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

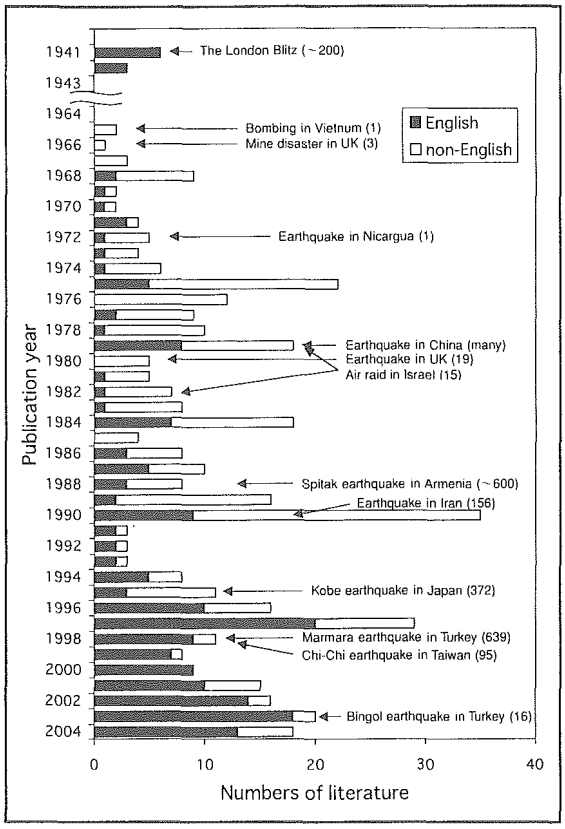

Fig. 1 Annual publication in the literature on "crush syndrome"

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

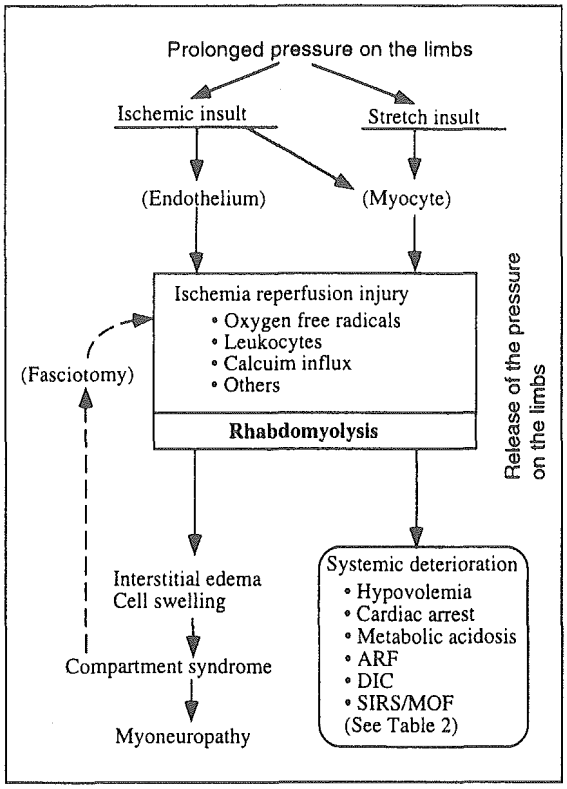

Fig. 2 Pathophysiology of the crush syndrome

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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スポーツドクターに必要な
救急医学の知識

第9回

JATEC (Japan Advanced Trauma Evaluation and Care)™

横田順一朗　市立堺病院

臨床スポーツ医学　第22巻　第12号　別刷

(平成17年12月)

第9回

JATEC(Japan Advanced Trauma Evaluation and Care)^{TM}

横田順一朗　市立堺病院

はじめに

外傷患者の急性期治療についてわが国は必ずしも先進国とはいえない．最近のアンケート調査によれば「防ぎ得た外傷死亡(PTD ; Preventable Trauma death)」が38%にも及ぶといわれている．これは外傷診療の先進国であるアメリカの約30年前の成績である．外傷診療の質を向上させるには，系統的な外傷教育や研修システム，外傷診療の標準，外傷に特化した救急医療体制，外傷専門の診療機関，診療実態や転帰に関するデータベース化，診療の機能評価などの充実が必要である．

わが国ではいずれの点においても整備が遅れていたが，近年，外傷診療の標準化や患者情報の登録について飛躍的な展開をみせている．その代表例の1つに外傷初期診療の標準として開発されたJATEC (Japan Advanced Trauma Evaluation and Care)^{TM}[1]があり，また，その研修システムとしてのJATEC^{TM}コースがある．本章ではJATEC^{TM}の概要とコースの現状を紹介する．

JATEC^{TM}が想定する医療

JATEC^{TM}が想定するのは外傷初期診療，すなわち外傷患者に対する急性期の診察と治療である．とくに，外傷患者を対象として救命救急と見落としのない損傷検索に焦点を絞っている．診療各科が行う専門的な根本治療を指導するものではない．診療科に関係なく，外傷患者を診る機会のある臨床家なら誰もが習得しておくべき知識と技能を教授するのが目的である．

またJATEC^{TM}を通して，病院前，救急外来，院内各科，さらには各医療機関が外傷初期診療に対して共通の認識をもつことを期待している．

JATEC^{TM}が教える外傷診療理論

以下に，JATEC^{TM}が教える外傷診療の理論を概説する．詳細は，「外傷初期診療ガイドライン JATEC^{TM}」[1]を参考にしてほしい．

1. 診療手順の2つのステップ

命を守るには確定診断より生命危機の状態を早く認知する．実践しやすいように診療手順を2つのステップで構成し，それぞれを外傷診療のprimary surveyおよびsecondary surveyと呼ぶ．前者は蘇生の必要性を判断する目的で生理学的な徴候を把握することであり，後者は治療を必要とする損傷を検索することである(**図-1**)．

2. Primary survey : ABCDEアプローチ

Primary surveyを以下に述べる英語の頭文字を組み合わせABCDEアプローチで行う．これは生命維持の仕組みと蘇生の観点から考案された線型のアルゴリズムであり，その誕生の背景は次の通りである．

生命は大気中の酸素を体内に取り込み，全身に酸素を供給する一連の作業によって維持されてい

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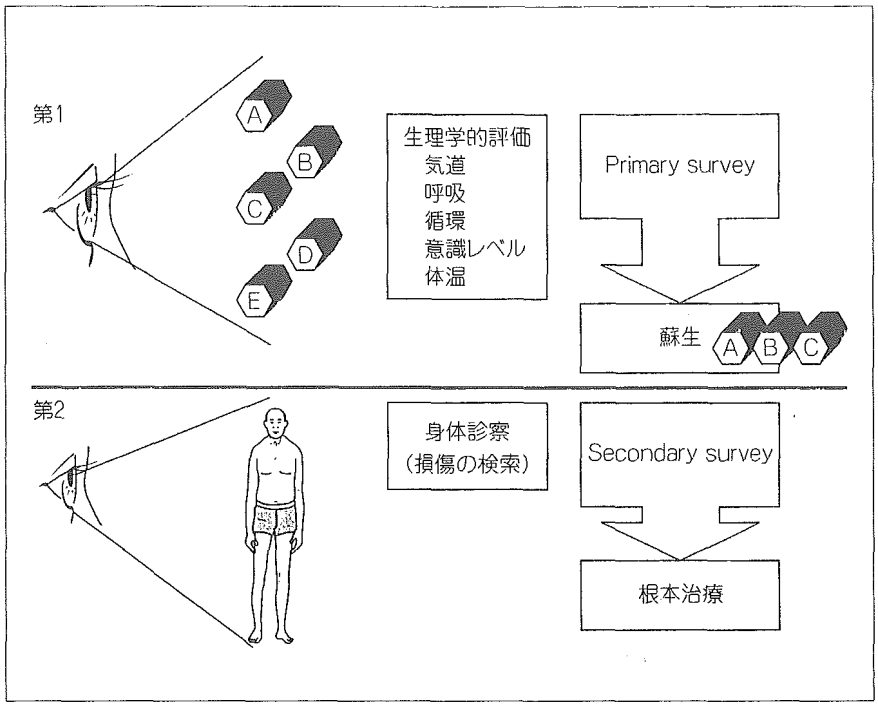

図-1　JATEC^{TM} の診療手順

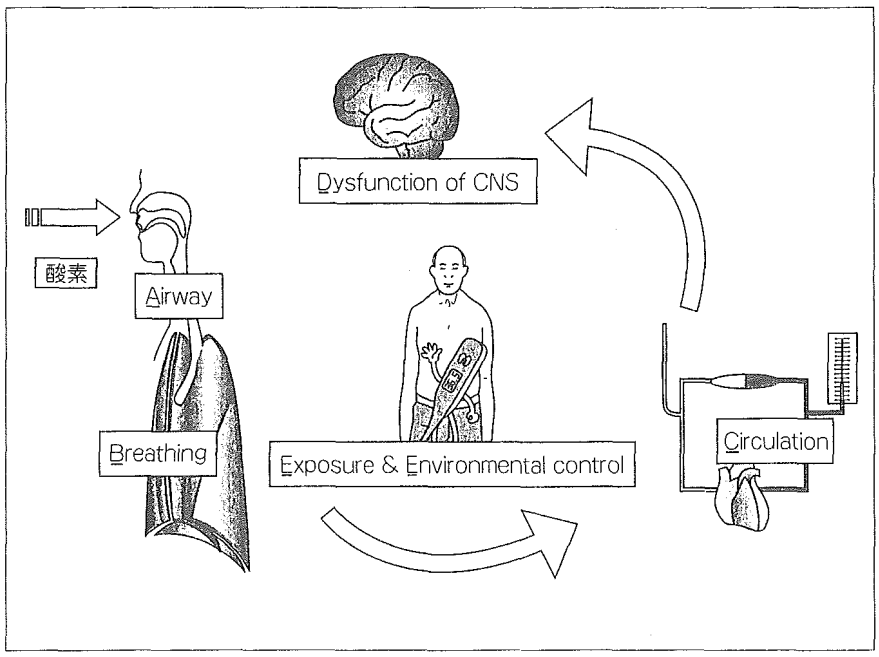

図-2　生命維持の仕組みとその生理機能

る．ことに中枢神経への酸素供給がかなうことで，呼吸の命令(自発呼吸)が発せられ，呼吸，循環を介する生命の輪を形成している(図-2)．

現在の医療レベルで迅速な支持療法が可能なのは呼吸管理と循環管理であり，中枢神経はこの呼吸と循環によって支えられる．したがって観察と蘇生の順番が気道の開放(A ： Airway)，人工呼吸(B ： Breathing)，循環管理(C ： Circulation)となる．外傷では呼吸，循環の評価に加え，頭蓋内損傷を疑う観察が必要である(D ： Dysfunction

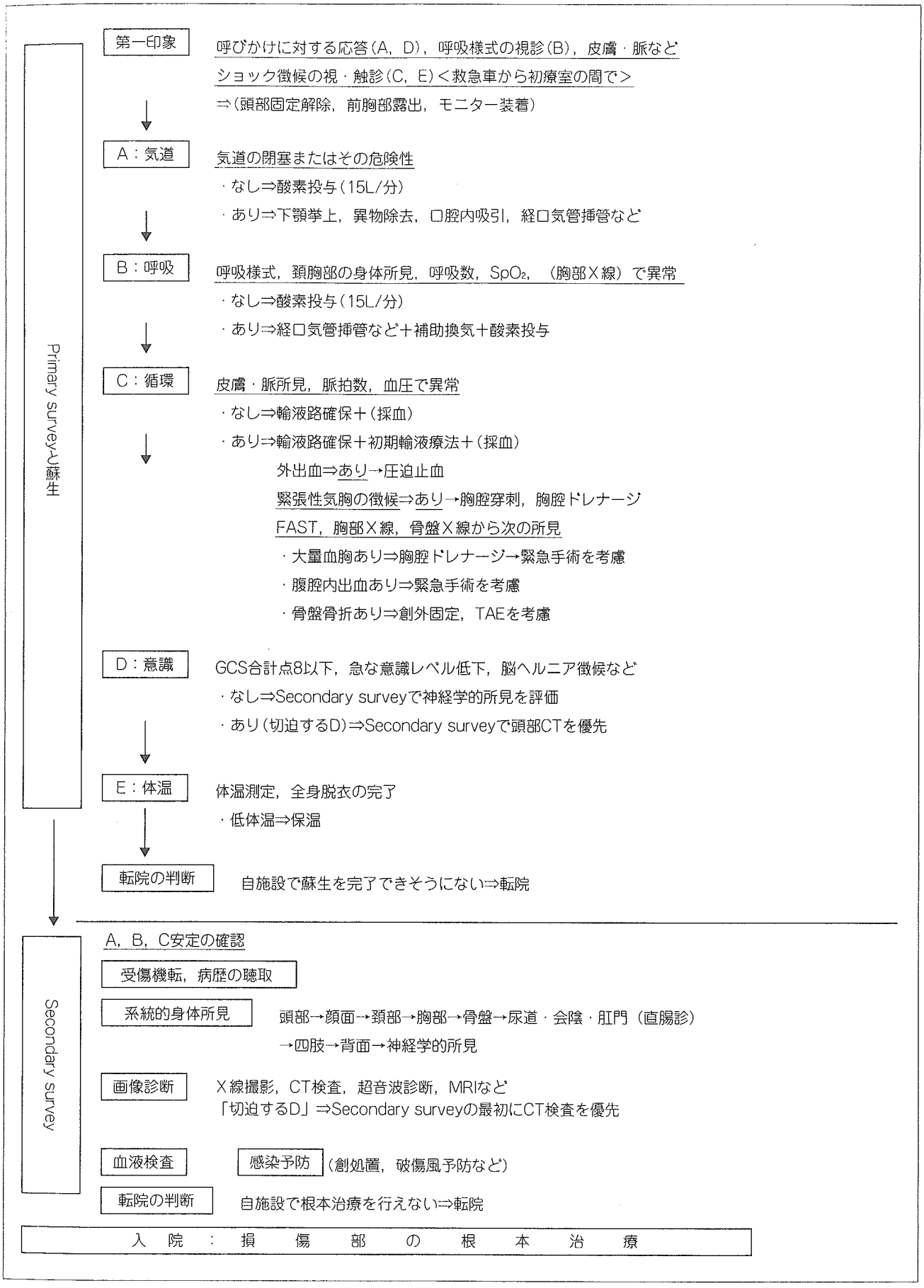

図-3 外傷初期診療アルゴリズム

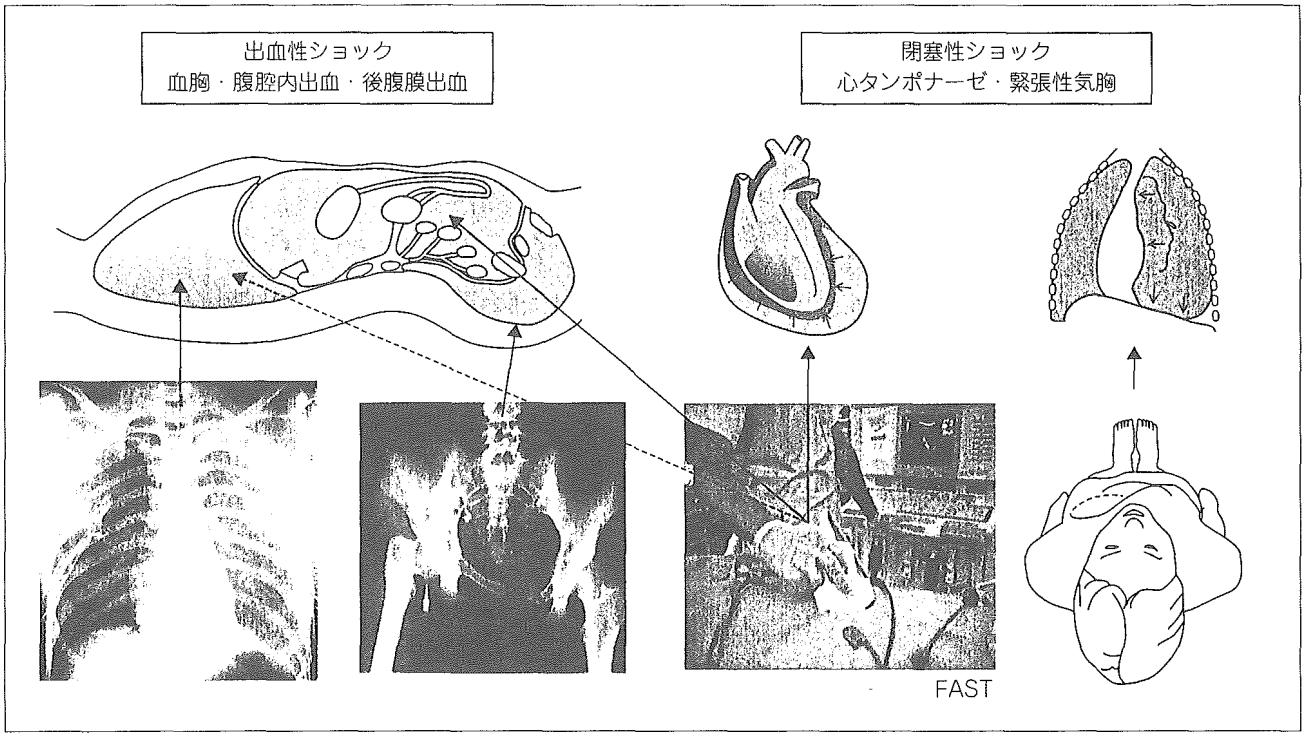

図-4　ショックの鑑別(FAST：本文参照)

of CNS)．これらと並行して全身を露出して診察を進めるが(E：Exposure)，その際，低体温を回避する努力が必要となる(E：Environmental control)．外傷初期診療の第一の目標が生命危機の回避であり，このため「Primary survey と蘇生」は省くことのできない診療手順である．

3．Secondary survey：系統的な損傷検索

外傷初期診療の第二の目標は，見落としのない全身の損傷検索と根本治療が必要性を判断することである．このステップを secondary survey といい，生命危機の状態を脱していることが絶対条件となる．

4．病院間搬送の的確な判断

JATEC^{TM} は助けられる外傷患者の救命を期待するものであって，個々の損傷に対する根本治療を求めているのではない．例えば，肝損傷の治療方法や骨折の処置の仕方を教授することを目的にしていない．むしろ，根本治療の必要とする損傷を見落とさず，適切な診療科へ紹介できることを期待している．状態の安定化を図ること，また，それを最大の担保に的確な転送の判断ができることを目指している．間違っても，自己の診療能力や自施設の機能を超えてまで外傷患者の診療に当たらないよう，コースで指導している．

初期診療の具体的な手順

具体的な手順は以下の通りで，そのアルゴリズムを**図-3**に示す．

1．Primary survey と蘇生

処置室に入り次第，直ちに primary survey を行い，必要なら蘇生を開始する．

a．A：気道評価・確保と頚椎保護

まず話しかけて気道の開放が確実かどうかを確認する．気道が開放されていれば100％酸素を10～15L/分で投与する．同時にパルスオキシメータを装着する．気道の閉塞，意識低下，酸素化が不十分なら気管挿管を行う．

全ての外傷患者には頚椎の損傷が隠れているものとして愛護的に扱い，カラー固定を続ける．

b．B：呼吸評価と致命的な胸部外傷の処置

頚胸部の視診，聴診，触診，打診を行い，呼吸様式の異常と胸部外傷を示唆する所見をとる．呼

表-1　Secondary survey の概要

	身体所見	検索すべき損傷	補助検査
頭顔	創傷，raccoon eye，Battle's sign，頭部陥没，顔面骨の変形，眼，口鼻腔，外耳道(髄液瘻)など	陥没骨折，頭蓋底骨折，顔面骨骨折，眼外傷，口・咽頭外傷	X線，CT
頚部	創傷，穿通創，増大する血腫，ベルト痕，圧痛，頚静脈怒張，血痰，嗄声，頚動脈雑音，皮下気腫，気管の変位，拍動する腫瘤など	喉頭・気管損傷，頚動脈損傷．閉塞性ショックの間接所見	X線，CT
頚椎	疼痛，運動痛，運動制限，棘突起圧痛，四肢のしびれ・麻痺，呼吸困難，腹式呼吸，持続勃起，神経原性ショックの所見(低血圧，徐脈)など	頚椎捻挫，頚椎脱臼骨折，頚髄損傷．〈頚椎カラーはクリアランスできるまで継続〉	頚椎X線3方向，CT，MRI
胸部	穿通創，呼吸困難，胸背部痛，打撲やベルト痕，呼吸様式，胸郭変形，胸郭動揺，軋轢音，呼吸音・鼓音・濁音およびこれらの左右差など	肺，大動脈，気管・気管支，心筋，食道，横隔膜の損傷と血気胸など	X線，CT，透視，内視鏡など
腹部	創傷，打撲やベルト痕，膨隆，呼吸様式など．圧痛，反跳痛，筋性防御(直腸診)	腹腔内出血と管腔臓器損傷．とくに，消化管(後腹膜穿破)，膵損傷，尿路損傷(溢尿)に注意	X線，CT，FAST (US)，DPL
骨盤会陰	腰殿部痛，股関節・大腿痛，股関節ROMの制限，下肢長差，下肢の異常肢位，会陰の皮下血腫，外尿道出血，腫脹，仙腸関節部や恥骨上圧痛など	運動器としての骨盤骨折(寛骨臼骨折など)と骨折に伴う合併損傷(後腹膜出血，尿路，直腸損傷)	X線，CT，血管造影，尿路造影
四肢	疼痛，運動制限，しびれ，創傷，皮膚欠損，変形，腫脹，蒼白，圧痛，運動域，末梢脈拍，冷感など	開放性骨折，整復の遅れる脱臼，疽血障害，筋区画症候群，広範囲皮膚欠損	X線，CT，血管造影
神経	GCS，瞳孔所見，筋力評価，知覚検査，深部反射などの異常	頭蓋内損傷，頚髄損傷，末梢神経損傷	CT，MRI

吸数とSpO_2をチェックする．異常があればポータブルで胸部X線を撮る．呼吸に異常を来す多くは胸部外傷に由来する気道出血，フレイルチェスト，緊張性気胸，開放性気胸，大量血胸などであり，これらの存在を絶えず念頭におく．処置として気道確保と人工呼吸，胸腔ドレナージなどが必要となる．

c．C：循環の評価および蘇生と止血

ショックの早期認知は脈拍，毛細血管再充満時間，皮膚所見および意識レベルなどで総合的に判断する．引き続き脈拍数と血圧をチェック，心電図とともに連続的にモニターする．ショックなら出血部位と閉塞性ショックの有無を検索する(**図-4**)．同時に初期輸液療法を開始する．

1)　外出血は直ちに止血

2)　静脈路の確保と初期輸液療法

保温した乳酸リンゲル液の急速投与(1～2Lまたは20mL/kg)を開始し，循環の反応で治療方針を決定する．

3)　出血源の検索と治療の選択

ショックに至る出血源は，外出血を除けば，主として胸腔，腹腔，後腹膜腔の3部位に多いため，胸部X線，骨盤X線および超音波検査(US)を駆使して検索と処置に精力を注ぐ(**図-4**)．USは，腹腔内出血のみならず心タンポナーデ，血胸まで診断できるすぐれた検査であり，FAST (Focused assessment with sonography for trauma)として初期診療での必須の手技である．

表-2 JATEC™ コースカリキュラム

時刻	内容
初日 800	受付・集合
810-820	挨拶
820-835(15)	JATEC 概要
835-855(20)	JATEC 理論
855-935(40)	初期診療総論
935-1005(30)	初期診療のデモ
1020-1040(20)	気道と呼吸
1040-1100(20)	ショック
1200-1910(50×8)	Skill station （4 人一組，8 グループ）
ST1	気道確保の実技
ST2	胸部外傷治療手技と胸部X線読影
ST3	ショック時の対応(骨髄内輸液，FAST)
ST4	骨盤外傷の扱いと骨盤画像読影
ST5	意識レベルの見方と頭部CT読影
ST6	頚椎保護と頚椎X線読影(クリアランス)
ST7	Primary survey の実技
ST8	Secondary survey の実技
2 日 800-820(20)	胸部外傷
820-845(25)	腹部骨盤外傷
845-905(20)	頭部外傷
905-925(20)	脊椎外傷
925-945(20)	四肢外傷
1000-1200(30×4)	ケースシナリオ/4 ステーション
1000-1200	ケーススタディ
1300-1500(30×4)	ケースシナリオ/4 ステーション
1300-1500	ポストテスト
1500-1600	質疑，認定証授与，閉会

（コースにより修正されることがある）

4) 閉塞性ショックの検索と解除

出血と輸液療法で説明のつかないショックでは閉塞性ショックの発見に努め，緊張性気胸なら脱気をし，心タンポナーデでは心嚢穿刺を行う．

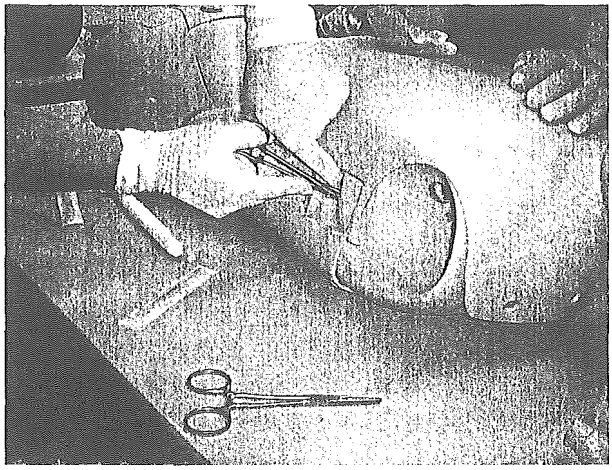

図-5 胸腔ドレナージ

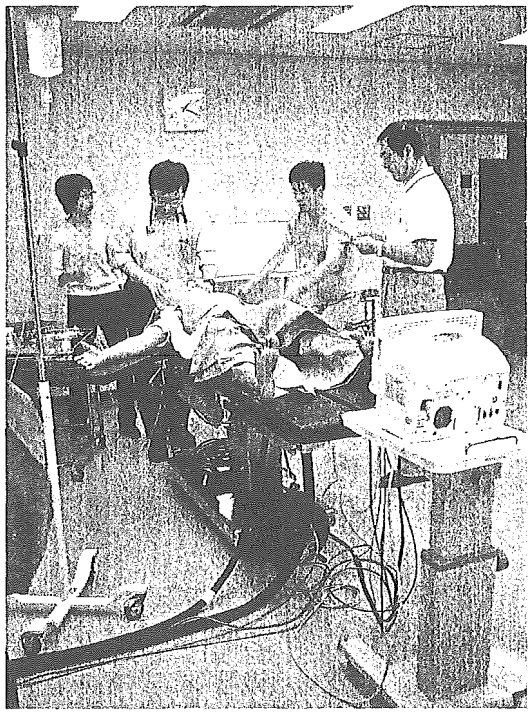

図-6 ケースシナリオ 1

d. D：生命を脅かす中枢神経障害の評価

意識レベル，瞳孔径，対光反射，四肢運動を診る．GCS ≦8（または JCS ≧30），急速な意識低下，ヘルニア徴候などを[切迫するD]と位置づけ，脳外科医のコールまたは転送判断の基準とする．当然，状態の安定化が確認できない時点での頭部 CT 検査は禁忌である．

e. E：脱衣と体温管理

着衣をとり，簡単な体表観察を行う．同時に体

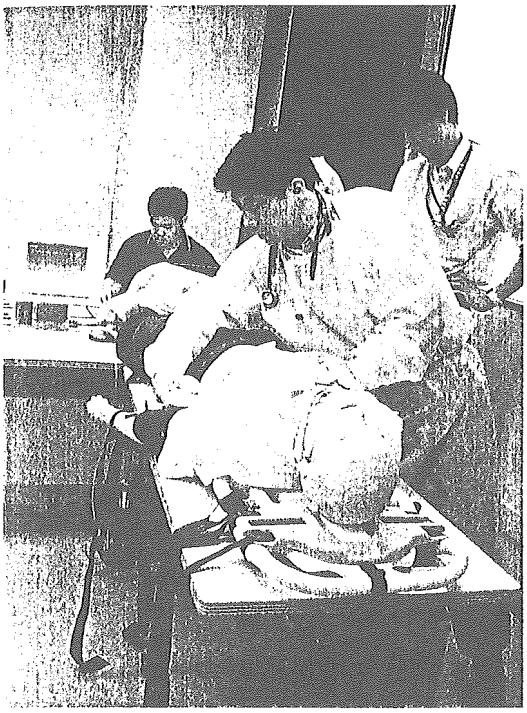

図-7　ケースシナリオ2

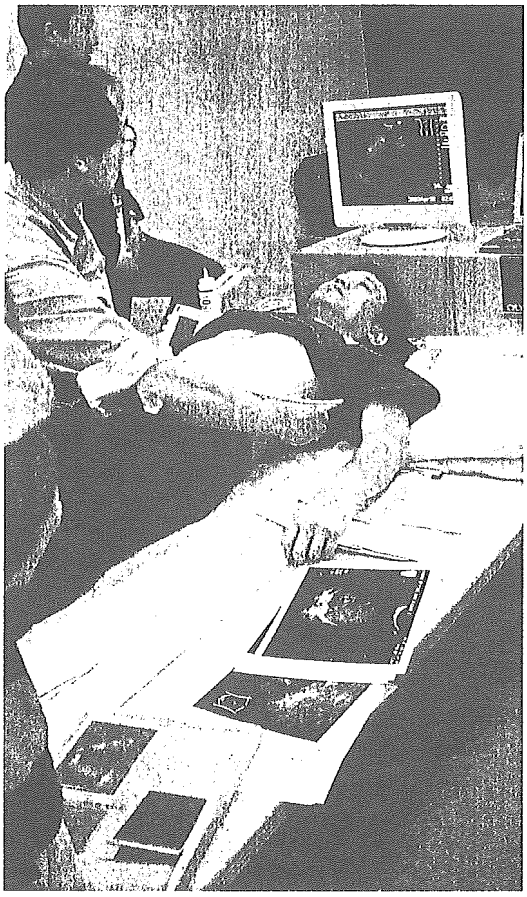

図-8　FASTの練習

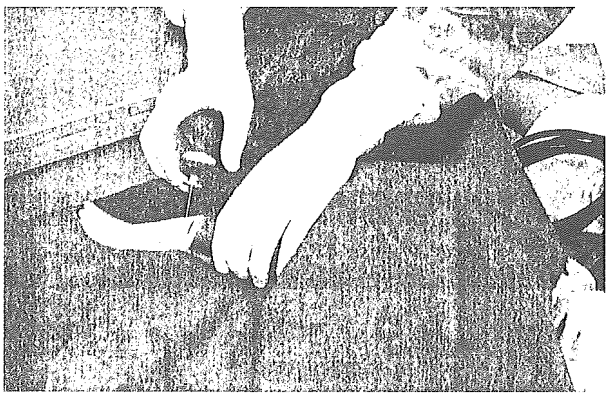

図-9　骨髄輸液の練習

温を測定し，低体温なら保温に努める．

以上，状態の安定を確認すれば，secondary survey に移ってよい．ただし，自施設で対応が困難であると予測すれば，可能な限りの蘇生に努め，この時点で転院搬送の準備にかかる．

2．Secondary survey

Primary survey と蘇生が完了し，患者のバイタルサインが安定してから開始する．Secondary survey は受傷機転や既往歴などの問診，“頭の天辺から足のつま先”までの身体所見，ABCDE の再評価からなる．

a．受傷機転や既往歴の聴取

病歴聴取からアレルギー，常用薬，既往歴，妊娠，最終食事時間，受傷機転などを聞き出し，診察上の危険因子をチェックする．とくに受傷機転は損傷部位を推定するのに役立つ．

b．系統的に診る身体所見

頭，上顎顔面，頚部，胸部，腹部，会陰・直腸・膣，四肢および神経系など詳細に診察する．背面など体位で隠れた部位にも目を通す．また口腔，鼻腔，外耳道を始め，肛門，尿道や膣などの“孔”は内在する損傷を示唆する情報を与える．したがって，指診し，挿入したチューブ内の性状を観察する．画像診断など必要とされる諸検査を行うが，突発的な急変に対応できる設備や熟練した医療従事者のもとで行う．**表-1**に要約する．

c．切迫するDを優先

Primary survey で前述した「切迫するD」を観察した場合，secondary survey を行う際には最優先して頭部外傷の精査を行う．頭部以外の系統的身体所見はCT検査後に行ってよい．

d. 根本治療，またはそのための転院

損傷の部位や程度，集中治療の要否，手術適応などで専門診療科への転床や別の医療機関への転送が必要かどうかを判断する．

模擬診療としてのJATEC™コース

標準化された「外傷初期診療ガイドラインJATEC™」を広く普及させるには，啓発活動が必要である．外傷診療には，外科学，脳神経外科学，整形外科学，麻酔科学や集中治療学などさまざまな分野を包括した知識と技能が要求される．それぞれの専門分野との連携も重要であり，いずれの領域の医師も関与しなければならない．しかし，それぞれの専門家ともなると，標準化された診療手順の学習には抵抗が生じる．このため，単なるセミナーや座学のみでは効果が期待できない．もちろん出版物としての「外傷初期診療ガイドライン」は最低必要な知識であるが，身につけるためには体を使い，手足を動かした体験学習がよい．いわゆる模擬診療やシミュレータによるトレーニングである．

現在，JATEC™コースは2日間で**表-2**に示すコースカリキュラムで，座学，技術・技能習得，ケースシナリオ(**図-5～9**)などをこなし，最後に学習効果を判定するためにOSCE（客観的臨床能力評価試験）を行っている．

なお，本コースは32名の受講に対し，約同人数の指導する講師陣が必要である．現在，日本救急医学会の支援で，講師陣の育成に努めている最中である．早晩，多くの医師に受講の機会を提供できるはずである．コース受講やインストラクターに関連した情報はJATEC™のホームページ(http://www.jatec-web.com/)から入手していただきたい．

文　献

1) 日本外傷学会・日本救急医学会監修：改訂外傷初期診療ガイドライン．へるす出版，東京，2004.