

HEAT STROKE.

A study of the effects of Heat and Altitude  
on the inhabitants of Utrecht, Natal.

Thesis submitted for the

M.D. Edinburgh

by

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### Introduction.

During the thirteen years I have been in practice in Utrecht, I have had a good opportunity of studying the effect of heat and altitude, not only upon the aborigines but also upon a population of some two thousand white people, most of whom are engaged on arduous work.

The district of Utrecht is some 1,800 square miles in area. I have been the Government District Surgeon for the past ten years. The native population is roughly 40,000; the white population is 1,000 in the town, and 1,000 scattered throughout the extensive district. As District Surgeon, I have to deal with all epidemic diseases; all cases of faction fights amongst the natives; murders, assaults, rapes, in fact all the medico-legal work. In addition, I am the Government Railway Medical Officer, and have the care of the health of all the employees at the railway station at Utrecht, and the twenty-eight miles of rail extending from Utrecht to Newcastle. Further, I am the Medical Officer to the Utrecht Collieries, where I have the care of the thirty-two white employees and a compound of about six hundred native employees.

My work is very diverse and interesting. We have a considerable number of lepers here. Typhus is endemic. Syphilis, especially congenital, is rife; Phthisis, amongst the native miners who have previously worked at the

Rand, Johannesburg, snake bite and malaria, all form part of the daily routine and although sunstroke and its various manifestations is perhaps not quite so common, I have had to deal with a certain number and have also interested myself in recent literature on the subject.

The elevation of Utrecht, of about 4,000 feet above sea level, appears to give it the air of a mountainous region. The North wind is the prevailing one. Next in order comes the North-East and South winds of about equal frequency, and next, with comparatively little difference, are winds from the North-West and South-West. The rain fall average is about 23 inches per annum, and from April to September, there is practically no rain fall. The barometric pressure appears to be at its maximum in June, in which month the reading is 26.177 inches, and the minimum 25.849 inches, occurred in January. The heat experienced in Utrecht is sometimes excessive. When I state that the maximum summer heat of the day, in the shade, during the months of November, December and January, ranges from 99 to 103, and in the winter months June, July and August 30 degrees minimum, the contrast in temperatures may be gauged.

The dust storms to which we are liable all the year round are our greatest trial. They sweep over the country burning, blinding and choking up everything in their course. In the summer months we have hail storms with stones of

extraordinary size, 2 to 2 $\frac{1}{2}$  inches in diameter. Terrific lightning storms, and occasionally cyclones.

From this description it will be seen we experience snow and frost in winter, and tropical heat with its attendant ills and discomforts in summer.

Heat stroke is considered to be the effect of exposure to excessive heat, but opinions vary greatly as to the way the elevated temperature acts in producing the peculiar symptoms present. The most convincing and simplest theory of the causation of heat stroke appears to be rather a loss of control by the heat regulating centre than an increased production of heat. Haldane noticed that at a wet bulb temperature of 78, an individual in light clothing experienced a slight rise in temperature, during moderate exercise. When the wet bulb temperature reached 88, the body temperature rose without exercise, but a slight current of air prevented this rise; when the wet bulb reached 93, the body temperature rose irrespective of slight air currents. This brings out the relationship of atmospheric humidity to sunstroke. It was found that heat stroke occurred at a much lower mean temperature when a higher degree of moisture was present. If a higher degree of moisture is present, causing interference with the evaporation of perspiration, then heat stroke may occur with a maximum air temperature of just about blood heat.



Whatever occurs within the body to cause symptoms of heat stroke or heat exhaustion, exposure to external heat is the exciting cause. The skin either fails to function properly, or, because of the environmental physical conditions, is unable to transfer heat rapidly enough to prevent an increasing rise in body temperature, and as Sutton has pointed out, a vicious circle is established. With the rise of internal temperature, oxidation in the system is increased with the production of more heat, and an even greater rise in body temperature. In sunstroke cases, the patient may have been sweating profusely, but with the failure of the skin to transfer heat rapidly enough to prevent a rise of body temperature, perspiration often ceases. Continued generation of heat within the body is an essential feature of thermal fever, but in heat exhaustion, at a certain point the production of heat in the tissues ceases or is greatly reduced. Acidosis, which develops as damage to the organism proceeds, because of an insufficient supply of bases to neutralise the acid ions rapidly freed with the increasing rate of metabolism and accentuated through excessive loss of salts in drenching sweat, may be an important factor.

Definition and Symptomatology.

With regard to the definition of heat stroke, Dr. D.W. Reid has a useful article in the Boston Medical and Surgical Journal, October 26th, 1911, based on his not inconsiderable experience of 158 cases during a heat wave in Boston, U.S.A. in July, 1911. The temperature was not too high; on four days of the period, 2nd to 13th of July, did it reach 100°F. and 80°F. at midnight was frequently recorded. The humidity varied from 56 to 76., Dr. Reid divides the cases in, for want of better terms he calls (1) Heat Exhaustion, (2) Heat Prostration, and (3) Heat Stroke. This will be admitted to be a useful clinical classification. He describes the symptoms of each class as follows:

As regard class (1), the Heat Exhaustion cases, these patients commonly present a moist, cool skin with subnormal temperature, occasionally as low as 95 and 96, the pulse is small and rapid and the patient is very pale and prostrated, not really unconscious. This patient has usually been subjected to long continued high temperature, not necessarily in the sun, combined with physical exertion.

Class (2). The so-called Heat Prostration cases comprise the largest number in the series and with the lowest mortality on record.

This patient had a temperature varying from normal to

102 or even to 103 degrees.

Many felt dizzy, nauseated and complained of headache; a few lost consciousness, apparently not to be distinguished from syncope and lasting for a short time only, while practically all felt prostrated.

As stated above, many of these patients had a normal temperature, while of those with fever, the skin retained the moisture and there was an absence of cyanosis or lividity. The circulatory condition of this type was generally good, only a moderate stimulation being used on 30 out of 78 patients.

Class (3). Which we term heat stroke, is by far the most serious and impressive of the three. There were sixty-one of these cases with a mortality of 38. These patients were generally unconscious, livid, and often cyanotic with a hot, dry skin, and a temperature ranging from 104 to 110 degrees. Many axillary temperatures registered at 110 degrees. The more serious of these cases were breathing stertorously, frothing at the mouth, and some vomited large amounts of dark semi-fluid material, almost faecal in character. This type all showed Venous engorgement with visible carotid pulsations in the sides of the neck, and a full, bounding pulse, excepting in the moribund cases, where the heart was giving out. The pupils were generally small, often pin-point and not reacting

to light. Knee jerks were frequently absent, and a large majority showed considerable muscular rigidity.

Duncan, in Vol. 5 of his Journal of Tropical Medicine, classifies the condition under two heads: (1) Heat collapse and (2) Heat stroke. In Heat Collapse, the patient suddenly turns giddy and falls. The skin is cold, breathing stertorous and hurried, the pulse is soft and small, pupils dilated, the temperature normal or sub-normal, no loss of consciousness and recovery is the rule.

In Heat Stroke he divides the forms as follows:

(1) Occurs in persons unaccustomed to marching, and attacks them especially when the air is moist. There is violent headache and oppression, followed by convulsions, loss of consciousness, difficult respiration, small and irregular pulse and often incontinence of urine.

(2) Is characterised by excessive sweating, pallor, cyanosis, shallow breathing, injected eyes, swollen veins and partial collapse without complete unconsciousness. Revival occurs under proper treatment.

(3) In this form no fatigue is complained of, but the patient is thirsty and suddenly falls forward, comatose. The coma may last 24 to 36 hours, and ends in death.

(4) After exertion and exposure to the sun, a racking headache sets in. This becomes intense and finally agonising.



Great intolerance of light ensues, followed perhaps in 48 hours by unconsciousness. If death does not occur, the intense pain in the head may last from six to eight weeks unrelieved by any drug, but there may be slight evening remissions. It then gradually abates.

(5) In direct Heat stroke, this is the syncopal form occurring, not in the open, but in bungalows. Manson Bahr & Stitt divide the condition into three symptom complexes:

1. Heat Exhaustion (Heat prostration).
2. Heat Hyperpyrexia (Heat stroke, insolation, siriasis, thermic fever).
3. Sun Traumatism (Sun stroke).

In Heat Exhaustion there is sudden faintness, or fainting brought about by exposure to high atmospheric temperature. It resembles surgical shock, and is caused by loss of heat from the body being prevented. The onset is sudden, accompanied by collapse and semi-consciousness, which usually lasts about one quarter of an hour, and is rarely fatal.

The patient is pale, with a small, soft, sometimes fluttering pulse. The breathing is shallow, sometimes sighing, but never stertorous. The pupils are dilated, the skin is cold and the temperature is subnormal.

The treatment consists of loosening his clothes, placing him in a cool spot, and spraying the face. Avoid

douching him, as one wants rather to stimulate than depress. Apply Ammonia to the nostrils, and give brandy per os or rectum. An injection of camphor and ether in oil may be given. Usually after a short time, he recovers, may have a splitting headache, but is usually fit for duty in twenty-four hours.

In Heat Hyperpyrexia we have an acute condition developing in the presence of high atmospheric temperature, and characterized by sudden incidence of hyperpyrexia, stoppage of perspiration, dry skin, coma and extreme pulmonary congestion and oedema.

Distribution.

Heat hyperpyrexia occurs in deep valleys, deltas of rivers and along shores. It never occurs on the high seas. It occurs especially in the valleys of the Euphrates and Tigris. It is not necessarily confined to the equator. It occurs in the cities of North America, and especially New York, on the shores of the Red Sea, Australia, South America, Mexico and the Panama.

Etiology.

Heat hyperpyrexia has generally been attributed to direct action of atmospheric or solar heat on the body. Many theories of the modus operandi of this assumed cause have been advanced; among these may be mentioned super-heating of the blood by the high temperature of the surrounding atmosphere. Most

authorities agree that a process of auto-intoxication occurs, as evidenced by the indicanuria. Hearn noticed that sweating is suppressed from 1 to 48 hours before an attack of heat hyperpyrexia. With sweating suppressed, the body temperature rises until, when 108°F or more is reached, unconsciousness and convulsions develop. He suggests that the inhibition of sweating is local in the sweat glands, and not central, as diaphoretics fail to produce sweating once it has stopped.

Symptoms.

There is usually a prodromal stage, characterised by listlessness, drowsiness, vertigo headache anorexia, thirst, injected conjunctival vessels, suppression of sweating. Urinary irritability is a valuable sign according to Mansno-Bahr. Willcox has drawn attention to the loss of knee-jerks which occurs, and their return is a favourable indication. In other cases, the first indication of anything wrong, may be a short stage of restlessness, which rapidly passes into coma and high fever, which may reach 110°F. The pupils are usually contracted until just before death, when they, in common with the sphincters, relax. The dejecta and skin of the patient emits a peculiar mousy odour. The respiration is usually singular and may even become Cheyne-stokes in type.

Willcox distinguishes different clinical types of heat hyperpyrexia.

1. Gastric type - a deceptive form where the axillary temperature is normal - the rectal temperature raised and gastric symptoms with congestion of the liver predominates - fatal hyperpyrexia may develop without previous warning.

2. Choleraic type - sudden onset with purging and general resemblance to true cholera - many times fatal within two or three days.

3. True heat hyperpyrexia - in which nervous symptoms predominate. It accounts for 70% of the cases. Patients have recovered even after the temperature has been 113°F. (Marshall).

Gennaris divides the condition into three types:

1. Asphyxial, 2. <sup>Pa</sup>Paralytic. 3. Psychopathic.

Curative treatment is discussed later.

Sunstroke. (Sun traumatism).

The consensus of opinion is that sunstroke is heat stroke resulting from direct exposure to the rays of the sun. The actinic rays are held by some to be the cause of this condition and they, no doubt, are important in its production. The symptoms produced are not due to heat, for such effects do not result from exposure to the heat of a furnace, however intense. The usual case is characterised by severe headache, hot, dry, red skin, rapid full pulse, intolerance of light, sound and movement. Frequently nausea, vomiting and varying degrees of



delirium and unconsciousness are accompanying features. The acute phases may be quickly recovered from, or may prove very persistent and last for days or weeks. It may leave no injurious after-effects, or may be followed by certain morbid conditions such as loss of memory and various other mental and nervous disturbances, for quite a period of time.

Treatment.

The first consideration is to remove the patient to a cool place protected from the sun, preferably where there is a good circulation of air, in a darkened room. The head should be shaved and cold applied to the scalp. The bowels must be free, food light, and alcohol forbidden. Restlessness and insomnia are treated by bromides. For a time the patient will be troubled with loss of memory, feebleness of intellectual power and the faculty of concentration - he may be irritable, liable to headache and extremely sensitive to heat, more particularly the heat and glare of the sun. He should be removed to a cooler climate as soon as he is sufficiently recovered to be moved.

Curative Treatment.

The patient should be placed in cool surroundings, and hydro-therapeutic measures immediately adopted. Spraying with ice cold water and ice applied to the back of the neck and head. Rectal injections of ice cold water are of value. Should the hyperpyrexia not quickly subside, or should it not be readily controlled, an intramuscular or intravenous injection of a solution of quinine byhydrochloride grains 10, should be given. Blood examination should be made, and if these show malarial infection, the quinine treatment should be continued, daily injections being given for three to four days and afterwards an oral course. Convulsions are best treated by a venesection of ten to twenty ounces, and a rectal injection of ice cold water containing two drams of bicarbonate of soda to the pint. In cases with convulsions, it is not advisable to give intravenous saline after venesection for the intravenous injection may cause recurrence of the convulsions. In some cases morphine hypodermically, or inhalations of chloroform may be required. Cardiac failure should be treated by digitalin, adrenalin, camphor and ether, or pituitrin hypodermically. Strychnine should be avoided owing to tendency to cause convulsions in these cases.

Anti-pyretics should be avoided owing to cardiac depression. Failure of respiration should be treated by artificial

respiration and oxygen bubbled through alcohol may be administered. Lobelin may be injected and inhalations of CO<sub>2</sub> may be tried. Note. Occasionally retention of urine may take place, and require catheterisation.

Leonard Hill noted the inefficiency of the application of ice to the hyperpyrexial body as compared with evaporation. He found that water evaporation at body temperature abstracts 0.59 calories per gram, while melting ice only takes away 0.08 calories, and that 70 grams of water evaporated from the skin takes away as much heat as 1,000 grams of ice water as an enema. The application of ice constricts the capillaries and interferes with evaporation. The best effect from evaporation can be produced by directing the current of air from a fan on to the body covered by a wet sheet. The sheet may be wet by diluted alcohol. A thermometer should be kept in the rectum because when the temperature starts to fall, it may do so with great rapidity, and collapse may result. The application of cooling measures should be discontinued when the temperature drops to 103°F. The patient is then removed from the cold sheet and wrapped in a dry blanket, when very likely sweating, a favourable event, will begin. When the temperature has dropped too rapidly, and too much as a result of the application of cooling measures and the patient is in danger of collapse, the use of stimulants may be necessary. Chandler, as the result of his large experience,

recommended the injection of 40 minims of tincture of digitalis. Failure of respiration will require artificial respiration maintained for half an hour or longer. Lumbar puncture is indicated in cerebral cases as a rational method of relieving intra-cranial pressure. Gastric cases should receive a liberal supply of bicarbonate of soda - 30 grs. every two hours.

E.H. Cluver, Health Officer for the Union of South Africa, has an interesting paper under the heading, "An analysis of 92 fatal heat stroke cases on the Witwatersrand Gold Mines". During the period under review, the number of native labourers employed on the Witwatersrand Gold fields has been more or less 200,000. The natives are distributed over forty-two mines. The "miners" are all European, and their duties are supervisory. The shovelling of loose rock, pushing of truck-loads along rails, drilling with machines and other manual work, is done by the "boys", i.e. native labourers. It is only among the native labourers that fatalities due to heat injuries have occurred. The European miners occasionally collapse underground as the result of the heat, but so far no fatal cases have been recorded among them.

Of the 209,500 native labourers employed, only 24,000 are employed on mines, having a maximum vertical depth of workings of under 2,500 feet; the remaining 185,500 are employed on mines having a greater vertical depth of workings. The greatest depth



at which work is at present being carried on is 7,640 feet.

The increase in temperature on penetrating the earth's crust is here remarkably low, being only 1°F. for every 200-220 feet. In Great Britain, the increase is usually about 1°F. for 60-70 feet.

In mine 1. at a vertical depth of 7,500 feet, the temperature of the virgin rock is found to be 99°F. It is therefore, impossible, without some form of artificial refrigeration underground, to attain atmospheric temperatures more than a few degrees below the rock temperature. Nevertheless, these atmospheric temperatures would not be excessive from the physiological standpoint, if it were not for the factor of humidity. The methods used for laying silica dust for combating silicosis, involve the copious use of water, with the result that the air in the slopes where the hard work is done, is usually all but saturated. There is seldom more than a degree or two difference between the dry bulb and the wet bulb thermometric readings. Unless very free air movement is provided, wet bulb temperatures of 86°F or over throw a great strain on the cooling mechanism of the body, and strenuous work easily results in heat stroke in the unacclimatized. From enquiry made in the eleven mines affected, it appeared that some 14,000 natives are at present employed in atmospheres with a wet bulb temperature of 86°F. and over.

Symptoms.

The usual history is as follows. Towards the end of the first or second shift, the boy feels thirsty, and complains of being tired. Complaint of fatigue may be followed by obvious weakness, and the boy may stagger and fall down. In a number of cases maniacal running about and struggling now takes place, and the boy has to be forcibly strapped to the stretcher. Vomiting generally occurs. Occasionally the skin is described as hot and dry, but more usually at the subsequent enquiry, boss-boys and underground officials maintain that sweating was normal and profuse. By now, the condition is easily recognisable, and the boy is hurriedly taken to a place with good air movement, and lobelin may be injected by a European underground official. The transport of a patient from a working place to hospital takes time seldom less than thirty minutes, and as much as three hours may elapse before his admission to hospital.

In most of the fatal cases, the patient, on admission to hospital, is unconscious, with clonic contractions and twitchings of muscles, and an axillary temperature of 107-108°F. Usually he dies within a few hours of admission to hospital. Occasionally there is some improvement, death occurring some days later from lobar pneumonia. In these latter cases, the condition might conceivably have been one of impending pneumonia, precipitated

by the work in an atmosphere with a low cooling power.

The first death occurred in November, 1924. The monthly distribution of the fatal cases from that date until the end of July of the present year (1931) is tabled below.

<u>Month.</u>	1924	1925	1926	1927	1928	1929	1930	1931	<u>Totals.</u>
Jan.	-	-	1	1	2	2	3	4	13.
Feb.	-	-	2	-	-	1	5	4	12.
March.	-	-	3	2	-	2	3	-	10.
April.	-	-	3	1	1	-	2	1	8.
May.	-	-	1	-	1	-	3	-	5.
June.	-	-	1	-	-	1	-	1	3.
July.	-	-	-	-	-	1	-	1	2.
Aug.	-	-	1	-	-	-	1	-	2.
Sept.	-	1	1	-	1	-	5	-	8.
Oct.	-	-	4	1	1	2	2	-	10.
Nov.	1	-	-	1	1	5	2	-	10.
Dec.	-	2	-	2	2	3	-	-	9.
Totals	1	3	17	8	9	17	26	11	92.

The great preponderance of cases in the summer (September - March) months can be most readily accounted for on the assumption that heat stroke is an auto-intoxication due to the impairment of the excretory organs, such as the kidney and the liver, and the increased production of toxic substances at high

body temperatures. In the winter months the daily cold periods spent on the surface allow of the complete elimination of such toxic substances.

Tribal Distribution. With a view to ascertaining whether any of the tribes employed on the gold-mines showed special susceptibility to heat injury, the tribal distribution was examined.

Tribal Distribution of Fatal Heat Stroke Cases.

	1924	1925	1926	1927	1928	1929	1930	1931	<u>Totals</u>
Eastern Cape Province	-	1	3	3	5	5	10	5	32
Mozambique	1	2	6	2	3	4	8	3	29
Basutoland	-	-	5	3	1	6	7	3	25
Other	-	-	3	-	-	2	1	-	6
	<u>1</u>	<u>3</u>	<u>17</u>	<u>8</u>	<u>9</u>	<u>17</u>	<u>26</u>	<u>11</u>	<u>92</u>

As mentioned before the number of natives at present employed in the hot zones of the eleven mines from which fatal cases have been recorded, was found to be approximately 14,000; a hot area was arbitrarily chosen as one having a wet bulb temperature of 80°F or over. The tribal distribution of the 14,000 natives in these hot zones was found to be approximately,

Eastern Cape (Hosa, Pondo, Fingo, Baca)	4,200
Mozambique (Shangaan, Fongo, Myambaan, Inchopi)	5,100
Basutoland	2,900
All other tribes	1,800



If we express the total heat stroke deaths among each of these four groups in rates per 10,000 of the present population employed in the hot zones, we get

Eastern Cape	-	76
Mozambique	-	57
Basutoland	-	86
Other tribes	-	33

These figures suggest that of the three main recruiting areas for the gold mines, natives from the Portuguese territory are least susceptible to heat injury; the Transkeian territories come next, the Basutos showing the greatest susceptibility. This is in keeping with the climatic condition in these areas.

In Mozambique the conditions are tropical, with a hot, humid atmosphere; the greater part of Basutoland is situated at a high altitude, with a cold, dry climate; the climate of the Eastern Cape is intermediate between these two.

A similar result was indicated in a study made by Wakefield & Hall (Heat Injuries, Journal Am. Med. Assoc. July 9th, 1927) of heat injuries in the United States Navy. Their analysis suggested that the incidence of heat injuries was greater in those born and reared in the Northern states.

Among our natives, this result, was however, somewhat unexpected. It had been variously suggested that natives harbouring parasites, such as those of malaria and ankylostomiasis, would be more likely to suffer from a breakdown of the

heat-regulating mechanism. The boys from Mozambique are to a very considerable extent infested with such parasites.

These figures are an interesting contribution to the controversy regarding the desirability of recruiting natives from north of latitude 22° South. Whatever the other dangers such tropical natives would be exposed to by working in these mines, it would appear that in deep level working, they would have a considerable advantage over those recruited from further South, because of their greater resistance to heat injury.

#### Underground Atmospheric Conditions.

With only four exceptions, temperature readings were taken at all the working places concerned soon after the fatal case occurred. As mentioned before, there is seldom more than a degree or two difference between the dry and wet bulb readings. Of the points where fatal cases occurred, 67 were found to have wet bulb readings of 86°F and over, and 21 were under this figure. The lowest wet bulb temperature at any of these places was 82°F and the highest 93°F.

Wet bulb thermometer readings were obtained at all but 7 of these places. In 62 there were under 7.5 millicalories per square centimetre per second. In the remaining 23 it was over that figure, the highest being 11.1.

Acclimatization.

As shown above, the tribal distribution of these cases suggests a greater susceptibility on the part of natives born and reared in cold, dry areas. This may be looked upon as inherent lack of acclimatization. Analysis of the mortality figures brings out further the extreme importance of temporary acclimatization acquired underground. This is fully realized in all our deep mines. As far as practicable, efforts are made to allow the recruit to get acclimatized to the low cooling powers of the atmosphere at deep levels before putting him on to strenuous work in a heat zone. He is usually required to serve a probationary period of a fortnight in a specially supervised gang, where over-exertion is prevented for example, by issuing only one shovel for two boys. Complete supervision and prevention of excessive effort is, however, impracticable, because of the conditions obtaining underground, and collapses occur both during probation and immediately after, as shown in the following table.

Fatal cases of Heat Stroke classified according to the number of shifts worked in a Hot place.

	<u>Shifts in Hot Place.</u>					Total cases.
	1st.	2nd.	3rd.	4th.	More than four.	
New Boys.	19	15	7	2	4	46
After Probation.	10	4	2	1	4	21
After Hospital.	4	3	1	1	1	10
Others.	<u>7</u>	<u>3</u>	<u>1</u>	<u>1</u>	<u>3</u>	<u>15</u>
Totals.	<u>40</u>	<u>25</u>	<u>11</u>	<u>5</u>	<u>11</u>	<u>92</u>

It will be seen that 34 of the boys were serving their first or second shift underground at the time of collapse. Most of these were in probationary gangs. The inadequacy of the spell, of probationary work is revealed in the case of 21 boys who collapsed shortly after being transferred from probationary gangs to more strenuous work in hotter working places. A period spent in hospital might render a boy susceptible to heat injury, either by removing temporarily acquired acclimatisation or because of incomplete recovery from a febrile condition.

Most of the cases are seen to have collapsed during their first four shifts worked in a hot area, of the eleven who collapsed after that period, the evidence in some suggests that they might be attributable to a temporary increase in effort or lowering of the atmospheric cooling power.



Summary.

The 92 fatal cases of heat stroke occurred in eleven of the gold mines of the Witwatersrand; 39 of them in three mines with a vertical depth of over 6000 feet.

Although there is little seasonal variation in deep-level atmospheric temperature, the great bulk of the cases occurred in the summer months. If the pathological condition in heat-stroke is accepted to be primarily an auto-intoxication, the paucity of cases in winter can be accounted for by the daily periods spent on the surface, allowing of elimination of toxic substances.

Relatively, most cases occurred among natives recruited from the highlands of Basutoland. The Eastern Cape recruiting area, with its milder climate, comes next, whilst tropical Mozambique shows the lowest rate. The degree of acclimatisation to low atmospheric cooling powers would thus appear to be of importance. Of the 92 cases 67 occurred in working places in which the wet bulb temperature reading was 86° F., or more; 21 were under this figure.

Of the 85 cases where wet Kata thermometer readings were taken, at the working place, the reading was under 7.5 in 62 of the places. In all but 11 of the 92 cases, collapse occurred during the first four shifts worked in a hot place.

Differential Diagnosis.

The coma of ~~ur~~raemia, diabetes, and drugs (morphia and alcohol) is known by the absence of high fever. In the coma of cerebral-haemorrhage into the pons, fever may occasionally be present, but it would not precede the onset of coma. The comatose form of malaria is recognised by finding the parasite in the blood and an enlarged spleen. Cerebro-spinal fever, may be recognised by the occipital retraction, the irregular pupils, frequent occurrence of strabismus, Kernig's sign, comparatively low and fluctuating temperature, the associated herpes, the initial rigor and its duration.

Pathology.

Rigor mortis occurs early and putrefactive changes set in within a few hours after death. Oedema and general hyperaemia of the brain and lepto-meninges occur and the nerve cells and the grey matter show marked degenerative changes. Petechiae occur in the skin and mucous membranes in severe cases; the right side of the heart is dilated and venous congestion is marked in all the organs. Signs of intercurrent diseases such as malaria and typhoid may be present. It has been stated that heat stroke is due to bacterial infection, but there is no recent evidence to support this view. In the Mesopotamian campaign a large

number of blood examinations were made during life in cases of heat stroke, but all gave a negative result.

Cayicol and Lapierre (Montreal Clinic 1898) found a micro organism in the blood of patients suffering from heat apoplexy and regarded it as the specific cause of the disease, but these findings have not been confirmed by later workers. The symptoms of heat stroke in its various forms all point to an auto-intoxication. The affect of high temperature on the brain and the central nervous system is to diminish its functional activity and likewise the excretory organs such as the liver and kidney are undoubtedly impaired in their excretory power. In addition, owing to the effect of heat on the muscles and tissues and the body generally there must be an increased production of toxic substances due to protein katabolic changes. These factors all tend to produce a marked auto intoxication which explains many of the symptoms of heat stroke. Indican is usually present in the urine in considerable excess and is evidence of an auto intoxication, Acetone and Diacetic acid are not found in appreciative excess in the majority of cases, though in a small percentage they are present in a moderate degree.

It has been shown by Dr. W. Cramer that Beta-tetrahydro-naphthylamine will cause hyperpyrexia in animals and it

is likely that substances having a similar action are produced as a result of the heat on the tissues. Suppression of sweating has been shown by Dr. K.G. Herne to be an important factor in the hyperpyrexia cases. The paralysis of the sweat secretion by the intense heat, leading to the rapid rise of body temperature. In hot climates the administration of atropine may be followed by hyperpyrexia from a similar cause. Suppression of sweating does not explain the causation of the gastric and choleraic type of heat stroke nor heat exhaustion.

Pathology (contd.).

A notable feature of fatal hyperpyrexia is the early appearance of rigor mortis. The blood is remarkably fluid and dark and pours from the phenomenally engorged lungs and other viscera on section. The red blood corpuscles are said to be crenated and do not form rouleaux. If the examination is made immediately after death, the heart, particularly the left ventricle will be found remarkably rigid, this rigidity is sometimes described as being of "wooden hardness". Microscopic examination shows necrotic changes in the ganglion cells, with chromatolysis of the nuclei. The cerebro-spinal fluid is clear and under



pressure. Cortical changes in the supra-renals have been described. The temperature of the cadava continues to rise after death and may reach 114°F.

Heat hyperpyrexia tends to produce muscle tetany and there seems to be sufficient clinical and pathological evidence to show that tetany of the heart muscle, which is affected by tetany along with the rest of the body musculature, is the immediate cause of sudden death in heat hyperpyrexia. The pathogenesis of this tetany appears to be incident to the profound metabolic disturbances resulting from thermoregulatory decompensation. An uncompensated acidosis with lowered p H value occurs in heat stroke. This may be due to some single acid body, Hall and Wakefield attribute the alterations found in heat stroke to lactic acid which was increased in the blood stream to three times normal in their experiments.

#### PREVENTIVE TREATMENT and ACCLIMATISATION.

Adequate protective covering for the head by a good cork helmet is essential, spinal pads for the protection of the spinal cord are valuable. The clothing should be light and loose and not too thin. A large amount of water should be drunk by those exposed to great heat as so much moisture is lost by the skin in regulating the body temperature.

Alcohol is to be avoided especially during the heat of the day. Also too much exercise and excessive food. Constipation is to be avoided and all errors of refraction should be corrected tinted glasses are of great value. New-comers and Europeans are more liable than natives or residents of long-standing. Men over forty are more susceptible than those of younger age. Apparently long residence, confers a relative though not an absolute immunity. The most important factors in determining whether or not man can adapt himself to a given climate, are temperature and humidity. Schilling holds that the equable temperature of the tropics may only affect the health of the white man adversely because it is almost always associated with a high degree of humidity. This moist heat renders the tropical resident very sensitive to relatively slight changes of temperature which would be readily borne in dryer climates.

Many writers have stated that a rise in body temperature of from  $0.5^{\circ}\text{F}$  to  $1.5^{\circ}\text{F}$ , the actual amount dependent upon the season of the year, occurs upon a change of residence from a temperate to a tropical climate. This elevation of body temperature is most noticeable in a white person during the first few days or weeks of tropical residence, but is not observed while the person is at rest. Other authorities have failed to confirm these views, amongst these are Castillani and Chalmers who have failed to show any change in the bodily

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temperature during residence within the tropics, or in passage to and from, provided the individuals observed were normal.

Body temperature is regulated largely by evaporation of water from the surface of the skin. As the brown skin absorbs more heat than the white skin, the point where perspiration shows is reached more rapidly by the brown skin than by the white skin under similar conditions, indicating that the regulatory apparatus of the brown is more sensitive, and works more promptly and successfully than the white skin. While the white man is perspiring profusely, there is only a fine layer of very small sweat drops on the skin of the brown man. As the latter cools even more rapidly than the white skin, it is evident that the determining factor is the invisible sweat, or the water evaporated, and not the water secreted, which is of value. Hypersecretion of sweat, is water lost from the body without the corresponding cooling effect. It is useless and deprives the body of water. The sweat secretion of the white man in the tropics goes beyond the limits of efficient heat regulation and becomes superfluous, so that the skin is moist, and this with the warm moist air increases the frequency of bacterial and mycotic skin infections, and ~~is~~ ~~called~~ ~~heat~~ ~~rash~~.

Eijkman found no perceptible difference in the number of

sweat glands between the brown and the white race, but counts made by Clark show that the darker skins contain a larger percentage of sweat glands than the lighter. Persons who do not perspire or who have a defective sudorific system are not suited for residence in the tropics.

Following the experiments of Aron, Shaklu (1917) showed that some acclimatization occurred in monkeys, after two days exposure to the sun, that the maximum was reached after two months, and that the temperature of a thoroughly acclimatized monkey exposed, in contact with a hot roof, to the sun day after day, rarely exceeds its normal maximum. While the sweat glands of monkeys are not so well developed and efficient as in man, Shaklu noted that there is a more copious secretion of sweat by the acclimatized monkey than by the unacclimatized one, when placed on a hot surface in the sun; that the effects of heat were inversely proportional to the amount of sweating, and that a small dose of atropin would cause the death of an acclimatized monkey by stopping the perspiration. He also noted that some forms of sickness, an unsuitable diet, and absorption of toxins from the intestine rendered the animals more susceptible to the effects of heat.

It is an error for a person to expose large areas of the body to direct rays of the sun in the tropics, as is often practised in the temperate zone for the purpose of producing



tanning or pigmentation of the skin. Excessive exposure by persons with fair complexion and very little pigment in the skin has been followed by constitutional symptoms such as chills, nausea, vomiting and exhaustion.

Pigmentation of the human skin has been said to be a sign of adaptation to special types of environment. According to Leonard Hill (1920) the primary function of pigment is to arrest excess of sunlight, which has a harmful effect. This is shown by the fact that the blackest races are to be found in dry sunny climates, with much sun and glare from the ground. Hill has remarked that climatic adaptation cannot depend merely upon pigment, for a very moderate pigmentation, such as the yellow of the Mongol, or the sunburn pigmentation of the white person, suffices to prevent the harmful effect of the sun. Moreover the pigmented Eskimo is not fitted by his pigment to stand a tropical climate. Accordingly there appears to be correlation between the metabolism and heat losing mechanism, which adjusts the race to the climate it has been evolved in.

From the general point of view acclimatisation may be construed as a common-sense adaptation, individual or racial, of body and mind, to unusual conditions of life, and the prevention or control of such conditions, which may produce consequences injurious to health and life, through measures

consistent with the performance by a normal man of ordinary physical labour irrespective of climatic conditions.

Therefore acclimatisation of natives living in the tropics may be assumed to be complete. With white races whose habitat is outside the tropics the problem is different, and whether they can exist, labour, thrive, and propagate in a tropical country has always been a debated question. The traditional and empirical view held by many, appears to be that it is very doubtful if the white man can perform manual work in the open under tropical conditions such as exists in the coastal plains and that, if he attempts to do so, he will assuredly degenerate.

Eijkman states that for more than three centuries there has been a continuous stream of hundreds of thousands of people going from Europe to oversea lands, and that everywhere, outside the torrid zone, where winter and summer alternate, the white race has gained a firm foothold, and eventually supplanted the native. In the hot regions, however, the thoroughbred white man has nowhere advanced further than to an aristocratic minority. He further states that whereas the white races dominate nearly everywhere in the temperate zone, in the tropics they advance no further than a numerical minority, and this proves that the native race can engage with more success in the struggle for life, and that the

superiority of the white race is only a relative one. Where the native population was thin-sown, it has been necessary to import men from elsewhere, negroes, Indian Coolies, Chinese and others who are able to perform hard toil in a hot climate in order to supply the urgent need of labour.

In direct opposition is the view that the ill effects of tropical residence are not due to the climate alone but to preventable diseases of protozoal, helminthic or bacterial origin, and that the insanitary conditions usually prevalent throughout the tropics offer exceedingly favourable opportunities for their propagation and spread. Manson Bahr states that the healthy human body when untrammelled by unsuitable clothing, when not exhausted by fatigue or excess, when not clogged by a surfeit of food, by alcoholic drinks or drugs, can support with impunity very high atmospheric temperatures.

Experience in the Panama Canal zone, in the Phillipines and other insular possessions of the United States, where modern sanitary and preventive measures are observed, indicates that those who are adaptable to the climate, who adopt reasonably satisfactory habits of life, remain free from tropical infections, maintain industrious habits, and keep steadily and properly at work, may be as healthy as in a

temperate climate and attain normal longevity. Those who are not adaptable to their environment or show indications of myelosthenia or other serious illness, should return without delay to a more agreeable climate.

Whatever proves to be the future for the tropics, whether the white race in the coming years settles these lands, whether they will come under an unprogressive native control, or whether coloured populations will be able to govern themselves, or be successfully organised under white leadership, protection against disease will always be a fundamental necessity. A tropical belt abandoned to a negligent native regime would be such a menace to mankind that concerted health measures would be inevitable.

The following are notes on some of the cases treated by me typical of all the others:-

G. - D. - A farmer aged 30, had been ploughing all day. That evening he complained of head-ache, which became increasingly severe; he also complained of great thirst. When I was called out to see him he was unconscious. His skin was hot and dried; his temperature was 103,; his pulse 106, and his respiration was quickened. He was treated according to the hydre-therapeutic measures outlined by me under the heading of curative treatment. His temperature fell to 101 and he made a complete and rapid recovery.



Mrs. A - M - , aged 47 years, had attended a bazaar on a hot summer morning held in a local hall, which was very crowded and poorly ventilated. She complained to her friend that she felt giddy and nauseated and shortly afterwards suddenly collapsed. When I saw her she was unconscious, her breathing was of a shallow and sighing nature. The pulse was irregular, small and soft. The pupils were dilated, the skin was cold and clammy. The temperature was subnormal. I had the patient removed to a cool dark room, all tight clothing was loosened. An injection of camphor and ether in oil was given, and hot water bottles were applied to her feet. Smelling salts were held to her nose, and she slowly regained consciousness. She still complained of a splitting headache and felt nauseated. The next day she had made a complete recovery, her temperature and pulse were normal, and she was free from headache.

Mr. D - B -, a farmer aged 35 had been assisting his boys in reaping lucerne. They worked all morning, at twelve o'clock they rested, and his wife brought him some lunch, but he would not take any as he said he had a bilious headache. He asked his wife to send him a drink of milk. He started work again reaping in the hot sun, and an hour later a boy came from the house with a drink of milk for him.

He drank this. Shortly afterwards the boys noticed him vomiting and he then collapsed. They carried him home and his wife put him to bed and sent for me. When I examined him I found his skin hot and dry, his temperature was 106, his pulse was 120, full and bounding, His breathing was stertorous and the pupils were contracted, he was delirious.

I had the patient placed on a mat, wrapped him in a sheet soaked with cold water to which brandy had been added and fanned him. Camphor and ether in oil was injected, also Digitalin, but the temperature remained at 106, and despite all possible treatment, the patient died three hours later. His temperature then was 107 despite all our efforts to bring it down. Note there was no ice available, and the water was merely cool not cold.

Though the wife was assured that her husband had died of sun-stroke, she was suspicious that the native boy had put poison in the milk he had taken to her husband, and she asked me to do a post mortem.

When I commenced to do the post mortem, the temperature was still 106°, and there was already a certain resistance in the limbs, which heralded an early onset of rigor mortis. The lungs were engorged with dark fluid blood which drained away readily. All the organs showed signs of venous congestion; the right side of the heart was dilated and of particular interest was the rigid hardness of the left ventricle. No signs of poisoning could be found.

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Additional Notes.

A. O. Dreosti, in his article on Pathological Reactions produced by work in hot and humid environment in the Witwatersrand Gold Mines, states that work in hot and humid environment produces a rise in body temperature, an increase in the rate of the heart beat, a fall both of systolic and diastolic pressure with an increase in pulse pressure and a considerable loss in weight due to sweating.

These physiological reactions vary greatly in degree in different subjects, some being naturally more tolerant to work in heat and humidity. Thus, some subjects showed a remarkable ability to control rise of body temperature, whereas in others the temperature rapidly rose to a hyperpyrexia. The increase in pulse rate also varied greatly in different subjects - some reaching over 160 beats per minute. The blood pressure changes showed similar variation, more particularly the fall of diastolic pressure. The amount of weight lost as the result of sweating varied from a few ounces to 4 lbs. after one hour's work in the experimental chamber. There was no correlation, however, between the loss in weight and the rise in body temperature. The amount of sweat produced appeared to be proportional to the original body weight of the subject.

An interesting observation was made during these investigations in regard to the pathological effects of work in such atmospheric conditions. Several subjects had to be



removed from the experimental chambers owing to collapse. In these cases there was a typical picture of cardiac syncope although in no case was the temperature of a hyperpyrexial nature. On the other hand many natives whose temperature had risen to 104° F. or over, felt no ill effects and appeared to be perfectly happy. These natives obviously were not suffering from cardiac embarrassment but rather from an inefficient control of rise in body temperature as compared with the great bulk of the subjects investigated. It will, therefore, be observed that two different conditions appear to be produced by work in hot and humid environment. Firstly, there is the condition when the subject collapses owing to cardiac syncope; secondly, there is the condition of inability to control body temperature as compared with the average subject.

1. Heat Collapse. (Heat Exhaustion or Heat Syncope.)

This condition is due to cardiac syncope and is brought about by exposure to, or work in, atmospheres with a low cooling power and is often accompanied by abnormal bodily health.

Aetiology.

The mine labourer, whose health is impaired by disease, is more likely to develop "heat collapse" when working underground. Even slight indispositions such as an empty stomach, an over-loaded stomach, alcoholic indiscretions, constipation, lack of drinking water, or any other cause

which temporarily lowers the vitality of an individual may precipitate an attack. Serious disease has frequently been observed in many of such cases admitted to hospital. Of 205 admitted between January, 1931, and January, 1934, 96 were suffering from a disease diagnosable on admission. Diseases found included pneumonia (lobar and broncho-(in all stages, enteric fever, dysentery, other intestinal affections, old cardiac lesions, malaria, chicken pox, multiple boils, acute tonsillitis, etc., etc. Pulmonary affections, the various forms of enteritis, and intestinal parasites such as tapeworm, roundworm and even hookworm, were the commonest maladies found in these cases. In some instances evidence of disease does not manifest itself for several days after the occurrence of heat collapse underground.

#### Symptomatology.

The symptoms are cardiac in type, i. e., those of fainting. The recognised symptoms and signs of fainting are sudden weakness, giddiness, pallor, weak pulse, cold clammy skin and very often a transitory period of unconsciousness. The same condition occurs underground. The native while at work becomes giddy or feels faint; he sits down or tries to stagger to some cooler part of the working place. His temperature is normal or subnormal but may be above normal if collapse occurs with an acute disease. The skin is cold and moist, and there is a weakening of the pulse. Unconsciousness may supervene as the result of this cardiac embarrass-

ment. Collapse may occur at any period during the native's contract from newly recruited to fully acclimatized natives.

2. Heat Stroke. (Heat-hyperpyrexia, Thermic fever, Siriasis.)

This condition involves acute mental excitement, delirium and convulsions, associated with a high body temperature and occurring in un-acclimatized natives exposed to underground atmospheres of low cooling power.

Aetiology.

Taylor (1919) found, in Mesopotamia, that air temperatures of over 110° F. dry bulb and 85° F. wet bulb produce heat-stroke. Apparently the critical shade temperature, where the humidity is low, is in the region of 119-120° F. (Morton, 1932). According to Pembrey (1913, 1914,) heat-stroke can also occur in temperate climates from improper clothing and strenuous work. Europeans working in dry mines in which the dry bulb temperature varies between 100.4° and 102.2° F., do not develop heat-stroke (Strauss and Schwarz, 1932). On the Rand mines, in which the air is practically saturated with moisture, heat-stroke occurs at temperatures of about 89° F. wet bulb and 90° F. dry bulb. The wet kata thermometer readings of the working places in which heat-stroke has occurred vary between 3.5 and 7.5, the average being about 5 m.c.

It must be remembered, however, that these readings do not take into consideration such important factors as individual output of work, movement of air created by muscular

work, acclimatisation and individual susceptibility. Shattuck and Hilferty (1932), in their review of mortality from "Heat Cases" in the United States from 1910 to 1928, came to the conclusion that industrial conditions and occupations were correlated with incidence. Similarly, the fact that the death rate from heat effects in states having a large coloured population is 2.4 times as high for coloured as for white, suggests a correlation with occupation. The type of work, therefore, is an important aetiological factor in the causation of heat-stroke.

On the Rand mines the native labourers are those most violently exposed to the pathological effects of heat, in the sense that all the most strenuous manual labour is performed by them. It is also significant that heat-stroke occurs amongst the natives employed on work requiring a great expenditure of energy. Such forms of work as "tramping" (pushing trucks) and "lashing" (shovelling rock) most frequently predispose to heat-stroke. It must be remembered that, with training, the amount of energy required will gradually decrease, which in consequence will decrease internal heat production. This lack of training in new natives together with the abnormal atmospheric conditions taxes the native's power of endurance to the maximum. It is not surprising, therefore, that the new un-acclimatised natives are the ones susceptible to heat-stroke.

The fact that the limit of heat endurance is associated



with a deranged heat-regulating mechanism is evidenced by the accompanying high body temperature. Hyper-pyrexia alone, however, cannot be the only aetiological factor since, in many cases which terminate in death, the rectal temperature never exceeds 107° F. Nine such fatal cases were observed in the series admitted to the City Deep hospital and reviewed here. Further, numerous cases of extreme hyper-pyrexia (over 110° F.) have been recorded in medical literature. Yet in these cases no evidence of heat-stroke was observed.

The responsible agent for precipitating an attack of heat-stroke is still not settled. Wilcox (1920) believes it to be an autointoxication. Hearne (1932) holds that suppression of sweat is primarily responsible. Other workers have concentrated on the blood chemistry of this condition. Hall and Wakefield (1927), as the result of their investigations of experimental heat-stroke in dogs, found a three-fold increase in the blood lactic acid. Marsh (1930) obtained similar results in animals in which heat-stroke was produced experimentally. Cramer's (1928) work shows that the thyroid-adrenal apparatus is intimately associated with body heat regulation, hyperpyrexia and heat-stroke.

Observations made of the physique of natives did not reveal any conclusive data. Mavrogordato and Pirow (1927) formed the opinion that the native with a poor physique is more liable to develop heat-stroke than another with a better

physique. Cluver (1932), on the other hand, states that the native affected is usually a "robust" young man of about 25 years of age."

All mine natives are weighed at least once a month and their weight is recorded on a special card. By this means it was possible to obtain the weights of the natives in whom heat-stroke had occurred. Only the weights of the cases that occurred on the City Deep mine could be obtained (46 cases). The data show one fatal case in a native of 107 lbs. body weight; another in a native whose weight was 175 lbs. The remainder of the cases had weights between these two extremes, the average being 138 lbs. This is also the average weight of all the underground native labourers.

The average age of the cases ranged between 25 and 35 years. The extremes of ages employed in the industry is 18 to 45 years. Heat-stroke, therefore, occurs in the normal well-built individual of average age, i.e., an individual capable of doing a hard day's muscular work.

Climatic conditions on the surface can be definitely correlated with heat-stroke incidence. Cluver found the great bulk of cases occurred during the South African Summer months. Our observations confirm this finding. The seasonal variation of temperature on the City Deep mine is less than 2° F. and the humidity shows no appreciable change. Apparently, therefore, even such slight changes in the underground atmospherec temperature are sufficient to upset the

balance between conditions that can be tolerated and those that produce pathological effects.

#### Symptomatology.

The prodromata described as usually preceding an attack of heat-stroke are weakness, frequency of micturition, restlessness and dryness of the skin. Hearne (1932) has been able to detect suppression of sweat as long as 48 hours before definite signs of heat-stroke become established. By anticipating the attack, immediate preventive measures can be instituted. These prodromata were not observed on the Rand, even by the European miners who were in charge of the natives and who were instructed to be on the look-out for them. This is also borne out by natives who recover from heat-stroke and from whom no history of these prodromata can be obtained. The onset of heat-stroke on the Rand is definitely more sudden in its onset and more acute in its course than that occurring in hot climates.

Wilcox (1920) describes three clinical types, namely, the "Gastric type," the "Choleric type," and the "Hyper-pyrexial type," which is accompanied by nervous symptoms. The type of heat-stroke admitted to this hospital corresponds to Wilcox's hyper-pyrexial type in which the nervous symptoms predominate.

#### Onset.

A typical description of the onset in such a case is as follows:- The native usually of good physique, while working

and without any premonitory signs or symptoms, suddenly develops a condition of acute mental excitement or wild delirium. He shouts and rushes about blindly, struggling violently if any attempt is made to restrain him. He becomes a danger both to himself and to those who are trying to control him. The European miner in charge of the native will report that the native was working quite normally and "suddenly went mad, requiring four or more men to control him in order to strap him on to the stretcher for transport." In some cases the onset is accompanied by a condition very much resembling extreme fear.

After recovery the native cannot recollect the onset of the attack. He remembers the work he was employed on and the events up to the time of the attack; apart from the heat, he did not feel any discomfort or premonition of impending danger. This condition of mental excitement is soon followed by muscular tremors going on to generalized muscular twitchings and often convulsions of an epileptiform type; coma supervenes and finally death may occur in the comatose condition. From the commencement of the attack to death is merely a matter of hours and in some cases death occurs while the patient is being transported to hospital.

On admission the patient may be in any of the conditions mentioned above.

Temperature.- The rectal temperature varies in different cases, sometimes reaching as high as 110° F. Occasionally,



owing to the wild excitability of the patient, it is impossible to take any temperature until after some form of sedative has been administered hypodermically. Table 1 shows the temperatures, on admission, of the cases which terminated fatally.

TABLE I.

Rectal Temperatures of 31 Heat-Stroke Cases  
on Admission to Hospital.

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Cases.	Temp. (°F.).
1	102
1	103
2	104
2	105
3	106
22	107 to 110

Not all cases that terminate fatally have, therefore, a true hyper-pyrexia.

Sweating.- The skin is hot and dry and there is no evidence of visible perspiration even when the patient is struggling violently or when convulsions are present. One of the first signs of recovery, however, is the appearance of perspiration.

The suppression of visible sweating and the onset of heat-stroke appear to be simultaneous, as no dryness of the skin prior to the attack has been observed underground. In view of the rapid return of normal sweating with recovery, the suppression during the attack cannot be caused by any serious damage to the sweat glands or depletion of body fluids.

Circulation.- The pulse is slow and full but, with the onset of coma, it becomes progressively weaker and faster, finally

disappearing completely at the wrist. The blood pressure does not show any profound changes. Both the systolic and diastolic pressure before the onset of coma are slightly lower than would be expected from the native's age. In view of the frequency of comparatively "low" blood pressure in native mine labourers and also the fall in blood pressure that occurs with a rise in body temperature, the changes in the blood pressure in heat-stroke cannot be regarded as a characteristic feature of the condition.

Urine.- There is incontinence of urine and faeces. The urine is acid in reaction and apart from slight traces of albumin occasionally found, is negative. Microscopical examination of the centrifugalised deposit shows a few spheroidal epithelial cells and a few granular casts. Cultivation gives a negative result.

Cerebro-spinal Fluid and Blood.- The cerebro-spinal fluid is clear and under pressure. Microscopical examination shows nothing abnormal. Globulin is not present in excess and quantitative estimations of sodium chloride and sugar are normal. Cultivations yield no growth. The Wassermann reaction, both of the cerebro-spinal fluid and of the blood, is negative. Blood smears, with the exception of one case in which a few malaria parasites were found present, are negative. Blood cultures are also negative. These examinations together with the absence of any clinical signs of co-existing disease are confirmed by by macro- and micro-pathological examination post-mortem. Co-existing disease,

therefore, cannot be regarded as a predisposing factor in heat-stroke.

Prognosis.- Apparently the death rate from heat-stroke varies in different parts of the world. Castellani (1931) puts the average death rate between 15% and 25%, but also mentions that the mortality may be as high as 51%. Of the 71 cases admitted to the City Deep hospital, 31 terminated fatally - a case mortality of 44%. In considering this apparent high-mortality, it must be remembered that only cases that were unconscious and presented the clinical picture described here have been classified as heat-stroke.

In cases that recover, consciousness is usually regained within 12 hours; there is an accompanying drop in body temperature which reaches normal within 48 hours. Cases with a rectal temperature of over 107° F. are almost invariably fatal although death has occurred in cases with rectal temperatures below this figure (vide Table I). The acuteness of the condition as met with on the Rand has already been emphasised. Death occurs within six hours of the onset of the attack; survival for a longer period makes the prognosis considerably brighter. One case died within ten minutes of arrival in hospital, although special arrangements were made for speeding his removal to hospital; from the time of onset of the attack to death was barely an hour. In three cases death occurred underground.

Natives who have survived one attack of heat-stroke

appear to be more susceptible to subsequent more severe attacks. This was shown tragically in one native who had recovered completely from a moderate attack and developed a second fatal attack on recommencing underground work. No similar cases have occurred because, as a precaution, heat-stroke recoveries are not returned to underground work.

Complications following an attack of heat-stroke are comparatively rare. Two cases contracted pneumonia after recovery from heat-stroke but before discharged from hospital. One case, twelve hours after recovery of consciousness and return of temperature to normal, suddenly passed into a state of coma and died shortly afterwards. Mental sequelae were observed in two cases after regaining consciousness and normal temperature. Both these natives developed a condition of mental confusion and delusions. The condition persisted for over a week in both cases, and then suddenly disappeared. There was no accompanying rise in temperature.

#### Prevention.

Heat-stroke on the Rand mines is a preventable affliction. When considering the Rand mines as a whole the incidence of fatal heat-stroke cases appears almost negligible, in comparison with deaths due to disease and accident. For the years 1926-1931, Cluver (1932) could find only 92 deaths which were accorded as due to heat affects. During this period the average daily complement of underground native



employees exceeded 200,000.

On the other hand these figures assume more significance when it is realised that of the 92 fatal cases recorded 69 had occurred in three mines. These three mines were all at depths of considerably over 6,000 feet and great difficulties were experienced regarding ventilation and atmospheric cooling powers.

To-day the position is more or less the same. Only 21 deaths from heat-stroke were recorded for the year 1936 out of an average daily complement of underground native employees of over 300,000.

Apart from adequate ventilation, heat-stroke is intimately related to the problem of acclimatisation. Heat-stroke invariably occurs in new unacclimatised natives, or in natives suddenly transferred from cool parts of the mine to hard work in hot parts of the mine. In both cases the native is not acclimatised for hard work in bad underground atmospheric environment.

Acclimatisation is rapidly acquired. Some natives can become used to hard work in hot underground conditions in a few days. There are some natives, however, who require much longer. Acclimatisation, however, though rapidly acquired is also rapidly lost (Dreosti, 1935a).

The difficulty and economic significance of acclimatising mine natives can be appreciated from the following. On the Rand mines natives contract for periods of work



varying from 1 to 18 months. On completion of their contracted period of service the natives return to their homes on a system of extended leave, and after an absence of six or more months return to the mines for re-employment.

During this absence from the mines the natives lose their acclimatisation and when re-engaged have to be treated as unacclimatised. Hence all natives employed for mine work, with or without previous underground experience, require acclimatisation.

For this purpose those mines on the Rand experiencing ventilation difficulties have introduced a procedure of practical acclimatisation in order to minimise the incidence of heat-stroke. The procedure consists of employing all new natives on light underground work for a week or more, and then only are the natives allowed to take on normal underground work. It has been found that practical underground acclimatisation must be carried out in a relatively hot working place, by increasing the work output from almost nil on the first day. Acclimatisation cannot be satisfactorily achieved by the subject working in a cool place.

It will be recalled that the native labour turnover is over 90% annually, e.g., a mine employing 100,000 underground native employees must engage 9,000 natives annually to maintain a steady complement. It is obvious therefore, that underground acclimatisation, as outlined above, can only be successful through constant conscientious and competent

supervision.

Since the period of acclimatisation required varies in different individuals, a procedure has been evolved on the City Deep mine for testing all new natives in specially erected heat chambers, under controlled conditions of work and atmospheric environment to determine beforehand the period of acclimatisation required by individual natives. This testing process apparently also protects new natives by stimulating their acclimatisation properties (Dreosti, 1936b).

Summary.

The underground atmospheric working environment of the Rand mines is hot and humid, and in some cases associated with stagnation of air. Hard manual labour in such conditions may produce pathological reactions, namely, Heat-collapse and Heat-stroke.

Heat-collapse is due to cardiac embarrassment brought about by the inability of the heart to cope with <sup>the</sup> increased physiological demands made upon it by the work and the environment. The symptoms and signs are those of a fatigued heart, namely, headache, dizziness, general feeling of weakness or collapse. The discomfort that these symptoms produce prevents further muscular work and therefore saves the already overworked heart from still further effort. Heat-collapse is therefore in a sense protective.

It is only natural that workers who are debilitated as

the result of disease are more prone to heat-collapse than normal healthy subjects. Further, the abnormal strain put on the heart, as the result of work in bad underground environment, which normally produces symptoms of heat-collapse, may in diseased subjects be sufficient to bring about a complete breakdown of the heart's action with subsequent death.

Heat-stroke is a condition of acute mental excitement with delirium, convulsions, muscular twitchings or tremors and is always associated with a high body temperature. The actual causative agent in the body which precipitates this condition has not yet been established. Hyper<sup>XR</sup>pyrexia is merely an accompaniment of the general bodily derangement and is not the causative agent. The invariable absence of diagnosable disease in cases of heat-stroke on admission to hospital, and the subsequent similar findings by Macro- and micro-pathological examination in cases that terminate fatally, is consistent with the fact that co-existing disease does not play a role in precipitating heat-stroke. Unlike heat-collapse, heat-stroke only occurs in <sup>un</sup>acclimatised subjects. Owing to the acute onset and course of the affliction, treatment directed at anticipating an attack is impossible.

Heat-stroke is preventable. Adequate control of the cooling power of the working environment together with a suitable form of underground acclimatisation will eliminate it.



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