and risk factors. In situations immediately after a disaster, stress reduction by improving the conditions of the post-disaster environment and managing stress-induced potentiation of risk factors may reduce prolonged increase in cardiovascular events for the surviving population.

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