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Case Report

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From Near Death to Recovery in Exertional Heat Stroke: Lessons Learned from a Long Distance Runner's Case

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Abstract

We report the case of a 26-year-old long-distance runner who survived three intubates after exertional heat stroke with multiple organ dysfunction, but left neurological damage. Heat stroke is a preventable illness, and thorough knowledge of the disorder can help to reduce mortality and morbidity. Timely management (the "golden hour") with rapid cooling and intravenous fluids is crucial to prevent irreversible and fatal organ damage. With this report, we want to remind emergency physicians or healthcare practitioners of the primary hospital that early aggressive cooling strategies can avoid EHS deterioration and even death.

Keywords: Heat; Exertional heat stroke; Runner; Central nervous system; Emergency

Introduction

Heat stroke is an acute disease caused by central dysfunction of body temperature regulation, sweat gland failure, excessive loss of water and electrolytes in the hot season, high temperature, and high

humidity environment. It is a form of hyperthermia associated with a systemic inflammatory response leading to a multiorgan dysfunction syndrome in which encephalopathy predominates [1]. Heat stroke, the most serious type of heat illness, is a fatal emergency, clinically divided into exertional and non-exertional heat stroke. Exertional heat stroke occurs mainly with endogenous excessive heat production in a high-temperature environment. It is more common in healthy young people with heavy physical labor, sports, or military training. The symptoms are hyperthermia, convulsions, coma, peripheral circulation failure, or shock. Rhabdomyolysis, acute renal failure, liver failure, DIC, or multiple organ failure are more likely to occur in exertional heat stroke patients, and the mortality rate is higher.

Case Presentation

A previously healthy 26-year-old long-distance male runner collapsed at the 300meters around the terminal of the 10-kilometer training on a hot summer morning in Yulin (Sanya, China), Ambient temperature, hygrometry, and wind speed were 28℃, 84%, and 9 km/h respectively. He was immediately delivered to a shady and cool place, the clothes removed and rapidly transferred to the medical room. Initial onsite evaluation showed a state of coma, high fever (axillary temperature 38.5°C), low blood pressure 80/40 mmHg, and he was infused with a 200 ml fluid. As the limited conditions, non-aggressive cooling therapy and fluid management were provided in the first 50 minutes until arriving at a primary hospital, with intermittent seizures in the ambulance. Exertional heat stroke was diagnosed, and the patient was immediately started with rapid cooling therapy including an ice blanket machine, and was put in a urinary catheter. He was transported to our hospital at 11:50 AM (UTC+8) for further treatment, unconscious with a high rectal temperature (39.6°C), blood glucose of 3.6 mmol/L, and 200 ml foul-smelling stool. On arrival, the patient was immediately received with intravenous 50% GS, rapid cooling therapy including ice packs, an ice blanket machine, an ice cap, aggressive fluid management (7800 ml crystalloids in 10 hours), and intubation. On admission to the intensive care unit at 13:30, his rectal temperature was 37.8℃, his blood pressure was 84/40 mm Hg, his pulse rate was 68/min, SPO2 99%, and ventilator-assisted breathing (FiO2 40%).

Laboratory tests showed elevated Aspartate Transaminase (AST; 65.2 U/L), elevated myoglobin quantification (746 ng/ml), elevated troponin T (0.376 ng/ml), elevated creatinine (157.8 μmol/L), elevated creatine kinase (370 U/L), elevated amylase (456 U/L), elevated lipase (71.4U/L). C-reactive protein was reported to be <0.5, procalcitonin was 0.555 ng/dl, and interleukin 6 was 550 pg/ml. Plasma D-dimer was considerably higher than the normal range (17769 ng/ml). The international normalized ratio was 1.31, the prothrombin time was 14.2s, and prothrombin activity was 67%. A Computed Tomography (CT) of the brain was taken to report multiple groups of sinusitis, excluding any cerebral parenchymal pathologies, and no specific parenchymal lesions were detected. CT of the lung examination revealed scattered inflammation in both lungs. Exertional heat stroke (extra-heavy) was diagnosed, and the patient was complicated with multiple organ dysfunction (eight organs) including central nervous system dysfunction, circulatory failure, Coagulation Disorders (DIC), hepatic dysfunction, acute kidney injury, exercise rhabdomyolysis syndrome, respiratory failure and functional injury of the pancreas. CT of the lung examination revealed scattered inflammation in both lungs on

June 17th on hospitalization. Multiple exudative lesions of both lungs increased significantly compared with the previous film and bilateral pleural effusion was added on June 23rd. On June 30th, both lungs scattered exudative lesions significantly reduced and a small amount of fluid was absorbed in the original bilateral pleural cavity **(Figure 1)**. The patient was in rapid progression, and the main results of a blood analysis are shown in **Table 1**. The patient was in rapid progression. On the second day of admission, platelets dropped from 171 to 33, decreasing by 80%. Blood amylase on admission day was 3 times the upper limit of normal reference value. The maximum myoglobin value was 856ng/ml on the second day of admission and exercise-induced rhabdomyolysis was diagnosed. Liver function deteriorated to the worst level on day 4 of admission. The inflammatory markers peaked on day 5 of admission.

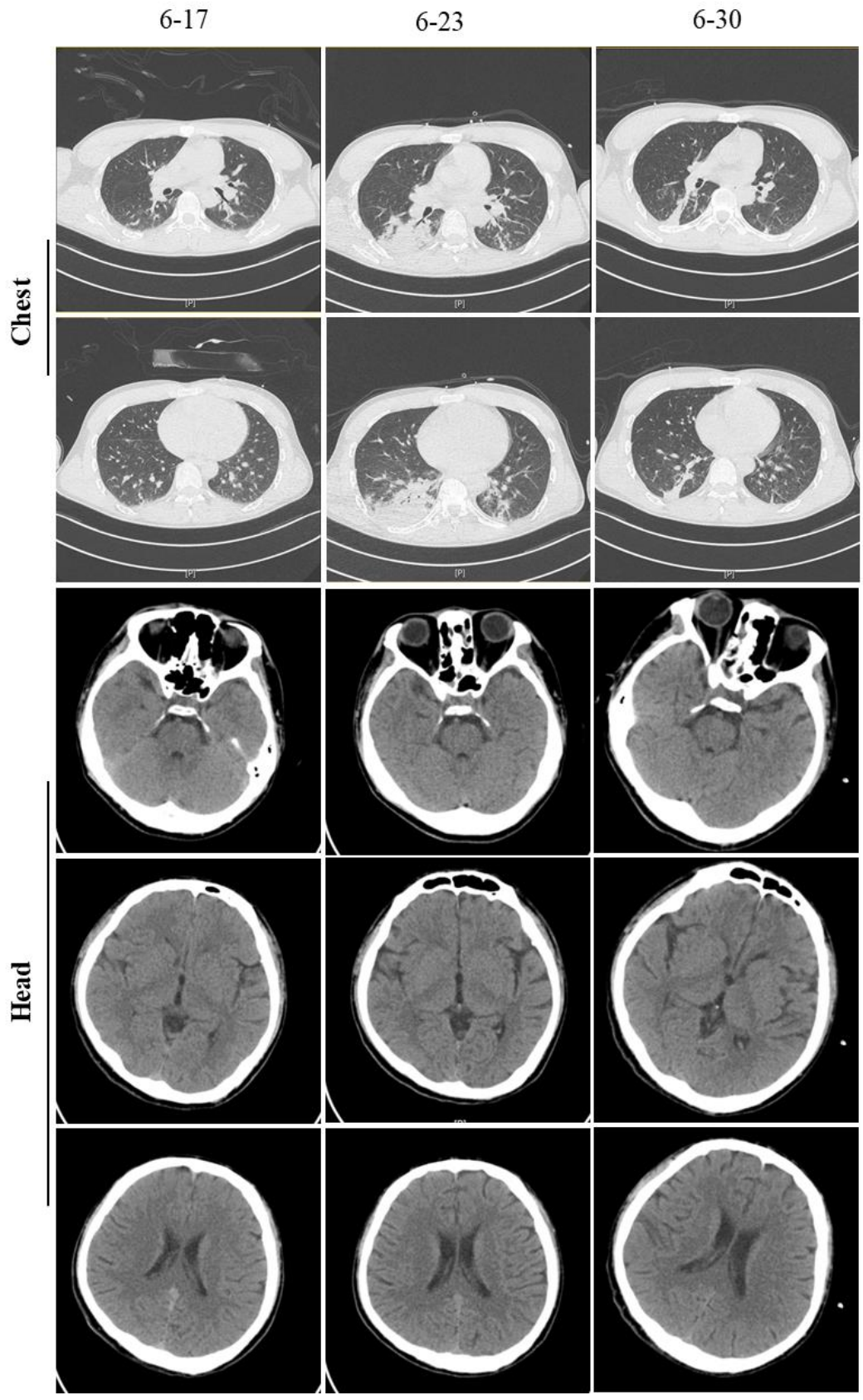

Figure 1: Computed tomography imaging examinations for Chest and head.

Table 1: Laboratory tests and results.

Laborator	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day9	Day11	Day13	Day15	Day17	Day24	Day31	Day43	Day63
WBC coun	12.48	8.01	9.14	8.48	7.18	6.89	5.74	8.59	11.99	10.9	11.63	16.53	10.33	8.3	3.87	3.59
Hemoglob	142	134	127	118	113	113	107	114	128	108	85	81	103	110	132	148
Platelet c	67	33	33	29	41	84	125	298	440	288	224	375	588	317	260	225
PCT (ng/m	0.986	0.933	0.607	0.431	0.733	0.461	0.381	0.163	1.81	0.421	0.245	0.184	0.114	-	0.051	0.046
PT (s)	21.6	21.9	20.8	18.4	14.7	12	11.8	12.3	12.9	12.2	12.2	11.6	12	11.7	11.7	11.3
APTT (s)	93.4	41.2	64.5	32	31.2	30.4	28.6	21.9	29.1	25.4	25.1	23.1	26.8	28	34.3	39.3
Fibrinoge	1.66	1.82	2.39	2.8	3.13	6.07	7	4.84	7.08	6.07	2.59	3.42	3.58	3.62	3.1	2.21
D-dimer I	17769	16867	16168	3721	4754	1583	1053	4044	1591	5448	15398	1356	-	877	774	355
BUN (mm	10	5.1	2.7	1.7	1.6	2.9	3.7	7.4	6.7	4.1	3.9	7.9	8.1	5.8	3.7	4.6
Creatinine	157.8	87.9	87.6	70.5	71.8	58.1	56.7	67.1	63.4	46	45.3	57.7	58.1	54.6	61.1	62.3
AST (U/L)	65.2	1505.9	1622.5	1137.9	730	275.7	181.1	116.3	78.6	268.3	364.1	155.2	66.8	29.8	27.5	31.7
ALT (U/L)	33.7	1176.4	1949.9	1896.7	1760.3	979.9	615.7	373.1	199.9	123.3	147.7	139.1	101	44.8	51.1	31.1
Tbil (μmo	22.4	59.5	82.3	71.9	46.7	23.9	16.4	16.8	17.5	11	13.4	10.7	8.9	8.9	7.5	10.1
LDH (IU/L	426	1441	979	680	307	322	331	355	303	509	927	843	718	306	219	259
CK (IU/L)	737	3570	3135	1191	1326	2984	2343	604	1130	5462	4588	2042	547	150	40	49
CK-Mb (U	32.8	65.9	55.5	28.1	23.8	47.1	29.8	16.3	16.1	75.4	71.1	40.4	23	13.7	9.7	22.3
cTnT (ng/	0.469	0.186	0.169	0.0791	0.0746	0.0305	0.015	0.0145	0.0151	0.0217	0.015	0.0218	0.0222	0.0258	0.0151	0.0117

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; APTT: Activated partial thromboplastin time; BUN: Blood urea nitrogen; CK: Creatine kinase; CK-MB: Creatine kinase-MB; cTnI: Cardiac troponin I; LDH: Lactate dehydrogenase; PCT: Procalcitonin; PT: Prothrombin time; SOFA: Sequential Organ Failure Assessment; WBC: White blood cell.

The patient was in deep sedation, norepinephrine was provided to maintain the blood pressure. Cooling was continued with a hibernation mixture (Dilantin 100 mg+ chlorpromazine 12.5 mg+ promethazine 25 mg), and hemofiltration (the temperature was normal in 10 hours). Fresh frozen plasma, human fibrinogen, and nadroparin calcium were administered. Ceftriaxone at first and vancomycin combined with cefoperazone sulbactam sodium later were used for anti-infection. The GS 200 ml nasal feeding pump was used for enteral nutrition. 40 methylprednisolone was used intermittently as an immune conditioning drug. An intermittent enema was administered for intestinal decontamination.

The patient survives in the end but sustains what may be permanent neurological damage. The brain CT and MRI examination was completed, and no abnormal signal changes were found in the brain **(Figure 1 and 2).** During the recovery period, the nervous system was damaged with seriously decreased myodynamia of the head and neck, like the dorsal muscles. The muscle strength of the limbs was grade 4, and the fine motor movements were poor and the patient could not write. In the recovery of strength and fine motor movement, the vision is still blurred during rehabilitation.

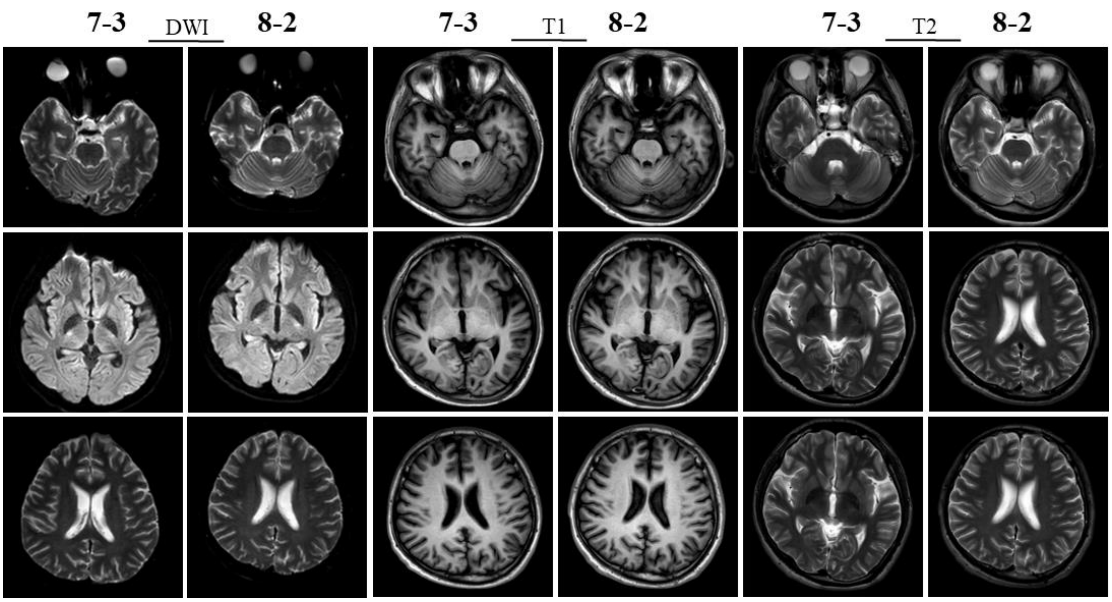

Figure 2: Head imaging examinations of Magnetic Resonance Imaging (MRI).

Discussion

Although the patient survived, the complicated treatment process is worth discussing. The patient's body temperature was repeatedly above 39°C during the 20 days of admission. Temperature management was difficult during the admission and was performed with ice blankets, brain protection, continuous hemofiltration, hibernation mixture, and non-steroidal anti-inflammatory drugs which were considered accompanied by pneumonia. As antipyretic agents such as aspirin and acetaminophen are not effective in patients with heat stroke since they raise the core body temperature through different physiological pathways [2]. Within 6 hours after the onset, the patient maintained a high rectal temperature (39.6°C) arriving at our hospital, and the cooling and fluid infusion prehospital was seriously insufficient, resulting in heat accumulation and possibly abnormal thermoregulation. The preoptic anterior hypothalamus is the thermoregulatory control center of the brain. If it becomes damaged or dysfunctional after a heat stroke episode, it will hamper the ability to thermoregulate adequately. Some studies show that the heat-caused thermoregulatory deficit could be caused by ischemia and oxidative damage to the hypothalamus in the mouse [3]. Maybe it was one of the reasons why the patient remained at a high core temperature except for the pulmonary infection factor, which contributed to fever. However, the hypothesis that hypothalamus damage is responsible for thermoregulation loss has not been proved [2,4]. This case may provide some reference value. Decreasing the body temperature as fast as possible below 39°C is the crucial goal in therapy and it is critical to obtain this temperature quickly to avoid cell damage [5]. The primary objective is the alleviation of hyperthermia; thus, “cool and run” rather than “scoop and run” is the slogan, which is different from other traumatic conditions [2,6,7]. A recent study of the effectiveness of Ice-Sheet Cooling (ISC) provides a safe and effective alternative for the field where cold-water immersion resources may not be readily available [8]. Some studies report that in patients with severe heat stroke and multiple organ dysfunction, the early use of active intravascular cooling is an aggressive and

effective cooling technique [9,10]. Some cases show a sensible application although intranasal cooling for heat stroke with imminent cerebral edema, which is mostly known for post-resuscitation care [11]. In this case, though the ambient temperature was not that high, the humidity was high, and the air surrounding the surface of the body was saturated with water, sweat can't vaporize and cool the body surface [12]. Sweating can result in 2 liters or more of salt and water being lost per hour, which must be balanced by generous salt supplementation to facilitate thermoregulation [13,14]. Dehydration and salt depletion impair thermoregulation [14]. Thus, insufficient fluid supplement out of the hospital at the beginning of the patient impaired his thermoregulation, and his temperature was turned over and over.

The patient tolerated weaning as he successfully passed an SBT (Spontaneous Breathing Trail) from ventilatory support but failed extubation for 3 times. It sustained for 42 hours at the first time, as the need for re-intubation within 24–72 hours of a planned extubation are defined as extubation failure [15]. For the second time, it sustained for 15 minutes, as the subglottic tracheal foreign body was found through laryngoscopy, and the extubation failure was considered to be related to vocal cord paralysis. The endotracheal tube near the anterior division of the recurrent laryngeal nerve (the branch of the vagus nerve that provides motor innervation to the larynx) can cause vocal cord dysfunction. Injury to the vagus nerve or the recurrent laryngeal nerve can cause vocal cord paralysis [16]. The third time at 16 days after hospitalization, the patient could cough and expectorate spontaneously and the extubation was finally successful. The most common causes for re-intubation include respiratory insufficiency, airway obstruction, bronchospasm, residual neuromuscular blockade, residual effects of sedatives/opioids, and aggressive fluid administration [17]. In addition, it is necessary to evaluate the patient's ability to protect their airway, the strength of the cough, the amount of airway secretions, and their level of consciousness [18,19]. Airway parameters, such as cough strength and endotracheal secretions, can reflect the patient's ability to generate an effective cough to expectorate endotracheal secretions and maintain a patent airway related to weaning and extubation success [20]. Several studies have assessed that cough reflex has the potential to predict successful extubation in patients who pass an SBT [21]. More recent attention has focused on the Rapid Shallow Breathing Index (RSBI), defined as the ratio of respiratory rate to tidal volume (f/VT), which seems to be an important measurement to predict extubation outcomes [22].

Heat stroke is a life-threatening disorder and the main clinical feature is central nervous system dysfunction. Yang Mengmeng et al. [23] studied the clinical data of 119 patients with HS and found that all patients had different degrees of CNS damage, of which 79.5% had moderate to severe damage and 20.5% had mild damage. Most heat stroke patients' lives are saved, but 20% ~ 30% of patients are still left with permanent central nervous system injury [23]. Some in vivo studies show that hyperbaric oxygen treatment can effectively ease the multiple oxygen dysfunction induced by heat stroke, reduce brain neuron damage, and alleviate brain edema [24,25]. In this case, the patient received active rehabilitation exercise, special training for the neck muscle group, nervous system, limbs muscles, core strength, swallowing, sputum, and other physiological functions under the multidisciplinary team treatment including acupuncture department, rehabilitation department, and traditional Chinese medicine department in the recovery after extubation. However, the patient remained at lower limb muscle strength level 5-, poor coordination of limbs, and indistinct speech, and was prescribed with

hyperbaric oxygen treatment.

Hyperbaric oxygen may improve outcomes of heat stroke by improving hypothalamic and thermoregulatory function by reducing hypothalamic ischemia and oxidative damage in mice. After the onset of heat stroke, overproduction of iNOS-dependent NO in the hypothalamus was observed, and increased production of reactive oxygen and nitrogen species has been reported to be directly involved in oxidative damage with cellular macromolecules in ischemic brain tissues, which leads to cell death [3]. In this case, the effect of hyperbaric oxygen therapy was limited, and the patient's nerve function was not fully recovered. Song Qing, the team leader of our medical group, put forward the cluster therapy for exertional heat stroke, which is ten early and one prohibition. Ten early stands for cooling, early dilation, blood purification, sedation, early tracheal intubation, anticoagulation and coagulation, anti-inflammatory, enteral nutrition, dehydration, immune conditioning, and intestinal decontamination. One prohibition is surgical treatment would be prohibited. In this case, the patient's early clinical index is extremely poor, and survival is almost impossible. After our treatment, he survived.

In conclusion, heat stroke can be fatal, and brain damage can be transient or eventually result in irreversible injury. Meanwhile, heat stroke is a preventable illness, the Israel Defense Force used a heat tolerance test as a screening tool to prevent exertional heat illness in the Sahara and Arabian deserts [26]. Even if heat stroke occurs, appropriate situational awareness and effective countermeasures can greatly reduce mortality and organ damage. Timely management with rapid cooling and intravenous fluids is crucial to prevent irreversible and fatal organ damage. Among all Song Qing's slogans, the foremost is that first-responder action must be an effective and adequate cooling strategy. In this case, the prehospital cooling strategies were inadequate, and even with effective intensive care treatment, the patient survived and remained with deformity. With this report, we want to remind healthcare practitioners that early aggressive cooling strategies can save lives.

Conclusions

Heat stroke is a preventable illness, and thorough knowledge of the disorder can help to reduce mortality and morbidity. Timely management (the "golden hour") with rapid cooling and intravenous fluids is crucial to prevent irreversible and fatal organ damage. With this report, we want to remind emergency physicians or healthcare practitioners of the primary hospital that early aggressive cooling strategies can avoid EHS deterioration and even death.

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References

1. Bouchama A, JP Knochel. Heat stroke. N Engl J Med. 2002;346(25):1978-88.
2. Epstein Y, R Yanovich. Heatstroke. N Engl J Med. 2019;380(25):2449-59.
3. Tai PA, et al. Reduction of Ischemic and Oxidative Damage to the Hypothalamus by Hyperbaric Oxygen in Heatstroke Mice. J Biomed Biotechnol. 2010:2010:609526.

4. Cramer MN, et al. Human temperature regulation under heat stress in health, disease, and injury. Physiol Rev. 2022;102(4):1907-89.
5. Shapiro Y, DS Seidman. Field and clinical observations of exertional heat stroke patients. Med Sci Sports Exerc. 1990;22(1):6-14.
6. Demartini JK, et al. Effectiveness of Cold Water Immersion in the Treatment of Exertional Heat Stroke at the Falmouth Road Race. Med Sci Sports Exerc. 2015;47(2):240-5.
7. Yeargin SW, et al. National Athletic Trainers' Association Position Statement: Exertional Heat Illnesses. J Athl Train. 2015;50(9):986-1000.
8. Caldwell AR, et al. The Effectiveness of a Standardized Ice-Sheet Cooling Method Following Exertional Hyperthermia. Mil Med. 2022;187(9-10):e1017-e1023.
9. Bursey MM, et al. Successful Management of Severe Exertional Heat Stroke with Endovascular Cooling After Failure of Standard Cooling Measures. J Emerg Med. 2019;57(2):e53-e56.
10. Hamaya H, et al. Successful management of heat stroke associated with multiple-organ dysfunction by active intravascular cooling. Am J Emerg Med. 2015;33(1):124.e5-7.
11. Manegold R, et al. Effective intranasal cooling in an 80 year old patient with heatstroke. Am J Emerg Med. 2020;38(11):2488.e1-2488.e2.
12. Nelson N, LW Eichna, et al. Thermal exchanges of man at high temperatures. Am J Physiol. 1947;151(2):626-52.
13. Adams WC, et al. Thermoregulation during marathon running in cool, moderate, and hot environments. J Appl Physiol. 1975;38(6):1030-7.
14. Deschamps A, et al. Effect of saline infusion on body temperature and endurance during heavy exercise. J Appl Physiol (1985). 1989;66(6):2799-804.
15. Epstein SK. Decision to extubate. Intensive Care Med. 2002;28(5):535-46.
16. Ellis PD, WK Pallister. Recurrent laryngeal nerve palsy and endotracheal intubation. J Laryngol Otol. 1975;89(8):823-6.
17. Cavallone LF, A Vannucci. Review article: Extubation of the difficult airway and extubation failure. Anesth Analg. 2013;116(2):368-83.
18. McConville JF, JP Kress. Weaning patients from the ventilator. N Engl J Med. 2012;367(23):2233-9.
19. Perren A, et al. Protocol-directed weaning from mechanical ventilation: clinical outcome in patients randomized for a 30-min or 120-min trial with pressure support ventilation. Intensive Care Med. 2002;28(8):1058-63.
20. Smina M, et al. Cough peak flows and extubation outcomes. Chest. 2003;124(1):262-8.
21. Su WL, et al. Involuntary cough strength and extubation outcomes for patients in an ICU. Chest. 2010;137(4):777-82.
22. Baptistella AR, et al. Predictive factors of weaning from mechanical ventilation and extubation outcome: A systematic review. J Crit Care. 2018:48:56-62.
23. Yang M, et al. Outcome and risk factors associated with extent of central nervous system injury due to exertional heat stroke. Medicine (Baltimore). 2017;96(44):e8417.

24. Niu KC, MT Lin, CP Chang. Hyperbaric oxygen improves survival in heatstroke rats by reducing multiorgan dysfunction and brain oxidative stress. Eur J Pharmacol. 2007;569(1-2):94-102.
25. Tsai HM, et al. Resuscitation from experimental heatstroke by hyperbaric oxygen therapy. Crit Care Med. 2005;33(4):813-8.
26. Yanovich R, et al. Probability of Heat Intolerance: Standardized Interpretation of Heat-Tolerance Testing Results Versus Specialist Judgment. J Athl Train. 2018;53(4):423-30.

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