

Exertional heat stroke: a lucky bunch of overly motivated policemen!

運動性中暑：一批幸運的激發過度的警察

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Introduction: Exertional heat stroke is a true medical emergency resulting from thermoregulatory failure. The risk of death is related directly to the peak temperature, duration of exposure and acclimatisation period. This case report illustrates the management of multiple casualties of heat stroke in the emergency department and their outcomes. **Clinical feature:** Nine policemen arrived in the emergency department in semiconscious state after being retrieved from a group of policemen who were loaded into a bus without proper ventilation during a selection program. All of them presented with hyperpyrexia, hypotension, tachycardia and altered sensorium. **Treatment:** Immediate evaporative cooling with support of organ-system dysfunction was commenced. Six patients were intubated and admitted to the intensive care unit. One patient was monitored in high dependency ward while the other two were admitted to the general medical ward. **Outcome:** All patients were extubated within 48 hours. None of them had residual neurological dysfunction. One patient developed coagulopathy which required blood product transfusion. Three patients were discharged in less than one week and the rest were observed in the general ward in stable condition for liver profile monitoring. (*Hong Kong j.emerg.med.* 2007;14:37-44)

簡介：運動性中暑是因體溫調節失效的真正急症。死亡的風險與最高的溫度，暴露的時間及適應氣候的時段有直接的關係。本個案報告舉例說明在急症室治療多名中暑的傷者及其結局。**臨床特徵：**一羣警察在一個挑選程序中被裝載進一架沒有適當通風系統的公共汽車上，其中 9 名警察被拯救後送達急症室時是半清醒的。所有病者呈現高熱、低血壓、心搏過速及神智不清。**治療：**立即開始蒸發性降溫及器官系統功能障礙的支持療法。6 名病者氣管插管後被收進深切治療部，一名病者在加護病房接受監察，而其他兩名被收進普通內科病房。**結局：**所有病者 48 小時內除管，沒有病者有神經功能障礙的後遺症，一名病者顯現凝血病需要輸入血的物質。3 名病者一星期內出院，其餘的情況穩定，在普通病房內觀察，以監察其肝功能剖析。

Keywords: Body temperature regulation, exercise, fever, rhabdomyolysis, seizures

關鍵詞：體溫調節、運動、發燒、橫紋肌溶解、癲癇發作

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Case report

On 30th May 2006, nine policemen who were allegedly involved in a special force selection program were brought to our Emergency Department (ED) in semiconscious state. They were part of a group of 50 policemen who were loaded into a bus without proper ventilation at a police training centre, approximately a 10-minute drive from the hospital.

All of them were pre-morbidly fit and well. They were all in their early 20s. They had been trained for the selection program for more than one week. On the day of the incident, they spent two hours immersed in a swimming pool from 4.00 to 6.00 a.m. After a short break, they took part in a running session from 7.00 to 9.00 a.m. They were ordered to stay in the bus from 10.00 a.m. to 2.00 p.m. as part of the selection program, but were given the options of staying or leaving. The patients arrived at the ED between 1.30 and 2.30 p.m. by police vehicles. No cooling intervention was initiated at the incident site and no notification to the ED was made prior to arrival.

They were immediately triaged to the critical zone upon arrival. However two patients were rerouted to the semi-critical zone due to bed constraint in the 11-bedded (including a paediatric resuscitation bed) critical zone. They were tagged by using the resuscitation bed number as no identification cards were available. All patients presented with altered

sensorium, hyperpyrexia, hypotension and tachycardia. Their vital signs are shown in Tables 1 and 2. Most of them had vomiting, diarrhoea, carpopedal spasm and abnormal postures. Three of them developed seizures in the ED (Table 3).

Treatment was initiated immediately with supplementary oxygen at 15 L/min via non-rebreathing mask, fluid resuscitation with crystalloid solution via two large bore cannulae and evaporative body cooling technique. All clothes were removed and tepid sponging with tap water was started. Electric fans were positioned into the patients' direction. Spraying of cold water was also done using the ice-water blowers. Ice packs were placed strategically at the neck, axillae and groins. Definitive airway management with endotracheal (ET) intubation was performed for patients with airway compromise, seizure, poor breathing effort and haemodynamic instability despite resuscitation. Six patients were eventually intubated and the rest were closely monitored. All patients were monitored with serial blood pressure, pulse rate, pulse

Table 1. Initial vital signs of the patients

Patient	1	2	3	4	5	6	7	8	9
Blood pressure (mmHg)	87/50	91/62	100/45	80/50	60/41	92/59	104/48	102/40	95/60
Pulse rate (beats/min)	122	118	125	123	125	117	114	120	130
Respiratory rate (breaths/min)	20	24	26	14	26	28	20	30	18
Temperature (°C)	40.0	39.4	38.5	39.5	39.6	40.0	39.7	38.8	42.0

Table 2. Initial Glasgow Coma Scale scores of the patients

Patient	1	2	3	4	5	6	7	8	9
Eye	2	2	3	1	1	1	1	3	2
Motor	3	2	2	2	1	1	2	2	1
Verbal	5	5	3	5	5	4	1	5	5
Total	10/15	9/15	8/15	8/15	7/15	6/15	4/15	11/15	8/15

Table 3. Clinical features at presentation

Feature	No. of patients	Feature	No. of patients
Abnormal posture	5	Vomiting	6
Confusion	5	Diarrhoea	6
Coma	4		
Seizure	3		

oximetry, ECG and serial core (rectal) temperature charting. Inotropic support was started for hypotensive patients refractory to fluid resuscitation. Adequate sedation with intravenous diazepam or midazolam was provided to reduce restlessness, shivering and carpedal spasm. To reduce the risk and complication of aspiration, intravenous antiemetics, H₂ antagonist and antibiotics (cefuroxime and metronidazole) were provided for patients who presented with coma or vomiting. Urinary catheters were inserted for all patients. All patients had clear but concentrated urine while they were in the ED. Minimal blood investigations were done to avoid confusion in labelling and delaying treatment. Initial arterial blood gases on non-rebreathing mask revealed a spectrum of normal, respiratory alkalosis and metabolic acidosis features (Table 4).

All patients achieved normal core temperature before being disposed from the ED. One patient was transferred from the critical to the semi-critical zone after being stabilised. Another patient was contrarily moved from the semi-critical to the critical zone for resuscitation when his condition deteriorated.

In anticipation of a mass casualty incident, additional personnel and equipment were simultaneously mobilised while the patients were being attended. The head of department of the ED and the hospital director were notified about the crisis. All available emergency physicians were summoned to the ED. The medical officers and paramedics who worked on the morning shift were retained to provide aid. Early referral to the Anaesthesia and Medical departments was also made to co-manage the patients. Mobilisation of the ED staff nurses and medical assistants (MA) was coordinated by the ED Head Nurse and MA supervisor respectively.

Apart from retaining the morning shift staff, the hospital matrons and supervisors were informed and they responded by mobilising staff nurses from the General Intensive Care Unit (ICU), Coronary Rehabilitation Ward and surgical wards down to the ED to provide additional help.

To ensure adequate beds, all in-patients in the critical and semi-critical zones were mobilised and admitted to the wards accordingly. Electric fans, ice-water blowers, crystalloid fluid and extra linen, gauze and gamgee were brought out from the storeroom. Ice was collected from the ice-making machine which was located in the ED. As there were limited numbers of ventilators and critical care beds available for these patients in the hospital, arrangements with other Klang Valley hospitals were carried out. Four patients were transferred by ambulance to two Klang Valley hospitals between 6.00 p.m. and 10.30 p.m. They were closely monitored by the ICU and ED staffs during the transfer. Two patients were admitted to the General Intensive Care Unit, one was observed in the High Dependency Ward and the other two were admitted to the general medical ward.

Within 24 hours, six patients had evidence of rhabdomyolysis (creatinine kinase >600 U/L) and two patients developed transient ECG changes in the form of 1st degree heart block and ST segment elevation. Both changes resolved without pharmacological treatment. One patient who was intubated had aspiration pneumonia. Another patient who developed seizure in the ED was found to have hyponatremia (serum sodium 115 mmol/L) which was probably the actual cause of the seizure. It was corrected accordingly without any consequence.

Table 4. Initial arterial blood gases on non-rebreathing mask with 15 L O₂/min

Patient	1	2	3	4	5	6	7	8	9
pH	7.32	7.29	7.34	7.34	7.40	7.55	7.35	7.38	7.35
PCO ₂ (mmHg)	35	32	33	39	31	27	34	24	36
PaO ₂ (mmHg)	135	150	120	89	228	161	160	237	115
HCO ₃ (mmol/L)	16.3	15.7	19.1	20.0	19.0	24.5	21.5	17.3	21.8
BE (mmol/L)	-7.5	-10.1	-6.9	-3.8	-7.2	3.5	-3.5	-9.8	-2.0
SpO ₂ (%)	100	98	100	94	99	99	99	100	98

Elevated liver enzymes, coagulation disorder, renal impairment and hypertension were the delayed complications of heat stroke (HS) seen in these patients. Four patients developed prolonged INR and one patient had bloody diarrhoea which resolved spontaneously (Table 5). All patients recovered with adequate rehydration without requiring replacement therapy.

The patients' clinical conditions were followed up for one week after their disposition from the ED. All patients were extubated within 48 hours of admission to ICU. None of the patients had residual neurological deficit. After one week, only one patient was still in the ICU due to coagulopathy and uncontrolled hypertension which required blood product transfusion every 12 hours and intravenous antihypertensive respectively. Three patients were discharged without further complications. The rest of the patients were observed in the ward for liver profile monitoring. They were stable with normal blood glucose and improving coagulation profile.

Discussion

Heat stroke is defined classically as a core temperature more than 40.5°C accompanied by central nervous system (CNS) dysfunction and anhidrosis.¹ It results from thermoregulatory failure coupled with an exaggerated acute phase response and possibly altered expression of heat-shock proteins.² Individuals may begin to sustain cellular damage anywhere from 45 minutes to 8 hours after exposure to a core temperature of 42°C.² The risk of death is related directly to peak temperature, duration of exposure and acclimatisation period.^{1,2} The mortality rate has been reported to be 10-70% with the highest number of deaths occurring when the treatment is delayed for more than two hours.²

It is a common problem in the tropics, and with the ever-increasing global warming its incidence is rising even in temperate climate.³

Two clinical presentations of HS are classic HS (CHS) and exertional HS (EHS), which are defined by the underlying aetiology but are clinically indistinguishable. CHS is usually seen in the extremes of age related to either limited mobility or chronic diseases and commonly develops during heat waves when the air temperature exceeds 39.2°C.² In August 2003, France experienced an unusual and severe heat wave which was responsible for more than 14,000 deaths during a period of 20 days. A retrospective analysis by Davido et al in an urban ED in Paris showed most of them were elderly patients, predominantly women and dependents.⁴

In contrary, EHS is an illness of the overly motivated people. Most victims of EHS are highly motivated, healthy young individuals who exert themselves beyond their physiological capability. In a review of 82 cases of EHS in the Israeli Defense Forces from 1988 to 1996, 57% of EHS occurred during basic training and 21% occurred during screening test for special forces – in which motivation is a key issue.⁵

EHS typically occurs over hours in patients who exercise at elevated temperature for a sufficient period of time to cause the rate of heat production to exceed the capacity of the body to dissipate heat. The cardiovascular system responds to heat stress by increasing the cardiac output to 20 L/min and shifts the heated blood from the core circulation to the peripheral circulation where heat is then dissipated to the environment.⁶ During strenuous exercise, blood shifts from the mesenteric circulation to the working muscles and skin, leading to ischaemia of the gut and

Table 5. Delayed complications

Complication	No. of patients	Complication	No. of patients
Elevated liver enzymes	7	Bloody diarrhoea	1
Thrombocytopenia	5	Hypertension	1
Coagulopathy	4		
Renal impairment	3		

intestinal hyperpermeability. Alteration in the immunologic and barrier function of the intestine allows leakage of endotoxins which triggers the production of cytokines that induce endothelial cell activation, and release of endothelial vasoactive factors. These factors interfere with normal thermoregulation by raising the set point at which sweating is activated and alter the vascular tone thereby precipitating hypotension, hyperthermia and heat stroke. Sweating may be profuse or absent, anhidrosis usually is a late finding due to profound volume depletion and sweat gland dysfunction.²

The ensuing multiorgan injury results from a complex interplay among the acute physiological alteration due to hyperthermia, the direct cytotoxic effect of heat and the inflammatory and coagulation response of the host.⁶ The plasma levels of inflammatory cytokines (tumour necrosis factor [TNF], interleukin-1 β , interferon- γ) and anti-inflammatory cytokines (interleukin-6, soluble TNF receptors, interleukin-10) are elevated in patients with heat stroke. An imbalance between these factors may result in either inflammation-associated injury or refractory immunosuppression. The onset of heat stroke also coincides with the activation of coagulation and fibrinolysis processes. This constellation of events leads to alterations in blood flow in the microcirculation and results in injury to the vascular endothelium and tissues. The serious complications of heat stroke include encephalopathy, rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial injury and disseminated intravascular injury. CNS dysfunction is attributed to cerebral oedema, metabolic derangement, ischaemia and is universal at a core temperature of more than 42°C.^{1,3}

Predisposing factors for EHS include the male sex, sleep deprivation, dehydration, alcohol abuse, skin diseases, febrile illness, drugs impairing normal thermoregulatory response and a past history of HS.^{3,7} As heat-shock proteins protect cells from damage by heat, ischaemia, hypoxia, endotoxin and inflammatory cytokines, conditions associated with low levels of expression of heat-shock proteins i.e. genetic polymorphisms and lack of acclimatisation, favour the progression of heat

stroke.⁶ These patients had indeed some degree of sleep deprivation and dehydration due to the activities they had undergone prior to the incident, thus increasing their risk to develop EHS.

Though heat stroke is a diagnosis of exclusion, the history of heat exposure and classic clinical presentation had prompted instant treatment aiming at immediate cooling in order to minimise end-organ damage. An ideal goal is to lower the patient's core temperature by 0.20°C/min until an endpoint of 38°C to avoid overshoot.² There is debate over the best method of body cooling. Evaporative cooling technique was chosen as it is safe, effective, easily accomplished and well tolerated.^{1,2,5} Tap water was readily available and did not require any complicated logistical arrangement. Electric fans were used to maximise evaporative heat loss as 1 g of water dissipates seven times as much heat as melting the same quantity of ice.² In addition, ice packs were strategically placed at the neck, axillae and groins to cool down the blood in the large jugular, axillary and femoral vessels.

Ice water immersion offers the best cooling time and rate, according to a review by Smith on the cooling methods basing on 17 articles published from 1966 to July 2003.⁸ Cooling with ice was found to reduce rectal temperature by 0.20°C/min, compared with the evaporative technique at the rate of 0.11°C/min. It is however associated with more complications than the evaporative method. Peripheral vasoconstriction shunts blood away from the periphery and may lead to shivering and less heat dissipation.^{1,5,8} It is uncomfortable for conscious patients, limiting the access for cardiac and vital signs monitoring and may cause hypothermic overshoot. It is also impractical to be utilised in these cases as most of the patients presented with diarrhoea, hence requiring disinfection of the ice water.

Invasive methods of cooling include gastric, peritoneal and bladder lavage with cold water. These have been investigated in animal models and have limited human experience.¹ Data in human subjects came from a case series and a single case report.⁸ The role of invasive cooling methods therefore has not been fully

established. The most rapid invasive cooling technique is cardiopulmonary bypass which may be required if a patient's condition is recalcitrant to all other measures.¹ A case reported by Broessner et al illustrated a successful endovascular cooling technique to combat hyperthermia and maintain normothermia in treating heat stroke with multiple organs dysfunction.⁹ However, nonsteroidal anti-inflammatory drug was used as the primary cooling method in this case and the rationale of the use was not provided.

Normalising the body temperature after the onset of HS may not prevent inflammation, coagulation and progression to multiorgan dysfunction.⁶ Therefore a new concept of treating HS with immunomodulators i.e. interleukin-1 receptor antagonists, antibodies to endotoxin and corticosteroid has been studied in animals and proven to improve survival. Replacement therapy with recombinant activated protein C reduces mortality in patients with severe sepsis and may be useful in patients with HS as well.

Supportive treatment was commenced while the temperature was being stabilised. The decision of performing endotracheal intubation on six patients was based on various reasons. Most patients had more than one indication. It was performed for patients who remained comatose to protect the lungs from aspiration. A patient developed ineffective breathing and ET intubation was required to maintain oxygen saturation at more than 95%. As the brain is very vulnerable to ischaemic injury due to its relatively high oxygen consumption, intubation was also carried out on the patients with haemodynamic instability for cerebral protection. Interruption of cerebral perfusion pressure or severe hypoxaemia will rapidly result in functional impairment and ruin the clearance of potentially toxic metabolites. Cerebral protection was also provided to the patients who developed seizure as cerebral oedema is a common finding in heat stroke casualties.¹ Intracranial bleeding could not be ruled out at that time. There was also a high chance of all these patients having some degree of hypoxic ischaemic encephalopathy based on the history of being confined in a crowded and poorly ventilated bus for a few hours. Relative tissue hypoxia could also occur due to

the increased cerebral metabolic rate from the hyperpyrexia. Hyperpyrexia per se could liquefy membrane lipid, denature enzymes and alter the molecular activities of the brain.¹⁰ Therefore ET intubation was essential to provide adequate cerebral perfusion pressure, decrease metabolic requirement and possibly block mediators of cellular injury.

All patients were successfully extubated after 12 to 48 hours without neurological consequences. None of the patients was subjected to CT scan of the brain due to improving neurological signs. The role of cerebral protection in these cases, whether preventive or therapeutic, remains hypothetical. Clearly, the most effective strategy is prevention of secondary insults to the brain, because once the injury has occurred measures aiming at cerebral protection will become less effective.

Hypotension refractory to fluid resuscitation and cooling measures are likely due to peripheral vasodilation (distributive shock) and primary myocardial dysfunction. Hence inotropic support was started and the fluid input and output were closely monitored. Benzodiazepine was used to treat seizure, restlessness and also shivering. As HS is a relatively immunosuppressed state, the chance of secondary infection exists. Mehta and Jaswal advocated antibiotics administration for HS patients particularly when aspiration pneumonia is suspected.³

No antipyretic was provided to reduce the core temperature in these patients. In contrast to a febrile patient who develops fever because of an elevated hypothalamic set point, the HS patient does not benefit from antipyretic therapy.^{1,2} Paracetamol in large doses can in fact worsen the hepatic damage. Ideally, emergent investigations should be done in the ED to detect and correct immediate HS complications.¹ However, only a few investigations which could change the therapeutic intervention in the ED were done. Serum enzymes to identify rhabdomyolysis and liver damage may be insensitive because of the delayed release of the enzymes into the circulation.¹¹ Hyponatremia in exercise or training is commonly produced by excessive water intake, fluid retention in the heat and may be worsened by

vomiting.¹¹ Hepatic transaminases are universally elevated in HS secondary to centrilobular necrosis as the result of direct thermal injury. Thermal injury to the vascular endothelium causes platelet aggregation and deactivation of protein plasma, resulting in thrombocytopenia and decreased level of clotting factors.¹ Renal impairment (serum creatinine >150 $\mu\text{mol/L}$) may be related to rhabdomyolysis, hypotension and direct heat injury to the renal parenchyma.

Although complete recovery is the rule in survivors of HS, deficit may persist in 20 percent of cases with the cerebellum being the most commonly affected.³ Our patients' favourable outcomes are possibly due to the immediate and effective cooling measures, adequate rehydration and aggressive treatment to prevent potential complications taken by all disciplines. They were also lucky since the police training centre was located only in a 10-minute drive from the receiving hospital, thus reducing the length of time between heat exposure and the initiation of cooling measures. Prognosis worsens proportional to the duration of time between the onset of elevated core temperature and commencement of cooling measures.^{2,7} Poor prognostic signs include coagulopathy with liver hepatocyte damage, rectal temperature more than 42.2°C, prolonged hyperthermia, residual brain damage and acute renal failure.^{1-3,12}

This case report would like to highlight the importance of personnel and equipment mobilisation and also external coordination in managing multiple casualties of heat stroke. The unannounced arrival of nine unstable patients within a short period of time was considered a mass-casualty incident as it disrupted the normal daily emergency department operation.¹³ Delayed notification of hospital staff, poor delineation of the command structure, broken communication and lack of supplies are the usual shortcomings of a hospital disaster plan.^{13,14} However these were avoided by the swift actions taken by the multiple disciplines and the high level of cooperation with other hospitals.

The management can be upgraded with a proper planning by the police department in executing the training program. Goodman et al had carried out an

epidemiologic investigation in 1988 following the outbreaks of exertional rhabdomyolysis with renal impairment among candidates for public safety position.¹⁵ As a result, the Massachusetts Criminal Justice Training Council extensively revised its police training program. The study recommended a specific physical fitness and medical standard before and during the training program, adequate hydration during the activities based on the intensity and duration of the activity and the prevailing environmental condition and also a clear administrative chain of responsibility and protocol for responding to injury or illness.

Exertional heat illness continues to be a military problem during training and operation. More than 5,000 soldiers were hospitalised and 37 died due to heat illness in the U.S. army from 1980 through 2002.¹⁶ In 1989, Sir Roger Bannister commented on EHS in the armed forces in a letter to The Times of London: *The notion that encouragement and esprit de corps can somehow defeat the principles of physiology is not only wrong but dangerously wrong; life can unnecessarily be lost.*¹⁷ The police or any training department should therefore liaise with the local Emergency Medical Services to help in preventing, recognising and treating EHS. Education of the participants to recognise the subtle behavioural signs possibly attributable to heat injury helps in the early detection of cases. Immediate on-site initiation of rapid cooling and concurrent resuscitation are the most critical steps in the management of EHS. A cooling body unit at site is essential. Nevertheless, it should not impede patient's transfer to the hospital if heat stroke is suspected.

Conclusion

Heat stroke is a true medical emergency, requiring rapid reduction of the core temperature simultaneously while the ABCs (airway, breathing, circulation) are being assessed and secured. Evaporative cooling is the technique of choice as it is simple and non-invasive. Hospital admission is mandatory. With the ever-increasing global warming, its incidence is rising even in temperate climates. An emergency department should have adequate equipment to provide effective

treatment for multiple heat stroke casualties. In situations where the number of cases exceeds the normal daily emergency department capability, internal and external personnel and equipment mobilisation are very crucial. Rapid intervention and effective communication with other relevant departments have proven to prevent mortality among these patients. Exertional heat stroke can be prevented with proper planning, identification of risk, rescheduling of strenuous physical activities, adequate hydration, pre-hospital body cooling system, efficient transportation and early notification to the hospital.

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