# THE MERCILESS SUN

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## SYNOPSIS

**A case of sun stroke with coma, high core temperature and watery diarrhoea is reported. The patient was an unfortunate victim who succumbed to the disastrous effects of a hot summer sun.**


## INTRODUCTION

Heat disorders embrace four clinical syndromes namely,

a. Heat stroke (sun stroke, Heat pyrexia or heat hyperpyrexia),

b Heat Exhaustion (Heat prostration or Heat collapse)

c Heat Cramps (miners cramps), and

d Heat syncopy

They cannot be compartmentalised due to considerable overlap among them.

Heat exchange of the body takes place by means of convection, radiation and evaporation and the co-efficient of these factors can be applied to calculate heat gain or loss, if the surrounding temperature, humidity, radiant heat, velocity of air movement and the rate of metabolic heat production are known.

Heat load on the body = Metabolic heat + Environmental heat.

The good old formula

$S = (M + C + R) - E$ still holds good where S stands for storage of heat, M for metabolism, C for convection, R for radiation and E for evaporation. S or Storage of Heat which represents heat gain or heat loss is zero as long as the body remains in thermal equilibrium. These physical properties are influenced by certain dynamic physiological effects like sweating and blood circulation.

## CASE REPORT

A twenty year old previously healthy male was found un-

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conscious at a running track on a summer morning after having completed seven out of ten kilometers of his exercise training programme.

On examination, he was found to be unconscious and his tight fitting uniform was soiled with sweat, urine and faeces. His rectal temperature was 41°C, he was dehydrated, had a tachycardia of 136 per minute, blood pressure of 110/60 mm Hg and his skin was dry due to cessation of sweating. He subsequently developed profuse watery diarrhoea and jerky movements of the limbs.

His haemoglobin was 8.5 gm per cent with a haematoerit value of 28. Total white cell count was 11,300 per c.m. with 72% polymorphs. Blood film for malarial parasites were persistently negative. Platelet count and clotting screen was normal except for a slightly prolonged prothrombin time. Urine examination revealed trace of albumen and casts. Stool was negative for vibrio cholera, vibro parahaemolyticus, salmonella, shigella and amoeba. Full liver functions tests, serum urea, creatinine, glucose and electrolytes were within normal range. ECG showed sinus tachycardia. Chest X-ray and cerebrospinal fluid examination including tests for acid fast bacilli and torula were negative. Prompt medication in the form of rapid body cooling by wet sheets and vigorous fanning, intravenous fluids, blood transfusion, parenteral chlorpromazine and ampicillin were instituted. The following day after forty eight hours of admission, he suddenly collapsed and did not recover despite heroic resuscitation measures. A post mortem was done and the findings were as follows:

> "Heart showed thin lymphocytic and mononuclear infiltrations in the interstitium with focal necrosis of myocardial cells. Lungs showed marked congestion with patchy bronchopneumonia and evidence of tracheobronchitis. Kidneys showed hydropic degeneration of the tubular epithelium with casts in the lumen. Liver showed normal architecture with centrilobular congestion and fatty changes. The brain was essentially normal.

## DISCUSSION

### 1 HEAT STROKE

It is a dangerous and often life threatening form of heat disorder due to continuous or excessive exposure to heat. There is an intrinsic breakdown of heat regulatory system whereby the central mechanism for cooling situated in the hypothalamus suddenly fail and the patient cease to sweat despite rising temperature. Susceptible population consist largely of the elderly people with degenerative vascular disease, young children, as well as soldiers with thick uniforms and athletes doing strenuous exercise without adequate prior conditioning. Consumption of moderate to large doses of anticholinergic drugs like atropine especially if combined with phenothiazines, high humidity, stagnant air and fever makes the situation worse. Congenital absence of sweat glands, anhydrotic ectodermal dysplasia and severe scleroderma with resultant difficulty to loose heat may drag the patient from frying pan to fire.

The patient may present with restlessness, irritability, drowsiness or giddiness. On the contrary, the onset may be sudden with fever, fits, delirium and coma or loss of consciousness may be the first mode of presentation. The syndrome is characterised by a high body temperature of at least 40.6°C and a rectal temperature more than 41°C (106°F) is not uncommon. Dilated pupils, suffused conjunctiva, hot dry skin with absence of sweating and flaccid muscles may be noted. Fatal cases may present with shock, cyanosis and sudden loss of consciousness.

Incontinence of urine and faeces and severe vomiting and diarrhoea simulating cholera may be called choleraic type of heat stroke.

Investigations may reveal leucocytosis, raised blood urea and diminished serum potassium. Clotting disorders in the form of prolonged bleeding time, clotting time, prothrombin time, thrombocytopenia and afibrinogenemia leading to disseminated intravascular coagulation and bleeding disorders may be seen. ECG may show sinus tachycardia, ST depression and T wave inversion or rarely evidence of myocardial infarction. Hepatic damage in the form of jaundice and enzyme changes as well as renal failure may occur one to two days after admission.

Differential diagnosis include all the causes of loss of consciousness including cerebral malaria and blood films for malarial parasites is mandatory in tropical countries. Potine haemorrhage is also associated with hyperpyrexia and coma, but then the pupils are pin point and lumbar puncture may reveal blood stained cerebrospinal fluid.

The prognosis depends largely on the speed with which therapy is instituted. In the choleraic type and in the comatose patient with temperature above 43°C (109°F) the outlook is grave. With prompt therapy, most patients will recover the acute episode. A few may die a few hours after

discovery and some may succumb several days or weeks later from myocardial infarction, heart failure, renal failure, complicating bronchopneumonia or septicemia. Autopsy may reveal widespread parenchymal damage and petechial haemorrhages in various organs including heart, liver, kidneys and brain. The overall mortality of heat stroke has been estimated to range from seven to seventy percent.

The aim of treatment is essentially to bring down the body temperature as fast as possible. If found on a roadside, place the victim under a shade, remove most of the garments and provide adequate ventilation including vigorous fanning, cover with a moist sheet, spray with cold water, apply ice to the head and neck if possible and transfer to the nearest hospital.

The best and most effective method of heat dissipation is to immerse the patient in a cold or ice water bath. Contrary to theoretical objections, an ice water immersion in this situation does not induce significant cutaneous vasoconstriction or shock. However, watch the patient constantly and monitor rectal temperature. Discontinue the bath when the rectal temperature reaches 38°C, but be alert to resume the same, should a febrile rebound occur. In some centres, gastric lavage with ice water and in desperate situations peritoneal dialysis with cold solution has been tried with success, but may be unnecessary. Rapid cooling of the skin may result in peripheral vasoconstriction and stagnation which may be corrected by gentle massage of the limbs. Metabolic heat production can be reduced by rest and sedation such as intravenous chlorpromazine 25-50 mg in 500 ml normal saline in an hour. Intravenous fluid regime should be started early to correct dehydration. IV normal saline 500 ml in first hour followed by 500 ml 3 to 4 hourly monitoring jugular venous pressure, lung bases, and urinary output or better still under central venous pressure line is essential. If acidotic give 4.2% sodium bicarbonate as required. If bleeding is a problem, plasma or fresh blood may be needed. Consumption coagulopathy necessitates heparin therapy and oliguria calls for peritoneal dialysis. Phenothiazines may be given to reduce shivering. When conscious and co-operative, saline drinks in the form of one to two grams of salt per pint of water flavoured with fruit juice orally or by nasogastric tube may suffice.

Prophylaxis consist of adequate sleep, and sufficient intake of salt and water prior to exposure to heat for a prolonged period. Those particularly susceptible must live in a cooler surrounding and must not wear tight fitting clothes. All activities must be limited to within safety level in a hot humid environment. It is equally important to restrict all physical activities as well as exposure to sunlight for a fortnight during the convalescent period.

## II HEAT EXHAUSTION

It is the most common form of heat disorder and it may affect both visitors as well as residents of hot humid tropical countries. There is a depletion of both salt and water resulting in decrease in blood volume with preservation of concentration of body fluids. It occurs insiduously or may be sudden with headache, fatigue, confusion, drowsiness, anorexia, visual disturbances and vomiting, which may lead to circulatory collapse. It occurs in heavy manual labourers, athletes taking part in long distance running or prolonged roving on a hot day. The skin may be cold and clammy and the pupils may be dilated. The patient is anxious and exhausted with tachycardia and hypotension. The body temperature may be normal or subnormal or it may be raised but not higher than 39°C. There is oliguria and the urine may contain albumen and casts. Shock and oliguria leading to fatal renal failure may be seen in severe cases.

Treatment consist of removal to a cool place, when spontaneous recovery usually takes place. Give 5 to 6 pints of water with 25-30 grammes of salt flavoured with fruit juice by mouth in the first 24 hours. In severe cases intravenous therapy and treatment of any concurrent infection may be needed.

Prophylaxis consist of maintenance of water and electrolyte balance in persons exposed to high temperature, even though the mechanism of heat exhaustion is not primarily a water and salt depletion.

## III HEAT SYNCOPE

Heat Syncope is a mild variant of heat exhaustion. It is a self limiting condition and it ranges from light headness to severe fatigue and transient loss of consciousness. Pallor, hypotension, elevated temperature up to 39°C, and flaccidity of the muscles may be observed. Removal to a cool place and rest in recombent position results in complete recovery in a few minutes.

## IV HEAT CRAMPS

It is the most benign heat syndrome and it occurs

in miners, furnacemen, ship firemen, stokers and engineers. It is the result of electrolyte imbalance alone and is often due to working at high temperature.and drinking large quantities of water without salt supplements. There is a dilutional effect on the body fluids simulating water intoxication. The skeletal muscles suddenly start fibrillating leading to very painful spasms. The episode usually occurs in the later part of a working day and some people regards it as an occupational hazard. Loss of chloride through excessive sweating is thought to be the cause.

The spasms start in the muscles of the calves and later may spread to the arms and the abdomen. The cramps are really painful and may last many hours. The body temperature is usually normal and direct exposure to the sun or high external temperature is not necessary. The spasms may be noticed during the attack. Haemoconcentration and decrease in sodium and chloride content of serum and urine are often seen. Treatment is essentially provision of sodium chloride by mouth or in severe cases by parenteral route. During acute episodes, analgesics including pethidine or morphine may be needed to combat painful spasms. Prophylaxis consist of sufficient intake of salt containing food or about 3 grams of extra salt per day. Limitation of water intake during work in a hot surrounding will certainly prevent cramps but may result in serious heat exhaustion or heat stroke which indeed is more disastrous than the cramps. Cramps involving abdominal muscles may mimic an acute abdomen, when adequate replacement of sodium chloride prior to an emergency laparotomy may prevent an unnecessary operation and will obviously save the reputation of an abdominal surgeon.

## CONCLUSION

The consequence of heat stroke include cerebellar ataxia, mental disorders, neuropathy and heat intolerance. In the tropics, the workers are inadvertently exposed to the combined bombardment of industrial and climatic heat. Healthy young adults including National Servicemen and athletes on special training programmes are not exempted from the sinister effects of moderate to severe heat wave especially before acclimatisation, if the state of previous health and the water and electrolyte requirements are not met with, by way of errors of commission or ommission.

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