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Editorial

Review Articles

Current Activities of JMA

Tendency for Increasing Severity of Natural Disasters and the Indian Ocean Tsunami Disaster

Yasuhiro Yamamoto*1

According to an old Japanese proverb, the four most fearful things in Japan are *Jishin*, *Kaminari*, *Kaji* and *Oyaji*. The first three, which translate as earthquakes, thunder and fires in English are understandable enough, but how about the fourth? There is, of course, the straightforward interpretation of *oyaji* as fathers. However, in the Edo Period (seventeenth century to mid-nineteenth century) Japanese society was largely matriarchal, the wife's power was absolute and the husband was always henpecked. The period in which the father figure was strong was from the Meiji Era to the second World War (late nineteenth century to mid-twentieth century) when the national policy was to enhance the wealth and military strength of Japan. It, therefore, generally used to be said that *oyaji* meant gales and typhoons. Whatever the case may be, recent natural disasters including typhoons and hurricanes are increasing severity and frequency.

The number of typhoons that hit Japan last year in 2004 was ten, more than four times as many as the average for the last decade of 2.6. Also in the United States, hurricanes are becoming increasingly severe, with Hurricane Ivan, which struck the United States and hung around for a long time last year, being the largest hurricane recently at Level 1. This trend is clearly connected with global warming and the fact that recently more and more typhoons in the sea off Japan veer north and hit Japan instead of turning west and heading towards China is also said to be attributable to global climatic change.

Some academics also say that there is a connection between the occurrence of earthquakes and global warming. They say that increasing thermal expansion of land and seas is causing greater slippage of plates, etc., which in turn results in earthquakes of greater magnitude.

Earthquakes directly or indirectly affecting Japan from last year to this are the Niigata Chuetsu Earthquake in October 2004, the Indian Ocean Earthquake and Tsunami in December and the earthquake off the western coast of Fukuoka in March 2005. Both of the earthquakes occurring in Japan caused serious damages. The earthquake off the coast of Sumatra and the Indian Ocean Tsunami were disasters of a magnitude said to occur only once every few centuries. The final death toll including those missing is reported to be between 320 and 350 thousand people.

The Japan Disaster Relief Team from the Ministry of Foreign Affairs and JICA sent a medical team to Sri Lanka on December 27 the day after the Indian Ocean Tsunami, and later dispatched a total of seven teams to four countries, Sri Lanka, Thailand, the Maldives, and Indonesia (Sumatra), where they proved enormously effective. From the end of January to the beginning of February I myself was assigned to medical support as Medical Team Leader of the third team to be dispatched to the Sumatran city of Banda Aceh. In Banda Aceh at the end of January one month had passed since the disaster, but operations to search for the missing were still underway, and from time to time a strange smelling truck would go by filled with twenty or thirty bodies in black plastic bags. At the Japanese Team's medical center, every day we divided 100 to 150 patients between booths offering surgical treatment and booths treating internal diseases and provided treatment between the hours of 9 and 12 in the morning and 2 and 4:30 in the afternoon. In the surgical booths there were lots of patients who had sustained fractures and broken bones from being swept up by the tsunami, many of whom had also developed secondary infections. In the booths treating internal diseases, many patients were suffering from respiratory infections from contaminated water, diarrhea, skin diseases from the tsunami water, insomnia and anxiety. And then, finally, the Japanese Self-Defence Forces medical team took over our site.

I would like to express my sincere thanks to the MFA, JICA and JMA for having supported these activities.

More efforts of health workers will continue to be required in the event of future natural disasters, and I hope that the feature articles of the present issue will generate many ideas and suggestions for disaster countermeasures in medical care.

*1 Department of Critical Care and Emergency Medicine, Nippon Medical School, Tokyo
Correspondence to: Yasuhiro Yamamoto MD, Department of Critical Care and Emergency Medicine, Nippon Medical School, 1-1-5, Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan.
Tel: 81-3-3822-2131, Fax: 81-3-3821-5102,
E-mail: yamamtoy@nms.ac.jp

Review Article

Lessons from Hanshin Awaji Earthquake
—Experience of a medical association—

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Yasuaki Kako*1

Abstract

An earthquake of unanticipated scale occurred just beneath an urban area that included the cities of Kobe (prefectural capital with 1.5 million inhabitants), Ashiya (90,000 inhabitants), and Nishinomiya (420,000 inhabitants). It was a terrible disaster that struck the narrow strip of land containing important east-west transportation routes squeezed between mountains to the north and the sea to the south. Interruption of information severely impeded cooperation among local governments and that between them and private organizations, and local entities had to act independently for a time. Medical institutions were also isolated due to the interruption of information, experiencing difficulty in communication with other hospitals for patient transportation. At the time, the concept of triage was understood and practiced by emergency personnel, but we were still uncertain about the general public's understanding of the concept. However, triage was accepted well at the hospitals performing it and no problems were encountered. In the extreme difficulties caused by the unexpected disaster, medical association members did their best with a firm sense of mission to help victims from the initial to the subacute and chronic phases, overcoming the lack of experience and bewilderment. However, it is also a fact that our response was not sufficient. This article provides some proposals based on this experience.

Key words Lifeline facilities, Acute care, Subacute care, Chronic care, Disaster medicine, Medical association

Introduction

At 5:46 a.m., January 17, 1995, there was a great rumbling of the earth followed by strong vertical shakes and then violent horizontal shakes. I could not stand up fully, and it was all I could do to prevent myself from falling over. After the shock subsided, I moved out of my house to see most of the houses in the neighborhood completely destroyed or half-collapsed. It was a miracle that my house stood apparently intact. The 2-storied apartment in front of my house had collapsed burying 2 children, who were soon rescued alive. An old woman died of compression in another collapsed house. Fortunately, the JR Shinkansen Line and other railways had not started the day's operation at the time of the earthquake, and there were no casualties from derailment or other railway accidents. However, fire in some areas killed people confined in collapsed houses, and the collapse of the expressway killed drivers in their cars. Tremendous losses resulted from the damage to medical institutions, impacts on medical workers, interruption of telecommunications, interruption of lifelines (electricity, gas, and water), disruption of road networks including expressways, and destruction of railway facilities.

Damage to Medical Institutions

January 15 and 16 were holidays. When the earthquake hit at 5:46 a.m. of January 17, only the personnel on night duty were in hospitals other than emergency hospitals. It was a time when hospitals were at their most understaffed. The situation was similar in smaller clinics. These also sustained

*1 Hyogo Prefectural Medical Association, Hyogo
Correspondence to: Yasuaki Kako MD, 3-40 Uedanakamachi, Nishinomiya-shi, Hyogo 663-8134, Japan.
Tel: 81-798-41-0123, Fax: 81-798-46-0956

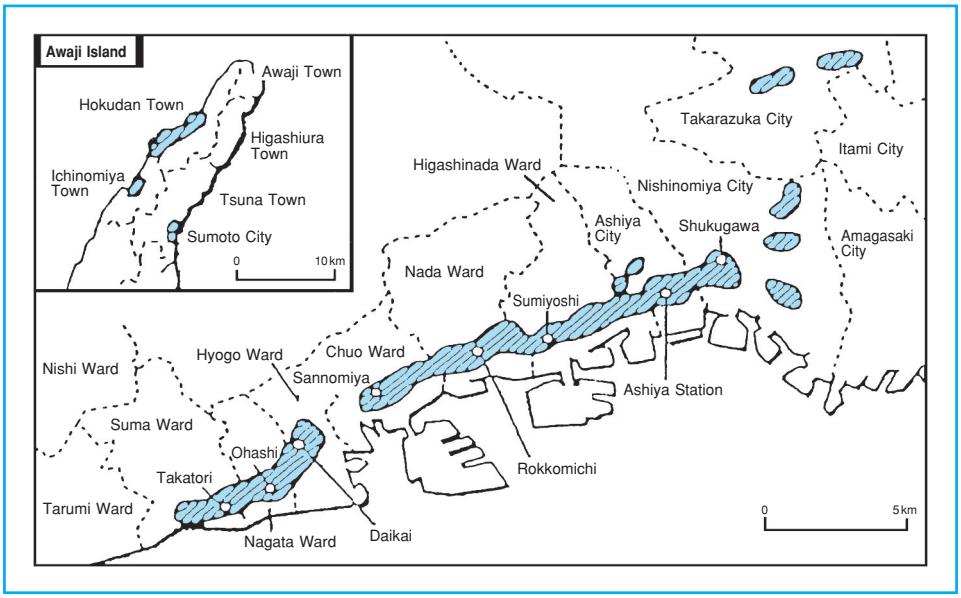

Fig. 1 Disaster areas of the Hanshin Awaji Earthquake

total and half collapse of buildings, destruction of medical equipment, and personal damage. The earthquake killed 9 members of the medical association, and very regrettably, there were also deaths among the families of members, employees, and the families of employees. These personal losses aggravated the impairment of clinical functions.

According to the survey of disaster medicine in Hyogo Prefecture (June 1995), the damage to medical institutions affiliated with the medical association was as follows. Of the 1,436 medical institutions in Kobe City, 40.6% sustained total or half collapse. The damage was particularly severe in the hardest-hit strip of land (seismic intensity 7) passing through the central part of Kobe from west to east and reaching Nishinomiya City. The percentage was 33.4% in Suma Ward, 62.3% in Nagata Ward, 53.8% in Hyogo Ward, 50.4% in Chuo Ward, 61.8% in Nada Ward, 46.7% in Higashinada Ward, 36.6% in Ashiya City, and 23.6% in Nishinomiya City. When adjacent areas are combined, 747 of the total 2,983 medical institutions sustained total or half collapse and lost the ability to perform medical functions. Other medical institutions that remained without major destruction also suffered from damage to some extent and their clinical functions were impaired.

Interruption of Electricity, City Gas, Water Supply, and Other Lifelines

Disruption of lifelines, including the stoppage of the water supply, breakdown of electric power supply, stoppage of city gas, and interruption and disturbance of telephone lines drastically affected not only medical care services, but also citizens' livelihood. The failure of electricity made it impossible to operate medical equipment and laboratory apparatuses using electric power, in addition to lighting. The stoppage of city gas hindered tasks involving boiling water and caused difficulties in preparing meals for in-patients. The lack of water supply caused a shortage of drinking water and water for general use, the latter required for hand washing and for flushing toilets. While the supply of drinking water was barely secured by the use of bottled mineral water, the shortage of water for general use caused difficulties beyond expectations. Because hemodialysis was impossible due to the disruption of the electricity and water supply, patients requiring hemodialysis had to be moved to hospitals that could perform this procedure.

As the then president of the Nishinomiya Medical Association, I asked the municipal government of Nishinomiya for a prioritized supply of drinking water to hospitals and hemodialysis

Table 1 Factors causing impairment of clinical functions

	Hospitals (163)	Clinics (1,528)
Interruption of water supply	73.6%	74.4%
Interruption and disturbance of telephone	60.1%	51.8%
Interruption of city gas	54.0%	49.3%
Shortage of medical workers	44.2%	47.9%
Damage to facilities and equipment	41.7%	40.9%
Interruption of electric power	33.1%	37.8%
Shortage of medical supplies	20.9%	20.4%

(Source: Survey of Disaster Medicine, Hyogo Prefecture, June 1995)

Table 2 Percentage of workers coming to work on the day of the earthquake

	Hospitals (163)	Clinics (1,528)
Physicians	58.4%	65.6%
Nurses	44.2%	39.3%
Clerks	31.0%	24.4%
Pharmacists	51.6%	51.6%
Radiological technologists	66.3%	38.3%
Other personnel	69.5%	—

(Source: Survey of Disaster Medicine, Hyogo Prefecture, June 1995)

centers. According to the announcement of the Nishinomiya City Disaster Response Headquarters, the restoration of lifelines in Nishinomiya City progressed as follows. Electricity was restored in the areas where transmission was possible on January 21, and restoration in other areas would be later depending on damage to electrical facilities. A fire was reported to have been caused by a short-circuit in a collapsed house when the power returned. While the telephone lines were restored completely by January 31, problems due to line congestions continued for an extended period of time. Cellular phones were sufficiently serviceable, as the total number of subscribers was small at the time.

Currently, cellular phones now suffer from problems due to line congestions, similarly to fixed telephones. Priority telephones designated for use in a disaster were useful for making calls, but they had the same difficulty as ordinary fixed telephones when receiving calls, and this resulted in the impediment of telephone communication. Internet communications did not work with sufficient performance due to damage to equipment and interruption of electricity, as reported later at the Hyogo Prefecture study meeting on the information network. This disruption of communications had considerable impact on medical care in the initial phases. Recovery of the water supply took more than a month, and temporary restoration was completed on February 28. City gas was not supplied for a long period until restoration was completed around April 11 after 3 months of interruption. Households got through this period using portable propane gas grills.

Impairment of Clinical Functions

According to the survey of disaster medicine conducted by Hyogo Prefecture (June 1995), major factors that caused impairment of clinical functions were as shown in Table 1. (Unless otherwise specified, descriptions in the following are based on this survey.)

The disruption of lifelines (water, city gas, and electricity) had considerable impact on the clini-

Table 3 Breakdown of patients treated at hospitals (Unit: persons)

		Serious	Severe	Slight	Hospitalized
Acute phase	1st Day	304	1,004	9,213	1,750
	2nd Day	68	477	7,411	1,251
Subacute phase	3rd Day	43	346	6,926	1,148
	4th Day	38	261	7,311	1,051
	5th Day	176	207	5,536	995
	6th Day	44	159	2,559	942
	7th Day	44	204	8,324	1,030
	Total	717	2,658	47,280	8,167

(Source: Survey of Disaster Medicine, Hyogo Prefecture, June 1995)

Table 4 Breakdown of patients treated at clinics (Unit: persons)

		Serious	Severe	Slight	Hospitalized
Acute phase	1st Day	121	590	7,303	334
	2nd Day	30	195	12,713	236
Subacute phase	3rd Day	17	254	13,107	221
	4th Day	21	310	19,566	223
	5th Day	11	221	17,713	241
	6th Day	8	75	7,191	183
	7th Day	13	249	23,732	280
	Total	221	1,894	101,325	1,718

(Source: Survey of Disaster Medicine, Hyogo Prefecture, June 1995)

cal functions of both hospitals and clinics. A shortage of medical workers was also reported by more than 40% of medical institutions. The survey also examined the workers' attendance at work on the day of the disaster (Table 2).

As mentioned above, many medical workers were prevented from going to work as a result of the damage suffered by them and their families, as well as the interruption of commuting routes. The data shown here are for the whole day, as opposed to the moment of the earthquake. Except for emergency hospitals, hospitals were very understaffed at the time of the earthquake. The rate of attendance at work was high among physicians working at clinics, probably reflecting the fact that many of them lived in the same places as the clinics. Other physicians, as well as most nurses and staff, lived in separate places, and many of them had difficulty in reaching the workplaces due to the damage from the disaster and interruption of transportation. Although the number is not known, there were casualties among employees and families. In such a situation we had to try our best to provide medical care during the initial phases of disaster.

Medical Care in the Acute Phase

We had to provide acute phase care in the situation outlined above. As the president of the Nishinomiya Medical Association, I rushed to the office of the Association with no means of obtaining a full picture of the situation. While the building of the Medical Association was unharmed, city gas was not supplied and air conditioning was not working. Electricity was available, but the water supply had stopped. We only had a limited amount of water remaining in the outdoor aboveground tank.

While trying to confirm the situation of medical institutions with no avail, due to the interruption of telephone services, I set up the Nishino-

miya Medical Association Disaster Response Headquarters. I instructed the small number of personnel appearing in the office to confirm the damage and casualties at medical institutions, and made contact with the Hyogo Prefectural Medical Association, Nishinomiya Health Center of Hyogo Prefecture, and Nishinomiya City Office. We had to cope with the situation without sufficient emergency management systems. The president of Hyogo Prefectural Medical Association instructed me to concentrate on the response in Nishinomiya City. To avoid telephone line congestions, we arranged to make subsequent contacts at fixed times late at night.

Casualties seeking medical care in this situation mostly visited medical institutions in their neighborhoods using their own means of transport, they had no information and patient transportation routes were disrupted. Calling an ambulance was impossible due to the interruption of telephone services. Ambulances were used mostly for relief activities rather than the transportation of emergency cases, and the Fire Department later identified this fact as a problem to be solved in the future. To handle the large number of patients, triage was practiced at some hospitals, while confusion developed at hospitals where triage was not performed. Victims seeking a sense of security stayed in some hospitals treating them as if they were refuge camps, and in some cases, they had to be removed by the police. Requests for medicines in short supply mainly included antibiotic injections, infusions, and local anesthetics. The breakdown of treatment was as shown in Tables 3 and 4.

The transportation of patients to hospitals outside the disaster area was almost impossible because of the interruption of telephone services and the destruction of roads. Only one patient was transported using a helicopter on the first day. In this case, the Nishinomiya Municipal Fire Department requested the transport of drugs, and the helicopter of the Osaka Municipal Fire Department that arrived in response to this request was used for carrying a patient with crush syndrome to a medical institution in Osaka. At that time, the procedure for requesting helicopter transport was complicated and not widely known. Hyogo Prefecture announced the procedure regarding the operation of helicopters on January 22. It was demanded that a physician should attend the patient in a helicopter, and this resulted in additional impairment of clinical functions at the front line of medical care.

Disaster relief teams from various organizations began to arrive on the 2nd day following the earthquake.

The total death toll from this earthquake was 6,433, including earthquake-related deaths. The police asked us to participate in post-mortem examinations of the deceased. While a medical examiner system was in place in the older parts of Kobe City, other areas did not have this system, and the police in such areas made the request to the local medical association. The number of post-mortem cases examined by medical association members totaled 2,654, as compared with 2,340 cases examined by medical examiners and 486 cases by volunteer physicians. This was also a duty of physicians, and was conducted with the participation of 388 medical association members (Survey by Hyogo Prefectural Police, June 9, 1995). The causes of death among the victims of earthquake were death from traumatic asphyxia in 4,224 cases, death due to fire and thermal injury in 504 cases, head and neck injury in 282 cases, visceral injury in 93 cases, traumatic shock in 63 cases, whole body contusion in 45 cases, crush syndrome in 15 cases, other in 123 cases, and unknown in 124 cases.

Refuge sites were opened on and after the day of the disaster and became the homes of a great many victims. Medical association members were sent to refuge sites as follows:

From hospitals: 71 persons on day 1 and 87 persons on day 2.

From clinics: 281 persons on day 1 and 293 persons on day 2.

People at various risk, such as the sick, injured, babies, the elderly, and those suffering from psychological shock, were forced to live in confined spaces with little privacy.

Medical Care in the Subacute Phase

Two days after the disaster, people were still being rescued from collapsed houses and patients with injury were being treated (Tables 3 and 4).

It is regrettable that these patients with serious and severe conditions could not be transported efficiently to the hospitals outside the disaster area, as a result of the disruption of communications and transportation routes. Although Osaka Medical Association provided about 500

beds to accept patients from the disaster area, only a part of these beds were used.

First-aid stations were opened in succession. Responding to the call of the Japan Medical Association, relief teams from prefectural medical associations, university medical schools, NPOs, the Japanese Red Cross Society, and many other organizations cooperated in providing care at first-aid stations and making rounds at refuge sites. I worked with Nishinomiya City authorities and the Nishinomiya Health Center of Hyogo Prefecture to facilitate cooperation among these teams. I also instructed medical association members to ensure early setup of clinical capabilities and to make rounds at refuge sites. While most of the relief teams were self-supported, we were perplexed by some teams that requested lodgings and meals.

Medical Care in the Chronic Phase

There were 984 refuge sites in the prefecture on January 23, and the number peaked at 1,153 with 316,678 refugees on January 23. As mentioned above, the group living with little privacy and no choice of meals was the worst situation for patients with chronic diseases, who also suffered from significant psychological shock and were in extremely insecure conditions. Starting from the subacute phase, the Nishinomiya Medical Association announced the list of local medical institutions that could provide medical care. Some of the patients with chronic diseases suffered from a shortage of drugs, because their physicians had stopped practicing or they could not visit hospitals due to the interruption of transport services. Some showed worsening of symptoms resulting from the interruption of medical treatment, change in diets, change in living environment, psychological shock, etc. Exacerbation of symptoms of mental illness, acute stress reaction due to mental stress from the disaster, alcohol dependence, and other problems were treated at first-aid stations and medical institutions that were operating.

With respect to psychological care, the director of the Nishinomiya Health Center was a psychiatrist. Psychiatrists and clinical psychologists supported by the help from neighboring prefectures performed visiting care at refuge sites from the subacute phase. In addition to medical care, public health is also an important problem at refuge sites. With mutual communication and cooperation with related organizations, we addressed the issues of privacy, post-traumatic stress syndrome, alcohol dependency, and other psychological problems, as well as health problems caused by maladaptation to the living environment and prevention of infectious diseases. It was a praiseworthy fact that there were no outbreaks of oral infectious diseases despite the continued presence of many refuge sites over a long period including the rainy season.

Proposals for Future Disaster Response

Based on this experience of an unexpected major disaster, I would like to make the following proposals:

(1) We strongly felt that physicians in general need to learn triage techniques. After the disaster, we visited various localities in response to lecture requests from local medical associations, and found to our delight that training using triage models was being performed in various departments regardless of their specialties. Training should be provided so that all physicians can perform triage. The difference between emergency medicine and disaster medicine and the purpose and meaning of triage should be announced to the general public in ordinary times to improve the people's understanding and recognition of triage.

(2) After triage, patients should be transported to medical institutions outside the disaster area according to the severity of conditions. At the time of this disaster, there were relatively few emergency helicopters in the country, helicopter transportation of patients was not widely recognized, and the procedure was complicated and time-consuming. While operation of emergency helicopters in Hyogo Prefecture was entrusted to the police before the earthquake, the organization was reformed with the establishment of the Air Rescue Team and streamlining of the procedure.

(3) Once a disaster takes place, the land transportation of patients will be impeded by the destruction of roads and other damage, and patients will concentrate at the nearest medical institutions. For this reason, first-aid stations should be established at as many loca-

tions as possible near the places suitable for the landing of a helicopter. The physicians attending first-aid stations should be assigned in ordinary times, and other physicians in the vicinity should attend to help out as long as possible in the event of a disaster.

(4) The earthquake resistance of houses must be ensured. Earthquake resistance diagnosis and reinforcement based on it are required. Furniture and equipment should be fixed to prevent from falling.

(5) Accurate information and the means of communication must be secured. Public announcement from governmental bodies via the TV, radio, etc. must be provided, and police information must be secured. While securing of the means of communication (wire, wireless, satellite telephone, the Internet, etc.) is one of the most important requirements, both fixed telephones and cellular phones are expected to suffer from inability of communication due to line congestions. Vehicle-installed satellite telephones would be the best option. The connection with the Disaster and Emergency Medical Information System must also be ensured.

(6) Lifelines must be secured and provided in redundant systems. The disruption of lifelines seriously affects clinical functions, as mentioned above. While the waterworks bureaus of some cities have recently begun replacing water supply pipes with earthquake-resistant pipes, the use of such pipes is still limited. The water required for drinking, cooking, and medical care should be stored. Use of well water should be considered with respect to water for general use, which is needed in large quantities. In this earthquake, most of the elevated water tanks installed on rooftops were damaged. In some cases, the water that spilled from the tank damaged the interior of the building. In particular, medical institutions performing hemodialysis must ensure the storage of dialysis fluid and the supply of water. While emergency generators are already used to backup the supply of electric power, the quantity of fuel in storage is limited by the Fire Services Law. In this earthquake, some generators ran out of fuel and were unable to supply power. We need to consider a means to ensure the fuel supply for power generation. As demonstrated in the experience of this earthquake, the stoppage of city gas affects a wide area and restoration takes much time. When city gas is used for cooking in hospitals, additional use of other fuels such as propane gas should be considered.

(7) Disaster response manuals must be developed and the ability to use such manuals must be ensured through repeated practice. In ordinary times, it is necessary to confirm what should be included in the emergency kit and what the roles of each staff member are. Manuals are of no use without repeated practice. A disaster like this earthquake generates a large number of deceased persons at one time, and general physicians also have to participate in post-mortem examination. Based on this experience, the Hyogo Prefectural Medical Association established the Hyogo Prefectural Medical Association Society of Police and Clinical Forensic Medicine, and is conducting training in cooperation with university departments of forensic medicine, medical examiners, and criminal identification sections of the police.

Conclusion

In this article, the lessons learned from the experience of the Hanshin Awaji Earthquake, which occurred more than 10 years ago has been described. I hope that these may be relevant today, although drastic changes in social situations have taken place during the last decade. Disasters strike when you least expect them, and the development of a situation may not go as planned in the manuals. Manuals are important, but what is more important is to take proper steps flexibly to meet the actual situation.

Lessons from the Niigata Chuetsu Earthquake in Japan

—Experience of a small medical association in hilly and mountainous areas—

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Masaaki Niwayama*1

Abstract

An Earthquake with a magnitude of 6.8 and a maximum seismic intensity of 7 hit the Chuetsu region of Niigata Prefecture in Japan on October 2004, causing tremendous impacts including the generation of about 100,000 evacuees, destruction of about 90,000 houses, and total damages exceeding 3 trillion yen (about US$27.6 billion) in the prefecture. In contrast to the Hanshin Awaji Earthquake of January 17, 1995, which mostly involved large cities on relatively flat land, the earthquake in Niigata affected hilly and mountainous areas with depopulation and aging of citizens. A lack of information, underdevelopment of communications media, and inequality in health care services emerged as significant problems, and caused confusion among rescue volunteers, who entered the disaster area in great numbers. The local medical association, without sufficient information, had to tackle the challenges of promoting cooperation with the medical teams from other areas and answering new problems peculiar to hilly and mountainous areas, such as death during overnight stays in cars. The experience from the 2 earthquakes, one in urban and the other in hilly and mountainous situations, should be utilized as typical reference information in future disaster measures not only in Japan, a country with frequent earthquakes, but also in other countries with the experience of earthquake disaster. This article does not intend to cover the details of all affected areas by this earthquake, but to outline the lessons learned from the activities of the Medical Association of Ojiya, Uonuma, and Kawaguchi following the earthquake.

Key words Disaster medicine, Earthquake, Medical team, Emergency medicine, Community medicine, Top-down, Line of command

Introduction

At 17:56, on October 23, 2004, an earthquake with a maximum seismic intensity of 7 occurred with its epicenter directly beneath the Chuetsu region, Niigata Prefecture. The areas under the coverage of our medical association (Ojiya City, Uonuma City, and Kawaguchi Town) were suddenly sent into a panic.

Niigata Prefecture is located near the middle of the coastline of Japan facing the Sea of Japan (land area 12,582 km^2, population 2.46 million). The inland region of the prefecture is surrounded by high mountains, and about a half of the total land area is classified as hilly and mountainous areas. The Chuetsu region, including the epicenter of this earthquake, consists mostly of hilly and mountainous areas. The damage from the earthquake in the whole of Niigata Prefecture is summarized in Table 1. Kawaguchi Town (land area 50 km^2, population 5,693) suffered disruption of roads, destruction of the 2 clinics, and a breakdown of lifelines, resulting in the loss of medical care and telecommunications functions. Shinkansen railway lines and expressways were

*1 Medical Association of Ojiya city, Uonuma city, and Kawaguchi town in Niigata Prefecture
Correspondence to: Masaaki Niwayama MD,
Tel: 81-25-792-5511, Fax: 81-25-792-7874, E-mail: niwayama@ams.odn.ne.jp

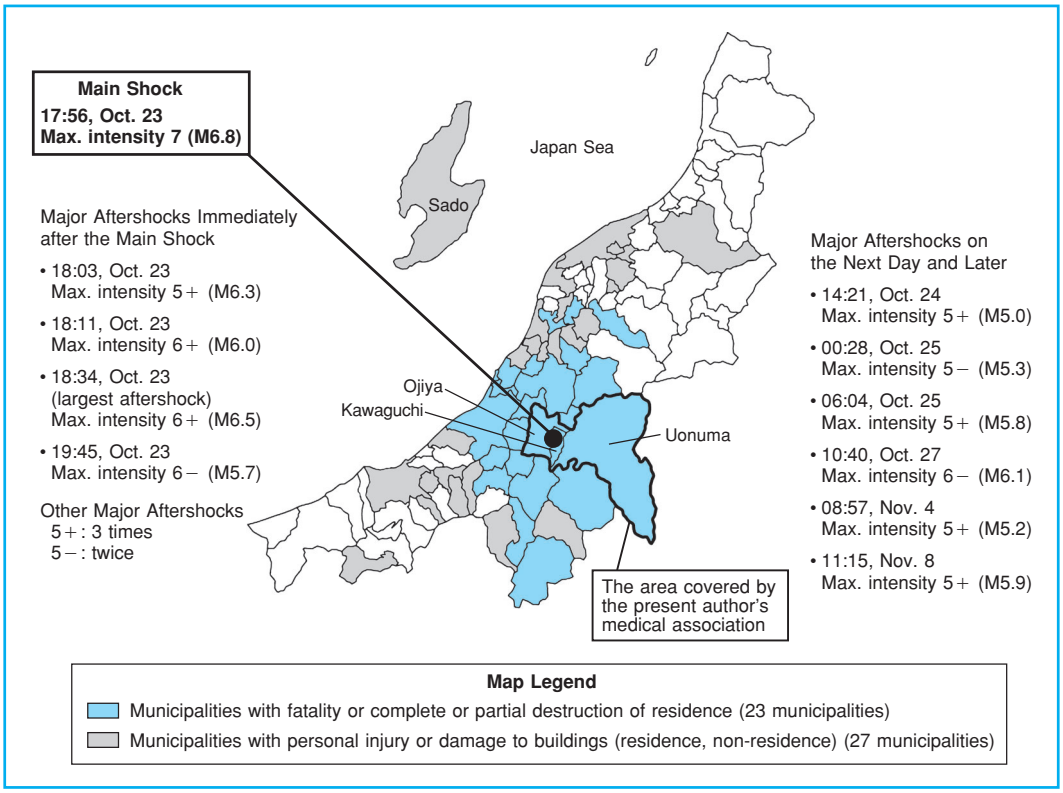

Fig. 1 Niigata Chuetsu Earthquake in 2004
Main Shock (17:56, Oct. 23; Intensity 7) and Major Aftershocks
(Press Release, Japan Meteorological Agency)

Table 1 Damage from the Niigata Chuetsu Earthquake in 2004

(Niigata Chuetsu Earthquake Disaster Response Headquarters
As of 14:00, June 22, 2005)

■ Personal Damage

Division	Death	Missing	Severe Injury	Slight Injury
Total in Niigata Prefecture	48 persons	0 persons	634 persons	4,160 persons

■ Damage to Residences

Division	Complete Destruction		Severe Partial Destruction		Partial Destruction		Slight Destruction	
Unit	Buildings	Households	Buildings	Households	Buildings	Households	Buildings	Households
Total in Niigata Prefecture	3,174	3,138	2,140	2,142	11,560	11,844	103,314	111,507

■ Damage to Non-residence Buildings and Structures

Division	Non-residence Building	Other Structure		
	Public Facility	Road	River	Landslide
Unit	Buildings	Locations	Locations	Locations
Total of Niigata Prefecture	40,368	6,064	229	442

also destroyed, and many medical rescue teams experienced considerable delay in reaching the disaster area.

The prefectural government of Niigata has been conducting regional disaster prevention meetings every year, based on the experience of the large earthquake that hit Niigata 40 years ago in June 1964 and the lessons from the Hanshin Awaji Earthquake in the western part of Japan in 1995. However, the actual response to the present emergency revealed the presence of many problems. These problems were partly derived from the geographical difference in that the former 2 disasters occurred mainly in urban areas and the present earthquake affected a large expanse of mountainous regions. The fundamental cause, however, was the disruption of the line of command.

As part of the efforts to cope with the disaster, the local medical association requested support from various medical teams and also established its own "Emergency Medical Headquarters" to support the provision of medical care.

Situations from Day 1 to Day 4

October 23

Immediately after the first earthquake, the local medical association completely lost all its functions due to the breakdown of lifelines, disruption of traffic means, and interruption of telephone services. It was impossible to perform routine medical care, collection of information, and confirmation of damage.

Most of the mass of casualties were sent to hospitals and clinics during the period from 2 hours after the shock to midnight. Wounds were treated under small floodlight lamps and flashlights. While treatment was continued 24 hours a day, prescription of drugs was impossible and there was no way to confirm any information.

October 24

In Ojiya City, the medical rescue team of the Japanese Red Cross Society started independent rescue activities in the morning by setting up headquarters in a room of the city hall and opening first-aid stations in the municipal sports hall and elementary and junior high schools in the city. While the local government sent out public health nurses and other health personnel for rescue and information collection, the local medical association was not informed of this fact. During the 2 days, Ojiya General Hospital and Uonuma Hospital received as many as 340 and 154 emergency patients, respectively.

In the communities that had lost all lifelines, inhabitants filled refuge sites illuminated with emergency power generation.

October 25

I established the disaster countermeasures office of the local medical association in Niigata Prefectural Koide Hospital, which sustained relatively little damage, and contacted the members of the medical association, as well as the responsible persons of health centers and fire departments. Activities included information exchange concerning damage to medical facilities, decision to use local practitioners in place of hospital physicians who could not go to work, and decision to start visits to refuge sites.

In Kawaguchi Town, many patients with chronic diseases and those seeking referral letters visited the hospital. The Self-Defense Forces were deployed and medical teams arrived, but their arrival was not informed to local hospitals. I established the "Emergency Medical Headquarters" in the Ojiya Medical Association to take command of activities in Ojiya City, and focused my work on Kawaguchi and Horinouchi Towns.

As the medical team of Tokyo Medical Center (6 persons including 1 surgeon, 1 physician, 1 pharmacist, 1 nurse, and 2 clerks) arrived, I explained the situation of the disaster and asked them to support the medical care in Horinouchi Town. The National Hospital Organization contacted us, and I asked for their support in Kawaguchi Town.

October 26

The Kan-etsu Expressway was passable, but cars had to use all 4 lanes to go (both north- and south-bound), switching from one lane to another in a zigzag manner. Only emergency vehicles were allowed to use the expressway. From this day on, the medical association joined the voluntary meeting held by the Japanese Red Cross Society, and the meeting started to function practically as the "medical support headquarters."

Problems in Current and Future Response to Disasters

1. Diseases and hygiene

Measures against food poisoning and other infections are required following the disaster. Because this earthquake occurred in winter, we placed emphasis on the control of influenza (Table 2). In view of the geographical features and climate of various locations throughout the world, a great variety of such measures are required according to the disaster situation. The large number of evacuees who stayed in their cars at night resulted in an epidemic of so-called economy-class syndrome, including cases of death. Response to this condition, as well as PTSD, will be required in future disasters.

As the earthquake occurred on a Saturday evening after the closing of local clinics, emergency patients mainly received treatment in hospitals. The 2 clinics in Kawaguchi Town treated casualties in an isolated situation, because road traffic was cut off.

While many patients visiting hospitals during the first 2 days following the disaster had external injuries, such as cuts from glass, burns, and fractures, the number of patients with the common cold and chronic diseases increased on later days. The action of JADP (Japanese Association of Dialysis Physicians) to patients requiring hemodialysis was particularly praiseworthy. The way of treatments to these patients will be a good reference for future disaster medicine.

2. Cooperation among medical teams

Saku Central Hospital in Nagano Prefecture sent medical teams nine times through the Japan Agricultural Co-operatives (JA). The relatively smooth continuation of medical care provided by these teams was supported by a number of facts. They brought 3 generators and many small floodlight lamps enabling emergency outpatient care at night from the very beginning. The medical teams also included nutrition staff and brought meals, canned food, water, LP gas, and portable cooking stoves to prepare food for 180 in-patients. This eliminated the need for transporting in-patients to other hospitals.

The Japan Red Cross team acted under an independent line of command separately from governmental agencies. Ten hours after the earth-

Table 2 Medical care and relief activities

(Health and Social Welfare Administration Department, Koide Regional Promotion Bureau, Niigata Prefecture)

■ Medical Care and Mental Care

Division	Persons Receiving Care from Medical Support Teams	Persons Receiving Examination or Consultation from Mental Care Teams
	From October 26 to December 22, 2004	From October 26, 2004 to January 22, 2005
Ojiya City	11,782 persons	1,690 persons
Uonuma City	1,713 persons	935 persons
Kawaguchi Town	2,497 persons	265 persons
Total in Region	15,992 persons	2,890 persons

■ Vaccination against Influenza

Division	Number of refuge sites at which vaccination was performed	Number of persons vaccinated at refuge sites	Number of persons vaccinated at medical institutions
	From November 4 to December 27, 2004		
Ojiya City	9 locations	200 persons	5,487 persons
Uonuma City	—	—	4,206 persons
Kawaguchi Town	4 locations	92 persons	850 persons
Total in Region	13 locations	292 persons	5,906 persons
		6,198 persons	

quake, the team established first-aid stations in various locations in Ojiya City near the epicenter in Kawaguchi Town, and the early provision of medical care was very effective.

One problem recognized from this disaster was the lack of sharing information among different medical teams to facilitate the appropriate allocation of human and material resources. Although it is difficult to take control the situation of all refuge sites, which numbered at least 168 places in Ojiya City alone, the highest priority should be placed on cooperation between the medical teams from outside and the members of the local medical association, who understand the health conditions of inhabitants.

With regard to the time of withdrawal, Japan Red Cross first decided to provide support for 2 weeks, based on their initial on-site survey of damage (actually the support continued for 4 weeks). On the other hand, Saku Central Hospital started support without specifying the period. When AMDA (an NGO) decided to support earthquake victims in Kobe, it planned to provide support until such time that 50% of medical institutions were restored. In this regard, the cooperation of local governments and medical associations is important when external medical teams do not have information on which to base their decision to leave.

3. Cooperation with government agencies

The information concerning the disaster area is essential to "acute phase medical care," and an indispensable prerequisite for providing this information is the close cooperation between the "Emergency Medical Headquarters" set by the medical association and the "Disaster Countermeasure Headquarters" set by the government. The lack of cooperation between both the headquarters caused several problems in the initial phase of the disaster. This experience revealed the importance of discussion and practice regarding the following matters:

(1) To secure means of communications and collect accurate information during emergency;
(2) To construct an information network using satellite telephones;
(3) To establish an organization in the role of headquarters to grasp the full picture of the disaster;
(4) To leave decision-making about disaster medicine to medical professionals;
(5) To ensure that the responsible officials of the Medical Headquarters of the medical association and those of the disaster countermeasure team of the government are stationed at the site of disaster;
(6) To handle the problems of allocation, lodging, etc. of many supporters from outside;
(7) To solve the problem of the cost of medical care given to victims; and
(8) To ask the mass media for prompt and appropriate response.

4. Proposals for disaster management

(1) To provide appropriate medical care to victims of disaster, it is necessary to estimate the human and material resources required and to ensure the appropriate allocation of medical resources. To this end, it is necessary to unify the flow of information and the line of command.
(2) As I experienced in the Great Niigata Earthquake in 1964, one of the most important issues was the management of water, including drinking water. In present-day Japan, we can expect to receive a sufficient supply of medical staff, drugs, water, food, etc. by the third day after the occurrence of disaster. However, situations differ in different parts of the world, and it is important to practice disaster management simulations including the supply of drinking water and utility water during normal times.
(3) There were cases where an outside medical team and a local hospital were working separately at a distance of less than 1 km from each other without mutual communication or sharing of disaster information. It is the responsibility of the Emergency Disaster Headquarters of the local government to assign appropriate locations to outside medical teams and to provide relevant information to both local hospitals and outside medical teams, but such activities were very difficult in the current situation of Japan, where the Emergency Disaster Headquarters did not include a medical professional. As shown in the statistics of medical workers and various organizations sent to the disaster area (Table 3), there were personnel and drugs in sufficient numbers, but they were not distributed to the areas where they were most needed during the "hyperacute phase."

It is desirable that medical professionals are stationed at the Emergency Disaster Headquarters and issue appropriate orders.

(4) The national government had already issued guidelines to strengthen medical care system at the time of disaster in 1986 (Table 4), but the opinions of physicians have not been reflected in these guidelines. As a result, local medical workers had to establish an independent support system based on the judgment of the president of the local medical association. A top-down approach is suitable for the provision of medical care in an emergency. For example, the Tokyo Metropolitan Government has issued a new rule that all participants of disaster-related medical care following a large disaster must work under the control of the Tokyo Medical Association. This should provide a good model for system development in various localities.

Conclusion

Based on my experience of earthquakes, I as a physician would like to add some suggestions as outlined below. What is important at times of

Table 3 Medical teams and public health nurses sent to the disaster area

(Health and Social Welfare Administration Department, Koide Regional Promotion Bureau, Niigata Prefecture)

■ Medical Teams Sent to the Disaster Area

Division	Medical Support Organizations and Total Operating Days		Nurses Sent from Japanese Nursing Association
Unit	Organizations	Operating days	Total person-days
Ojiya City	32	352	327
Uonuma City	16	71	130
Kawaguchi Town	10	116	404
Total	58	539	861

■ Public Health Nurses Sent to the Disaster Area

Division	Public Health Nurses Sent via Ministry of Health, Labour and Welfare		Public Health Nurses Sent by Niigata Prefecture
Unit	Local governments	Total person-days	Person-days
Ojiya City	20	1,371	55
Uonuma City	7	385	94
Kawaguchi Town	14	946	48
Total in Region	41	2,702	197

Table 4 Strengthening of early emergency medical care system at the time of disaster

1. Attendance at Regional Disaster Management Meetings
2. Conclusion of mutual disaster support agreements
3. Development of independent support systems
4. Development of key emergency hospitals
5. Strengthening of health center functions related to disaster medicine
6. Publicity, education, and training concerning disaster medicine
7. Development of disaster management manuals
8. Cooperation with fire-fighting organizations
9. System for identifying deceased disaster victims

(Health Policy Bureau, Ministry of Health and Welfare, May 1986)

disaster is, among others, the close relationships that exist between the people living in that area, regardless of how many advanced technologies they possess. The people living in these areas who have overcome the disadvantages from snowdrifts of over two meters in winter for years are kind and considerate in nature. I would say that it is this human nature that helps them surmount the various difficulties they humbly faced at the time of the big earthquake when otherwise great confusion could have occurred in society. The emergency system to cope with medical care and disasters can be technically and legally established. But the trustful relations among the people cannot be created by laws. It gives me much joy together with the other physicians involved in the rescue of the disaster victims to be able to live among the local inhabitants who exhibited the most beautiful aspects of the human spirit in helping each other while under the threat of subsequent and frequent aftershocks. While disasters almost always end in tragedy, they can also highlight the wonderful nature of people.

Although no one knows where and when, we can say a next big disaster will surely occur in the future. I really hope that the damages expected in future disasters will be minimized by the best and brightest human minds.

Review Article

Crush Syndrome in Disaster

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Junichiro Yokota*1

Abstract

Crush syndrome is a condition observed in patients who have been buried under collapsed buildings or rubble. It is characterized by rhabdomyolysis developing shortly after rescue and subsequent hyperkalemia, shock, acute renal failure, and other systemic symptoms. The development of acute renal failure can be avoided if fluid therapy is initiated early and diuresis can be induced. In severe cases, intensive care including hemodialysis, prevention of compartment syndrome, and infection control is effective in reducing the mortality. However, actual treatment involves considerable difficulties because we must deal with a large number of patients at the time of a disaster. Even in such demanding situations, we should be able to save the lives of as many patients as possible by predicting the development of crush syndrome, initiating fluid therapy as part of confined space medicine, practicing appropriate triage, and transporting patients to high-level medical institutions.

Key words Traumatic rhabdomyolysis, Ischemia reperfusion syndrome, Acute renal failure, Fluid therapy, Hemodialysis, Compartment syndrome

Introduction

The Hanshin-Awaji Earthquake (also called the Kobe Earthquake) in 1995 caused a great many cases of crush syndrome, which we rarely encounter in daily clinical practice.[1] Patients who had been rescued in apparently good condition suddenly died or gradually developed severe systemic symptoms, to the astonishment of many healthcare workers. In fact, most physicians in Japan at that time lacked sufficient understanding of this syndrome, as well as of its pathophysiology and treatment options. After this experience, they have become aware of this syndrome through seminars on disaster medicine, academic meetings, and publications. Management of injuries leading to this syndrome is not always difficult, provided that adequate care is initiated early. Rather, factors arising between rescue and transportation and initial treatment determine the outcome. The problem is that numerous cases of this syndrome occur at the time of disaster such as earthquakes when prompt treatment is often difficult. Therefore, we need to have not only a full understanding of the pathophysiology and treatment of this condition, but also to be prepared to treat numerous patients at the time of a massive disaster.

Definition of Crush Syndrome

Crush syndrome (CS) is a condition in which rhabdomyolysis develops rapidly after the skeletal muscles are released from prolonged pressure, resulting in shock, acute renal failure, and other systemic symptoms. CS develops when the limbs are subjected to prolonged pressure or tightly restrained and the patient is rescued alive. This syndrome is sometimes referred to as "traumatic rhabdomyolysis" in English language papers. In Japanese translation, the old term, "zametsu" syndrome for CS has been replaced by the new term, "atsuza" syndrome, because the former implies association with highly destructive crush injury, which is not consistent with the clinical

*1 Sakai Municipal Hospital, Osaka
Correspondence to: Junichiro Yokota MD, PhD, Sakai Municipal Hospital, 1-1-1 Minamiyasui-cho, Sakai-city, Osaka 590-0064, Japan.
Tel: 81-72-221-1700, Fax: 81-72-225-3404, E-mail: yokota-j@city.sakai.osaka.jp

appearance of injury leading to crush syndrome. While "crush injury" refers to wounds caused by or showing signs of crushing of the human body, this does not necessarily describe the clinical appearance of injury leading to crush syndrome.

Historical Overview and Epidemiology

The earliest description of CS is considered to have appeared in the German language literature in 1910, which reported "rhabdomyolysis with the triple symptoms of myalgia, loss of muscle power, and dark brown urine" observed following the Sicily Earthquake in 1909.[2,3] German medical books contain descriptions of similar symptoms observed in World War I soldiers who were buried under debris or confined in bomb shelters and then rescued. Bywaters in the U.K. first used the English term "crush syndrome" in his 1941 paper that delineated the pathogenesis of CS and established guidelines for the management of casualties.[4] He observed many civilian victims of the London Blitz, who were rescued from collapsed houses but presented remarkable swelling of wounded limbs and died from acute renal failure. He treated nearly 200 patients with CS before the end of the war.[5] Analyzing clinical cases and studies on myonecrosis and acute renal failure, he established a largely complete clinical picture of this syndrome after the war.[6–8]

Clinical cases of CS related to the Vietnam War[9] and coalmine accidents[10] appeared in the literature in the latter half of the 1960s. Later reports related to earthquakes,[11,12] local conflicts,[13,14] mine accidents,[15] railway accidents,[16] collapse of old houses[17] occasionally appeared following disasters.[18] When the earthquake in Armenia in 1989 caused about 300 cases of CS, case reports and results of animal experiments were published,[19,20] but we do not have access to the details, because the reports were mostly written in Russian. After the Hanshin-Awaji Earthquake in Japan, there have been increasing numbers of English language papers dealing with casualties in the Marmara Earthquake[21] and the Bingol Earthquake[22] in Turkey, the Chi-Chi Earthquake in Taiwan,[23] etc. (Fig. 1).

Although a majority of reported CS cases are associated with disasters involving large numbers of victims, CS may also be seen in daily practice in various situations, such as

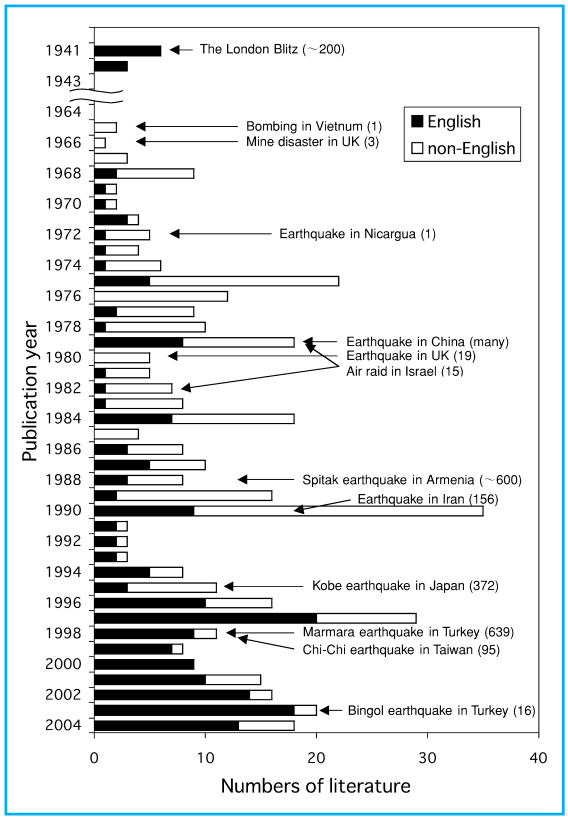

Fig. 1 Annual publication in the literature on "crush syndrome"

Articles on crush syndrome were searched with PubMed and cited reference. Remarkable disasters in relation to crush syndrome are listed chronologically.

torture involving hitting with blunt instruments,[24] comatose patients,[25] patients receiving surgery in tight body positions,[26] difficult rescue cases in traffic accidents,[27] complications with the use of MAST suits,[28] injury from using immobilizing bandaging,[29] and injury due to automatically cycled blood pressure cuff.[30] The occurrence of CS is not clear, because it frequently occurs in disasters where accurate medical statistics are difficult to obtain. CS is reported to have occurred in 7.6% of all traumatic cases in the Spitak Earthquake,[31] 13.7% of all traumatic hospitalized patients in the Kobe Earthquake,[32] and 1.4% of all hospitalized patients in the Marmara Earthquake.[21] The development of CS is considered to vary depending on the structure of the building, injury conditions, and rescue situation. A study on the Kobe Earthquake showed a significant positive correlation between

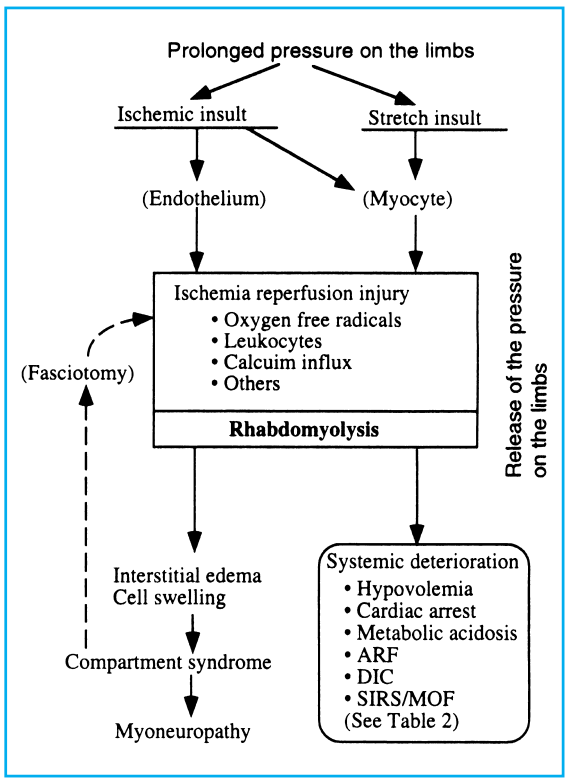

Fig. 2 Pathophysiology of the crush syndrome

the number of collapsed houses and the number of CS patients.[1]

Pathology

Locally, CS presents signs of compartment syndrome and anesthekinesia.[33] Systemically, the central pathological feature is acute renal failure[34] arising from rhabdomyolysis[35] (Fig. 2). This clinical picture resembles the systemic symptoms observed following the reperfusion of an ischemic limb.

Damage to skeletal muscles from compression

In discussing the pathophysiology of CS, it is important to consider what triggers the breakdown of the skeletal muscles. There are different theories based on similarities with other diseases; one considers that partial ischemia may be the cause, and another postulates injury to the cell membranes of the skeletal muscles due to physical force.[36]

a) Compression injury to the skeletal muscles: Stretch myopathy

Stretching of the cell membranes may initiate injury to the skeletal muscles. When the cell membranes are stretched, Ca channels are opened. The cell tries to maintain the potential difference by temporarily allowing the outflow of K, which maintains the cell volume. On the other hand, the inflowing Ca is buffered by adsorption to the organelles and the pumping function of the cell membranes. This process consumes ATP not only by the activation of Ca-ATPase but also by the reuptake of K that has been lost as a result of Na influx due to Ca/Na exchange. The increase in intracellular Ca level causes the activation of protease, phospholipase, and a wide variety of other enzymes, and furthermore, the deposition of Ca to mitochondria weakens their activity.[37] The cascade of these events causes shortage of energy in the cell and attenuation of the Na gradient, resulting in the development of cellular edema.

b) Ischemic injury to skeletal muscles

The parts of the limbs located peripheral to the sites of pressure naturally become ischemic. Skeletal muscles in complete ischemia develop edema and lysosome degranulation within about 30 min, and undergo irreversible morphological changes leading to necrosis within 4 to 6 hours at normal temperature.[38] Although such necrosis may be partially present in limbs receiving external crushing force, most parts of the limbs remain in a condition of incomplete ischemia because of the presence of collateral circulation or weakness of compression. While intracellular energy flow may be barely maintained by anaerobic metabolism, the Na permeability of the cell membranes is increased and Na-K-ATPase is activated to enhance the pumping out of Na, resulting in accelerated ATP consumption. Gradually, Ca flows into the cell with the development of cellular edema due to ATP shortage. Such damage to the cell membranes tends to occur more readily in incomplete ischemia than in complete ischemia.[39] In summary, the progression of membrane damage is promoted by the temporal contiguity or concurrence of ischemia and reperfusion taking place in the compressed parts of the muscles and their vicinity due to the presence of collateral circulation, accompanied by the mechanism of reperfusion injury discussed below.[40]

c) Propagation of intracompartment pressure
Skeletal muscles are covered with fasciae and bones to form muscle compartments. Because of this peculiar anatomy of skeletal muscles, even localized compression or unnatural posture may cause a substantial increase in intracompartmental pressure (ICP) leading to widespread muscle injury.[41] When edema develops in myocytes for the above-mentioned reasons, elevation in ICP may result and all skeletal muscles in the same compartment are affected. If ICP is elevated to 30–50 mmHg or higher, this factor alone causes skeletal muscle ischemia within 4 to 8 hours, resulting in the so-called compartment syndrome.[42] In compartment syndrome due to ordinary trauma, muscle damage is caused by factors outside the muscle, such as bone fracture, hematoma, and circulation impairment. In contrast, muscle damage in CS is caused by edema in the muscle itself.[43]

Local changes after release of pressure (rescue)

Following the release of pressure, the myocytes damaged from being stretched and by ischemia rapidly develop edema and gradually necrotize. This process involves reperfusion injury at the level of microcirculation and compartment syndrome specific to skeletal muscles.

a) Ischemia reperfusion injury
The rapid reestablishment of blood circulation following release of pressure may impair microcirculation and cause tissue injury. This condition, called ischemia reperfusion injury, expands ischemic damage through interaction between the leukocytes and endothelial cells. Many researchers have pointed out the involvement of reactive oxygen species in this condition.[44] Reactive oxygen species impair skeletal muscles and vascular endothelial cells through peroxidation of the cell membranes and the membranes of organelles.[45] Leukocytes adhere to the damaged endothelial cells and impair microcirculation, aggravating the hypoxic condition of myocytes. There is evidence suggesting that reactive oxygen species may damage myocyte cell membranes even before the shortage of intracellular energy occurs.[46]

b) Involvement of compartment syndrome
Reperfusion increases the volume of the parts affected by pressure and ischemia. In addition, edema of the skeletal muscles resulting from ischemia reperfusion is further enhanced. Cellular edema and the increase in vascular permeability causes the rapid rise in ICP, and compartment syndrome develops in the parts that have not shown overt signs of injury.

Systemic changes after release of pressure (rescue)

a) Fluid shift and hyperkalemia
As water and various substances flow into and out of the damaged myocytes (Table 1),[47] blood flow in the vicinity promotes the rapid development of systemic symptoms. We need to pay particular attention to the nonfunctional extracellular fluid and K outflow occurring relatively early following reperfusion, because hypovolemic shock and hyperkalemia are major causes of early death. Analysis of fatal CS cases following the Kobe Earthquake showed that 70% of the 27 fatalities from circulatory failure took place within 3 days. While there were 11 fatalities from hyperkalemia, 8 of them occurred within 3 days.[1] It has also been demonstrated that severe cases with a peak CK value of 75,000 U/L or more show abnormal values (Hct 52%, BE −10.2 mEq/L, and K 6.4 mEq/L) at the time of initial examination.[48] There is a case report providing detailed description of cardiac arrest due to hyperkalemia.[17] Experiments suggest the factors causing circulatory failure include not only dehydration and hyperkalemia but also loss of cardiac function. In addition, sympathetic hypertonia and sudden electrolyte abnormalities cause functional and organic changes in the myocardium.[49]

b) Development of acute renal failure
Multiple factors including a drop in renal blood flow and renal tubular ischemia due to dehydration,[7] myoglobin,[50] acidosis,[51] tension of the renal nerves,[8] azotemia and hyperphosphatemia[47] contribute to the development of acute renal failure. While myoglobinuria is certainly a central factor, few consider that it is the sole cause. When human myoglobin is injected into rabbits, renal failure may not be induced without the presence of dehydration and acidosis.[51] This fact provides the basis for advocating the importance of body fluid control and alkalizer treatment. In addition to tubular obstruction and tubular toxicity caused by myoglobin, iron ions derived from myoglobin are considered to promote the generation of reactive oxygen species and inhibit

Table 1 Flow of solutes and water across skeletal-muscle-cell membrane in rhabdomyolysis

	Consequence
Influx from extracellular compartment into muscle cells	
Water, sodium chloride, and calcium	Hypovolemia and hypodynamic shock, prerenal and later acute renal failure; hypocalcemia, aggravated hyperkalemic cardiotoxicity; increased cytosolic calcium; activation of cytotoxic proteases
Efflux from damaged muscle cell	
Potassium	Hyperkalemia and cardiotoxicity aggravated by hypocalcemia and hypotension
Purines from disintegrating cell nuclei	Hyperuricemia, nephrotoxicity
Phosphate	Hyperphosphatemia, aggravation of hypocalcemia, and matastatic calcification, including the kidney
Lactic and other organic acids	Metabolic acidosis and aciduria
Myoglobin	Nephrotoxicity, particularly with coexisting oliguria, aciduria, and hyperuricosuria
Thromboplastin	Disseminated intravascular coagulation
Creatine kinase	Extreme elevation of serum creatine kinase level
Creatinine	Increased serum creatinine-urea ratio

(From Better OS[47])

the action of vasodilator factors.[52]

c) Changes in serum calcium and phosphorus

The phosphorus flowing out of the cells tends to combine with calcium and be deposited in the body as a result of lowered renal function, and this sometimes appears as calcification in X-ray observation.[53] This deposition is reported to appear more clearly on CT images of affected limbs.[54] Combined with the influx of Ca into the damaged cells, Ca deposition causes remarkable hypocalcemia during the oliguric phase. In contrast, hypercalcemia develops when the patient enters the diuretic phase.

d) SIRS or sepsis

This syndrome causes gradual strengthening of systemic inflammatory response in addition to body fluid movement and renal failure. Leukocytosis, CRP increase, and fever are observed when no infection foci are expected to occur, and the patient often presents the remote organ failure such as DIC, respiratory failure, or liver impairment. While the most significant cause of death during the initial 2 weeks is acute renal failure, later deaths are caused chiefly by multiple organ failure.[48] Considering the recent concept of systemic inflammatory response syndrome (SIRS), it is possible that the condition involves various mediators derived from leukocyte activation. However, one study reported the lack of significant differences in TNF-alpha and IL-1 beta compared with healthy persons, and there is no evidence supporting this possibility.[55] Studies in the former USSR include a paper stating that early hypercatecholaminemia is involved in shock, organ failure, and depression of immunity.[56] Extreme tension of the sympathetic nerves due to pain and mental stress has already developed when the body is being compressed. Catecholamine suppresses tissue perfusion, promoting tissue damage and depressing the monocytic phagocyte system and immune system. The author of the above paper discusses decompression after rescue leading to hypercatecholaminemia, fluid shift, and intoxication with myolysis and pathogenic microflora products, resulting in shock, organ impairment, infection, DIC, etc. On the other hand, it has been pointed out that fasciotomy for compartment syndrome tends to be a cause of infection and sepsis.[57]

Disaster Medicine to Cope with Crush Syndrome

Rescue and on-the-spot treatment

It is important to expect that a patient buried under debris or a collapsed house to develop CS

Table 2 Clinical manifestation of crush syndrome

Immediately following extrication (on the spot)
1. Stable vital sign
2. Clear conciousness, unless head injury
3. Emotional complaint, but no physical complaint
4. Numbness of the involved limbs, exception for a short time of pain after extrication
5. Flaccid paralysis of the injured limb
6. A patchy pattern of sensory loss, mainly to pain and touch
7. Patches of erythematous skin, delineating accurately the areas of compression
8. No limb edema initially
Several hours to a couple of days after extrication (e.g. on admission)
1. Hypovolemia and hypodynamic shock; hemoconcentration
2. Hyperkalemic cardiotoxity
3. Metabolic acidosis
4. Oliguria, myoglobinuria; prerenal and later acute renal failure
5. Insensitive and paralyzed limbs
6. Compartment syndrome following gross edema of the injured limb
7. Present distal pulses of the edematous limb
8. Blister formation of the erythematous skin, mistaken for burns
Following fluid therapy
1. Hemodilution
2. Weight gain and sequestration of external cellular fluid
3. Congestive lung, ARDS
4. DIC
5. SIRS
6. Sepsis

from the rescue stage. Table 2 summarizes the physical findings to be examined as the basis for diagnosis. Unless complicated by other injury, the patient is fully conscious and vital signs are stable at the time of rescue. Therefore, severity evaluation and triage based on vital signs alone tend to result in underestimation of the patient's condition, and much attention must be paid to the injury mechanism and physical findings in the limbs.[43] Even if the affected limb has no swelling or skin damage, motor paralysis and paresthesia are always observed. Paresthesia often presents an irregular map-like appearance. While the skin is sometimes intact, cases of protracted compression show pale skin at the center with circulation impairment, and blisters are observed.

Cases with accompanying head and trunk injury or bone fracture in the limbs present complicated clinical symptoms. In addition, it is important to understand that clinical symptoms change depending on the time after rescue.

Recently, the term "confined space rescue" has been used to describe the extrication of victims confined in closed or small spaces, and medical practice conducted in such situations is called "confined space medicine".[3] Confined space medicine is not a pure medical discipline, but a form of practical medicine striving to incorporate medical treatment into the process of difficult rescue. Confined space rescue is characterized by risk involved in rescue activities arising from the presence of hazardous substances (carbon monoxide, toxic gas, etc.), oxygen-depleted air, the possibility of explosion, the collapse of housing structures, etc. As a result, rescue activities may take long time to complete, and only limited, basic medical care can be provided in the process. Victims of disasters with a high probability of developing CS are in fact confined in such dangerous situations.

Efforts to rescue victims should not be abandoned for at least the first 5 days.[58] In the case of the Marmara Earthquake, the longest time before the rescue of live victims was 135

Table 3 Infusion therapy

On the spot
1. Normal saline should be infused at 1.5 liters/h.
2. Continuous infusion should be secured by the time of arrival at a hospital.
In the hospital
1. A standard solution of 75 mEq/L NaCl in 5% dextrose*1 should be started at 500 mL/h.
2. If a diuretic response of more than 300 mL/h is not achieved, and CVP rises by more than 5 cm H_2O, the infusion should be stopped and manitol, 1 g/kg of body weight, as a 20% solution should be administered IV.
3. Once a diuresis of more 300 mL is established, fluids excreted in the urine should be replaced with a solution of 5% dextrose with the sodium and potassium content adjusted, on the basis of measurements made on the previous six-hour urinary collection.
4. Sodium bicarbonate, 44 mEq/L, should be added to every other 500 mL bottle of the standard NaCl in 5% dextrose solution.*2 The dose of sodium bicarbonate will be adjusted to maintain urinary pH above 6.5.
5. Acetazolamide (Diamox) should be administered in a dose of 250 mg IV if plasma pH approaches 7.45.
6. Disappearance of visible myoglobinuria and a leveling off of the negative potassium balance will indicate a cessation of this treatment protocol.
(The urinary pH is measured hourly. Six hourly collections of urine should be assayed for sodium content, potassium content. Blood gases, plasma pH, and serum electrolytes are similarly measured every six hours.)

This protocol[35] is modified from D. Ron.[13]
*1: Solution with a similar composition in Japan is KN1A.
*2: The solution will contain of 150 mEq/L of Na^+, 69 mEq/L of Cl^-, and 81 mEq/L of HCO_3^-.
A solution with a similar composition in Japan will be equivalent to 40 mL of sodium bicarbonate added to a 500 mL bottle of KN1A. It will contain of 145 mEq/L of Na^+, 71 mEq/L of Cl^-, and 74 mEq/L of HCO_3^-.

hours, and victims with less severe injury are expected to withstand longer before rescue and survive.

Initiation of fluid therapy

Fluid therapy is the first choice in the management of CS, because the development of shock and acute renal failure can be avoided by the early provision of fluid resuscitation, such as the initiation of fluid infusion on the spot before rescue. As early as 1943, the UK Department of Health directed that air-raid victims be given large quantities of water containing sodium bicarbonate before rescue.[5] The importance of pre-rescue and on-the-spot fluid therapy was later emphasized by the US armed forces during the Vietnam War,[9] urologists in former East Germany,[59] a review in Australia,[60] those by a group in Israel,[61,62] and study reports on the Kobe Earthquake[63] and the Bingol Earthquake in Turkey.[22] The initiation of infusion before rescue is particularly recommended, but the decision should be made considering the safety of activity in a confined space. Since the infusion route established on the spot of disaster is liable to the risk of infection, it should be replaced soon after rescue. Due to the risk of inadvertent aspiration, oral feeding is now considered an option to be selected only when infusion is impossible.

The purposes of fluid therapy in CS are: (1) to replenish the shortage of extracellular fluid: (2) to promote the renal excretion of potassium; and (3) to avoid acute renal failure. On the spot of disaster, the rapid administration of physiological saline is conducted at a rate of 1.5 L/h (10–20 mL/kg/h for children), and an infusion cocktail containing sodium bicarbonate 1 A and mannitol 10 g per 1 L of infused fluid is recommended (3). No consensus has been reached concerning the use of lactate Ringer solution or acetate Ringer solution.

Mannitol is effective in improving blood pressure through the increase in extracellular fluid and strengthening of the contracting power of the myocardium. It also protects the kidneys through various mechanisms such as dilation of glomerular blood vessels, enhancement of filtration pressure, increase in tubular flow, and inhibition of damage from reactive oxygen

species.[64] In addition, it retards the progression of compartment syndrome via an action resembling the mechanism for the suppression of brain edema.[65] In addition to the osmotic effect, this efficacy is considered to involve the action of mannitol as a scavenger for reactive oxygen species involved in cell membrane impairment.[66]

Sodium bicarbonate improves hyperkalemia and metabolic acidosis, and prevents myoglobin and uric acid deposition in the renal tubules.[62] However, alkalosis tends to cause ectopic calcification (deposition of calcium phosphate), and this must be corrected by the use of acetazolamide.

If fluid therapy is performed in a medical institution equipped for drug preparation, a protocol modified from the formula of Ron et al.[13] may be considered (Table 3). The principle of this protocol is the use of a starting fluid to avoid potassium load and the use of an alkaline isotonic electrolyte fluid with sodium bicarbonate adjustment. The goals of fluid therapy are stabilization of circulation, hourly urine volume of 200 to 300 mL, blood pH<7.5, and urine pH between 6 and 7.

If fluid therapy is not initiated early, the patient may suddenly die from shock and hyperkalemia. Avoidance of acute renal failure is usually difficult unless fluid therapy is initiated within 6 hours. Even if the patient does not develop severe conditions, the patient presents dark brown urine (mainly myoglobinuria) due to oliguria several hours after rescue, and gradually develops hyperkalemia, hyperphosphatemia, hypocalcemia, azotemia, metabolic acidosis, and high CK blood levels.

Triage and severity evaluation

Unless complicated by other injury, the patient shows relatively stable vital signs at the time of rescue. In fact, a review of CS cases following the Kobe Earthquake showed that initial measurements of blood pressure and heart rate indicated no abnormalities predicting circulatory failure.[1] Therefore, patients are rarely classified as having an immediate life threat (red) at initial triage using START (Simple Triage and Rapid Treatment) or the UK Triage Sieve, and they are likely to be undertriaged. Because patients with CS are likely to take a sudden turn for the worse at any time from immediately after rescue and management of acute renal failure will be eventually needed, we need appropriate triage criteria to avoid the preventable death of CS patients. For this purpose, we need to improve Step 2 anatomical criteria and Step 3 mechanistic criteria in secondary triage. Specifically, "paralysis of limbs" should be added to the anatomical criteria and "confinement in a closed space or burial under debris" should be added to the mechanistic criteria, and patients meeting these criteria should be considered as having CS.

According to an experimental study, the severity of CS is proportional to the time of compression and the amount of injured muscles.[67] However, in actual disasters, no correlation is found between the time to rescue and severity.[68] This may reflect the fact that less severe cases withstand longer before rescue. There is certainly a correlation between the volume of injured muscles and severity. The extent of injury can be evaluated by CK level,[1] blood myoglobin level,[69] the number of parts with compartment syndrome,[33] and the number of limbs affected by compression.[48] Oda et al. found that patients with a larger number of injured parts had higher CK levels, and the CK level was higher than 250,000 U/L when injury involved both lower limbs and the trunk. The CK level is elevated by approximately 50,000 U/L for each affected limb. Therefore, it is reasonable to evaluate severity based on the number of affected limbs on the spot of disaster.

Establishment of hemodialysis

In the Kobe Earthquake, only 25% of the patients who received infusion within 40 hours after disaster developed renal failure, while all patients in which infusion was initiated more than 40 hours after disaster developed renal failure.[63] Early fluid therapy increases the frequency of cases not requiring hemodialysis, but even with such efforts, about 40% of patients with CS following a disaster need hemodialysis. Of the 639 patients with CS following the Marmara Earthquake, 477 (74.6%) needed hemodialysis.[21] During treatment, patients with CS often develop multiple organ impairment and sepsis in addition to acute renal failure. Surgical treatment of compartment syndrome and necrotic tissues may also become necessary. Therefore, many hospitals with hemodialysis, intensive care, and orthopedic surgery capability must be made available, and casualties must be transported to

such hospitals. If diuresis is not achieved by fluid therapy, precautions should be taken during transportation to prevent congestive heart failure, pulmonary edema, and hyperkalemia due to excessive infusion. Portable analyzers are useful for monitoring electrolytes and other parameters at first-aid stations and during transportation.[70]

However, the strategy based on the transportation of casualties has limitations both in the capacity of transportation and in the availability of medical facilities providing hemodialysis. Following the Spitak Earthquake in Armenia in 1988, many patients requiring hemodialysis were transported to hospitals, but some patients were unable to receive treatment because of the limited number of hemodialyzers. Learning from this incident, the International Society of Nephrology (ISN) in Europe established the Renal Disaster Relief Task Force (RDRTF) in 1995.[71] RDRTF launched a program to send a team of medical staff specializing in hemodialysis and hemodialysis equipment. In fact, the team began operation within 6 hours after the Marmara Earthquake and treated 462 cases of acute renal failure. The mortality rate among these patients was 19%. Thus, we need activities following the example of RDRTF in parallel with the transportation of patients to non-disaster areas.

Selecting blood purification methods other than hemodialysis is still controversial. Because the clearance of myoglobin is not affected even by the use of methods other than HD, such as PE and CHDF, blood purification in CS should be regarded as the means for treating acute renal failure rather than the elimination of myoglobin.[72]

Treatment of compartment syndrome

No consensus has been reached concerning whether or not fasciotomy should be performed to treat compartment syndrome in CS. Early treatment certainly improves chances of preservation of the functions of affected limbs and avoidance of amputation, but the inevitable development of infection worsens life prognosis.[36,43,73] Many reports have pointed out the risk of uncontrollable hemorrhage and infection associated with fasciotomy in CS. Incision causes hemorrhage from muscles even in parts considered necrotic, and physicians often hesitate to conduct debridement, resulting in further progression of necrosis due to increased swelling. In this condition, wound closure is impossible, and the wound eventually becomes the focus of septic infection, necessitating radical debridement and amputation.[74] Fedorov et al. warned that inadequate surgical treatment in the early periods (complete closure of open wounds, failure to perform the debridement of fat and soft tissues, etc.) leads to severe wound infection.[75] Zimina et al. identified decompressing wounds as a cause of death from sepsis or infection, in addition to shunts and catheters.[76] Decompressing incision was performed in 49 (13%) of 372 cases following the Kobe Earthquake. Wound infection occurred in 12 cases (24%) and 2 patients died from sepsis. Following the Chi-Chi Earthquake in Taiwan in 1999, fasciotomy was performed in 35 patients, resulting in wound infection in 8 cases, deep infection in 16 cases, and amputation of affected limbs in 6 cases.[23] Of the 639 cases treated following the Marmara Earthquake, infection occurred in 223 cases (34.9%) and sepsis developed in 121 cases (18.9%). An analysis of the correlation between sepsis and fasciotomy showed a significant difference ($P<0.01$) between the 24.8% (80/323) and 13.0% (41/316) occurrence rate among fasciotomized and non-fasciotomized cases, respectively. Erek et al. also concluded that fasciotomy was a factor inducing sepsis.[21]

The fact that most neurologic symptoms improve after follow-up observation without incision provides the basis for rejecting aggressive treatment. In particular, as paresthesia resolves almost completely, conservative treatment is expected to achieve higher quality in ADL than fasciotomy or amputation, although some ROM restriction due to contracture may remain.[13,14] With some victims of the Kobe Earthquake, there were some cases in which it was difficult to conclude whether peripheral paralysis of the lower limps was caused by ischemic injury due to compression of the nerves or by complications with compartment syndrome. These patients showed remarkable recovery of muscle power within 8 to 9 months without decompressing incision, although recovery in the area around the peroneal nerve was retarded.[77] Matsuoka et al. studied the 2-year functional outcome of the 58 limbs affected by compartment syndrome of the victims of the Kobe Earthquake with CS. They obtained no evidence that fasciotomy improves outcome. Delayed rescue, delayed decompression, and radical debridement after

fasciotomy were identified as negative factors. They concluded that fasciotomy is indicated for patients that have been rescued early, and surgical treatment in the acute phase should be as minimal as possible.[78] Fasciotomy requires measurement of intracompartmental pressure, but hygienic manipulation is difficult to perform on the spot of disaster or at first-aid stations. For the reasons discussed above, many physicians are cautious about the use of fasciotomy for compartment syndrome in CS patients following a disaster.

Treatment after transportation to hospital

Fluid therapy and hemodialysis for acute renal failure are the central part of treatment in the early periods after injury. However, severe cases require intensive care to cope with various complications such as ARDS, DIC, infection, and sepsis. Patients with open wounds, those with ischemic necrosis in the soft tissues, and those receiving fasciotomy inevitably develop infection, requiring repeated debridement and often amputation of the affected limb. As discussed above, we need to remember that late deaths are caused by sepsis and multiple organ impairment. A review of the 97 fatalities following the Marmara Earthquake (mortality rate 15.2% = 97/639) also demonstrated that the main causes of death were complications with sepsis, thrombocytopenia, DIC, acute respiratory distress syndrome (ARDS), and thoracoabdominal trauma, emphasizing the importance of the clinical capacity to treat these injuries and organ impairment. A study of the 6,107 patients hospitalized in 95 hospitals over 15 days following the Kobe Earthquake compared treatment outcome among patients treated in hospitals in disaster areas and those in non-disaster areas.[32] The patients treated in hospitals in disaster areas showed a higher mortality rate from CS and trauma than the other group of patients. This suggests the need for treatment at high-level medical institutions.

Conclusion

CS is not a serious disease, provided that it occurs sporadically at ordinary times. However, the large number of patients and the limited medical treatment available in major disasters make the treatment of this syndrome a considerable challenge. Even in such demanding situations, we should be able to save the lives of as many patients as possible by predicting the development of CS, initiating fluid therapy as part of confined space medicine, practicing appropriate triage, and transporting patients to high-level medical institutions.

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Review Article

Posttraumatic Stress Disorder after Disaster: Issues of screening and early support

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Noriko Setou,[*1] Soichiro Maruyama,[*2] Kanehisa Morimoto[*3]

Abstract

Facilitation of spontaneous recovery from psychic trauma and prevention of severe, chronic stress reactions is important in providing psychological support for disaster victims. To this end, it is necessary to appropriately screen victims for the risk of chronic mental diseases such as PTSD. This document outlines current problems particularly in the screening and diagnosis of PTSD, and discusses early support and mental health care at the time of the disaster. In addition, given the behavioral characteristics of Japanese people during and after a disaster, it is emphasized that psychosomatic approaches consider not only the psychological aspects of PTSD but also somatization of symptoms in providing early support for disaster victims.

Key words PTSD (posttraumatic stress disorder), Disaster, Risk factor, Screening, Early support

Introduction

Research into psychiatric assistance for disaster victims is still in the early stage in Japan. In this country, posttraumatic stress disorder (PTSD) rarely has been a topic of discussion even among specialists in the field, since it was categorized and criteria specified in DSM-III in 1980. However, psychic trauma and the care of individuals with this condition has been attracting increasing attention over the past 10 years, largely through social interest in providing psychological support to victims of disasters including the Mt. Unzen-Fugen eruption in 1990, the Hokkaido Nansei-Oki (Okushiri Island) earthquake in 1993, the Great Hanshin-Awaji Earthquake, and the sarin attack in 1995. More recently, the need for psychological care immediately after a disaster has been highlighted by the Niigata Chuetsu earthquake of 2004, the earthquake west of Fukuoka, and the JR West Amagasaki accident in 2005. The memory is still fresh in the minds of the Japanese population that mental health professionals were sent to these disaster sites, and consultation services were provided early in the postdisaster phase.

Such immediate action is of great importance in providing victims and mental health professionals in the field with accurate information about the psychological reactions that occur after disasters and in motivating therapeutic intervention if necessary. On the other hand, there is the risk of confusing normal stress reactions and awareness of injuries with morbid conditions such as PTSD, which can be based on a tacit understanding that victims are unlikely to recover without support.[1]

According to epidemiological surveys in the US,[2,3] about a half the population encounters at least one traumatic event in their lifetime. Therefore, it is not uncommon for individuals to suffer

*1 Department of Medicine Faculty of Health Science, Kobe University Graduate School of Medicine, Kobe
Department of Pediatrics, Kobe University Hospital, Kobe
*2 Department of Clinical Psychology, Graduate School of Literature, Kobe-Shinwa Women's University, Kobe
*3 Department of Social and Environmental Medicine, Osaka University Graduate School of Medicine, Osaka
Correspondence to: Kanehisa Morimoto DMSc, Department of Social and Environmental Medicine, Osaka University Graduate School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan.
Tel: 81-6-6879-3920, Fax: 81-6-6879-3929, E-mail: morimoto@envi.med.osaka-u.ac.jp

extreme shock and distress from traumatic experiences such as natural disasters, accidents, terrorism, sexual violence, and the sudden death of someone close. Victims often show psychological reactions in the acute posttraumatic phase that include emotional numbing, insomnia, intrusive thoughts of the event, irritability, and avoidance behaviors.[4,5] Many of them, however, show marked spontaneous recovery with the passage of time through their ability to heal themselves. PTSD remains a chronic health problem in 8–9% of those who experience trauma,[2,3] whereas many of the initial psychological reactions are considered "normal reactions" that attenuate within a few months. Even if initial, temporary symptoms meet the diagnostic criteria for PTSD, nearly 70% of such symptoms ameliorate spontaneously within 6 years. Many of the remaining 30% of symptoms will follow the course of refractory illness regardless of whether or not treatment is provided.[3] Although it is necessary to give serious attention to the risk that PTSD may induce chronic psychosocial malfunction, there are diverse ways of recovering from trauma, and not all distress and emotional disturbance are unfavorable. In some cases the traumatic event can lead the victim's personal growth as an individual or result in stronger relationships with loved ones.[6] The main issue to be addressed is not necessarily the acute-phase symptoms but the severe, chronic symptoms.

From the standpoint of disaster psychiatry, facilitation of spontaneous recovery and the prevention of severe, chronic change in the stress reaction are two important goals. The latter in particular calls into question how to identify and screen for high-risk individuals. However, there have not been sufficient empirical and epidemiological studies of this field even in Europe and North America. Much remains to be clarified as to proper early assistance after a disaster.

This paper outlines issues involved in the screening and diagnosis of PTSD in relation to the psychological support of disaster victims and discusses the ideal way to provide early mental health support in disaster situations. The behavioral characteristics of the Japanese people during and after a disaster are also addressed.

Factors Affecting Traumatic Reactions

According to DSM-IV-TR,[7] a diagnosis of PTSD requires the development of characteristic symptoms following exposure to an extreme traumatic stressor. A diagnosis of PTSD is made when the characteristic symptoms—including persistent re-experiencing of the traumatic event, persistent avoidance of stimuli associated with the trauma, and persistent symptoms of increased arousal—are present for more than one month, causing clinically significant distress or impairment in social, occupational, or other important areas of functioning. Although various factors are involved in the prognosis of PTSD, one of the most important is the type and intensity of the traumatic experience itself.

Table 1 Simple versus complicated trauma

Simple Trauma	Complicated Trauma
Involves single event	Involves several repetitive events
Is of brief duration	Occurs over a long period of time
Occurs late in life	Occurs early in life
Contains no violence created by human beings	Involves violence created by human beings
Allows patients an active role	Allows patients a passive role
Carries advance warning	Occurs suddenly, with no advance warning
Has time-limited symptoms	Has long-lasting symptoms; can produce personality or neurophysiologic changes; causes some symptoms that may be reversible
Symptoms resolve spontaneously or with support in the recovery environment	Professional intervention is usually necessary for symptom resolution

(Excerpted from Peebles-Kleiger MJ et al. Postgrad Med. 1998.[8])

The prevalence and clinical course of PTSD and its effects on personality and neurophysiology depend on whether the traumatic experience is a single exposure to, for example, a natural disaster, accident, or fire, or repetitive or persistent exposure to abuse, torture, or extended warfare (Table 1[8]). Psychological consequences experienced by the victim are more likely to be prolonged after human-inflicted or interpersonal trauma than after natural disasters.[9]

In addition, there is a dose-response relationship in most cases as the incidence of PTSD increases with increasing severity of the experience or stressor (e.g., danger, threat to life, or other traumatic events). However, the relationship is not a simple causal one. Subjective cognitions and reactions to the event, rather than the objective severity of the event itself, have enormous influence.[10] The severity of the stressor is related to the degree of adversity of the event as well as the number of exposures to the stressor. An event characterized by malicious and grotesque trauma can cause the onset of PTSD even in a person who has been only a witness to it, and the risk of PTSD is further increased if the person is directly involved.[11]

People may or may not develop PTSD with exposure to the same type and degree of traumatic event, depending on the vulnerability of the person. The vulnerability mentioned here includes a past history or family history of mental disease and predispositions of personality. Other factors that render the individual vulnerable are poor coping and adaptation skills, a low level of support, and unskillful actions and/or improper treatment after trauma.[12]

Economic hardship and lack of social support are social factors that interfere with recovery from trauma. Social recognition of the trauma and giving meaning to the traumatic experience greatly influence the perception, assessment, and handling of symptoms by the victims themselves. Therefore, these variables should also be taken into consideration.[13]

Although the mechanisms of onset of PTSD in the presence of these risk factors have yet to be clearly elucidated, it is important to identify high-risk individuals in the context of the traumatic experience of victims when considering posttraumatic measures in mental health.[12] For example, in victims of human acts such as terrorism, accidents, and sexual violence, loss of interpersonal reliance or sense of security lowers the coping skills of the individual; with stigma and lack of understanding by the victim's caretakers or social network, it becomes difficult for the victim to receive adequate support. On the other hand, in cases of natural disasters, damage from the loss of home, property, and community is very large. Therefore, problems come to the surface as secondary stress in daily living rather than as psychological reactions to trauma.

Issues Involved in Diagnostic Screening

Although there has not been sufficient research documenting that early psychological intervention after disasters prevents chronic mental health problems, posttraumatic stress victims, in most cases, are in need of psychological information and guidance, social support, and someone to listen to their feelings and concerns.[14] Most victims follow a course of spontaneous recovery following the simple provision of information and social support. Although early intervention by specialists does not have an immediate therapeutic impact, it is beneficial in that it allows for the screening of high-risk individuals and can provide appropriate management.[13,15] People who expect recovery to be difficult should be subject to screening.[16] However, the following issues in diagnosis at the time of screening have recently been elucidated.

a. Acute stress disorder (ASD)

In DSM-IV[17] and DSM-VI-TR,[7] acute stress disorder (ASD) is defined as a stress reaction that develops within one month of trauma and persists for at least 2 days and at most 4 weeks. The two major diagnostic differences between ASD and PTSD are the duration of clinical manifestation and the dissociation characteristic of ASD (Table 2[18]). ASD fulfills two roles in the diagnostic criteria: one is to characterize the diagnostic interstice of the first month of posttraumatic stress reaction, and the other is to provide a distinction between transient stress reaction and the chronic traumatic reaction that develops into PTSD.[19]

However, there are various criticisms of the DSM positioning of ASD. The reasons cited include 1) the paucity of evidence and discussion concerning ASD itself; 2) the risk of providing a non-objective diagnosis of ASD without con-

sidering other diagnoses; 3) doubt as to whether or not the emphasis on dissociation is appropriate; 4) the risk that a diagnosis of ASD will predict other diseases in addition to PTSD; 5) the risk of misdiagnosing a normal reaction as morbid; and 6) doubt as to whether or not ASD and PTSD are actually distinguishable by the duration of clinical manifestation.[20]

It has long been indicated that symptoms of dissociation exert an important influence on adaptation after traumatic experience, and prospective studies have provided the supportive finding that dissociation may mask the memory of traumatic experience and interfere with the recovery process, thus leading to PTSD.[21,22] However, an opposing view takes the position that symptoms of dissociation immediately after trauma do not necessarily persist or lead to subsequent morbidity.[23] Bryant provided interesting data as to whether ASD allows the prediction of PTSD (Table 3[20]). Although there are certain variations in data from different studies because of issues of methodology, the table shows that ASD generally predicts PTSD, but a diagnosis of PTSD does not necessarily mean that there was preceding ASD. In other words, persons with ASD were at risk of developing PTSD, but those who developed PTSD included many individuals who did not meet the criteria for ASD. Bryant pointed out that the presence of symptoms of dissociation, which should be a key factor in predicting PTSD, may lead to individuals at high risk

Table 2 Diagnostic criteria for ASD and PTSD

Criteria	ASD	PTSD
Stressor	Both: Threatening event Fear, helplessness, or horror	Both: Threatening event Fear, helplessness, or horror
Dissociation	Minimum three of: Numbing Reduced awareness Depersonalization Derealization Amnesia	
Reexperiencing	Minimum one of: Recurrent images, thoughts, or distress Consequent distress not prescribed Intrusive nature not prescribed	Minimum one of: Recurrent images, thoughts, or distress Consequent distress prescribed Intrusive nature prescribed
Avoidance	"Marked" avoidance of: Thoughts, feelings, or places	Minimum Three of: Avoid thoughts or conversations Avoid people or places Amnesia Diminished interest Estrangement from others Restricted affect Sense of shortened future
Arousal	"Marked" arousal, including: Restlessness, insomnia, irritability, hypervigilance, and concentration difficulties	Minimum two of: Insomnia Irritability Concentration deficits Hypervigilance Elevated startle response
Duration	At least 2 days and less than 1 month posttrauma Dissociative symptoms may be present only during trauma	At least 1 month posttrauma
Impairment	Impairment functioning	Impairment functioning

(Excerpted from Bryant RA et al. Cli Psycho Rev. 1997.[18])

for PTSD being overlooked.[20]

On the other hand, acute stress reaction (ASR) is described in ICD-10[24] as a serious transitory disorder occurring in response to extreme stress. However, the time of onset and duration of symptoms are different from those described in DSM-VI. In ICD-10, ASR is described as usually occurring within a few minutes after the shock of a stressful stimulus or event and disappearing within 2 or 3 days (often within a few hours), whereas PTSD occurs after a latent period of several weeks to several months after a traumatic event. Although the description of the period is clearer in DSM-VI, congruent descriptions are needed in DSM-VI and ICD-10, since laws and systems related to workers' compensation and relevant national examinations generally use ICD-10 of the World Health Organization in Japan.

b. Comorbidity (presence of concomitant mental diseases)

Patients with PTSD often experience mental comorbidities including major depression, anxiety disorders such as panic disorder, and other mental diseases such as alcoholism.[3,25] In addition, traumatic experience exerts a great deal of influence on both the mental and physical health of the victim, with a close relation between PTSD and psychosomatic disorders being suggested.[26] In the clinical setting, it is more common for physicians to see individuals with PTSD who also have such comorbidities rather than those who have pure PTSD symptoms alone. In these situations, the question is how to diagnose such conditions, since findings related to the dual diagnosis of PTSD are insufficient.[27]

For example, when a patient with PTSD has concomitant major depression, the disorder that is more closely involved in the current symptoms should be identified, and the order of priority for treatment should be determined to develop an intervention program that facilitates improvement of the coexistent symptoms.[27] In patients with marked depression, the depression may mask avoidance or dissociation, and, therefore, the patients themselves may not recognize the PTSD symptoms, which eventually may be overlooked in the diagnostic process. On the other hand, even in patients with marked symptoms of PTSD, such symptoms are not simply followed by other morbid conditions. Other symptoms may appear in a delayed manner when the memory of the traumatic event is triggered, or with the complication that self-esteem was threatened in the traumatic experience. It is possible that the chronic changes of stress reaction cause changes in the endocrine secretion of catecholamine and neurophysiological changes in the body, leading to a variety of mental symptoms.[28]

Kato[29] reported that complaints characteristic of PTSD were unexpectedly rare among victims

Table 3 Summary of prospective studies of acute stress disorder

Trauma Type	Study	Proportion of ASD who develop PTSD	Proportion of PTSD who had ASD
MVA	Harvey & Bryant (1998a)	78%	39%
Brain injury	Bryant & Harvey (1998)	83%	40%
Assault	Brewin et al. (1999)	83%	57%
MVA	Holeva et al. (2001)	72%	59%
MVA	O'Donell et al. (2001)	30%	34%
MVA	Schnyder et al. (2001)	34%	10%
Typhoon	Staab et al. (1996)	30%	37%
Cancer	Kangas & Bryant (2002)	53%	61%
MVA	Harvey & Bryant (1999b)	82%	29%
Brain Injury	Harvey & Bryant (2000)	80%	72%

MVA = motor vehicle accident

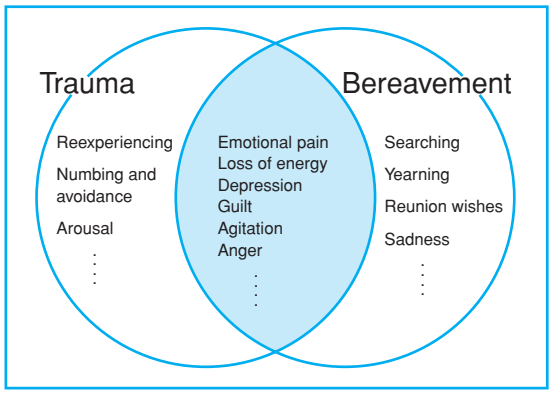
(Excerpted from Bryant RA. Early Intervention for Trauma and Traumatic Loss. 2004.[20])

of the Hanshin-Awaji earthquake who visited mental care centers for counseling, and stated that focusing attention only on PTSD is impractical. Kadokura[30] stated that not only PTSD but all mental disorders should be taken into consideration when determining the mental influences of disaster, on the basis of the finding that victims poisoned in the sarin gas attack on the Tokyo subway system who satisfied the diagnostic criteria for PTSD showed significantly higher scores on both the GHQ (General Health Questionnaire) and SDS (Self-rating Depression Scale) and had more physical symptoms in response to the disaster. The long-term effect of PTSD on concomitant disorders and inhibition of the recovery process from PTSD by concomitant disorders remain unclear. As time passes after a disaster, the mental health status of victims becomes individualized, and generalizations are difficult. The issue of diagnosing comorbidities will be the subject of future studies of trauma in view of long-term measures against PTSD.

c. Traumatic bereavement

The main focus of discussion in the psychiatry of disaster victims has so far been the issue of PTSD. However, many people experience not only danger or threat to their lives but also the death of someone close at the time of the disaster. At the time of the September 11, 2001, terrorist attack in the US, a rough estimate of 6 million people experienced the death of someone close because of the event.[31] Losing someone close sometimes causes greater distress than a personal trauma.

Grief over a loss, such as bereavement, may develop into pathologic or complicated grief, not necessarily after traumatic bereavement. It may also happen in cases of death from a natural cause.[32] Grief over a loss is essentially a "normal reaction." Many individuals recover spontaneously from such grief, without the help of specialists.



(Nader KO. Death and Trauma. 1997[36] modified.)

Fig. 1 Interaction of grief and trauma

Table 4 Phenomena of bereavement and posttraumatic reactions

Posttraumatic phenomena	Bereavement phenomena
Cognitive phenomena	
• Intrusion of scene of trauma • Preoccupation with the traumatic event • Reexperiencing of threatening aspects of the event	• Image of lost person constantly comes to mind Associated with yearning or longing • Preoccupations with the lost person and loved images of him or her
Affective phenomena	
Anxiety • Anxiety as the principal affect General and generated by threat Fearful and threat/danger	• Anxiety, when present, is separation anxiety Generated by imagined futures without lost person
Sadness • Sadness not commonly described	• Sadness frequent and profound
Avoidance phenomena	
• Avoids reminders of event, including places	• May try to avoid remainders of the absence of the lost person
• May have great difficulty talking of event	• May be very driven to talk of lost person
• Oriented to threat and danger	• Oriented to lost person

(Partly excerpted from Raphael B et al. Assessing Psychological Trauma and PTSD. 2004.[34])

However, recent attention has been paid to the finding that people who have lost someone close to them exhibit complicated symptoms that show a mixture of psychic trauma and grief.[33,34] Raphael[34] called an event consisting of both bereavement and trauma "traumatic bereavement."

Psychic trauma and grief show overlapping symptoms, such as pain, loss of energy, depression, and feelings of guilt[35] (Fig. 1[36]). These conditions have a common feature in that interpersonal relationships and feelings of security are threatened. Horowitz,[37] who attached particular importance to this common feature, regarded the cognitive and affective phenomena occurring after psychic trauma or loss as one concept, "stress response syndrome." As he noted, there are very similar aspects in dealing with psychic trauma and grief.

However, when providing clinical intervention to individuals suffering from traumatic bereavement, it is important to evaluate symptoms of grief and psychic trauma separately, while bearing in mind the differences between the two. The most important difference between psychic trauma and grief is the fact that trauma is based on fear for or threat from the "scene" of the traumatic experience, whereas grief is grounded on the emotion of longing and desire for the "lost person." Differences in bereavement and posttraumatic phenomena have been outlined by Raphael et al.[13,34] (Table 4), and have previously been introduced to Japan by Kin[38] and Shirai.[39] Although the stressor of bereavement obviously increases the degree of PTSD reactions and symptoms, and causes them to persist,[40] Gray warned that making a rash diagnosis of PTSD without considering the pathologic features of complicated grief may interfere with the course of treatment.[41]

With regard to how traumatic bereavement is related to subsequent mental health, Green[42] cited bereavement, physical injury, and life threat as factors affecting the degree of risk of trauma. Hull et al.[43] also found that injury and survivor guilt were closely related to prolonged symptoms of psychic trauma in a long-term follow-up study of survivors of a disaster.

In Japan, Nakane et al.[44] reported that, in a survey of survivors of the atomic bombing conducted 22 years after the event, those who had lost relatives and friends, those who had been exposed within 2 km of the explosion, and those who had developed acute physical symptoms immediately after exposure were found to have high GHQ scores. In addition, in a follow-up survey of victims of the Garuda air crash in 1996,[45] three types of victims, i.e., those who had lost friends and acquaintances, those who were 65 years old or older, and those who had been admitted to hospital because of physical injury were also found to have high GHQ scores and thus to be at high risk.

In research on trauma, the results may vary according to the time and method of evaluation of symptoms, and therefore further accumulation of data on risk factors and predictors associated with influences on posttraumatic mental health is necessary. In the screening of victims following a disaster, careful attention should be paid particularly to individuals who have experienced traumatic bereavement or injury.

Early Support after Psychic Trauma

For evident PTSD symptoms after trauma, various treatments including drug therapy, EMDR (eye movement desensitization and reprocessing), and cognitive behavior therapy are available,[46] and practice and research are also progressing in Japan. However, as mentioned previously, many psychological reactions after traumatic experience are normal, and the screening of high-risk individuals rather than therapeutic intervention is suitable in dealing with marked symptoms in the acute phase.

It may be common to think that psychological intervention should be given to victims as soon after a disaster as possible, but more than a few researchers in Europe and North America take the position not to interfere with spontaneous recovery.[46] For example, Litz recommends that, with the current lack of evidence for screening and intervention, victims should be supported by a method that respects their own resources while avoiding invasive intervention if possible.[14] Although it is important to accept and support victims, so-called psychological debriefing, by which a group of victims talk about their thoughts and feelings in the early phase after a disaster, has not proved effective. It is said that superficial intervention and compulsory debriefing are meaningless or even harmful.[47]

The fact that many victims request social and economic support and primary medical care is an

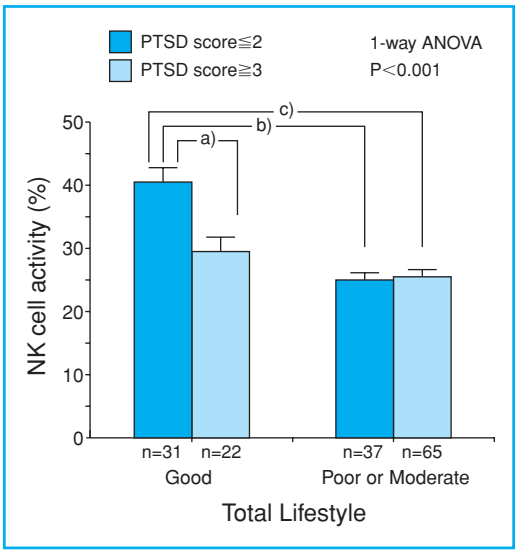
important aspect that should be borne in mind when considering early support after a disaster. Hobfoll[48] considers that many of the stresses following a disaster result from the loss of resources, and he advocates conservation of resources (COR) theory in which importance is attached to conserving and securing resources. Resources include object resources such as housing and household goods, condition resources such as work and family relations, personal resources such as self-esteem and feelings of security and peace of mind, and energy resources such as economic sufficiency. Hobfoll regarded recovery of these resources before dealing with psychological problems as important. A survey of earthquake victims performed by Kwon et al.[49] provided the finding that individuals with severe PTSD symptoms had experienced significantly more earthquake-related changes in life events such as a disrupted lifeline, damaged home, decreased income, and altered interpersonal relationships in the neighborhood.

Initial activities such as those carried out by people who contact victims soon after a disaster, e.g., fire fighters, police officers, rescue personnel, emergency medical personnel, and primary care physicians, can provide psychological support for victims.[50] It has been shown that Japanese victims tend to consult their family doctors, unwilling to see mental health specialists even if they have evident symptoms.[51] Therefore, it is extremely important that personnel involved in initial activities have some knowledge of disaster psychiatry, including PTSD, and are ready to provide appropriate management in close cooperation with mental health experts.

In cases of large-scale disasters such as earthquakes, it is important to set up an inquiry counter that is easily accessible by individuals who have difficulties and to provide outreach to high-risk individuals.[1] On such occasions, it is also important to identify the areas where intensive assistance is necessary. Maruyama et al.[52,53] found that, in the Hanshin-Awaji earthquake, victims in places of higher earthquake intensity showed higher GHQ and SDS scores, as well as higher PTSD scores, after the disaster. Based on this finding, they reported the possibility that an objective index such as earthquake intensity may be of use to evaluate the intensity of the event, which then could be used effectively for support activities.

Behavioral Characteristics of Japanese People During and After a Disaster

Goto[54] has reported interesting behavioral characteristics of Japanese people during and after a disaster. She stated that the Japanese tend not to ask for help for their own mental distress, valuing patience as a virtue, and tend not to disclose painful feelings although they encourage mutuality. Further, they often appear to maintain calm in crisis situations. Goto explained the background as follows: there is a strong stigma to having a mental disease, which is considered equivalent to being a weak person, and there is a cultural norm that precludes sharing feelings with others or receiving assistance for mental health problems. A number of researchers have previously suspected that psychosomatic symptoms are likely



(Excerpted from Inoue-Sakurai C et al. Prev Med. 1999.[58])

Fig. 2 NK cell activity in the groups divided by lifestyles and PTSD score

Data are presented as means and standard errors.

a) Subjects with good lifestyle and few or no PTSD symptoms vs subjects with good lifestyle and many PTSD symptoms ($P<0.05$ by Bonferroni's multiple comparison test).

b) Subjects with good lifestyle and few or no PTSD symptoms vs subjects with poor or moderate lifestyle and few or no PTSD symptoms ($P<0.05$ by Bonferroni's multiple comparison test).

c) Subjects with good lifestyle and few or no PTSD symptoms vs subjects with poor or moderate lifestyle and many PTSD symptoms ($P<0.05$ by Bonferroni's multiple comparison test).

to occur as the final pathway among Japanese people.[55,56]

Recent years have seen several reports documenting a psychosomatic correlation after disaster from the standpoint of neuropsychoimmunology. Among survivors of the Hanshin-Awaji earthquake, Morimoto et al.[57] found that residents of greatly damaged areas, particularly those who were mentally unstable, had decreased natural killer (NK) cell activity, and Inoue-Sakurai et al.[58] observed that individuals who had a poor lifestyle and strong PTSD tendency had significantly decreased NK cell activity (Fig. 2). In addition, Fukuda et al.[59] reported that, among survivors in areas where more than half of all houses were completely or partially destroyed, those with a strong PTSD tendency had higher salivary cortisol levels. Stress from disaster affects the nervous and endocrine systems, and consequently decreases immune strength. In addition, Japanese people are particularly likely to somatize the symptoms of stress. In this regard, it is necessary to provide help for PTSD, considering the wide view of psychosomatic medicine and not limiting oneself to simple "psychological care."

Conclusion

This paper has discussed issues of screening in the early postdisaster phase to prevent chronic PTSD among survivors and the desirability of early support. When dealing with issues related to psychic trauma, a long-term perspective is necessary when providing early support. At present, even in Western countries, attention is paid to ASD and PTSD symptoms in the early postdisaster phase, but secondary prevention in individuals who show the severe, chronic changes of such symptoms is attended to less seriously, and support tends to taper off.[14] This trend is more prominent in Japan, which lags behind in disaster psychiatry as a whole.

There is no markedly effective treatment for traumatic reactions such as PTSD if they become chronic. The best form of support for individuals with such reactions during the course from immediately after disaster to recovery and adaptation remains a matter of discussion. In addition, secondary traumatic stress in disaster rescuers, although not addressed in this paper, is another problem inseparable from PTSD. Many issues remain unsolved, including accumulation of evidence about the pathologic condition, diagnosis, prevention, intervention, and treatment of PTSD and the need for a system of disaster mental health services to provide long-term follow-up of disaster victims.[60]

Looking back over the 10 years since the Great Hanshin-Awaji Earthquake, the authors feel that the survivors' mission of renewal, which grew out of their extreme trauma and loss, has facilitated the restoration of Kobe, the city affected by this major disaster. Although the price in lives that could not be saved was high, the mission entrusted to us may be to ensure that every medical care professional has some interest in disaster psychiatry and supports or participates in disaster medical care in some way.

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Review Article

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

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Kazuomi Kario*1, Kazuyuki Shimada*1, Fumimaro Takaku*1

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

*1 COE (Center of Excellence) Program, Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical School, Tochigi
Correspondence to: Kazuomi Kario MD, FACC, FACP, FAHA, COE (Center of Excellence) Program, Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical School, 3311-1, Yakushiji, Minamikawachi, Kawachi, Tochigi 329-0498, Japan.
Tel: 81-285-58-7344, Fax: 81-285-44-2132, E-mail: kkario@jichi.ac.jp

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 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).

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.

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-

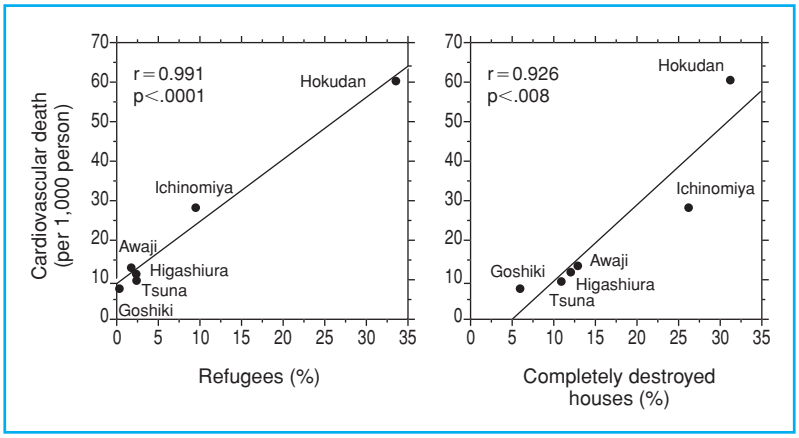


Fig. 1 Correlation between damage of the Hanshin-Awaji earthquake and cardiovascular death around the epicenter

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.

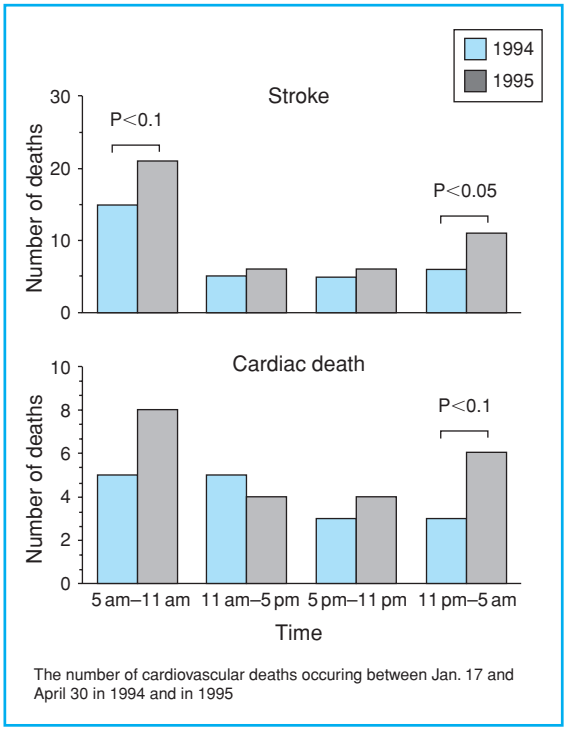
These results indicate the impact of chronic stress by environmental change on CVD and the importance of the immediate reduction of chronic stress to achieve effective prevention of disaster-induced CVD.

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).[1–8]

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



The number of cardiovascular deaths occuring between Jan. 17 and April 30 in 1994 and in 1995

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	• 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	• hypercoagulable and hyperfibrinolytic state • increased blood viscosity (hematocrit and fibrinogen) • endothelial cell dysfunction • poorly controlled in those treated with anticoagulation (warfarin)
Inflammation	• increased inflammatory reaction
Lipid profile	• unchanged in relatively shorter term (may worse in longer term)
Blood sugar	• increase in plasma glucose and new-onset diabetes • poorly controlled in treated diabetics

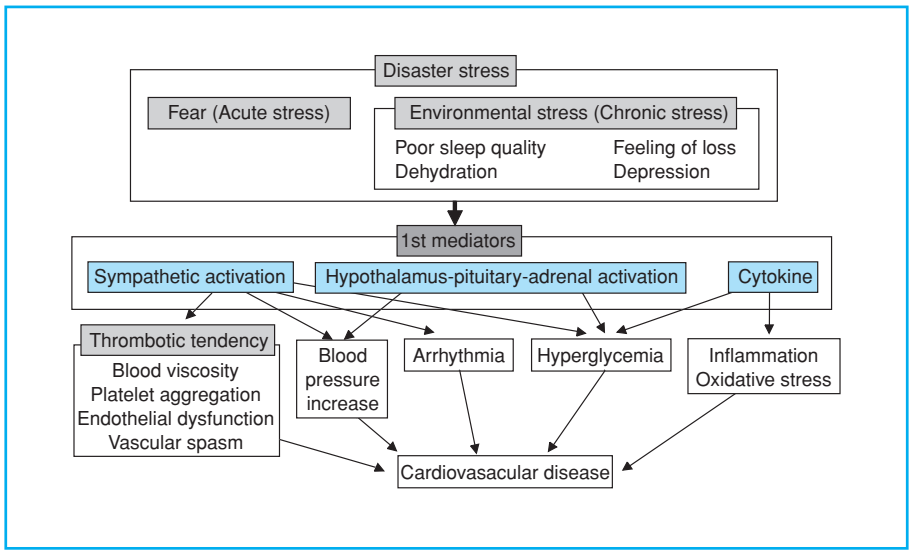


Fig. 3 Mechanism of disaster-induced cardiovascular disease

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 earthquake[24] (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.)

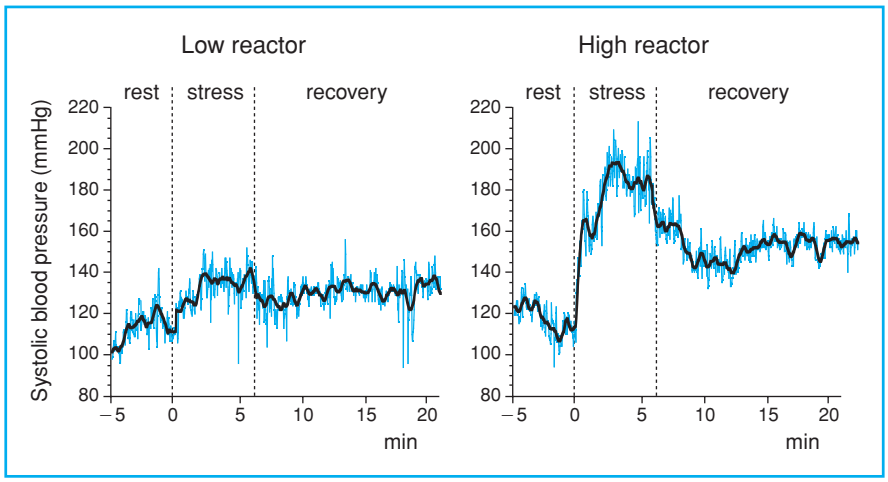


Fig. 4 Blood pressure change during acute mental stress

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 persisted 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

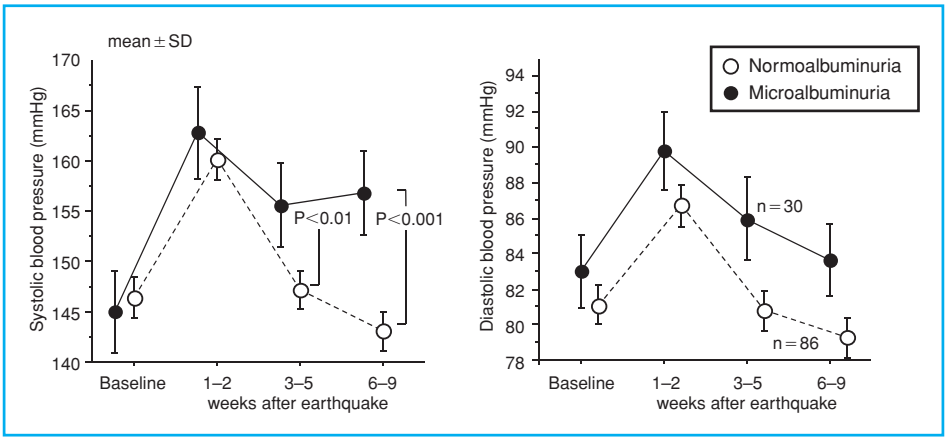


Fig. 5 Earthquake-induced blood pressure change in hypertensives with and without microalbuminuria

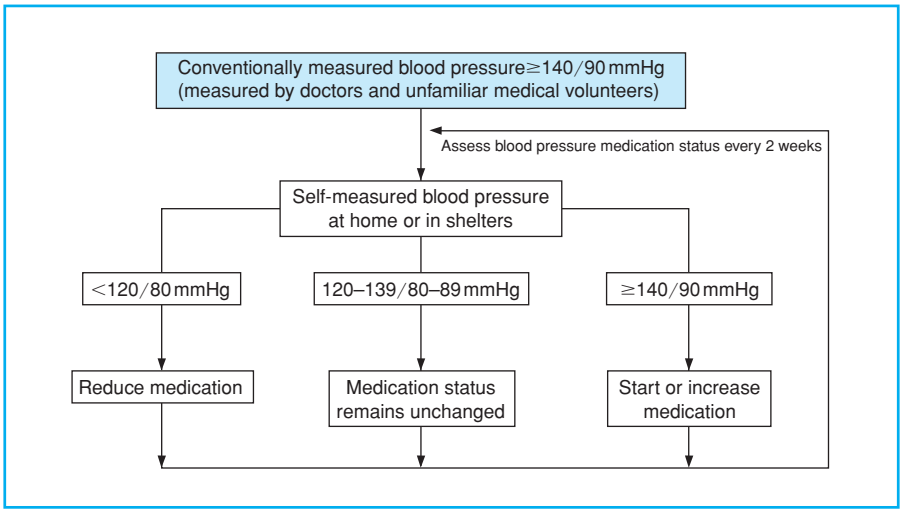


Fig. 6 Management of blood pressure in disaster

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

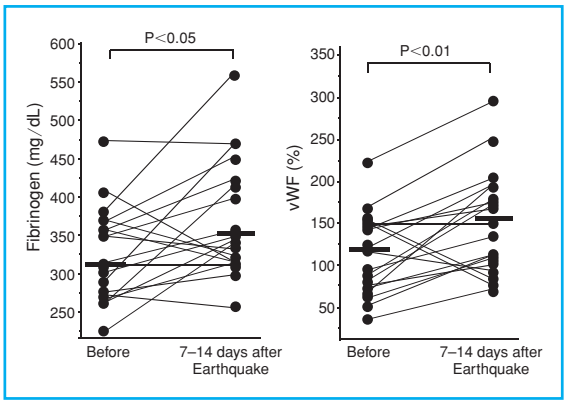


Fig. 7 Earthquake-induced change in fibrinogen and von Willebrand factor in high-stress hypertensives*

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

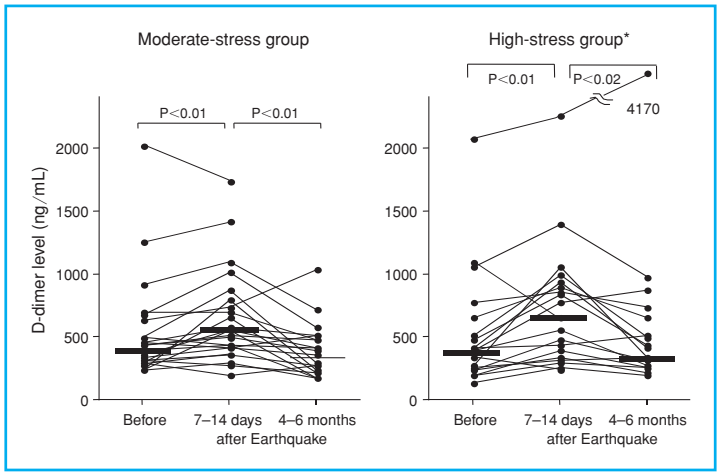


Fig. 8 Earthquake-induced change in D-dimer in high-stress hypertensives*

* 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 anticoagulant 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

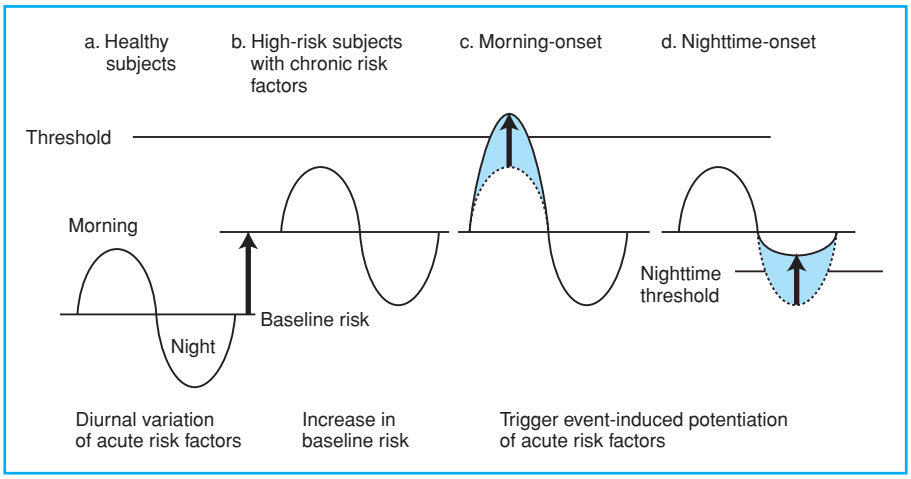


Fig. 9 Diurnal variation of disaster-induced risk factor and onset of cardiovascular disease

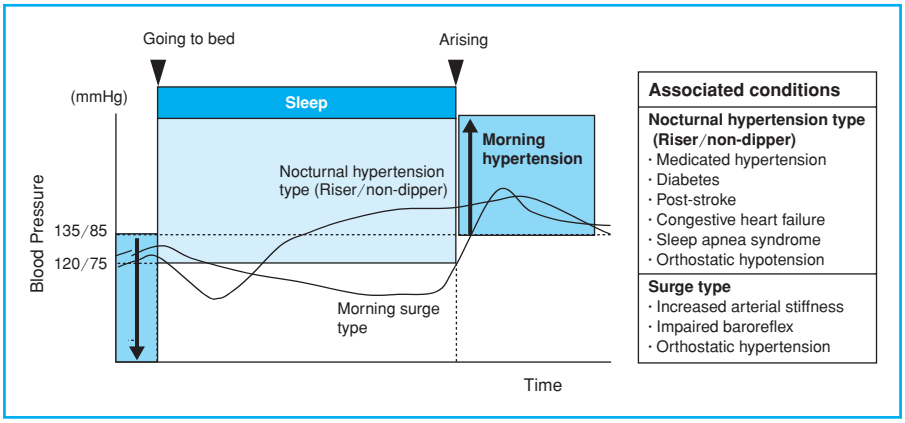


Fig. 10 Morning hypertension and diurnal blood pressure variation

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-alpha$_2$/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 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	• Turn off lights in shelters at night • Ensure privacy in shelters
Blood pressure	• 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	• 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	• distribute gauze masks • ensure a hygienic environment
Lipid profile	• reduce lipid-rich diet
Blood sugar	• 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 night[94] (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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Review Article

Public Health Impact of Disaster on Children

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Yasuhide Nakamura*1

Abstract

Disasters have the greatest affect on the most vulnerable groups—especially children. Children are particular at risk because they are developing in both physical and psychological aspects. Most young children depend on routine and consistency in the environment, home life and in relationships with their family. Disasters have affects on public health such as infectious diseases including diarrhea, acute respiratory infection, measles and malaria. Psychosocial support to children and their family is essential from the viewpoint of mental health. In refugee camps, playgrounds for children are set up even in the emergency phase.

Japan has developed good practice from its lessons learned in disaster medicine, and should contribute to international cooperation for children in disasters.

Key words Public health, Disaster, Children, Refugee, Mental health, Psychosocial support

Introduction

It is a myth that disasters are random killers: "Disasters strike hardest at the most vulnerable groups—the poor, and especially women, children and the elderly."[1]

Because children are in the process of physical and mental development, they are at particularly high risk of harm from a disaster. As compared with adults, children depend more strongly on stability in their daily lives and environment, and they are more vulnerable to the direct consequences of a disaster, including shortage of food and potable water, coldness due to poor sheltering, and a loss of protection in the family.

Natural disasters, as well as political turmoil and conflicts, affect people all over the world every year, resulting in the generation of international refugees and internally displaced persons. The situations requiring disaster health care can be divided into natural disasters (storm, flood, earthquake and tsunami, volcanic eruption, etc.), man-made disasters (fire, chemical explosion, train accident, etc.), and the presence of refugees. Different types of disasters pose different needs in the provision of health care support for children with different degrees of urgency. The common fact is that disasters affect the lives and physical, mental, and psychological health of numerous children every year worldwide.

This article analyzes the issues of children's health following disasters, both in Japan and overseas, chiefly from the standpoint of public health. The discussion is based on the lessons learned from my experience in the Hanshin Awaji Earthquake, as well as the experience in humanitarian assistance provided in overseas refugee camps and disaster relief activities. In concluding, I make recommendations concerning disaster health care for children.

General Characteristics and Public Health Effects of Disaster on Children

A major disaster hits the entire group of victims at the same time. For example, all people in the affected area experience the earthquake simulta-

*1 Graduate School of Human Sciences, Osaka University, Osaka
Correspondence to: Yasuhide Nakamura MD, PhD, Department of International Collaboration, Research Center for Civil Society, Graduate School of Human Sciences, Osaka University, 1-2, Yamadaoka, Suita City, Osaka 565-0871, Japan.
Tel: 81-6-6879-4033, Fax: 81-6-6879-8064, E-mail: Yastisch@aol.com

neously and become victims. This equality is a characteristic of large earthquakes. However, there are differences reflecting socioeconomic strata, such as that a reinforced concrete condominium may remain intact after a large earthquake next to wooden flat that collapses. In addition, infants and children suffer qualitatively different effects as compared with adults.

In the case of the Hanshin Awaji Earthquake in January 1995, victims had to look after themselves immediately after the disaster. Some people who lost their houses left temporary refuges opting to receive support from their relatives, companies, and friends. My colleagues and I examined the health of children living in refuges 2 weeks after the earthquake.[2] On this occasion, many severely affected children had evacuated using personal connections, while many of those staying in refuges (elementary school buildings) had problems in finding temporary shelters. The presence or absence of a private support network directly affected their standard of living following the earthquake.

This section outlines the characteristics of health care for children following disasters.[3] The issues regarding mental care and psychosocial support are detailed in the next section.

1. Death of children

The causes of death among children vary greatly depending on the type of disaster, such as earthquake, flood, etc. Following the Hanshin Awaji Earthquake, an overwhelming majority of the children under the age of 15 who died lost their lives due to suffocation (55.7%), crushing (12.3%), and contusion (15.3%). Nearly all of these cases were reported to have been almost instantaneous death.[4]

2. Health care for children following disaster

A major disaster also causes serious damage to medical institutions. Physical damage including collapse and destruction of hospitals and clinics, as well as death and injury of personnel, may be considerable. Disruption of transportation systems may prevent physicians and nurses in the disaster area from going to work. Damage to the operational aspects may also be serious, such as the breakdown of information systems interrupting communication needed for the transportation of patients. Following the Hanshin Awaji Earthquake, we encountered a number of problems with emergency facilities depending on the supply of water and electricity, such as that the emergency power source did not operate because it was water-cooled, the pumps sending water to rooftop cisterns did not work, etc.

According to the experience in the Hanshin Awaji Earthquake, the medical services provided during the 2 days following the disaster mainly consisted of emergency surgical care. Because many hospitals were keeping a lean inventory, there was a significant shortage of supplies for surgical treatment, such as bandages, gauze, and injection fluids. In the later periods, the focus moved to the treatment of infections (acute respiratory infection, diarrhea, etc.) and acute post-disaster stress arising from lack of sleep and abnormal excitation. At least in the case of disasters in Japan, many of the families with infants and children evacuate relatively early after a disaster occurs, and the number of patients visiting pediatric outpatient departments tends to decrease over time.[4]

3. System of health care for children in refuges

In preparing for disasters, it is important to establish a system of health care for children in refuges. The systems for health care in refuges are operated mainly by administrative bodies and local hospitals. In many cases of recent disasters in Japan, medical care in refuges is often provided by medical volunteer groups, including Japanese Red Cross Society, volunteer organizations, and local administrative and hospitals in other areas. However, a lack of good coordination between refuge health care systems and volunteer activities has sometimes caused problems. In the Niigata Chuetsu Earthquake in 2004, volunteer physicians administered influenza vaccination to children in refuges, but this caused a problem in identifying who received a vaccination and who did not, because the volunteer physicians failed to keep vaccination records.

Medical activities in refugee camps in developing countries are conducted by clinics without the facilities for hospitalized care and the hospitals in the camps. Pediatric patients requiring major surgery or special treatment in ophthalmology, ENT, etc. need to be transported to regional hospitals outside the camps. Because some people are not in the habit of visiting hos-

pitals or clinics in illness, pediatric patients may be cared for at home until the condition becomes severe. This underscores the importance of outreach activities. While some NGOs are staffed with many physicians and nurses and are equipped with simple operating rooms, others are small groups of several persons carrying limited medical instruments and drugs. In a refugee camp, medical NGOs of various sizes from various countries coexist and cooperate. Usually, regular meetings of health professionals are held in each camp to facilitate information exchange and work sharing among NGOs and international organizations.

4. Infections

The risk for the incidence of infectious diseases increases due to deteriorated hygiene and overcrowding within several days after a disaster. Thereafter, depending on the level of hygiene at the refugee camp, the risk for various infections has been shown to increase (Table 1). The most common types of infections observed in refugee camps include diarrhea, acute respiratory infection (ARI), measles, and malaria, which are called the 4 major killers.[5,6] In addition, increases have been reported in various infections, such as the epidemic of tuberculosis due to crowded living conditions, tetanus arising from unsanitary treatment of injury and childbirth, various parasitic infections,[7] and scabies due to the shortage of water.

(1) Diarrhea

The key to prevention is the supply of safe drinking water and food and the improvement of environmental hygiene. While the treatment of human wastes and garbage is important, attention should also be paid to the drainage facilities for gray water used in cooking and laundry. The causes of diarrhea include bacteria (pathogenic Escherichia coli, Campylobacter, Vibrio parahaemolyticus, Salmonella, dysentery, and cholera), viruses (Rotavirus), and parasites (dysentery ameba and Giardia). In principle, diarrhea is treated with oral rehydration salts (ORS). Cases with severe dehydration require intravenous fluid therapy.

The most important prevention measures are the provision of a safe water supply and sanitary toilets. The need for these measures is common to disasters in both Japan and refugee camps in developing countries.

(2) Acute respiratory infection (ARI)

The chance of contracting respiratory infections increases as a result of the high population density in refugee camps, poor ventilation in tents, and shortage of blankets and clothes. Persons in a state of malnutrition easily develop from simple cold and upper respiratory infections to lower respiratory infections, such as pneumonia and bronchitis. Effective treatment strategies are early detection and the use of antibiotics. The selection of antibiotics should comply with the first-choice agent specified in each country (co-trimoxazole is the first-choice antibiotic in many countries).

Prevention is achieved by the distribution of blankets and clothes and the provision of the minimal required space for living. According to UNHCR, each person requires the living space of 3.5 m^2 (in tropical areas) or 4.5–5.5 m^2 (in temperate and cold areas).[5] However, provision of sufficient living space is difficult in practice. Very few of the refuge shelters following the Hanshin Awaji Earthquake satisfied this criterion.

Table 1 Common infectious diseases in camps

DISEASES	FACTORS	PREVENTION
Diarrhea	Contamination of food/water	Safe water supply
ARI	Lack of shelter and blanket	Shelter and clothes
Malaria	New environment	Mosquito net
Measles	Over-crowding	Immunization
Tuberculosis	Over-crowding	Early detection/treatment
Tetanus	Injury or delivery	Clean delivery/treatment
Parasite diseases	Contamination of water	Safe water supply
Scab	Lack of water	Water supply and soap

(3) Measles
This disease is likely to occur in large-scale epidemics in group living conditions, and once an outbreak takes place, it threatens the lives of many infants and toddlers. The setting for an epidemic is created when people who had been living separately start to live in a group. In an immunization program for children, the highest priority should be placed on measles. Immunization against measles should be started as early as possible after the setting up of a camp.
(4) Malaria
This disease, transmitted by Anopheles mosquitos (Anopheles), is a representative tropical infection distributed widely in tropical and subtropical regions. There are 4 types of malarial parasites affecting human beings: tropical malaria (Plasmodium falciparum), tertian malaria (P. vivax), ovale malaria (P. ovale), and quartan malaria (P. malariae). Symptoms vary considerably depending on the history (primary infection or reinfection), the age of patient, and the type of pathogen. In particular, children often do not show typical fever patterns. A massive epidemic can occur when a large number of inhabitants have moved into a malaria endemic area. In the case of the Rwanda refugees, 1 or 2 in 10 persons contracted malaria, and the mortality rate was reported to be as high as from 10 to 30%.[8]

The recommended prevention measures are elimination of water pools producing mosquitos and the distribution of mosquito nets. Treatment is the administration of antimalarial agents, which should be selected considering the possibility of drug resistance.

Psychosocial Support to Children

Recently, the importance of psychosocial care for persons affected by disasters has been highlighted. Victims of a conflict do not easily recover from the sad experience of losing their close relatives, witnessing the killing of familiar persons, and being raped or nearly killed. The sense of material and mental loss may also be tremendous, when people have lost property, houses, and jobs, and have been forced to leave their beloved home behind. After experiencing life-threatening situations, some survivors and refugees develop the symptoms of post-traumatic stress disorder (PTSD).

Children are no exception. Pynoos described that a strong positive correlation was found between the proximity to the epicenter and the overall severity of post-traumatic stress reaction and indicated that after a catastrophic natural disaster post-traumatic reactions in children may reach epidemic proportions, remain high for a prolonged period, and jeopardize the well-being of the child population over a large region.[9]

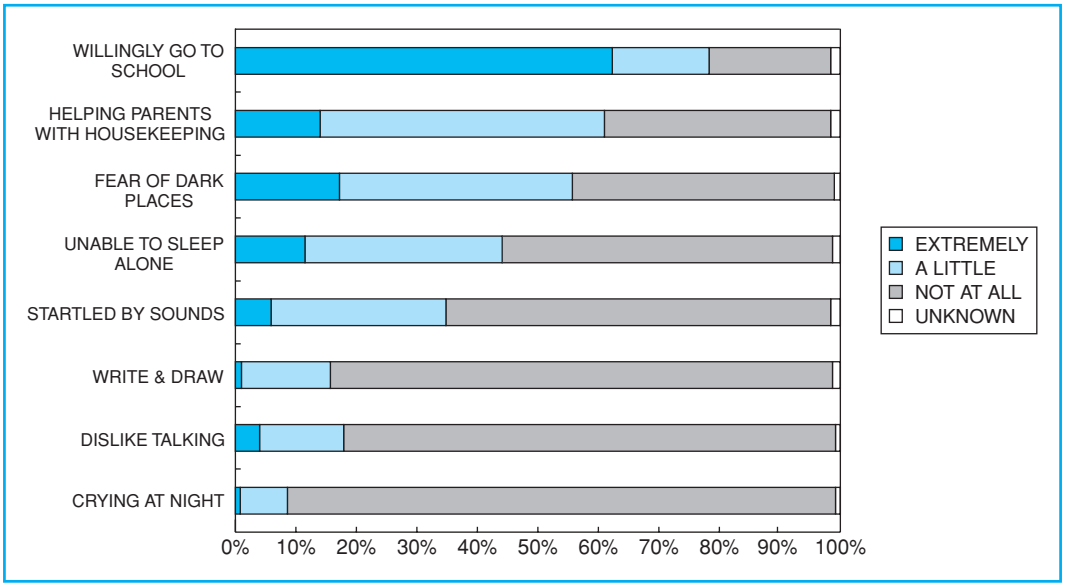

Fig. 1 Behavioral changes of children in Kobe

Following the Hanshin Awaji Earthquake, many of the children who lost their parents were reported to have psychological problems for a prolonged period.[10] On the other hand, better tendencies toward psychological recovery have been reported among the children who experienced the disaster but did not suffer psychological trauma such as the death of family members. Our questionnaire survey targeted mothers of preschoolers conducted 6 months after the Hanshin Awaji Earthquake produced the following results.[11] Many children in Kobe city demonstrated regressive behavior, separation anxiety, the re-experience and the elevation of arousal level, which are categorized as the symptoms of PTSD. However, 61.0% of them helped parents with housekeeping and 78.3% willingly went to school or kindergarten (Fig. 1).

Recently, humanitarian relief organizations in Europe and the U.S. have begun improving the provision of mental care through various measures such as the inclusion of psychological counselors in emergency support teams. The importance of psychosocial support in disaster response is also recognized in Japan. However, it is in fact difficult to find appropriate workers such as psychological counselors for children. It is most important to provide the children with the normal environment of everyday living, as opposed to just having children draw pictures. To this end,

Table 2 Phases related disasters

General Care	Human Care	Family	Community	Local Government	Child Health Care
Preparedness Phase					
Disaster plan	Disaster drills	Family stockpiling	Disaster & fire organizations	Local disaster plan	Disaster plan
Preparation of manuals	Disaster education	Everyday preparedness		Safe city planning	Disaster drills
Emergency Phase (about 72 hours after a disaster)					
Information collection	Life saving	Confirmation of survival status	Life saving	Establishment of disaster response headquarters	Damage survey
Traffic control	Emphasis on basic human needs	Mutual support in family	Fire fighting	Quick damage survey	Care for hospitalized patients
Fire fighting	(Securing of water, food, & shelter)		Search and rescue of vulnerable persons	Request for support from other local governments	Emergency care for children
	Emergency care services			Confirmation of the survival status of personnel	Home medical care
	Securing of safety			Beginning of reception of volunteers	
Relief Phase (about 1–2 weeks after a disaster)					
Establishment of lifelines	Provision of health care services	Return to work	Autonomy in refuge sites	Construction of local network	Vaccination & health examination
Information collection and supply	Measures for vulnerable persons	School reopening	Safety patrol in community	Cooperation with other local governments	Care for chronic patients
Prevention of secondary disasters	Improvement of refuge sites	Mutual support in family	Support to vulnerable persons	Establishment of a volunteer system	Psychosocial support
Beginning of restoration work	Care for evacuated persons			Establishment of information network	Care for disabled children
Damage survey					School reopening
Reconstruction Phase					
Full-scale restoration work	Environmental improvement of refuge sites	Return to normal living	Community meeting	Active use of volunteers	Enrichment of psychological counseling
Support to local organizations	Psychosocial support			Cooperation among local governments	Cooperation with schools
Construction of temporary housing	Enrichment of health care services				Psychosocial support
Development Phase					
Building a city to withstand disasters	Psychosocial support		Participation in restoration plan	Restoration plan involving inhabitants	Psychosocial support

education and training concerning the importance of psychosocial support should be given to specialists maintaining regular contact with children, including school teachers, nursery teachers, and school nurses. It is also important to facilitate the alleviation of psychological stress of children by providing playgrounds and securing places for group play in schools and nursery schools.

In April 1999, the refugee camp for Kosovo people in Macedonia already had playgrounds constructed for children when minimal living conditions including the supply of food and drinking water had just been ensured. In a corner of the refuge camp, there were swings and a jungle gym made of whatever timber was available. Children laughed as they played, and adults around them were smiling brightly. I was very impressed by the fact that providing children with an environment similar to their everyday lives could make them look so cheerful. Understanding the importance of providing warm food and a restful environment is the starting point of psychosocial support.[12]

Recommendation for Child Health System of Disaster

The most urgent tasks in disaster response are life saving and searching for survivors. For surviving children, it is most important that basic human needs (BHN) are satisfied, including drinking water, food, and shelter. There are various needs within the care for children. Groups of children who are vulnerable to the impact of disaster, such as infants, disabled children, and children of different nationalities, need special responses. Psychosocial support following disaster is also necessary. The health care system for children in a disaster should be developed as part of the overall framework for disaster response and preparedness. In addition to general care and human care, the roles of families, communities, and administrative bodies are particularly important in the care for children. These roles are summarized in Table 2 according to the stage of disaster.

In the preparedness phase, disaster prevention/response plans and disaster drills targeted at children are important. Signs of evacuation routes should be illustrated so that children may clearly understand where to go. During the emergency phase, pediatric emergency care and hospitalized care are important. Care for children receiving artificial respiration and oxygen therapy at home is also important. During the relief phase, vaccination against measles, health examination of infants, and individualized care for children with chronic diseases and disability are important too. In addition, it is essential to provide psychosocial support to children, includ-

Table 3 The code of conduct for the International Red Cross and Red Crescent Movement and NGOs in disaster relief

Principle Commitments:
1. The Humanitarian imperative comes first.
2. Aid is given regardless of the race, creed or nationality of the recipients and without adverse distinction of any kind. Aid priorities are calculated on the basis of need alone.
3. Aid will not be used to further a particular political or religious standpoint.
4. We shall endeavour not to act as instruments of government foreign policy.
5. We shall respect culture and custom.
6. We shall attempt to build disaster response on local capacities.
7. Ways shall be found to involve programme beneficiaries in the management of relief aid.
8. Relief aid must strive to reduce future vulnerabilities to disaster as well as meeting basic needs.
9. We hold ourselves accountable to both those we seek to assist and those from whom we accept resources.
10. In our information, publicity and advertising activities, we shall recognise disaster victims as dignified human beings, not hopeless objects.

Source: International Federation of Red Cross and Red Crescent Societies: Code of conduct for the International Red Cross and Red Crescent Movement and Non-Governmental Organizations (NGOs) in disaster relief, International Federation of Red Cross and Red Crescent Societies, Geneva, 1994.

ing those staying in refuge shelters. As schools and nursery schools reopen, attention should be paid to the environment of the everyday life of children through the close cooperation of school teachers, nursery teachers, etc. Finally in the reconstruction phase, psychiatric care and psychological counseling should be given to children with severe psychological trauma and those showing the symptoms of PTSD, separately from the psychosocial support to children as general. Continuation of prolonged psychosocial support is needed during the redevelopment after the disaster.

As discussed above, humanitarian assistance following a disaster involves the participation of various organizations including volunteers, and hence many arrangements have been made internationally. These arrangements were made on the assumption that the acts of outsiders performed with good intentions do not necessarily benefit the victims of disaster. In 1994, the Code of Conduct for the International Red Cross and Red Crescent Movement and NGOs in Disaster Relief was promulgated (Table 3).[13] Furthermore, the Sphere Project was launched in 1997 for the purpose of defining the minimal standards concerning the various aspects of humanitarian assistance. A revision was published in 2004, including the minimal standards concerning water supply, hygiene, nutrition, food distribution, health care services, etc.[14] In Japan also, administrative bodies, medical institutions, volunteers, and other relevant parties should share a code of conduct regarding disaster relief. In the present situation where much of the support comes from outside the disaster area, sharing of minimal standards and the code of conduct should be an effective means of protecting the human rights of children.

Summary

Shortly after the Hanshin Awaji Earthquake, children attending an elementary school in Kobe wrote essays.[15]

"When I went out, I saw a collapsed house. A dog and an old man were under the house. The old man was dead, but the dog was alive."

"Looking at my house, the second floor was now the ground floor, and the ground floor was a mess."

Six months later, the same child had grown to write the following letter:

"I'm no longer afraid of aftershocks. I feel safe. I sometimes feel like forgetting the earthquake, but I should not forget, because I was helped by many people.

When we hear the news of the war in Rwanda, our first instinct is to think that it is nothing to do with us, although we too suffered hardship from that big earthquake. All right? No, it's not all right at all."

Six months after the personal experience of the earthquake, the child had sublimated the experience of disaster, and was thinking sympathetically about the refugee camps on a different continent. Many schools and communities in the disaster area had steadily been supporting children who suffered extreme harm. These efforts led to the rebuilding of the minds of children themselves.

Disasters do not strike suddenly. On a national or global scale, disasters are "repeated" regularly. Learning from experience of the many disasters in Japan, we should strive to develop disaster health care that helps children to survive strongly after the tragic experience of disasters. This is the most important contribution that Japan has to offer to the world.

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Position of the Japan Medical Association Concerning Organ Transplants Based on the Judgment of Brain Death

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Hideki Miyazaki*1

Key words Organ transplantation, Brain death, Organ donation, Child abuse

The law concerning organ transplantation (Law on Organ Transplant) was enforced in October 1997. Appendix Article 2 of this Law provided that a revision should be made approximately 3 years after enforcement of the Law. However, no revision has been conducted up to the present. The progress of organ transplantation in Japan has lagged considerably during this period. It is widely recognized that there are defects in the Law and problems in the use of the Law. We demand that this Law, enacted at the insistence of House members, be discussed by the legislative body, reviewed promptly, and amended as soon as possible.

About Organ Transplantation under the Age of 15

(1) Concerning the determination of brain death and organ donation under the age of 15, the intention of the donor should be respected, but as long as no refusal has been expressed, these should be allowed based on the consent of close relatives. The present rules in Japan require that organ donors must indicate their will before their death, and the indication of will to donate organs is effective only if it is made at the age of 15 or over, as this age defines the ability to leave a will. As a result, persons under the age of 15 are not permitted to donate organs. This means that we must depend on overseas resources for organ transplantation in children, and this situation must be amended.

(2) Infants within 12 weeks after birth should be excluded from the determination of brain death, according to the criteria for the determination of brain death reported by the Study Group of the Ministry of Health, Labor and Welfare (MHLW). The 1999 report of the Study Group on the Criteria for the Determination of Brain Death in Children, MHLW, lists the exclusion of infants within 12 weeks of birth as an example of exclusion based on age. The Japan Medical Association has adopted the same treatment as this report.

(3) The involvement of child abuse must be addressed carefully. In Japan, a significant number of cases of child abuse are committed by persons in parental authority, and the child's right to self-determination would be infringed if organ donation is made based on the proxy consent of the person in parental authority who caused the death of the child. To prevent this situation, adequate caution must be paid to the treatment of possibly abused children.

*1 Japan Medical Association, Tokyo
Correspondence to: Hideki Miyazaki MD, PhD, Japan Medical Association, 2-28-16, Honkomagome, Bunkyo-ku, Tokyo 113-8621, Japan.
Tel: 81-3-3946-2121, Fax: 81-3-3946-6295, E-mail: jmaintl@po.med.or.jp

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For the reader's reference, please see the article entitled "Problems of the Law on Organ Transplant and the Situation of Organ Transplantation in Japan" (JMAJ 48(6): 318–323)) written by the same author.

About Organ Donation Facilities

While there were 323 potential donors who had donor cards indicating their willingness to donate organs and became brain dead by 2002, more than a half of these cases (162 cases) were excluded because brain death occurred in institutions other than designated organ donation facilities. The standards for designating organ donation facilities should be loosened so that the current 4 categories of these facilities may be expanded to include other similar facilities. It is a problem to be unable to respect the will of donors.

(1) While organ donation facilities are limited to 4 categories (306 facilities) at present, requirements concerning the designation of such facilities should be eased to allow the addition of more facilities.

(2) The determination of brain death conducted at any of the newly added medical institutions, other than those falling under the current 4 categories, must be conducted by more than one physician according to the same criteria as those used at present.

In the field of education and promotion activities concerning organ transplantation, donor cards should be made available at any time at the counters of medical institutions, and a rule should be made to allow an indication of whether or not each person has expressed the will to donate organs on insurance cards, driver's licenses, etc. In addition, the central and local governments should try to enhance citizens' understanding of and interest in transplant medicine at every opportunity.