Lessons learned about stress and the heart after major earthquakes

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There is evidence that certain stressors can trigger cardiovascular events. Several studies have now demonstrated an increase in major adverse cardiac events associated with natural disasters such as an earthquake. The purpose of this paper is to review the literature on earthquakes and cardiovascular events. Reports from 13 major quakes were reported. Earthquakes have been associated with a number of cardiac events including sudden cardiac death, fatal myocardial infarction (MI), myocardial infarction, stress cardiomyopathy, heart failure, stroke, arrhythmias, hypertension and pulmonary embolism. Most reports were associated with earthquakes of magnitude 6.0 or greater. Cardiac events were reported within hours of the quakes. In some reports there was a sharp spike in cardiac events followed by a decrease; but in other quakes the increases in cardiac events lasted weeks, months and even years. There often was an association between the cardiac events and amount of personal property loss. The Great East Japan Earthquake was an unusual event in that it was associated with a major tsunami and cardiac events appeared worse in inundated areas due to flooding. Some but not all reports suggested more MIs associated with early morning earthquakes that woke up the population. Hospitals in earthquake-prone areas should consider developing plans for handling increases in myocardial infarctions and other cardiac events that are associated with earthquakes. (Am Heart J 2019;215:20-6.)

There is mounting evidence that stress is bad for the heart. Emotional and physical stress can trigger major adverse cardiovascular events. Studies by Muller et al showed that a substantial percentage of myocardial infarctions were preceded by some type of stress. Stresses associated with myocardial infarctions included heavy or moderate physical activity, emotional stress, lack of sleep, overeating, and others.[1,2] For example after an episode of anger there is a several fold increased risk of myocardial infarction that lasts for a few hours.[3] Other types of emotional and physical stress associated with triggering of cardiovascular events include grief, financial stress, the stress of starting work on a Monday morning, the stress of watching a beloved sports team lose, the stress of the Winter holiday season, the stress of cold weather and the stress of heat waves.[4-6] Certain toxins can also precipitate a cardiac event including marijuana, cocaine, and air pollution.[7-9] Even the stress of waking up and moving around has been associated with cardiac events.[10] Perhaps one of the most well- studied triggers of acute cardiac events are earthquakes as these natural disasters affect large populations simultaneously. A large literature on the effect of earthquakes on major cardiovascular events has been accumulating. Our research group, which was based in Los Angeles during the Northridge earthquake of Jan 17, 1994, became interested in the concept because the authors experienced the event first hand.[11-13] This author (RAK) lived close to the epicenter and recalls the sheer terror of being jolted out of bed at 4: 30 AM in the morning with the sense that a bomb had gone off in the house. There was no question that with the initial tremor and subsequent aftershocks that my heart rate increased. The "fight or flight" response that I experienced could not have been good for those with a compromised coronary artery anatomy. These responses are associated with a stimulation of the sympathetic nervous system and release of catecholamines that increase oxygen demand (increased heart rate, blood pressure, and contractility of the ventricles) at the same time having the potential to reduce oxygen delivery by constricting some vascular beds, causing coronary spasm, rupturing atherosclerotic plaques, and increasing the propensity to develop thrombi (increased platelet aggregation and reduced thrombolysis). This imbalance of the oxygen supply/demand equation can result in myocardial ischemia, myocardial infarction and also contribute to lethal ventricular arrhythmias.[14] The purpose of the following discussion is to review the history showing the evidence that major earthquakes are a trigger of adverse cardiovascular events and to determine what lessons these events have taught us about the concept of stress

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Table I. Summary of earthquakes reported

Name of EQ	Location	Date	Time	Mag*	Increase in CV events/MI	References
Thessaloniki	Thessaloniki, Greece	6/20/1978	11:03 PM	6.4	Increase cardiac mortality	15
Athens	Athens, Greece	12/24/1981	~ 11 PM	6.7	Increase in cardiac death	16
Armenia (Spitak)	Armenia	12/7/1988	11:41 AM	6.8	Increase in heart disease risk over 6 months in those with higher levels of loss of material possessions and family members	48
Loma Prieta	San Francisco, CA USA	10/17/1989	5:04 PM	6.9	No increase in hospital admission for MI	18
Newcastle	Newcastle, Australia	12/28/1989	10:27 AM	5.6	Increase fatal MI's; increase MI's; increase in hospitalization for MI's	17
Northridge	Northridge (LA) California USA	1/17/1994	4:30 AM	6.7	Increase sudden death; increase in MI admissions; increase CV death; no increase death due to stroke or cardiomyopathy	18,11-13
Great Hanshin-Awaji	Hanshin-Awaji, China	1/17/1995	5:46 AM	7.0	Increase in mortality from MI and strokes; 3-fold increase in the number of MIs; increase in strokes; increase in blood pressure; increase in MI death at 2 months; correlation between seismic intensity and MI mortality	20-22
Niigatu-Chuetsu	Niigatu-Chuetsu, Japan	10/23/2004	5:56 PM	6.6	Increase mortality rates from MI in both men and women; increase in pulmonary embolism	24-26
Noto Peninsula	Noto Peninsula, Japan	3/25/2007	9:45 AM	6.9	Increase acute coronary syndrome; increase cerebral hemorrhage	27
Great Sichuan or Wenchuan	N-NW of Chengdu, China	5/12/2008	2:28 PM	8.0	Increase in blood pressure; increase in hemodynamically unstable VT; increase in myocardial ischemia; increase in heart failure	28,29
Christchurch, New Zealand	Christchurch, New Zealand	9/4/2010	4:36 AM	7.1	Increase hospital admissions; increase in MI's; increase in noncardiac chest pain; increase in MI's and CV mortality correlate with increased damage to homes	30-32
Christchurch, New Zealand	Christchurch, New Zealand	2/22/2011	12:51 PM	6.3	Increase stress cardiomyopathy	30-32
Great East Japan	Pacific coast of Tohuko, Japan	3/11/2011	2:46 PM	~ 9.0	Complicated by tsunami and nuclear meltdown; increase in sudden death; increase in MI that was prolonged in several studies; increase in mortality for MI and stroke; some but not all studies reported increases in strokes; increase in heart failure and arterial stiffness; increase in MI correlated with seismic activity in some but not all studies; increase in fatal MI correlated with percent of inundated area due to tsunami	22,33-36

Major earthquakes in which cardiovascular events were reported. EQ, earthquake; Mag*, magnitude of the earthquake, usually listed as per the Richter scale or Mw (Moment magnitude scale).

and heart disease. A literature review of Earthquakes and cardiac events, Earthquakes and myocardial infarction, Earthquakes and cardiovascular events and Earthquakes and cardiac death was performed from PUBMED to obtain the key papers for the following discussion. No extramural funding was used to support this work. The author is solely responsible for the design and conduct of the study, all study analyses, the drafting and editing of the paper and its final content. (See Table I.)

Reports of cardiac events occurring with major earthquakes since the late 1970s

The more prominent reports in the literature are described below.

One of the first reports of an association between cardiac mortality and earthquakes was reported by Katsouyanni et al in 1986.[15] They reported an association of 2 strong earthquakes in the city of Thessaloniki, Greece on June 19 and 20, 1978, that measured 5.2 and 6.4 (at 11:03 PM) in Magnitude respectively, with an increase in cardiac and total mortality compared to control days. The relative risk of dying from atherosclerotic heart disease on June 19, 20 and 21 was increased at 3.0 (1.5–5.9) and there was also an increase in death from all other causes as well. They did not find differences in age or gender in this analysis. An earthquake in Athens, Greece on Feb 24, 1981, at about 11 PM and listed as 6.7 in magnitude was associated with an increase in cardiac mortality.[16]

Dobson et al[17] reported findings associated with the December 28, 1989, Newcastle Earthquake (Australia) that struck at 10:27 AM and registered 5.6 in magnitude. The relative risk of fatal myocardial infarction (MI) and coronary death was increased during the period of December 28–31 compared to the usual number of deaths at that time of year (relative risk = 1.67; 95% confidence interval 0.72–3.17); and there was an increase in the relative risk of non-fatal definite MI (1.05), non- fatal possible MI (1.34); and hospital admission for MI or ischemic heart disease (1.27). In people under the age of 70 there were 6 fatal MIs which was a higher than usual number for that time of year ($P = 0.016$). During the subsequent 4 months following the earthquake there was no increased risk of MIs.

Dr Leor and I reported 3 studies regarding the Northridge Earthquake.[11-13] This quake occurred on Jan 17, 1994, at 4: 30 AM (and thus awaking a large population from sleep) and registered 6.7 on the Richter scale. As it occurred in the Los Angeles area it affected at least 10 million individuals in Los Angeles County and we saw this as an unusual opportunity to study the natural experiment of stressing a large population simultaneously (as well as suddenly awaking a large population at the same time). In one study we examined the records of the Department of the Coroner of Los Angeles County for the times including the week before the earthquake, the day of the event, the 6 days following the quake, and for corresponding control periods in the 3 previous years before the earthquake. There was a sharp increase in the number of sudden cardiac deaths on the day of the earthquake (n = 24) compared to the preceding week (daily average of 4.6; $P < 0.001$). The typical victim complained of chest pain or died within the first hour of the quake. Only 3 occurred in association with physical activity, suggesting that the emotional stress of the earthquake was a precipitating factor. Of note, during the 6 days after the event, there was a decrease in the number of sudden cardiac deaths to below the pre-quake baseline (2.7 per day). This post-quake reduction suggests that perhaps a certain number of people who may have been destined to die of sudden cardiac death did so, prematurely because of the trigger of the earthquake. An alternative explanation is that perhaps the quake preconditioned some individuals against sudden death. Or it is possible that both phenomena played a role in the subsequent decrease in sudden death. In another analysis, we canvassed coronary care units in Los Angeles County. There was a 35% increase in the number of admissions for MI the week of the quake compared to the week before the quake (201 versus 149; $P = .01$). The percentage of coronary units with increases in the number of MIs during the week of the quake was greater for those hospitals within a 15 mile radius of the epicenter, compared to those hospitals beyond a 15 mile radius of the epicenter of the quake. In a third study (13) we reviewed death certificate data from Los Angeles County during the entire month of the earthquake (January 1994) and compared it to control years of January 1992 and 1993. From January 1 to 16th (control period before the earthquake) there was an average of 73 deaths per day due to ischemic heart disease and atherosclerotic cardiovascular disease. On the day of the Northridge earthquake (Jan 17, 1994) there was a significant increase to 125 deaths ($P < 0.00001$); this number then decreased to 57 deaths per day from January 18 to 31 (significantly different from day of earthquake and control days before earthquake). Deaths during the control years of 1992 and 1993 remained relatively flat throughout the month of January. The reduction in number of deaths after the earthquake overcompensated for the increase on the day of the earthquake, suggesting a possible preconditioning effect to stress. The increase in deaths on the day of the quake was associated with increases in death due to MI and trauma but not stroke, hypertensive heart disease, cardiomyopathy or non-cardiovascular cause. Besides the temporal relationship between the stress of the earthquake and cardiac events, there was also a spatial relationship. A geographical analysis of Los Angeles County revealed that there was a redistribution of deaths related to ischemic heart disease and atherosclerosis, towards the epicenter of the earthquake on the day of the event. Our results suggest that adverse cardiovascular events can be triggered in an entire population by the stress of a natural disaster; however, this was followed by a decrease in events, which may have been due to moving up the day of inevitable death by the trigger or some element of preconditioning. Brown[18] also studied the effects of the Northridge quake on MI but contrasted it to another California earthquake, the Loma Prieta quake (San Francisco Bay area) of 1989. Brown analyzed California-hospital discharge records the week before, the day of and 6 days after the 2 quakes. Similar to our findings, he described a significant increase in admission rates for MI related to the Northridge earthquake compared to control days. However, he did not observe an increase in MI admissions in the San Francisco Bay area associated with the Loma Prieta quake. What could explain the differences? The Loma Prieta quake struck at 5:04 PM in the afternoon when the population was already up and about; while the Northridge quake struck at about 4:30 AM, jolting millions to awaken. Studies on circadian rhythm have shown that the wake up time itself is a trigger of MI. It may be that the sympathetic surge in the morning of waking up, changing posture, getting ready to take on a new day, is enough to trigger cardiac events. Superimposing the emotional and/or physical stress of an earthquake on top of waking up might have been the reason that the Northridge but not the Loma Prieta quake was associated with an increase in MIs. We commented on this paper in an editorial called "Natural disaster plus wake-up time: A deadly combination of triggers".[19] In reviewing additional data on earthquakes and the time of day they occur, it is true that many of the ones that occurred in the

morning were associated with an increase in MIs (Christchurch, New Zealand on Sep 4, 2010, at 4:36 AM, Northridge on Jan 17, 1994, at 4:30 AM, Great Hanshin-Awaji Jan 17, 1995, at 5:46 AM; Noto Peninsula, Japan, March 25, 2007, at 9:45 AM; but others associated with cardiac events occurred at later times: Niigata-Chuetse, Oct 23, 2004, at 5:56 PM, Wenchuan, China May 12, 2008, at 2:28 PM; Thessaloniki, Greece, June 20, 1978 at 11:03 PM, New Castle, Australia on Dec 28, 1989 at 10:27 AM, Armenia, December 7, 1988 at 11:41 AM; Great Eastern Japan on March 11, 2011, at 2:46 PM). Thus it is not clear that all earthquakes associated with cardiac events necessarily occur around the wake up time.

The Great Hanshin-Awaji, Japan Earthquake occurred on January 17, 1995, at 5:46 AM, one year after the Northridge earthquake. It registered 7. Ogawa et al[20] reported that there was a significant increase in mortality from acute MI which continued for about 8 weeks after the quake. There was regional variability in extent and duration of this increased mortality, but there was a positive correlation between the increase in standardized mortality ratio of MI and the percentage of houses in the region that were completely destroyed. The authors commented that this increased mortality was longer than seen with most previous earthquakes, perhaps due to the extensive damage to housing and the emotional stress that would have followed. Kario et al[21] also reported an increase in cardiovascular events with this earthquake including a 3-fold increase in MIs in the population living close to the epicenter, especially in women, with an increase in nighttime-onset events. They also noted a doubling of strokes, an increase in blood pressure and an increase in D-dimer, von Willebrand factor and tissue-type plasminogen activator antigen. Takegemi et al[22] compared the Great Hanshin- Awaji Earthquake to the more recent Great East Japan Earthquake, described in more detail below. Whereas both natural disasters were associated with a sharp increase in cardiovascular mortality, the Hanshin-Awaji quake, according to these authors, was associated with a more prolonged (at least 2 months) increase in MI deaths. Seismic intensity correlated with an increase in MI mortality for 2 weeks after both quakes. Yamabe et al[23] reported 6 patients who presented with chest pain who had deep T wave inversion on the electrocardiogram (5 were post-menopausal women) with the Great Hanshin-Awaji earthquake. Three of these patients had normal or minimal coronary artery disease on angiography. It is possible that these cases represented aspects of Takotsubo or stress cardiomyopathy. Takotsubo cardiomyopathy has been recognized to occur with other earthquakes as describe in more detail below, but may not always be identified. Perhaps limited resources during the course of the earthquake or confusion with myocardial infarction are more likely to occur with the chaos associated with natural disasters.

Nakagawa et al[24] reported the long term effects of the Niigata-Chuetsu Earthquake in Japan on acute myocardial infarction mortality. This earthquake occurred on Oct 23, 2004, struck at 5:56 PM and registered 6.6 in magnitude. An increase in mortality rate (14%) from MI was observed over 3 years after the quake compared to the 5 years before, in both men and women. There were no changes in these mortality rates in control areas. The authors concluded that the" Niigata-Chuetsu quake significantly increased long-term mortality from acute MI in both men and women." Another report concerning this earthquake described an increased incidence of pulmonary embolism in victims experiencing this earthquake and suggested that prolonged immobilization from taking refuge in their cars contributed to this phenomenon.[25] Watanabe et al,[26] reported that this quake was also associated with an increase in cases of Takotsubo cardiomyopathy, a condition that is often reported to occur after severe emotional stress.

The Noto Peninsula Earthquake in Japan occurred on March 25, 2007, at 9:45 AM, registered 6.9 in magnitude, and affected primarily a rural area. Tsuchida et al[27] noted that there were few data in the literature on the effect of an earthquake in rural areas. They reported a case of acute coronary syndrome occurring about 15 minutes after the quake. They also reported that in the first 35 days after the quake, there were 5 patients with acute coronary syndrome and 8 with cerebral hemorrhage which was greater than the averages for the same time period in the previous 3 control years (closer to 2 for both conditions). The authors made the point that even in rural areas a severe earthquake can increase the incidence of cardio and cerebrovascular events. They also noted that most of the acute coronary syndrome events occurred within 7 days of the disaster; while the cerebral events occurred 35 days after the quake.

The Great Sichuan or Wenchuan Earthquake occurred in China on May 12, 2008, at 2:28 PM and registered 8 in magnitude. Y Chen et al[28] reported the results of ambulatory blood pressure monitoring in 11 patients who happened to be wearing an ambulatory blood pressure monitor when the earthquake struck. Mean blood pressure rose rapidly from 126/72 mmHg to 150/98 mmHg in about 14 minutes after the first tremor. Blood pressure remained elevated for 6 hours after the start of the quake. Mean blood pressure during the day was greater after the earthquake (139/82 mmHg) than before the earthquake (130/77 mmHg). The normal circadian rhythm whereby nighttime blood pressure falls, was lost, with a night time decrease of less than 10%. The authors postulated that the post-earthquake elevation in blood pressure and abnormal circadian rhythm could contribute to the known occurrence of adverse cardiovascular events following an earthquake. In another report regarding this earthquake, XQ Zhang et al[29] reviewed medical records in the cardiovascular department of a hospital during the month of the earthquake and during 2 control periods. The rate of hemodynamically unstable ventricular tachycardia was significantly higher during the earthquake month than

the control periods (67 events per 10,000 person-days versus 7 and 14 events per 10,000 person-days; both statistically significant). Most ventricular tachycardia events occurred in the afternoon hours and followed strong seismic activity; rather than the usual diurnal distribution of ventricular arrhythmias. Hypokalemia, acute myocardial ischemia, and heart failure without ischemia were associated with the arrhythmias during the earthquake month.

Christchurch, New Zealand, was hit by 2 major earthquakes that have shed some light on the concept of timing of earthquakes and the type of cardiovascular event that may occur. Chan et al[30] described the cardiovascular outcome differences related to 2 fairly recent earthquakes in Christchurch. One occurred on September 4, 2010, at 4:36 AM and was 7.1 in magnitude. It was associated with an increase in hospital admissions, ST elevation myocardial infarctions (P <0 .016), and chest pain. The second earthquake was on Feb 22, 2011, at 12:51 PM and registered at a magnitude 6.3. The afternoon earthquake in contrast to the early morning one, was not associated with the same pattern of increases in myocardial infarction and chest pain. Rather it was associated with an increase in stress cardiomyopathy (Takotsubo cardiomyopathy). There were 21 hospital admissions over 4 days for stress cardiomyopathy associated with the afternoon earthquake; but only 6 associated with the morning earthquake (P <0 .05). Thus these 2 major earthquakes occurring at different times of day were associated with differing cardiovascular presentations. In another study, dealing with this earthquake, Zarifeh et al[31] described that women with non-cardiac chest pain following the September earthquake were more likely to have higher anxiety than women who presented with either MI or stress cardiomyopathy. Similar to other reports, Teng et al[32] also reported that rates of MI and cardiovascular admissions to hospitals were increased for victims living in regions where there were more severely damaged homes.

The Great East Japan Earthquake was an unusual event; it was really a triple disaster: an earthquake, a tsunami and a nuclear melt-down at a nuclear power plant. The earthquake occurred on March 11, 2011, at 2:46 PM in the Pacific Coast off of Tohuko, Japan and was magnitude 9.0–9.1. There have been many papers describing an increase in major adverse cardiovascular events; not all have been agreement regarding certain details of these events, but all reports have reported some increase in cardiovascular events temporally related to the events of March 11, 2011. There were reports of an increase in sudden cardiac deaths for at least 4 weeks, especially among Tsunami victims.[33] One paper reported an increase in out of hospital cardiac arrest for a year.[34] Nakamura et al reported a 3- fold increase in acute myocardial infarction in one hospital for at least 3 weeks and in these patients, a higher increase in CK-MB levels than usually seen, suggesting larger infarct sizes.[35] Yamaki et al stated that the increase in acute myocardial infarctions was limited to a specific area (Iwaki district) over 1 year.[36] Itoh reported an increase in in-hospital mortality for ST elevation MI in the Tsunami area.[37] A study reported that there was an increase in post- traumatic stress disorder, and that this disorder was associate with an increase in MIs, strokes and heart failure.[38] Tanaka et al described a relative risk of MI of 2.03 for the 4 week period after the event versus the previous year. The increased incidence of acute MI was positively correlated with seismic scale and aftershocks.[39] Takegami et al compared the Great East Japan Earthquake with the Hanshin-Awaji quake and reported an increase in monthly acute MI related and stroke related deaths for both earthquakes.[22] Acute MI deaths were increased at 2 months in the Hanshin-Awaji quake but not increased after 2 months with the Great East Japan quake. Seismic intensity was associated with MI mortality with the Great East Japan quake, but less so compared to the Hanshin-Awaji earthquake. In another study of the Great East Japan Earthquake, for fatal myocardial infarction, the standardized incidence ratio was stable in low-impact zones (based on the severity of the Tsunami damage) but increased in the high impact zone for 3 years. The standardized incidence ratio of fatal myocardial infarction correlated with the percent of the inundated area and number of deaths directly due to the tsunami but in contrast to some of the other papers, not the seismic activity[40] . At least 3 papers showed that the Great East Japan quake was associated with an increase in heart failure; some showed no increase in arrhythmias or strokes.[38,41,42] One paper reported a sharp but transient increase in all cause death within a month of the earthquake but no long-term prognostic impact.[43] A study showed an increase in arterial stiffness[44] and another suggested an increase in blood pressure.[45] Therefore multiple reports described increases in cardiovascular events over longer periods of time compared to other earthquakes alone, such as the Northridge quake in which there was really only a 1 day spike.

Limitations of our analysis include the issue that reports are not available on all earthquakes. For example, out of 50 seismic events with magnitude greater or equal to 6.0, which occurred in high-income countries between 1990 to 2012, only 11 were investigated or published for association with cardiovascular events.[46] Another example is the 2010 earthquake in Haiti (magnitude 7.0 (Moment magnitude [Mw] scale), with a death toll of about 230,000 in which no specific information on death due to cardiovascular disease has been published, to the best of this author's knowledge. In addition not all investigators have assessed the same endpoints, and how myocardial infarction was diagnosed is not always clear. It is possible that some of the cases reported as myocardial infarction were actually stress cardiomyopathy (Takotsubo cardiomyopathy). Some of the findings (such as that in the Newcastle earthquake, 5.6 Mw, showed trends but did not reach statistical significance). In addition there may be bias as larger earthquakes associated with reporting of

cardiovascular events are more likely to make it into the medical literature, whereas larger earthquakes without reports of cardiovascular events are less likely to be reported in the medical literature. Despite these limitations, there is enough in common in these reports to come to some useful common sense conclusions. In addition, there are several other recent reviews to which the reader is referred that clearly show an increase in cardiovascular events associated with earthquakes.[46,47] In a meta-analysis[46] assessing 13 earthquakes in 8 countries showed that people exposed to an earthquake had a 2% higher all-cause mortality, a 36% greater mortality rate from MI and a 37% greater mortality from stroke. Table I summarizes the recent major earthquakes since the late 70s and reported cardiovascular events.

Discussion

What are the overall lessons learned then from these recent earthquakes and cardiovascular events? One is that most of these earthquakes were magnitude 6 or greater. Therefore, when earthquakes of this magnitude strike, health care workers should be aware that they are more likely going to be responding and treating cardiac events and MIs. Second, earthquakes can be associated with several manifestations of increased cardiac events: increase in MIs, fatal MIs, sudden death, heart failure, stress cardiomyopathy, high blood pressure, ventricular arrhythmias, arterial stiffness, and pulmonary embolism. Increases in strokes appeared to be more variable. There may be regional distributions of earthquakes that center on those areas of most destruction of property; or flooding of property as with the Tsunami associated with the Great East Japan Earthquake. It is likely that not only the initial fight or flight response contributes to cardiac events but the stress of property loss and loss of family and friends contributes to earthquake related stress that may persist for weeks to months and in some studies, years. Whereas some of the earthquakes were associated with a sharp increase in cardiac deaths and then a fall (Northridge) perhaps due to early death of those that would inevitably die and/or preconditioning, other earthquakes were associated with continuing cardiac events that lasted long term, and again these may have been related to ongoing aftershocks, property damage, flooding, loss of family and friends. There might be a difference in presentation depending upon the time of day the earthquake strikes. Comparing the Loma Prieta quake in California to the Northridge earthquake revealed that an earthquake occurring during the late afternoon when people are already up and about may be less likely to be associated with major adverse cardiac events compared to an earthquake that occurs early in the morning and jolts people out of sleep. In addition an afternoon earthquake in Christchurch, New Zealand was more likely to be associated with stress cardiomyopathy (Takotsubo cardiomyopathy); while a morning earthquake was more likely to be associated with acute MI. However, these circadian variations in presentation of MI associated with an earthquake have not always held true. Several later-in- the- day earthquakes have been associated with MI and acute coronary syndromes. Several studies showed a correlation between the seismic activity and intensity and the development of acute coronary syndromes; some did not. In the earthquake associated with a Tsunami, the degree of flooding may be a factor in cardiovascular event triggering.

Conclusions

Although there have been some differences in the details of the relationship between earthquakes and cardiovascular events, there is enough in the literature showing an association that hospitals and clinics in earthquake prone areas should consider having plans in place on how to deal with the likely increase in cardiac events that are triggered by the natural disaster of an earthquake. In addition it is clear that Takotsubo or stress cardiomyopathy may be another manifestation of earthquake stress-induced cardiac disorders, and physicians should become aware of the possibility of this entity may present following an earthquake.

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