

Cardiovascular reactivity to earthquake

Reaktivitas Kardiovaskular terhadap Gempa Bumi

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Abstrak

Gempa bumi merupakan suatu stressor mental yang terjadi secara alamiah. Belum diketahui bagaimana respon kardiovaskular terhadap gempa bumi, berapa lama respon tersebut berlangsung, dan seberapa jauh akibat yang ditimbulkan oleh gempa bumi terhadap sistem kardiovaskular. Tujuan penulisan naskah ini adalah untuk mengetahui gambaran dan durasi serta potensial dari pengaruh reaktivitas Kardiovaskuler terhadap gempa bumi.

Metode penelitian dilakukan dengan studi literatur terhadap penelitian-penelitian mengenai efek gempa bumi terhadap sistem kardiovaskular yang telah dipublikasikan pada MEDLINE.

Tekanan darah dan frekuensi denyut jantung mulai berubah pada awal gempa bumi, dan kemudian naik sebesar 20% untuk tekanan darah sistolik, 46% untuk tekanan darah diastolik, dan 79% untuk frekuensi denyut jantung. Tingginya tekanan darah tersebut bertahan 1-2 minggu pasca gempa bumi, kemudian turun secara bertahap dalam kurun waktu 2 minggu. Peningkatan tekanan darah memanjang hingga 2 bulan pada pasien-pasien dengan mikroalbuminuria. Sementara itu, peningkatan tekanan darah tidak begitu tajam pada pasien-pasien yang mengkonsumsi obat-obat α - dan β -bloker. Frekuensi denyut jantung kembali ke nilai awal lebih cepat daripada tekanan darah. Kejadian infark miokard meningkat 3 kali lipat penduduk yang tinggal dekat dengan pusat gempa daripada penduduk yang tinggal jauh dari pusat gempa.

Terdapat hiperreaktivitas kardiovaskular terhadap gempa bumi. Hiperreaktivitas kardiovaskular terhadap gempa bumi tersebut bersifat akut dan berpotensi menimbulkan komplikasi infark miokard.

Kata kunci: frekuensi denyut jantung, gempa bumi, infark miokard, reaktivitas kardiovaskular, tekanan darah

Abstract

Earthquake is a naturally occurring mental challenge. It potentially exerts adverse effects on the cardiovascular system, thus may contribute to the development of cardiovascular diseases. To know pattern and duration of the effect of earthquake on cardiovascular reactivity, and to know the potential effect of earthquake on the cardiovascular system.

We did literature search on studies published in MEDLINE database that reported changes in cardiovascular parameters among subjects lived in earthquake affected area.

The result of blood pressure and heart rate started to change at the initial trembling preceded the earthquake. Then at the strongest shock, systolic blood pressure increased 20%, diastolic blood pressure rose 46%, and heartbeat was up to 79%. Blood pressure remained high in 1-2 weeks after the quake. It then gradually returned to the baseline by 4 weeks (3 – 5 weeks) after the disaster. This increased blood pressure was prolonged for at least until 2

months aftermath in patients with microalbuminuria. However, it was less pronounced in patients who treated with α - and β -blocker. The heart rate returned to the baseline level more promptly than the blood pressure. The events of myocardial infarction increased 3-fold in people who lived close to the epicentre. The conclusion is cardiovascular hyperreactivity to earthquake has cardiac and vascular pattern. Exaggerated cardiovascular reactivity to earthquake is short term response. Cardiovascular reactivity to earthquake potentially leads to myocardial infarction.

Keywords: blood pressure, cardiovascular reactivity, heart rate, myocardial infarction, natural disaster

Introduction

Cardiovascular reactivity is a set of changes in blood pressure, heart rate, and other hemodynamic parameters in response to a stimulus.¹ Basically, cardiovascular reactivity is a reflex.² Therefore, it depends on condition of the structures along the reflex arc as well as on the nature and strength of the stimulus.³

Cardiovascular reactivity can be used as a non-invasive method to identify the pre-clinical state of cardiovascular disease. Pre-clinical cardiovascular disease state is the pathogenic change in the cardiovascular structure and function that, if continued, will often progress to manifestations of cardiovascular disease, such as hypertension, myocardial infarct, and stroke.⁴

Cardiovascular reactivity to a variety of mental stress was correlated to subsequent hypertension. A study in among 3364 normotensive young adults total (comprised of 910 white men, 909 white women, 678 black men, and 867 black women) showed that increased systolic blood pressure (SBP) reactivity to video game – a laboratory mental test – was significantly associated ($p < .0001$) with subsequent 5-year SBP, independent of resting SBP.⁵ A study done in among 206 middle-aged adults showed that larger systolic and diastolic blood pressure responses to a combination of mental and physical challenges were associated with higher subsequent resting diastolic blood pressure 6.5 years later, after adjustment for age, resting blood pressure, and body mass index at study entry.⁶

Earthquake is a naturally occurring mental stressor. Therefore, it potentially exerts adverse effects to the cardiovascular system and thus may contribute to the development of cardiovascular diseases.

Material and Methods

The objectives of this study are to know the pattern and the duration of the cardiovascular reactivity to earthquake, and also to know the potential effect effects of earthquake on the cardiovascular system. Published studies in MEDLINE database, that is maintained by The United States National Library of Medicine at the National Institutes of Health, that reported changes in cardiovascular parameters among subjects lived in earthquake affected area was collected. Most evidences were found came from the earthquake that struck the Hanshin-Awaji district, Kobe, Japan at 5.46 am on January 17, 1995 (magnitude 7.2 on the Richter scale), that trembled the Marche and Umbria region, Central Italia, at 5.26 pm on March 26, 1998 (magnitude 4.7 on the Richter scale), and that affected Los Angeles County, USA, at 4.31 am on January 17, 1994.

Results

In the case when initial trembling preceded the earthquake, blood pressure and heart rate were started to change, even if it was only of mild-to-moderate intensity. At the strongest shock, blood pressure and heart rate rose up markedly. As reported by Parati et al. systolic blood pressure rose from 130 mmHg to 150 mmHg (20% increase), diastolic blood pressure rose from 85 mmHg to 122 mmHg (46%), and

heartbeat rose from 83 bpm to 150 bpm (79%) in a borderline hypertensive patient

measured using a 24-hour ambulatory blood pressure monitoring.⁷

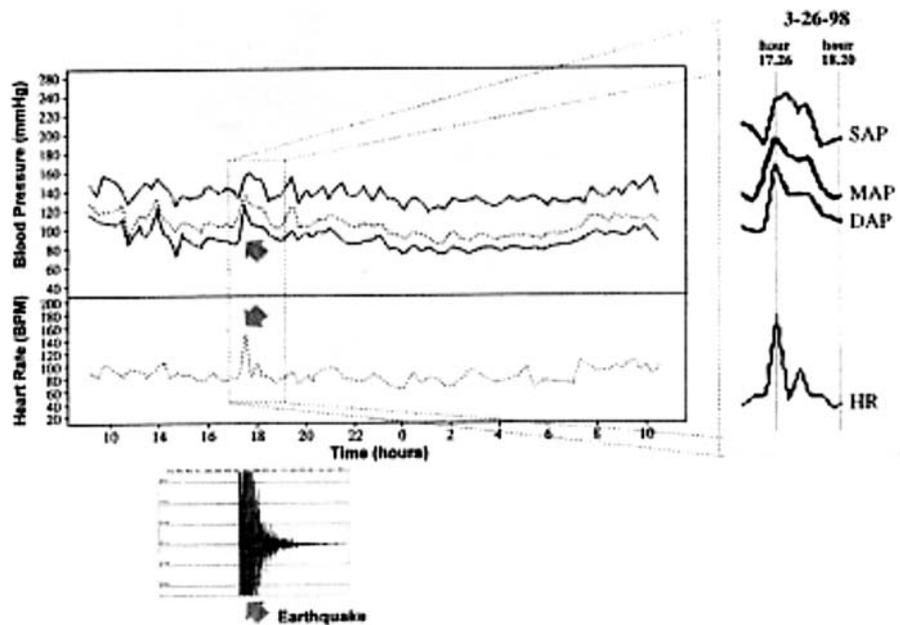


Figure 1. A record from 24-hour ambulatory blood pressure monitoring in a borderline hypertensive patient during an earthquake in the Marche and Umbria region, Central Italia, on March 26, 1998 (the seismograph recording is below the ABPM recording).⁷

Based on literature review showed that abnormal blood pressure persisted for 1 hour after the quake.⁷ Throughout the following 6 hours, blood pressure variability was enhanced.⁷ Blood pressure remained high in 1-2 weeks aftermath, as shown by office blood pressure⁸, home blood pressure⁹ and 24-hour ambulatory blood pressure.¹⁰ It was still high in the first to second week after the quake.⁸⁻¹⁰ The blood pressure then gradually returned to the baseline by 4 weeks (range 3 to 5 weeks) after the quake⁹⁻¹⁰, but it returned to the baseline within 6 weeks after the disaster.¹¹

As compared to the blood pressure, the heart rate returned to the baseline more promptly.⁷ The heightened blood pressure response was prolonged for at least until 2 months after the quake in patients with microalbuminuria.¹⁰ However, blood pressure increase related to earthquake was less pronounced in patients who had been treated with β -blocker¹⁰ and α -blocker.¹¹

After earthquake, blood viscosity is increased due to hemoconcentration. Hemostasis mechanism also changed as fibrinogen level, von Willebrand factor, tissue-type plasminogen (tPA) activator and D-dimer were increased.¹² Lipid profile was reported either increased for short-term period¹³ or did not change significantly.¹⁴

Factors associated with increased blood pressure due to earthquake were age, body mass index¹⁰, and gender.¹⁴ For heart rate reactivity, the influenced factors were financial loss, distance from relatives/friends, and decreased visiting after the quake.¹⁵ It was reported that women exerted greater blood pressure response to earthquake than men.¹⁴ Distance from the centre of the quake was also affects cardiovascular reactivity to earthquake. People who lived in the severe affected area had increased blood pressure greater than those who lived in the surrounding area.¹¹ The events of myocardial infarction increased 3-fold in people who lived close

to the epicentre¹⁴, whereas home blood pressure did not change significantly in hypertensive patients who lived farther than 50 kilometres from the epicentre.⁹ Incidence of fatal heart attack was increased on the day of earthquake, and it was decreased 6 days aftermath.¹⁵

Discussion

In response to mental challenge, cerebral cortex stimulates nucleus tractus solitarius in the medulla oblongata. Nucleus tractus solitarius in turn will activate cardiac-acceleratory centre and inhibits cardiac-inhibitory centre. Therefore, sympathetic activity to the heart increases, vagal inhibition to the heart decreases, and as the consequence, heart rate is increased. Sympathetic tone to the blood vessels will also increase, resulted in vasoconstriction and venoconstriction. Vasoconstriction occurs in the kidneys, splanchnic organs, and skin. Venoconstriction will raise central venous pressure; consequently, diastolic filling and stroke volume are increased.

Thus, during mental challenge, as heart rate and stroke volume increase, cardiac output is increased.¹⁶ As sympathetic activity to adrenal medulla is enhanced, epinephrine is released to portal vein. Epinephrine will bind to α_1 adrenergic receptors on the heart to enhance cardiac contractility and beat, thus increases further stroke volume and cardiac output.

Epinephrine will bind also to the α_2 adrenergic receptors on the blood vessels of the skeletal muscle to cause vasodilatation.¹⁷ Vasodilatation will also occur in the brain and heart as the result of metabolic hyperaemia. Blood is redistributed from the kidneys, splanchnic organs, and skin to the heart, brain, and skeletal muscle. Indeed, redistribution of the blood flow also occurs among skeletal muscles. Blood flow to the skeletal muscle in the arm is greater than that in the leg whereas the sympathetic activity to the skin of the arm is less than that of the leg.¹⁸ In response to mental stress, vasodilatation outweighs vasoconstriction, resulting in no changes or even slightly decreases in total

peripheral resistance. Thus, mental challenge is a cardiac stimulator, but not a vascular stimulator, since it increases heart rate, stroke volume, and cardiac output but decreases total peripheral resistance. Overall, it elevates blood pressure.¹⁶ Aftermath, blood pressure will suddenly drop and then almost immediately reach a plateau at a level that is somewhat higher than the baseline level.¹⁹

Earthquake is natural mental challenge, thus its nature is a cardiac stimulator. Therefore, as well as other mental challenges, it increases heart rate and elevates blood pressure. However, earthquake is also a vascular stimulator, in which diastolic blood pressure rises markedly than systolic blood pressure. The marked increase in diastolic blood pressure may indicate a pronounced peripheral vasoconstriction. Moreover, patients who had been treated with β -blocker¹⁰ and α -blocker¹¹, the blood pressure reactivity to earthquake was less pronounced.

Frightening, depression and anxiety accompanying the people who suffered from the quake disrupt the noradrenergic system to cause vascular hyperresponsiveness.²⁰ Marked increase in diastolic blood pressure may also reflect a reaction to immobile confrontation. Immobile confrontation is a condition when the subject is confronted by a challenge without any possibility of either attacking or escaping, thus without any chance of to fight or flight.⁷ The "bi-faces" of earthquake, i.e. as cardiac and vascular stimulator, differentiates it with other mental challenges, i.e. cardiac stimulators only.

The nearest the people lived from the epicentre, the greater the prevalence myocardial infarction after the quake.¹⁴ It was reported that exaggerated blood pressure reactivity correlates with intima-media thickness, the extent and progression of atherosclerosis in carotid artery, and left ventricular mass.⁴ Mental challenge increases oxygen demand but decreases oxygen supply. Oxygen demand is increased as systolic blood pressure and heart rate rises to cause an increase in rate

pressure product, which is a surrogate marker for cardiac oxygen demand²¹. Oxygen supply to cardiac muscle is decreased as epicardial coronary artery constricts and small resistance vessels in the heart fail to dilate. Acute psychological stress and imbalance of the autonomic nerve system to the heart contributes to the heart attack. The increased sympathetic activity and decreased parasympathetic activity to the heart contributes to cardiac arrhythmia. Anxiety and depression is related with regulatory failure of the serotonergic system, which in turn exerts influence to vasoconstriction.^{20,22} The increased hematocrit, fibrinogen level, vWF, tPA activator and D-dimer¹² will lead to thrombosis, which in turn is responsible for acute coronary syndrome. Mental challenge increases sympathetic activity, which in turn attenuates blood flow, resulting in platelet activation and thrombi formation.²²

The increased blood pressure dropped gradually after the quake.⁸⁻¹⁰ Cardiovascular reactivity varies on the magnitude of the responses & also in the extent to which it continues after the stressor is stopped (carryover effect).²³ After mental challenge, blood pressure should drop suddenly, although it reaches a plateau at a level higher than the baseline.¹⁹ Stress and depression will impair depressor mechanism and delay cardiovascular recovery.²⁰ Delayed cardiovascular recovery itself was associated also with future hypertension.^{4,19}

Conclusion

Cardiovascular reactivity to earthquake has cardiac and vascular pattern, includes in the increase in heart rate, blood pressure, peripheral resistance, and cardiac work. Exaggerated cardiovascular reactivity to earthquake is short term responses. Cardiovascular reactivity to earthquake potentially leads to cardiac events and hypertension in the long run for people who had survived from the earthquake.

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