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JUNE, 1941

Vol. XXXVIII. No. 6.

The Antiseptic

A Monthly Medical Journal

Edited by

Dr. U. Rama Rau, M.L.C. & U. Krishna Rau, M.B., B.S.

17 JUL 1941

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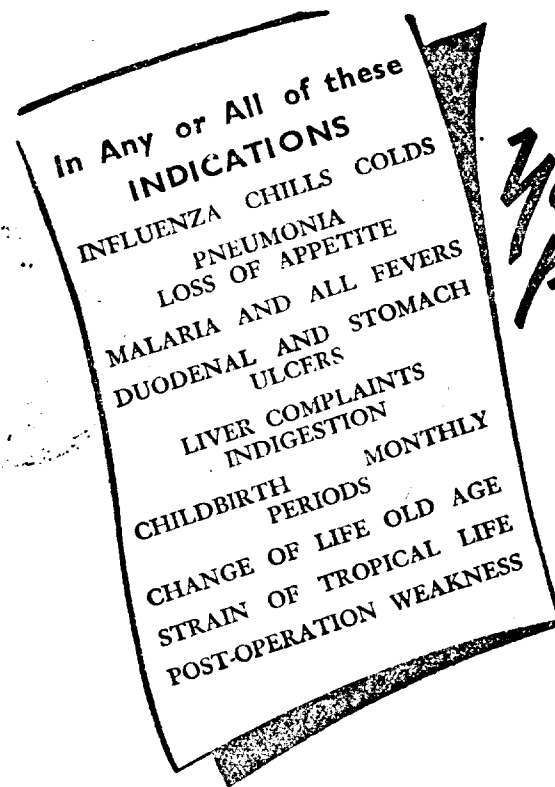
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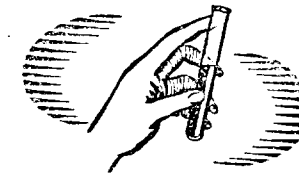
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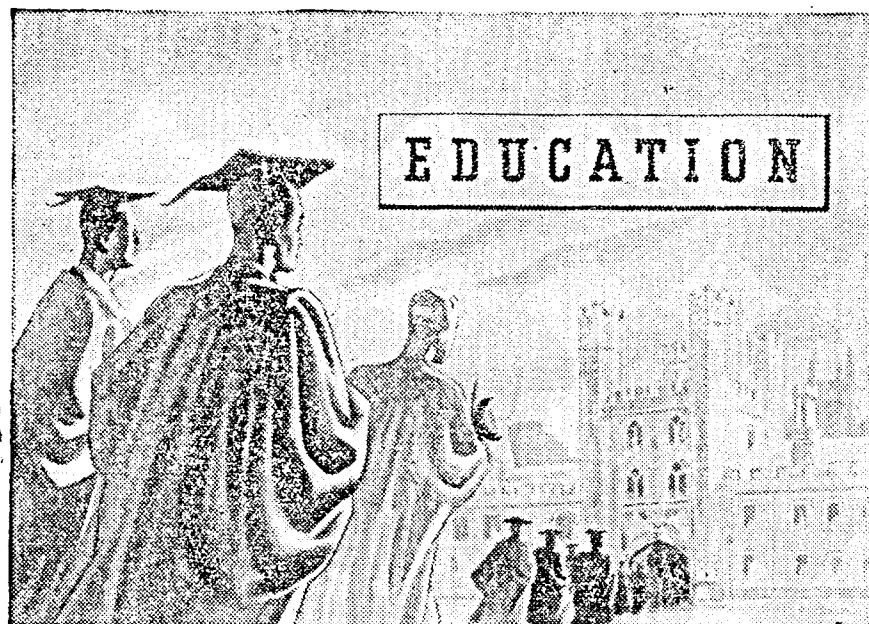
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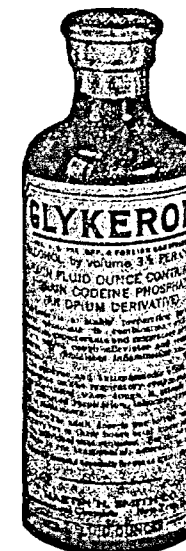
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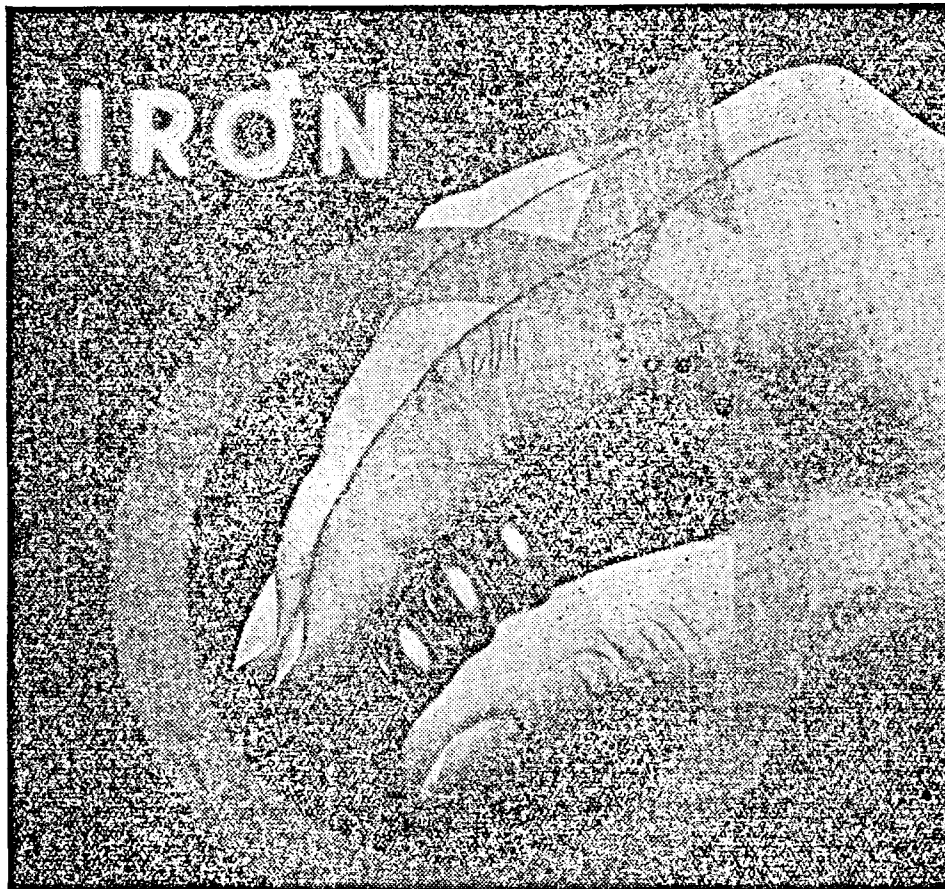
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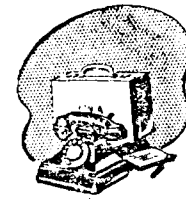
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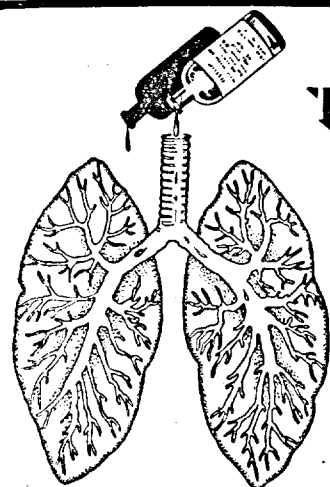
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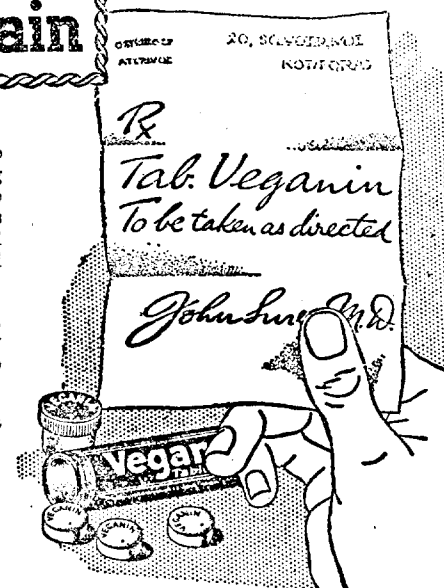
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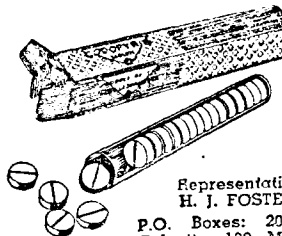
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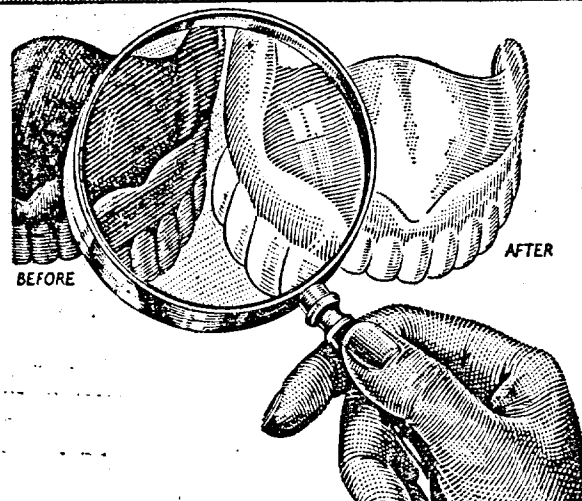
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Influence of Climate on Health and Disease

BY

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THE special number of *The Antiseptic* on 'Summer Complaints' is a timely and opportune publication of topical and seasonal interest, as summer is now on and is making itself felt acutely here in the plains of the tropics. Summer follows spring and precedes autumn. When winter comes, can spring be far behind? Summer is but a passing phase in the year with its procession of nights and days, weeks and months and alternation of seasons. It is but an infinitesimal part of the ceaseless cyclical phenomenon of the Universe. The wheel revolves perpetually with the zenith and nadir changing positions. As summer will soon pass off, it occurred to the author that it would be appropriate to have an introduction to this number on the influence of climate on health and disease in general.

The life of man is cast in a world comprising variegated fauna and enveloped by atmosphere, hydrosphere, trophosphere, stratosphere, ether and cosmic rays. He is a part of the Universe; his well-being depends on the aggregate influence he is subjected to by each and all of them.

Health can be defined as a general state of soundness of the body, mind and spirit. It is not simply and merely freedom from disease. It is not a negative attribute, but a positive dynamic state. It signifies that man is active, agile and alert, brimming and bubbling with vim, vigour and vitality. It is a state of exuberance. Languor, lassitude and lethargy indicate disease. Health does not pertain to the body only; it includes mind and also the subtle soul or spirit. Man is not merely a lump of flesh and blood. He is a sentient and sapient being, imbued with emotions and feelings, passions and sentiments, imagination and volition. It is fundamental to have this concept of man as a whole, in considering problems of health and disease.

Climate and health.—The climate of a place is the atmospheric condition, depending on its distance from the equator and the sea, its altitude,

the direction of the prevailing winds and the mean temperature. There are, besides, many other obscure, occult and ill-understood factors of an electrical and magnetic nature and extra-mundane influences such as solar, lunar and sidereal. The earth has been divided by geographers into as many as twelve regions having different climates. A shorter and simpler classification of climate into tropical, temperate and polar has been given by PHILIPS; the two former are further sub-divided by him into continental, insular and mountain. The mean temperature of the air is determined by the latitude, altitude, proportion of land and water, aspect and nature of the soil. It may be stressed that besides good soil, wholesome food, fresh air and pure water, it is the weather that moulds the life of man, that makes some people black, others tan, some tall and others short, some docile and subservient, others aggressive and overbearing, that makes the pigmy of the Belgian Congo dress in leaves, whilst the Eskimo goes in furs and friezes. It is the soil and climate that determine the distinctive attributes and characteristics of races and nations. The dark, damp, dismal, densely-wooded equatorial forests with their miasmatic exhalations are peopled by the backward, stunted and dwarfed pigmies. Living is precarious in such tracts. The Norwegians, Britons and the Japanese have developed, by reason of their environment, into great and hardy sea-faring nations. The fertile and temperate mediterranean littoral has been the home of great civilisations. A high state of prosperity and advancement is found in many monsoon lands. Man is thus a creature of environment and is controlled to a great extent by the climate and vegetation of the countries in which he lives. Healthy and fertile tracts, producing an abundance of food grains and cattle, release and emancipate man from the incessant struggle and toil of having to go about in quest of food for the appeasement of the primal instinct of hunger. The problem of food with the ancillary problem of economics is solved to a great extent by congenial climate. The quality of the food is dependent on the soil and fauna of the locality. Shortage of food consequent on drought and failing rains is the cause of famine with its concomitant of pestilence. The agency tracts of Vizagapatam and Godavari comprise densely wooded forests wherein the air is generally stagnant. Rain-fall is abundant. They are notorious for malaria and black water fever. Riparian tracts are hot and humid. Rajahmundry and Bezwada, situated on the banks of the Godavari and the Krishna, are unbearable during summer. Littoral areas like Vizagapatam, Gopalpur and Puri have an equable climate. Hills are resorted to in hot weather, owing to the advantageous conditions prevailing there, such as increased sunlight, greater movement of air which is free from dust, lessened humidity, presence of ozone in small quantities and a lowered temperature. The climate is salubrious, bracing and invigorating. At high altitudes, there is increased hæmopœsis. Those resorting to the hills develop a bloom—a crimson on their cheeks within a few months of their stay there. There is, besides, the psychological effect of a change of scene and surroundings and of respite from the monotonous routine and regimen. Some cannot stand high altitudes owing to the extreme rarefaction and diminished atmospheric pressure. They suffer from mountain sickness, hæmorrhages and other troubles. Acclimatisation is the process by which plants and animals become remarkably adapted to withstand and resist the effects of a climate different from their own. Man is endowed with marvellous powers of adaptability to environment; but for this, conquests, colonisa-

tions, explorations and other adventures would not have been possible. According to ARNOLD, a race is said to be acclimatised when it preserves its natural increase of population, its normal longevity and its aptitude for physical and mental work. Indian history abounds in instances of acclimatisation of foreign immigrant settlers.

Seasons are caused by the tilt of the earth with reference to its orbit round the sun. It is because of this that the vertical rays of the sun play on the tropics of Cancer and Capricorn. This also accounts for the summer and winter solstices and the vernal and autumnal equinoxes. There is the well-known four-fold division of the year into four seasons of three months each. The celebrated American humorist, SAMUEL CLEMENS, better known by the pseudonym of MARK TWAIN, refers to the climate of India as nine months hot and three months hotter. According to the Indian almanac, there are six seasons in the year—each of two months' duration. They are: *Vasant* (spring) *Greeshma* (summer) *Varsha* (monsoon) *Sharad* (autumn) *Hemantha* (cold) and *Shishira* (frigid). The glory of spring has been the favourite theme of poets and singers all the world over. They have vividly described its charms and beauties. Nature is in her exuberant mood in this part of the year. The trees put on their verdant mantle and burgeon and blossom out into sweet smelling flowers and luscious fruits. The skies are cloudless, clear and starry. Cool, gentle zephyrs laden with fragrance blow. The feathered choristers of the air pour forth their mellifluous melodies. The butterflies in their gorgeous multi-coloured hues flit from tree to tree. The peacock, with its resplendent tail, dances in sheer joy. The air is thick with the buzz of the bee and the drone of the beetle. The house martins and sparrows chirp incessantly. It is the mating and nesting season. The harvest is reaped, the granary full and the larder well-stocked with provisions. There is peace and contentment. It is the marriage season. There is feasting and rejoicing, music and mirth, merriment and hilarity everywhere. The nights are short, the days are long. The advent of spring, the best season of the year, is celebrated with great éclat in northern India, on the fifth day of the bright half of the month of *Magh*—*Basant Panchami*—coinciding with the month of March. The festival of festivals in the Andhra-desh is the harvest festival of *Pongal*, which falls in the middle of January when the sun touches the tropic of Capricorn. It signifies the end of the bitterly chill and unhealthy cold weather. Houses are white-washed. It is the equivalent of the spring-cleaning done in the West. Prayers are offered to the deities and demiurges to propitiate and appease them. Epidemics are thought to be due to incurring their displeasure and wrath. New clothes are worn. This is followed by summer (May & June) with its scorching and sweltering heat. The days become insufferably hot and close, stifling and oppressive; perspiration pours in torrents. Tempers get frayed. The tanks and lakes, pools and ponds, rivers and canals and wells dry up. Scarcity of water is felt in most of the places. Swarm of flies and gnats are in evidence everywhere. The opulent section plan their programme for holidays on the hills. This is succeeded by the welcome and refreshing rainy season, which greatly mitigates the rigour of the heat. Rivers are in spate. Sky is overcast with dark, rain-bearing clouds. There is great rejoicing among the farmers and cultivators. Jupiter Pluvius, *Varuna* of Indian mythology, is the God of the agriculturist. Autumn comes next. The bare trees, denuded of leaves, are forbidding in their appearance. The ground is strewn with dry and

withered leaves. It is the period of the sere and yellow leaf. There is dew-fall at nights. The autumnal moon is cool, clear and bright. "Live a hundred autumns" is the time-honoured old Vedic blessing. This is succeeded in turn by the coldest season of the year—*Hemantha*. This is believed to be the unhealthiest and worst of the six seasons. Prayers and poojahs are offered in temples in the small hours of the morning, for warding off disease. This is a trying season for the poor and the destitute. *Shishira ruthu* comes last. This is again an unhealthy season of the year. The new year that follows ushers in the spring. Nature puts on different seasons. These changes escape the eye of the dweller of the city with its din and bustle, congestion and overcrowding, heat and dust, buildings and traffic. The inhabitants of the fields and forests do react to the varying panorama, unfolded to them one after the other, in the course of the year.

Summer.—The first half of the solar year beginning from about the middle of January till the middle of July is spoken of as *Uttarayana* during which, the sun moves north to the tropic of Cancer. This is supposed to be the brighter, the better and the more auspicious half of the year, eminently suitable for the celebration of weddings and sacred rites and rituals. This is the healthier half of the year. Mortality is lower. Summer, notwithstanding the heat, is not altogether a bad season. The sun's rays have potent bactericidal properties. Excessive heat is no doubt enervating, exhausting and even maddening, especially to such as are not acclimatised. Even the poikilothermic reptiles go into a state of suspended animation—astivation or summer sleep consequent on scarcity of food. The sleep comes to an end with the breaking out of monsoon rains. Other reptiles and some mammals have their winter sleep, hibernation. The human infant at birth has a mass of fat and lymphoid tissue in the posterior triangle of the neck. HATAI regards this as the representative of the interspecular hibernating gland of insectivora and cheiroptera. Birds put on a brilliant plumage during summer. The same can be said of some animals also. A rise of atmospheric temperature is associated with an increase in the prevalence of gastro-intestinal affections such as diarrhoea and dysentery. These two are related to the bionomics of the flies. The principal summer complaints are mainly of the integumentary and alimentary systems. The skin bears the brunt of the function of excretion and elimination of waste products. The kidneys are relieved of strain during summer. There appears to be a rhythmic periodicity even in the functioning of organs. The skin which is the uninervated nervous system and which has been described as the largest and most versatile sense organ fulfils various functions in the economy. It is a remarkably tough and highly elastic fabric, capable of withstanding heat and cold, wet and drought, acid and alkali, microbic invasion and the wear and tear of three score years and ten, and even more. It is "the regulator of temperature and the principal excretory organ in summer. It is credited with the production of Vitamin D by irradiation with sunlight through the presence of Ergosterol in its substance. The melanin found in the malpighian layers of the epidermis protects the individual from the deleterious effects of the ultra-violet rays of the sun. It is well-known that blondes suffer far more than brunettes. Desquamation occurs to a greater extent during summer. The skin becomes vulnerable to microbic and fungoid infections. Prickly heat, furnuncles, carbuncles, dermatitis herpetiformis, ringworm, intertrigo etc., are the common cutaneous astival affections. The conjunctiva which is an epithelial structure of

ectodermal origin allied to the skin is again the site of inflammatory infections. This is due to the eye-fly which hovers about the face and eyes in the most annoying manner during summer. There is depletion of fluids from the system in large quantities which induces thirst. This craving accounts for the popularity of soda fountains with their stock of squashes, ales, ades, creams, cordials and other iced beverages. The cheap aerated and coloured waters sold in the streets are sources of infection, as impure unfiltered water is used in their manufacture. During hot weather, metabolism is decreased as there is little demand made on the heat-producing mechanism. Less solids and more liquids are required during summer. Humidity of the atmosphere in summer hinders perspiration and free evaporation. Heat stroke is common when humidity is high. Dry heat, though severe, is borne better than moist heat. The deltaic areas are as a rule hot, humid and unhealthy. From personal experience, the writer testifies to the excellence of Bellary district which is remarkably dry and is admirably suited to the convalescents. In winter, more food is necessary for stocking up the central heating apparatus. Hence, there is a craving for starch, sugary and fatty foods in cold weather.

Periodicity of epidemics.—Ancients recognised that certain infectious diseases recur at definite periods of the year. ARRHENIUS and SYDENHAM have drawn attention to the fact that contagious diseases are influenced by atmospheric (cosmic) influences. PASTEUR has referred to the strengthening and weakening of viruses brought on by changes in environment. VON PETTENKOPF was of opinion that the cause of epidemics lay in the soil and variations in the level of sub-soil water. The quantum theory of infection postulates that all epidemical phenomena are dependent on a change in the relationship of the infection quantum and the immunity quantum. The crux of the problem lies in the determination of the factors that raise the immunity quantum. The susceptibility of the masses to infection during some parts of the year is due to extra corporeal influences to which the transmission factor is exposed. Seasonal changes of climate attended with the seasonal oscillations of temperature have a great influence on the intermediary hosts concerned in the dissemination of parasites. Insect-life attains the maximum expression of prolificity in the astivo-autumnal seasons in the tropics. The chief enemies of mosquitoes are bats, birds, fishes, spiders. Their services have to be availed of for keeping the mosquito pest under control. These diminutive winged dragons are responsible for the causation of the much dreaded scourges of malaria, filaria, dengue and yellow fever. It need hardly be pointed out that certain diseases are peculiar to the tropics, whilst others are confined to the temperate zones; it has, however, to be borne in mind that modern facilities of rapid transport are potential factors in the implantation and dissemination of diseases from one part of the globe to the other. Though the parasitical diseases are relatively fewer in number in the temperate and sub-tropical zones, they tend to exhibit epidemical rather than endemical qualities. Cold and damp favour the spread of tuberculosis, rheumatic fever, measles and diphtheria. Intense cold and moist are conducive to the transmission of pneumonic plague. Bubonic plague usually breaks out during the cold season in tropical countries. Malarial epidemics are common soon after the rains.

Influence of season on war, crime and sex.—Wars, revolutions, mutinies, revolts and insurrections break out mostly in hot weather. The month

of March which was the first month of the ancient Roman calendar and the third one of the Julian calendar of the present day, is dedicated to Mars—the God of War. HERR HITLER is said to believe in the superstition that campaigns undertaken in this month will prove victorious. 'Beware the ides of March' is a familiar Shakespearean quotation. In ancient Rome, 'ides' meant the fifteenth day of March. 'Mad as a March hare' is a trite phrase, conveying the idea that even rodents are affected by intense heat. The phrenetic and orgiastic bacchanalia saturnalia, spring festivals and macaberesque and other terpsichorean frivolities characterised by unrestrained license and revelry are but the expression of exuberance of the hot weather. The boisterousness and rambustiousness incidental to them are the result of generous imbibition of spirituous liquors. The curves of rape, murder, running amok, rise in the hot weather. Health, strength and sexual vigour are supposed to vary with the waxing and waning moon. Full moon is said to exert an erigent influence on man. The largest number of conceptions are said to occur in the tropical and subtropical zones during the coldest month of the year. The term lunacy significantly points to the influence of the moon in the causation of mental aberrations. Sun-spots may also have a similar effect on man. There are some ultra-rationalists who scoff and sneer at the idea that man is affected by planets. They are to be pitied for their ignorance of extra-mundane phenomena. It is a matter of common knowledge that there is a feeling of gloom and depression on a dark cloudy day and a feeling of elation on a bright sunny day. Patients suffer far more at nights than during the day. It may be because of the dread of the sombre, solemn and silent darkness. Some individuals are hypersensitive to variations in the weather. They are human barometers. A wet and windy day will precipitate an attack of asthma and urticaria in them. A few can even predict the onset of seismic convulsions, storms and cyclones. The science of epidemiology, though it is yet in its infancy, has already wrought wonders. The epoch-making introduction of preventive and prophylactic inoculation and the study of the bionomics of the insect vectors have considerably reduced the incidence of small-pox, plague, cholera and other epidemics. It is now possible to live in the so-called unhealthy parts of the world. The strides of science may, perhaps, in the course of a few more decades, render the sandy Sahara fertile and even the frigid Tundras habitable.

To sum up, this short paper aims at establishing the thesis that climate is not only a potent factor in determining racial characters and attributes, but also one profoundly influencing health and disease. It is beyond the scope of this brief and limited survey to deal in detail with the vast and complicated problem of the influence of climate in the causation of various diseases. Man is but a chain in the link of the Universe. To change the metaphor, he is but a part of the gigantic cosmic web which depends for its integrity on a well-ordered scheme of conservation and maintenance of an equilibrium and a balance wherein "the smallest insect, the ultra-microscopic virus and the tiniest atom are of no less consequence, significance and importance than the mighty oak and elephant and the most magnificent nebula".

Maharani, Vizag.

Effects of the Sun's Rays on the Human System

BY

G. R. RAO, D.T.M.,

Jharia Mines Board of Health, Dhanbad, E.I.R.

Introduction.—Of all the complaints prevalent in summer, the group of 'symptom-complexes' of varying degrees of severity with different types of clinical manifestations, that are severally designated as 'heat-stroke', 'sun-stroke', or 'siriass', 'insolation', 'heat-exhaustion', and 'heat-cramps', etc., presents a chapter of modern medicine as yet partly-understood (but nevertheless serious in pathological significance). That the life-giving sun's rays have also a very potent destructive force, has been probably realised, from the dawn of human intelligence, as both reverence and awe have been shown in the hymns of praise addressed to the sun by our ancient progenitors. Some of these symptom-complexes, (these are not yet elevated to the dignity of definite disease-entities), as for instance, the condition known as insolation or sun-stroke, prove quickly fatal in susceptible individuals, while others like heat-exhaustion or heat-prostration, reduce the individual sufferer, more or less to the state of an invalid, for some time at least and in a few cases, effectively preclude the active participation in strenuous activities, by the sufferers, who have once been attacked. Hence, no apology is needed for including a brief discussion of our present-day knowledge regarding these symptom-complexes, in a special number devoted to 'Summer Complaints'.

Effects of the sun's rays on the human system.—It is a matter of common knowledge that the sun's rays have three well-defined properties or effects: calorific or heating, illuminant or lighting, and actinic or chemical. Within certain limits, all the three properties of the sun's rays are utilised for the good of human beings in various ways. But what we are concerned with, here, is their pathological effects.

Calorific or heating:—Taking the calorific effect first, a certain quantum of solar-heat is considered absolutely essential for the continuance in life of at least the higher forms of all terrestrial fauna and even the higher order of the 'flora' thrive only in an atmosphere pervaded with solar heat. But, when the quantum of this heat is exceeded, the living tissue gets heated and katabolism increases with the result that waste-products accumulate more rapidly than the system can deal with; and finally, these accumulated katabolic products damage the delicate nerve-centres. The thermic centre in the brain of human-beings is quickly upset and ceases functioning, with the result that the temperature of the affected individual rises to a height incompatible with life. That heat (solar) accelerates chemical action, is well known and that the deleterious action of katabolic products is also hastened by the same heat needs no emphasis. The interaction between the continued calorific effects of the sun's rays and the increased katabolic activities further leads to an enhanced virulence of the baneful effects exerted by the accumulated waste-products on the highly evolved nerve-tissues and centres. Also, the liberation of organic acids from heated tissues, in larger quantities than in a normal state, leads to a disturbance in the acid-base equilibrium of the blood which also profoundly affects the course of life. The action of buffers like the phosphates comes to the rescue

and prevents, up to a certain extent, the upsetting of this equilibrium, but a time comes when any amount of buffer action will not restore the acid-base equilibrium to normal. In short, the continued calorific effects of the sun's rays are said to precipitate such a condition in which the vast accumulations of katabolic products themselves induced by the heat, upset the acid-base equilibrium profoundly and life becomes incompatible with an acid-reaction of the blood. The more highly evolved the tissue, the more quickly and the more intensively do the pathological effects, described above, appear in it.

Illuminant or Lighting:—Now, coming to the second property of the sun's rays, *viz.*, the illuminant or lighting, a prismatic analysis shows that the ordinary ray of light is really composed of at least seven visible colours, each having a different wave-length. These seven colours constitute what is commonly and scientifically spoken of as the normal solar spectrum. Whether these colours collectively or severally have any pathological effects on the human system, is rather a difficult question to answer, in the present state of our knowledge. That these different colours are therapeutically utilised in Heliotherapy and probably also in Solaria, is very well known, but beyond this, our knowledge does not go. Whether each colour has a selective action on a particular tissue and whether, by a combination of two or more different colours, a definite pathological effect can be obtained, are questions that call for an answer. This much is, however, now known definitely, that beyond the visible ends of the normal solar spectrum, on either side, are the really potent rays that profoundly affect all living creatures. At the one end are the ultra-violet rays and at the other end, the infra-red rays. The former are already therapeutically employed, and this needs no special mention here; but that they have a well-marked pathological effect also and can destroy living tissues quickly, needs emphasizing. Whether these ultra-violet rays, by their diffusion in the atmosphere, contribute to the production of the symptom-complexes which form the subject-matter of this note, under certain conditions of atmospheric temperature, pressure and humidity, is a question that cannot be answered off hand, and needs careful pondering and further research. That these particular rays have a certain penetrating power, has been realised at the cost of the lives of a few scientific martyrs, who were pioneers in this type of investigation. But, recent researches in solar physics, have shown that the other end of the invisible spectrum contains rays which are more penetrating in their effects and are known as infra-red rays. These rays are said to possess very great penetrative powers leading to a quick destruction of living tissues. And they are also credited with intense thermal effects on living tissues. Whether these effects *per se* can cause heat or sun-stroke is a point that merits our attention. Certain experimental investigations into this aspect of the subject, conducted in the Philippines, have shown that definite pathological effects such as heat-retention or failure to radiate heat and destruction of tissues can be and are caused by the infra-red rays. Whether these infra-red rays are really responsible for the so-called actinic or chemical effect (the third property of the solar rays) is a subject that is being investigated. Already, some knowledge seems to have been gained, as these rays have been utilised industrially, as for instance, in infra-red photography and for appliances utilising infra-red rays to detect flaws in gems and high quality precision tools made of special metallic alloys, etc.

Actinic or chemical:—Finally, taking the third effect—actinic or chemical—of the solar rays, our knowledge is still in its infancy. That the sun's rays do initiate as well as accelerate chemical reactions, is too well known to need any special mention, but what is not so well known is the actual identity of the particular component part of the solar rays, that plays this role. Can photo-activity or heat-activity effects of the sun's rays be dismissed out of consideration, when we are studying the actinic effects? To my mind, it appears that these two effects are also contributory factors to the bringing into existence of the actinic effects. It is not known with any degree of certainty as to whether there are clear-cut chemical effects on the living tissues, exercised by the so-called actinic property of the solar rays *per se*. How far the actinic effect alone (if it can be separated and studied) can cause the pathological conditions under reference in this note, or contribute to their onset, is an interesting speculation.

Summing up, I think, as our knowledge stands at present, it is permissible to conclude that the calorific and illuminant effects of the sun's rays and particularly the infra-red rays of the invisible end of the solar spectrum, have a direct causative relationship with the pathological conditions under consideration here.

Mechanism of heat regulation in the human system.—The human system, like that of the warm-blooded mammalia in general, maintains normally a fairly steady temperature, by a constant combustion of waste-products, through the agency of Oxy-hæmoglobin; and it is conceivable that the amount of combustion will vary in accordance with the quantum of waste-products produced and the respiratory activity of the individual, with special reference to the degree of oxygen-saturation of the surrounding atmosphere. Any deficiency in the oxygen-content of the atmosphere, is sought to be counter-balanced by the increased respiratory efforts and any excessive intake of oxygen resulting in increased combustion, is sought to be offset, by the enhanced activity of the peripheral vasomotor system operating quickly through the medium of the sweat glands. Under normal circumstances, therefore, there is a fairly constant quantity of heat produced inside the body, and if an excess of heat is produced, it is sought to be got rid of by internal regulation. Now, the environment in which the system has to function and maintain its being, exerts its own effects and these vary from time to time according to season, climate, altitude, and meteorological factors, such as atmospheric temperature and humidity. Some agency is, therefore, necessary to maintain the balance between the forces exerted by the externally operative environmental factors and those put forth by the internally operative ones. And this agency is said to be the special thermotaxic centre in the medulla.

How does this thermotaxic or thermic centre work? At the outset, it has to be confessed that the exact mode of its action is not yet clearly understood. But, sufficient knowledge has accumulated, of late, to indicate roughly the channels through which this centre operates to reduce excessive heat, either generated internally or derived from the environment. Radiation, conduction and evaporation are the means adopted in normal circumstances to regulate the heat; and the former two (radiation and conduction) are said to promote heat-loss to the extent of 73%, and other emunctories, including the respiratory organs, account for the remaining 27%. Roughly, half of the burden thrown on the

emunctories, is borne by the skin, specially by the sweat-glands. But when radiation and conduction, which play the major role in heat-regulation normally, are rendered impossible by high atmospheric temperature (an unfavourable environmental factor) the system resorts to evaporation, chiefly through the medium of the skin and its appendage, the sweat-glands, which are stimulated by the peripheral vasomotor mechanism. And when this evaporation is also rendered difficult or impaired by a high degree of atmospheric humidity plus other constitutional factors, the system is unable to regulate the heat. And the thermic centre becomes practically defunct for the time being at least. From what has been said so far, it will be evident that heat-loss is promoted not a little by the emunctories, the functional integrity of which, should always be maintained at a very high level. It is only when emunctories fail to rise to the occasion (owing to constitutional factors) and the environmental factors (physical) continue to be unfavourable, *i.e.*, persistent high temperature and high humidity of the atmosphere prevail, that the failure to regulate the heat becomes apparent. And depending upon the degree and duration of such failure, different trains of symptoms and signs are produced giving rise to the various symptom-complexes, which, in the ultimate analysis, appear to be only variations in the degree of reaction or response shown by the individual human body, to unfavourable physical environmental factors. With the back-ground thus provided by the foregoing discussion, in general outline, of the various factors, involved in the production of the symptom-complexes under consideration in this note, let us now consider in detail, their clinical aspects and treatment, as well as their prevention. The milder ones are dealt with first, and the severer ones later.

Heat-prostration or heat-exhaustion.—DEFINITION :—This may be defined as a sudden attack of fainting with a preceding feeling of giddiness or dizziness, brought about by exposure to high atmospheric temperature or to direct solar heat.

Heat-exhaustion may also be brought about by exposure to highly heated atmosphere in the shade, and in fact, a good number of cases seem to develop the symptoms and signs of exhaustion while in the shade. High atmospheric heat alone does not seem to precipitate the condition, if the heat is of the dry type. High relative humidity of the atmosphere such as that obtaining in an overcrowded, stuffy office-building, especially during the onset of the South-west monsoon, seems to be a necessary contributory factor.

PREDISPOSING CAUSES:—The general health of the individual affected seems to act as a predisposing factor. Alcoholism, gluttony, unsuitable tight clothings necessitated by convention or fashion, a dirty skin, sluggish kidneys, costive bowels, previous attacks of malaria or filarial fever, narcotic addiction, untreated acquired syphilis, and habitual abuse of cardiac stimulants, are all known to act as predisposing factors both in cases of heat-exhaustion as well as heat-stroke. And it is obvious that persons with organic diseases of the heart, lungs, kidneys or liver or with such constitutional anomalies as hæmophilia, status thymico-lymphaticus and/or leukæmia also seem to be very prone to attacks of heat-exhaustion or heat-stroke. Diabetics and persons suffering from high blood pressure are also rather prone to suffer from heat-stroke or heat-exhaustion, under certain conditions of high solar heat or atmospheric heat and relative humidity.

Now, taking the predisposing causes, one by one, let us see how each contributes to the onset of heat-exhaustion or heat-stroke. Alcoholism is blamed first. But alcoholism evidently means excessive consumption of the highly potent and flavoured kinds of liquors, as for example—Brandy, Whisky, Sherry, Port wine, Claret, Gin, Rum, and Stout, etc. Those who consume liquors with a minimum alcohol-content (*e.g.* Beer) do not seem to be affected adversely. Aboriginal tribes of the Chota Nagpur plateau—the Orans, the Mundas and the Hos, who consume a home-brewed rice-beer called *Pachwai* or an aromatic alcoholic drink, distilled from the flowers of *bassia latifolia* (mahuwa *இலுப்பை*) do not seem to be prone to suffer from the effects of high atmospheric temperature with or without high humidity. No case of heat-stroke or heat-exhaustion has been reported from them. The epicurean connoisseur, frequently indulging in high-quality fiery liquid refreshments, seems to be very prone to suffer. As for gluttony, the reason is obvious, embarrassment to the heart or insufficiency of the fluid intake, precluding replacement of the fluid lost by perspiration.

Unsuitable and tight clothings impede free circulation of air and evaporation from the skin. The effect is the same as when there is a high humidity in the atmosphere, preventing evaporation of the sweat. Also, the free circulation of blood is interfered with, throwing an extra strain on the heart. A relative anoxæmia (deficient oxygenation of the blood) too, is induced by tight clothings, especially around the neck and chest. A dirty skin does not promote free sweating by reason of the clogged pores, and evaporation of the perspiration, which is one of the very important means of regulation of the body temperature, fails, leading to heat-retention.

Sluggish kidneys and costive bowels lead to a chronic intoxication, which, in the presence of a high atmospheric temperature and humidity, seems to favour either heat-retention or enhance the toxic effects of the katabolic products formed by the heated tissues and thus render the sufferer prone to attacks of heat-exhaustion or heat-stroke.

Previous attacks of malaria or filarial fever, apart from the anaemia caused by them, appear to devitalise the individual and impair the smooth functioning of the thermic centre, by inducing a state of relative thermic imbalance.

Addicts to narcotics always have a sort of subnormal temperature and this is probably caused by the depressant effect of narcotics on the brain and the cardiac as well as respiratory systems.

Untreated acquired syphilis has been first detected, after an attack of heat-stroke or heat-exhaustion in some cases. Whether such attacks are really helped by the untreated syphilitic factor, is a debatable point. More probably, some definite organic leutic lesion of the heart, liver, brain meninges, kidneys or the vascular system, may have been present and overlooked.

As regards the other predisposing factors enumerated, the reason why they act as such, is too obvious to need any elaborate explanation here. It must be remembered that heated tissues disturb the acid-base equilibrium, by the accumulation of katabolic waste-products and a relative acidosis is produced thereby. Diabetics are, therefore, very prone to suffer from the effects of high solar or atmospheric heat and high humidity.

SYMPTOMS AND SIGNS:—These are predominantly syncopal in character. A weak, thready pulse, probably imperceptible at the wrist, and sometimes so fluttering as to be uncountable, pallor of the face and skin of the whole body—cold and clammy, loss of consciousness, partly or wholly,

shallow breathing, dilated pupils, and a subnormal temperature are the usual symptoms and signs met with. Very commonly, the patient first complains of dizziness and fainting, staggers and falls down. Gradually, recovery ensues, the patient first complaining of a splitting head-ache and extreme prostration, and develops indistinct speech with a partial loss of memory in some cases, especially in alcoholics and narcotic addicts.

DIAGNOSIS:—Any condition, in which syncope is the predominant underlying pathological factor, may be mistaken for heat-exhaustion or heat-prostration. Coronary thrombosis, partial heart-block, narcotic-poisoning, diabetic coma, head-injuries leading to concussion, uraemia, algid malaria, cerebro-spinal meningitis etc., have all been mistaken for heat-exhaustion. In coronary thrombosis, there is usually a well marked præcordial distress or a shooting pain towards the left shoulder or axilla. Marked arrhythmia of the heart is also evident, specially of the type of auricular fibrillation, and cyanosis with dyspnœa and orthopnœa (when the patient is seated) are present.

Partial heart-block, either due to damaged myocardium or cardiovascular syphilis or any other cause, might be mistaken for heat-exhaustion. Quick recovery of the pulse in genuine cases of the latter, resumption of the normal cardiac rhythm, and the circumstances in which the attack of syncope occurred and the previous clinical history of the patient, may help in the diagnosis.

Narcotic-poisoning cases, specially with opium, may and do cause difficulty in diagnosis. In opium-poisoning, contracted pupils, stertorous breathing and profound coma are the unmistakable signs, and the smell of opium may be detected.

In diabetic coma, the fruity odour of the Aceto-acetic acid and B-hydroxy-butyric acid, in the breath of the patient, may be detected. The coma is deeper than the partial loss of consciousness seen in uncomplicated cases of heat-exhaustion. Previous history of diabetes, if it can be elicited, may help in diagnosis.

As for head-injuries, with concussion of the brain, the history of injury or the signs thereof, will be a help.

Uraemia generally causes stertorous breathing at first and later on, the breathing assumes the characteristics of the Cheyne-Stokes type. Total suppression of urine, precedent to the attack of fainting, will help in the diagnosis, if such a history could be elicited. The pulse is rather heightened in uraemia, though very irregular.

Algid malaria is really very difficult to distinguish from heat-prostration. An immediate examination of the blood may not be possible always. And even if possible, it may not help the diagnosis, if the results are negative to malarial parasites. Previous attacks of shivering and fever, if it can be elicited from the history, may help. Enlarged spleen, if present, will afford a good deal of help.

Cerebro-spinal meningitis can be easily distinguished by the presence of retraction of the head and rigidity of the neck. Kernig's sign can be elicited. High temperature and unequal pupils, with exaggerated respirations and deep coma, also afford diagnostic assistance.

TREATMENT. (Immediate):—Remove the patient from the environment, in which he got the attack, to a cooler and preferably a darker and quieter place. Strip off all the tight clothings, especially those around the neck

and the chest. Dash some cold water on to the face, and fan the patient. Administer stimulants, Ammonia to the nostrils, and Camphor-in-ether or oil, subcutaneously or better still, if available, Coramine in doses of 1 c.c. every half an hour, until the heart regains its normal rate and rhythm and the patient feels better. Friction with massage and warm-packs all over the body (to raise the temperature of the body to the normal) may be necessary, if the temperature remains subnormal. Rectal temperature will be a better guide in these cases. Needless to add, absolute rest in bed is necessary for a day or two after the attack. On recovering from the attack, splitting head-ache may require treatment. Avoid coal-tar products at any cost. Pyramidon or Anacin tablets may be given for the head-ache. Spt. Ammonia Aromatic with Coramine or Caffeine Citras and Tr. of Indian Ephedrine with or without Tr. Strophanthus may be administered for 3 or 4 days after the attack.

Mediate:—The patient should be advised to avoid muscular exertions during the middle of the day; unsuitable tight clothings, over-eating, consumption of alcoholic drinks and narcotics are to be avoided at least during the hot hours of the day, and also direct exposure to the sun's rays or a stay in stuffy, over-crowded rooms for a long time, as far as possible. One attack generally predisposes to others and hence the necessity for the after-care and mediate treatment.

Heat-cramp.—This is a pathological condition, characterised by a transient attack of myokymia and myotonia, lasting usually for not more than 24 hours and caused by exposure to high temperature, atmospheric or occupational, the severity of the symptoms and signs depending upon the degree and duration of exposure to the high temperature and the height of the temperature itself. The intensity of the muscular activities undertaken, has of course, a very important bearing on the production of this condition. Such workers as furnacemen, smelters, welders, glass-blowers, tin-workers and stokers as well as fire-men are apt to suffer from this malady.

SYMPTOMS AND SIGNS:—Muscular spasms like those in tetany, start suddenly and the patient winces with pain. The fingers are affected first, and later on, the calves. Gradually, the spasms involve the thighs and abdominal muscles also. Phantom-tumours may also appear in the course of the rectus abdominis muscles and may be mistaken for intestinal cramps induced by drinking a large quantity of cold water suddenly after enduring a prolonged thirst. In the intervals between the spasms, a tonic condition of the affected muscles may be noticed. Even after the attack subsides, spasms may be induced for a day or two by gentle stimulation of the muscles. During the attack, there may be a slight pyrexia also. If the cramps involve the cardiac musculature, death may ensue; or if the accessory muscles of respiration are involved, respiratory embarrassment may be caused.

DIAGNOSIS:—This condition can be diagnosed from tetany, by the nature of the spasm and the occupation of the sufferer. Tetany, usually involves one particular group of muscles, say the upper extremities only at a time, whereas in heat-cramps, several groups of muscles are involved. Tetany in children usually induces convulsions also, whereas heat cramps are met with in adults alone. The muscle-movements in heat-cramps are fibrillary or twitching in character and recur in short spasms, whereas, in tetany, they are contractile and tonic in character. No sensory changes or exaggerated reflexes are seen in heat-cramps and this may be sufficient to

diagnose it from chorea, disseminated sclerosis and alcoholic tremors etc. From tetanus, it can be differentiated easily by the absence of lock-jaw and the nature of the spasms.

TREATMENT:—Dehydration of the muscles caused by continued exposure to high temperature plus some other toxic product produced internally, appear to be the causative factors and treatment should aim at the removal of both the factors. To afford immediate relief, rest, warm-bath (to relax the affected muscles) and mild sedatives may be tried. Later on, intravenous or subcutaneous Glucose cum Saline may be given to remedy the dehydration.

AFTER-TREATMENT:—A change of occupation, if possible, or the intake of frequent draughts of citreous drinks with *Glucose* and increased Calcium intake may all be tried to prevent a recurrence of the cramps, after recovery from the first attack. Mild sedatives may be taken to lessen the excitability of the muscles. Avoid all wind-producing food-stuffs and prolonged muscular efforts.

Heat-stroke or Sun-stroke.—(Synonyms) *Siriasis*, insolation, thermic fever etc.

DEFINITION:—An acute disease with a sudden onset characterised by hyperpyrexia and shock, brought about by exposure to direct solar-heat or sun's rays, and high atmospheric temperature. From the definition, it will be evident that heat-stroke is only a severer manifestation of the effects of exposure to direct sun's rays and high atmospheric temperature; or in other words, a severer type of heat-exhaustion brought on by long-continued exposure to the sun's rays directly. In this disease, the symptoms and signs, though varying from those of heat-exhaustion, have the same underlying pathological basis, as discussed fully in the introductory section of this note. Hyperpyrexia and shock, rather than syncope, appear to be the predominant clinical signs in this condition. Evidently, the thermic centre becomes defunct in heat-stroke, unlike in heat-exhaustion. To put it in a nutshell, heat-exhaustion is syncopal, while heat-stroke is hyperpyrexial, as well as shock-inducing.

PREDISPOSING CAUSES:—These have already been discussed in detail under heat-exhaustion and hold good in this disease as well. In the interest of space, they are not repeated. Habitual deficiency in fluid-intake acts as a very important predisposing factor in heat-stroke and this deserves special mention here.

SYMPTOMS AND SIGNS:—Usually, there are some prodromal signs and symptoms, headache, dizziness, frequent micturition, disturbances of vision, hot-skin and a feeling of some impending calamity or presentiment. The patient suddenly faints and falls down. The skin feels very hot and the face is flushed. If the individual has long, dark, bushy hairs on the scalp, the temperature of the head may be higher than that of the body. The temperature shoots up to 105° or even higher, say, up to 110° F. Further developments of the clinical signs and symptoms depend upon the previous general health of the sufferer and the system that is predominantly involved by the stroke. Usually, 3 well-defined types of cases are described:—(i) Asphyxial, (ii) Paralytic and (iii) Psychopathic. In the first type, cyanosis, stertorous breathing at first but later on, Cheyne-Stokes breathing, irregular pulse, and præcordial distress are present. In the second, deep coma with convulsions or fibrillary twitching of voluntary muscles; passage of urine and fæces involuntarily, unequal pupils, and suffused

face with stertorous breathing are the usual signs. In the third, delirium alternating with coma, delusions of persecution and suicidal tendencies with variable pupils are seen. Hyperpyrexia is a common sign for all the 3 types, and varying degrees of shock, brought about by dehydration, also seem to be present in all the 3 types of cases. The pulse which is at first rapid, full and bounding, becomes thready and uncountable later on. Respiration is deep, stertorous or sighing at first and later on, becomes Cheyne-Stokes, especially if the case is going to have a fatal end. Recovering cases, however, show a gradual return to the normal type of breathing. After the acute attack passes off, (it may be within a few hours or after 24 hours even) the sufferer gradually regains consciousness, but even then, impaired memory and depressed mental functions may persist for days together. And permanent amnesia may result in some cases, especially of the alcoholic or narcotic-addict class. Recovery generally depends upon the previous general health of the patient and upon the degree and duration of the exposure to solar-rays. Alcoholics, diabetics, narcotic-addicts, and sufferers from organic diseases of the heart, kidneys, lung, liver and cardiovascular syphilis, have invariably a bad prognosis. Even in otherwise fairly healthy subjects, the disease may prove fatal, if the exposure has been too long and if no immediate appropriate first aid be available.

DIAGNOSIS:—Most of the diseases, which can be mistaken for heat-stroke, have already been dealt with under 'heat-exhaustion' and as such are not repeated here. But there are a few other diseases that can be mistaken for heat-stroke and are worth mentioning. Hyper-pyrexial type of malignant malaria offers the greatest difficulty in diagnosis. The history of previous attacks of shivering and fever (if available) and the condition of the spleen are aids in diagnosis. Concussion of the brain may cause some difficulty but the history of the injury and other signs may help. Apoplexy also may cause confusion in diagnosis, but the age of the patient and one-sided facial paralysis together with deviation of the tongue to one side, will help in the diagnosis. Cerebral hæmorrhage of the pontine region, may cause real difficulty as it induces hyperpyrexia but in this case, the hyperpyrexia comes later on and coma is the first sign, whereas in heat-stroke, hyperpyrexia is present from the very commencement and precedes coma.

TREATMENT (Immediate):—Remove the patient at once to a cooler, shady and preferably darker place. Strip off all the clothings, especially those around the neck and chest. Apply bits of ice all over the body and rub in vigorously; or ice-cold water may be poured from a height of 4 or 5 ft. on to the forehead in a steady stream. Bathe the whole body with ice-cold water. A sheet, wet with dilute Alcohol, may be used to cover the whole body. Ice-cold wet packs also may be used all over the body and vigorous fanning will, by introducing a current of air, materially help to reduce the temperature. Ice-bag, if available, may be tied on to the scalp, if the individual has thick, long and dark, bushy, tufts of hair, retaining heat. It must be remembered that most of these cases of heat-stroke occur in places far away from hospital, and where immediate qualified medical help may not be available. First aid is, therefore, of very great importance.

Venesection may be necessary in asphyxial type of cases, to relieve the cardiac embarrassment. Sedatives may be required in the psychopathic type of cases to quieten the delirious patient. But the introduction of fluid to combat the shock, is necessary in all cases. In asphyxial

cases, the introduction of fluid should be done after venesection. Normal Saline with Glucose and 1 % to 2 % of Sodii Bicarbonas may be injected intravenously as in cholera. Usually, one to two litres would suffice, to restore the patient, and this should be followed by subsequent oral Glucose cum alkaline drinks or, if necessary, by subcutaneous infusion. Asphyxial cases may also need artificial respiration for some time. Oxygen, if available, may be administered, until the breathing becomes normal. In paralytic cases, the tongue needs attention: it should be pulled out and held, so that it may not retract and fall down on to the glottis. In psychopathic cases, the patient will have to be watched and prevented from committing suicide or injuring himself. The application of ice-baths, ice rubbing, ice-cap to the scalp and bathing the body with ice-cold water should be stopped when the temperature (rectal) comes down to 103° F. *and should not be continued thereafter*, as otherwise, collapse may result from a critical fall of temperature. If the temperature on the contrary still remains high, iced enemata may be tried.

Mediate:—After the acute phase passes off, which may be after a few hours to a few days, impaired memory and depressed mental functions, persistent headache and meningeal congestion will need appropriate treatment. Insanity may supervene, especially in the psychopathic type of cases. Hemiplegia, as after true apoplexy, may result in the paralytic type of cases. The asphyxial type of cases will need treatment as for passive congestion or decompensated cardiac failure; or cedema of the lungs may require urgent attention. Nervous sedatives, Digitalis, Caffeine, purgatives and diuretics may be administered according to the requirements of each case.

If possible, the sufferer should have a change to a cooler climate; avoid all mental and physical exertions for a few months, and should have only mild recreations. Strict attention to general hygiene, especially clothings, diet, fluid intake and avoidance of Alcohol or narcotics, is necessary. Copious drinks of citreous fluid should be encouraged and avoidance of direct contact with the sun's rays, especially during the hot hours of mid-day, by means of a light, cool head-gear, is necessary. Strenuous muscular or even mental activities should be prohibited during the hot midday hours. The clothings should be light and loose-fitting, so as to promote a free circulation of air inside and thereby effect quick evaporation of the perspiration.

Prognosis.—In alcoholics, narcotic-drug-addicts and in persons with organic diseases of the heart, lungs, liver and kidneys and in diabetics, the disease may prove fatal immediately. In otherwise healthy subjects, recovery ensues after a variable period, but loss of memory or impaired mental function or headache or vertigo may persist for a few months. One attack may predispose to further attacks also, if the individual continues to live and work in the same environment in which he first got the attack.

PREVENTION:—Avoidance of (a) strenuous muscular and mental activities during the hot hours of the day (b) tight and ill-fitting clothings (c) direct exposure to the sun's rays or continuous stay in a stuffy overcrowded room, (d) deficient fluid intake (e) gluttony (f) alcohol or any narcotics and (g) a clogged dirty skin, is necessary. In other-words, strict attention to general hygiene and avoidance of direct solar rays and solar heat in the shade, are the only preventive measures needed.

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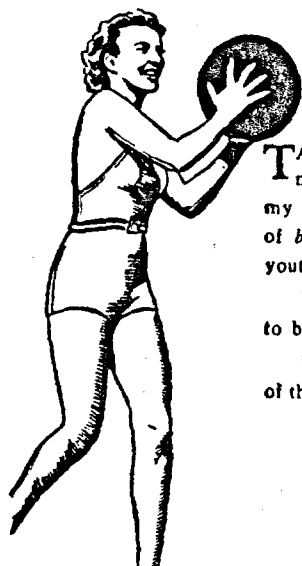
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Notes on Heat Effects

BY

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HEAT-STROKE includes various clinical entities, and it is not always easy to be satisfied as to heat being the only aetiological factor, determining the condition in certain patients; for, it is quite common to pass off any condition, occurring after exposure to great heat, as being due to heat, and proper clinical and laboratory investigations are not often carried out. As an instance, a child, who had been running about in the sun during the hottest part of the day, thus exposing herself to great heat, had developed increasing loss of appetite, a coated tongue, and when she began to vomit almost everything she took, she was examined by a doctor, who promptly took it as a condition of heat-stroke whereas a later examination showed it to be due to ascariasis. Such instances easily lead us to suppose that an infection (or infestation) and exposure to heat form a very unholy alliance. It is also a fact, as may be gathered from the incidence of heat-stroke amongst the army, that only about 2 to 3 per cent succumb, and necessarily, we have to look for individual predisposing factors which make him an easy prey. However, it may be said, at the same time, that there may be almost an epidemic form of incidence of heat-strokes, from mildest to the severest forms, during a sudden heat wave which suddenly bursts upon us like a *blitz*.

We all know the physiological considerations of heat effects. When the temperature of the atmosphere rises, we find that:—

- (1) Skin vessels react by dilatation.
- (2) Sweating takes place.

3. Internal viscera (brain and abdominal) generally diminish in size. These reactions all go to serve the heat-regulating mechanism of the hypothalamus so that by loss of heat, through conduction and evaporation, the body temperature is kept at the normal level in men (isothermic animals).

The role of thyroid, adrenal, pituitary, islets of Langerhans, is not very well known but that their work seems to become depressed is the opinion of some workers.

If, however, atmospheric humidity, clothing, stillness of air, inherent defect in sweat glands or intoxicants prevent proper evaporation and cooling, the increased supply of blood in the cutaneous dilated capillaries gets heated and leads to increase in pulse rate, respiration rate and an increase in the internal temperature. However, in the absence of any debilitating conditions and after only short exposures and particularly in the absence of physical exertions, the normal conditions become established soon, and biochemistry and metabolism are not deranged to any serious degree.

If, however, the body is exposed to dry heat as in a Turkish bath, there occurs a loss of weight through loss of fluid as sweat. But this does not lead to a concentration of the blood, as the fluid reserves of the body in the muscles, liver, interstitial spaces etc., are drawn upon. The role of the pituitary in this connection is problematic, but surely this is very important

and we may suppose that if the pituitary or the hypothalamus for that matter becomes deranged, this normal response may not hold and instead, a concentration of the blood takes place as is to be found in cases of heat-stroke.

So, we must suppose that besides the physiological reactions to dry or moist heat, there must occur certain other pathological conditions, affecting either the nervous system or the glands of internal secretion or processes of metabolism and excretions for bringing about such serious and frequent fatal consequences.

Incidence.—Cases generally occur during the hottest months of the year and begin to appear from April to July, but here in Bankura, a single shower cools the temperature so effectively that in June and July, incidence of cases falls very quickly. It is in April and May that the largest number of cases occur in contradistinction to other places, like the trans-gangetic plains where June and July are the worst months. Some of the cases, however, might have been really cases of meningitis, cerebral malaria, syphilis, and apoplexy, as two of the cases turned out, one due to encephalitis following an arsenical injection in a case of syphilis, and the other due to cerebro-spinal meningitis.

Predisposing causes.—1. *Alcohol* is certainly one of the important ones; it not only predisposes, but also makes the prognosis of these cases worse than that of others.

2. *Opium* is another very important cause.

3. *Belladonna*.

4. *Any fever, particularly malaria*:—In this connection, one may recapitulate that a patient with pyrexia, if exposed to the direct rays of the sun, may turn for the worse whereas, people with artificially cooled bodies, may be exposed to the most severe heat without any effect.

5. *Severe exertion* with uncomfortable clothing which prevents proper cooling of the body by evaporation of the sweat.

6. *Want of proper drinking supply*:—In this connection, the role of NaCl should be remembered.

7. *Heart disease*:—We know that (a) any increase in temperature of the blood increases the pulse rate and (b) that for the proper maintenance of the circulation through the dilated cutaneous capillaries, the heart must pump well to keep the heat-regulatory mechanism effective and (c) that the increased temperature of the blood may actually bring about changes of the heart muscle and a temperature of 115° F. may coagulate it, (d) and that metabolism is actually increased in cases of heart disease.

8. *Vascular disease*:—From similar considerations, we see that unless there is proper supply to the skin vessels and unless the elasticity of the arteries remains good enough for the maintenance of proper circulation, the regulatory mechanism may fail quickly.

9. *Kidney disease*:—Like chronic interstitial nephritis, malignant hypertension, senile kidney which cannot work at a pressure, and conditions of uræmia stand heat very badly. This leads us to suppose that either the disturbance of the acid base balance or the perverted metabolism of proteins due to heat proves a heavy burden for the kidney. Want of proper supply of chlorides may also take place. In this connection, I like

to mention the popular belief that taking tea is a preventive against heat effects. It is, however, known that Caffein (or Thein) of tea increases elimination of chlorides, even if the body is in a negative balance as to chlorides, as when undergoing a salt-free diet, and besides, hot fluids only help to add to the heat, increase sweating i.e. loss of more fluid and chloride than taken. I should think, therefore, that there is nothing, if not danger, behind this popular belief.

10. *Intestinal parasites*:—Ascariasis appears to be a very much predisposing cause amongst children. It is not uncommon at all to see heat effects amongst children and it is remarkable that, amongst a group of children who fall victim to heat, a history of ascariasis is frequent. I cannot, however, say that any predisposing cause obtains in cases of pin-worms and tape-worms.

11. *Lung diseases*:—We know that hyperpnœa and even dyspnœa follow exposure to heat, and that the heat-regulatory mechanism partly works through the respiration. We may also suppose that the accumulation of Lactic acid requires more Oxygen, and if the lungs, disabled through emphysema, adhesions, pneumoconiosis etc., cannot do their job properly, effects of heat may manifest themselves quicker. I have not, however, been able to detect evidences of heat effects amongst tubercular patients who may rather improve in these hot days, but it is common knowledge that cases of chronic bronchitis, congenital cystic disease or bronchiectasis and emphysema fare better in summer than in winter. In a series of post-mortems on heat-stroke, an incidence of 36% of pleural adhesions were detected, but in any other series, not including heat strokes, we find these adhesions quite as frequently. We cannot regard adhesions as predisposing to, or as an effect of, heat stroke. It is remarkable, however, that a sudden increase of attacks of the so-called "cold" "influenza" "rhinitis" becomes always evident during a heat-wave. In this connection, the reaction of the nasal mucous membrane to infra-red heat, as shown by L-HILL, may be remembered and may go to explain some attacks of nasal congestion which is very frequent at this time of the year. That all of them cannot be reflex but are certainly due to irritation by dust and infection, I have no doubt.

12. *Diabetics* do not stand heat well and their condition generally gets worse with greater danger of ketosis and coma. It may be supposed that the internal glands are depressed as a result of heat effect aggravating the diabetic condition.

Physical signs.—The appearance of the face in cases of heat-exhaustion is pale, shrunken, bedewed with sweat, but in other cases, the face may appear to be suffused or even cyanosed, and the eyes may be extremely congested. The pulse is generally fast and of low tension, but in cases of pyrexial forms, it may be bounding. The tension, however, need not be low in all cases. Respiration is generally irregular, deep and sighing, unless he is comatose, when it is stertorous. The heart generally does not show any enlargement unless it is actually dilating before terminal failure. The sounds are anything if not accentuated but the mitral first sound may lose its proper tone and it may be gathered from the sound that the left ventricle may be driving less blood than it should as in cases of mitral stenosis. The neck veins and the liver show congestion as also the lungs, pointing to a failure of both sides of the heart at the same time. Cyanosis is frequently present.

The reflexes.—Pupils may remain contracted but some are found dilated before death. The light reflex remains even if sluggish. Corneal reflex is generally present unless sometime before exitus lithalis. The superficial abdominal reflexes are generally absent. Knee jerks may be exaggerated but frequently lost. It is said that loss of knee jerks is one of the earliest manifestations, but I have not been able to corroborate it. I have found it exaggerated in early cases and only in grave cases, it is found lost. In my own personal case, as a result of working hard in a very hot day in unsuitable clothing, I had almost all the premonitory symptoms as (1) scanty, difficult, high-coloured urination (2) a sense of suffocation (3) a bad headache (4) a general restlessness (5) unquenchable thirst and (6) no taste for food. I could feel that everything was dry inside me and even the breath was hot and dry and the eyes were dry and burning. At this stage, I found that my knee jerks were exaggerated than lost. A Babinski may occasionally be obtained, but on this point it may be said that the sole is generally found so thick that no response is almost the rule.

Two findings should be emphasised: (1) Low blood pressure and (2) low urinary chlorides. For finding low or absent chlorides, the ordinary qualitative examination generally suffices. The addition of Silver Nitrate solution (3%) to filtered and albumin-free urine to which concentrated Nitric acid has been added generally shows only an opacity when the chlorides are low. The chlorides may be altogether absent. The urinary findings may be varied and may show, in bad cases, the presence of albumin and some casts. Indican may be detected. I suppose that the ketone bodies may only appear if the body supplies fat for burning in the absence of properly metabolised carbohydrate. Therefore, their presence should lead us to examine for hyper-glycaemia as indicative of disorder of carbohydrate metabolism and should point to the pancreas, the pituitary etc. Sugar itself may occasionally be detected but these are generally comatose cases or in convulsions. Uric acid and Calcium oxalate crystals may be found. The blood pressure may, however, be normal, particularly afterwards.

The blood in bad cases may show a concentration so that there may be polycythaemia, counts of over five millions not being unusual. The percentage of Hb may be like 100% or over. A leucocytosis in the beginning (counts of 10 to 15 thousand) is detected (which later falls off) with no significant increase in the differential count.

The hæmorrhages should lead us to investigate the platelet count (which appeared to be much below normal in these cases) coagulation time, fibrinogen contents etc. Loss of coagulability of the blood (GIBON) is a striking feature.

Cerebrospinal fluid is generally under increased pressure and on examination may show some increase of the albumin content. The cell content shows at first a poly-morphonuclear increase. Later, the cell content may show an increase in the lymphocyte like cells. The fluid is clear and does not form any coagulum on standing. The sugar appears to be diminished. The urea and chloride content have not been estimated.

Cramp, if present, is a very valuable corroborative sign. It is not to be mistaken for fibrillary twitchings, live flesh or myoclonus.

The blood should always be examined for malarial parasites and a Widal examination is occasionally necessary.

Lungs may show all kinds of rales and rhonchi which are generally bilateral and dispersed towards the bases. The breath sound may be weak or even absent. But occasionally, there may be localised patches of weak breath sounds with persistent crepitations with slight or no change after coughing and resonant rhonchi which prove that they are areas of congestion leading to consolidation. Slight dulness may be found also over these places, indicating areas of collapse with secondary infection or adhesion.

Occasionally, we find acute emphysema of the lungs with hyper-resonance and rhonchi and rales scattered here and there with cyanosed face and prominent neck and scalp veins. The heart in these emphysematous cases does not appear to be dilated nor the sounds weak so that it may be concluded that sometimes the lungs are responsible for increased venous pressure.

The liver, which might have been perfectly normal, may suddenly begin to get enlarged, hard and may appear to form a tumour across the upper part of the abdomen. The intestines generally are found tympanitic.

The spleen may be palpated occasionally but in these cases it is better to look for some acute infection, particularly malaria. But it should be remembered, at the same time, that malaria itself predisposes to serious heat effects, and finding of the parasite should not exclude the probability of heat effects producing a complex condition and the therapy should, therefore, be moulded accordingly. It may be noted in this connection, that ordinary thin film examination may prove negative for a few days sometimes in these serious cases of heat effects with malaria, and may lead to a serious error so that a thick and thin film examination, following KNOWLES' technique should always be done for at least two films taken at least twelve hours apart; for I have frequently noted that whereas one film in cases of malignant malaria, taken at a particular phase of the fever, does not show the parasites, another film taken some hours afterwards (*i.e.* during a different stage of parasites) demonstrates their presence. It may be that the free sporozoites in the blood are more difficult to detect than the intracorpuseular forms which are a little more definite in outline and have a definite relation with the red cells (*accolle* or *intra*).

Post-mortem.—The postmortem appearance of cases, dying of heat effects, generally show a generalised condition of congestion throughout all organs of the body with even petechial hæmorrhages in serous membranes, muscles and subcutaneous tissues. MAC COLLUM concludes from these appearances that "these features together with acute splenic tumour suggest the type of changes usually found in an acute infection," and that nothing definite can be found to explain such a death. We may say, however, that the theory of an acute bacterial infection (SAMBON) has long been discarded.

The blood is found dark and fluid in veins and the heart contracted in systole and empty. The acute congestion in the meninges, oedema and petechial hæmorrhages in the brain are other important features, particularly marked in cases of sun-traumatism. The findings may be interpreted as distinctive evidences of a lepto-meningo-encephalitis. But why evidences of an inflammatory reaction will be found only in the brain and only evidences of congestion and degeneration in other organs is difficult to explain.

Two cases of heat-stroke which came under autopsy, conducted by DR. P. CHOUDHURY, M.B., Civil Surgeon, Bankura, who kindly supplied me with the necessary P.M. appearances of viscera and kindly allowed me to publish these findings in my article, are given below.

Case No. 1. A.B. Hindu male, age 50, admitted to hospital at 12 noon on 1-4-41 and died at 1-30 p.m. the same day.

SYMPTOMS:—Comatose stage, conjunctivae congested, pupil contracted, pulse rapid and weak; patient restless; temperature 99.6° F. (axillary); respiration deep and hurried; slight convulsive movement of face and arms; spleen and liver normal; no M.P. found.

P. M. appearances (at 12 noon 2-4-41):—R.M. present in lower limbs; body poorly nourished; brain congested with a layer of coagulated lymph on it; sanious fluid came out on opening the membrane; both lungs congested and adhesions present. Pericardium contained half an ounce of sanious fluid; heart both sides empty; right ventricle contained two A.M. clots; stomach contained a few ounces of blackish fluid with a few particles of digested rice and chillies; small intestine empty; large intestine thick faecal matter; liver congested; gall bladder full; kidneys congested; urinary bladder empty.

Case No. 2. A.C. Hindu male, age 45, admitted on 13-4-41. (7-45 p.m.) died on 14-4-41 at 10 a.m.

SYMPTOMS:—Unconscious; conjunctivae congested, pupil contracted. No corneal reflex; pulse bounding; respiration deep and jerky; temperature 101.6 F. (axillary); restless; liver and spleen normal; no M.P. found.

P. M. appearances (at 5 p.m. 14-4-41): body poorly nourished; scalp congested; membranes highly congested with coagulated lymph in certain places; brain highly congested; lungs slightly congested; left heart empty and contracted; right side full of A.M. clots and contracted; stomach contained about three ounces of slightly yellow fluid. Intestine distended with gas and faeces; liver, highly congested; gall bladder seemed enlarged and distended; spleen congested; kidney congested; urinary bladder empty.

Neither the condition of the adrenals nor the histopathology was noted. Cramer's work on the adrenals in such a condition is highly stimulating and suggestive. The microscopic changes will be—if they are only due to heat effects—certainly of the nature of coagulation and necessary changes afterwards, but the change in the lipid content of the cortex is not easily understood. Can it be an acute failure of the cortical hormone?

Pathogenesis and biochemistry.—The theory of a bacterial infection is no longer in favour, as various considerations and negative cultures go to show.

It is being regarded more and more as a condition of auto-intoxication either—

(1) due to a perverted protein metabolism or,

(2) due to a failure on the part of the excretory channels, particularly the kidneys, to properly function and so get rid of the noxious by-products of protein catabolism,

(3) or a combination of both.

(4) It is not improbable that the symptoms of heat effects are

due to a depressed state of the centres of the brain and a consequent change in the water, chloride and metabolism as a whole, and as the control goes out of order, everything falls foul with the ordinary economy of the organism, leading to changes in all organs and particularly in the thyroid-adrenal-pituitary apparatus. MAC COLLUM thinks that "the centres of the medulla are directly impaired and that the extreme hyperpyrexia which follows the disordering of the regulating mechanism as well as the disturbances of respiration and circulation are of central origin."

(5) I think that the glands of internal secretion have a very important role and either the pituitary or the adrenal cortex has much to do with the pathogenesis. It is well known that when the adrenal cortical hormone is cut off, the changes like (a) blood concentration (b) rise of urea and non-protein nitrogen (c) a decrease of the plasma bicarbonate (d) some rise in the pH concentration of the blood, are found. They are exactly what happen also in a condition of heat-stroke.

The bio-chemical changes in the blood that occur in cases of heat-stroke are as follows:—

(a) Dehydration and concentration of blood as demonstrated by R. B. C. count and Hb percentage.

(b) An occasional rise of the urea and non-protein Nitrogen content of blood.

(c) A decrease of the plasma bi-carbonate.

(d) pH value of blood increases as due to accumulation of Lactic acid.

These are an exact replica of adrenalectomy picture.

A great difference, however, exists in that, in adrenalectomy the metabolism gets very low with a low temperature, but in heat effects, though a low temperature and characteristic muscular weakness, low B.P., nausea, and vomiting of adrenalectomy may be seen in heat-exhaustion, we never can explain the hyperpyrexia as due to adrenal failure. So that a combined disorder of the hypothalamous and adrenal cortex seems to be necessary.

It may be remembered that after Peptone intravenous injection, we have hyperpyrexia, loss of coagulability of blood which resemble effects of heat and this gives colour to the hypothesis of auto-intoxication by intermediary products of the protein metabolism.

(6) Water intoxication. Though it is true that some of the symptoms of water intoxication like anorexia, nausea, vomiting, headache, convulsion, coma and the intra-cranial pathology like oedema of brain and increase of intra-cranial pressure resemble those of heat effects and heat cramps are certainly due to increased intake of water and huge loss of chlorides, we do not find the same bio-chemical changes, dehydration etc. in water intoxication.

Clinical picture.—The clinical picture of heat effects is too well known for repetition and is ordinarily divided into hyperpyrexial exhaustion and sun-trammatism forms. But here, I like to stress upon some of the premonitory symptoms which are more important for taking proper precaution against the more serious sequelae which may be very fatal in the hyperpyrexial forms.

Premonitory early symptoms.—1. *Nervous symptoms*:—(a) *Dizziness*: It suddenly appears when lying on bed or, more commonly, when walking which may cause the patient to fall down wherever he might be. It is not only seen amongst people of advanced age of over 40 particularly, but also amongst adults; but children do not generally complain of dizziness. I suppose that it is not merely due to the effect of heat on the centres or of ionic imbalance upon the heart that such symptoms are caused, but the condition of the peripheral arteries determines to a great degree the incidence of such symptoms. The role of the adrenal cortex is not definitely agreed upon, and I have not tried the effect of Eschatin, Eucortone or any other such preparation on these patients as the cost is prohibitive and the people generally affected are of the indigent hospital class, who can never afford such medication. The dizziness may appear at any time of the day but, of course, it is particularly remarkable during the hot hours of the day. Dizziness due to arteriosclerosis of the brain vessels or hyperpiesia becomes highly exaggerated during these months of the year and may become so severe as to incapacitate the person, confining him to bed. People addicted to Alcohol, Opium, Belladonna, Cannabis Indica, suffer most from dizziness.

(b) *Headache*:—This may be of any degree of severity and may be occipital, temporal, frontal, and patients describe it in various ways. It may be accompanied with nausea, dizziness and some dimness of vision and its bursting character worsened by the coughing, straining, crying, laughing (QUECKENSTEDT's sign) irresistibly lead us to conjecture some increase in the intra-cranial pressure. It generally appears towards evening and after a night's rest, frequently disappears, but may continue for a few days, persisting throughout 24 hours or re-appearing every evening. But when the headache is of a persisting type, one has to examine the urine, the blood pressure, the jerks, the fundus oculi for proper therapy. Even then, some cases may persist for weeks with frequent exacerbations on exposure to heat or physical or even mental exertion. Gastro-intestinal disturbance is a frequent predisposing cause of headache in these cases.

(c) *Weakness and lethargy*, disinclination to work, both mentally and physically, and sleepiness are other early manifestations.

(d) *Restlessness and insomnia* are the two very important premonitory symptoms.

2. *Gastro-intestinal symptoms*:—A disinclination for solid food and an excessive thirst for cold drinks are the commonest of the premonitory digestive disturbances. But they may develop into anorexia, nausea and vomiting. A taste for acid fruits and acid drinks, is generally developed amongst people affected by heat.

An aggravation of any existing infection like dysentery, ascariasis, is always to be expected and necessary examinations are to be undertaken for proper diagnosis.

3. *Circulatory*:—A sense of heaviness, a distress, is sometimes complained of in or about the præcordium. It is more often felt on the left mammary region than in substernal area, and the heart does not show any abnormality as far as a clinical examination determines, but an occasional extra-systole may be found. The pressure is frequently low and may be unstable. But this is not the rule. A constant pressure of 130 to 140 mm. Hg.

systolic with 85 to 95 diastolic may show no variation for days even when the patient may be actually suffering from heat effects.

Besides blanching of face as a sign of collapse, a suffused appearance of the skin of the cheeks is quite frequently to be seen. The eyes may show injection and it may be a local reaction of the capillaries. There may be a peculiar reaction of these capillaries on the application of cold water to the head in that they become suddenly congested and may remain so dilated and congested for hours.

4. *Respiratory*:—Epistaxis is not an uncommon symptom amongst people exposed to the sun or heat. That sun or heat is only responsible in causing epistaxis cannot be definitely said. But the dry wind with its dust acts as an irritant to the nasal mucous membrane and so may precipitate an attack of epistaxis. Dr. HILL's contention mentioned before should be remembered in this connection. Whether there is any disturbance of the coagulability of the blood (GIBON) or platelet count or any damage to capillaries cannot be surely asserted. It occurs not only in children amongst whom it is quite common, and not due to local trauma, but also amongst adults and old people without arteriosclerosis or high pressure or kidney disease.

Dyspnoea and a sense of suffocation, are not uncommon. A remarkable case of acute dyspnoea, like the panting of a dog, except for short periods of apnoea in an inspiratory phase, resulting in cyanosis, distension of neck and head veins, acute emphysema of lungs, no clinical changes of the heart, were observed, and the patient told that such occasional attacks occurred only in these hot months of the year. There was no sweating at the time of during dyspnoea. The B.P. was normal (135-85). The urine showed no abnormality. Sodi Bi-carbonate in generous doses cured him quickly.

Frequency of micturition.—This is really a very important and a very common premonitory complaint. It appears almost at once after the exposure to heat or sun. The urine is generally very high coloured, small in quantity and may be just a few drops. There is a sense of discomfort or actual pain in the lower abdomen and in the perineum and is particularly felt during and after micturition. It is ordinarily known as "Rukshi" locally. At the end of micturition, the patient feels as if proper emptying has not been obtained and so may wait a good long time, straining to see that proper emptying takes place. These symptoms of urinary frequency, scanty urine, pain in the perineum and hypogastrium, tenesmus, burning in the urethra etc., may disappear during the night's rest only to re-appear the following day or they may appear only in the morning as the first symptoms. They may continue for a pretty long time provided undue exposure, muscular exertion, infection etc., do not precipitate a serious attack of hyperpyrexia. Pyelitis and lithiasis are not uncommon sequelæ.

Cramps.—Cramps of muscles like the calf muscles, abdominal muscles and muscles of the trunk and upper limbs are quite common, and we should always be cautious about them as indicating serious sequelæ. Miner's cramps are extremely painful and distressing to look at. They affect generally those muscles that are commonly used.

Dysphagia has been seen in a few cases.

Disorders of sweating as a certain failure of sweat are always ominous. It is almost pathognomonic. Skin eruptions like pemphigoid and herpetic

eruption are sometimes to be seen. But whether they denote any subsequent attack of heat-stroke or not is not determined. A smell almost musty is sometimes to be noted in certain cases.

Definitely developed pictures like heat-stroke, heat-exhaustion, sun-traumatism, are too well known for any repetition. Nor do I propose to enter into a consideration of the differential diagnosis of the various forms. However, I like to draw attention to some of the important sequelæ that may follow heat-effects.

1. Continuous dyspepsia with frequent attacks of diarrhoea may immediately follow or may appear after the lapse of a few days, following attacks of heat-effects. In these cases, no parasites nor any infection may be demonstrated. The cases gradually come round with careful dieting only.

2. Conditions of extreme exhaustion, so that the patient practically remains confined to bed, and may not move without severe sensation of dizziness and dissolution. These conditions are extremely slow to recover, and take months before the patients regain their former vigour of body and mind. These cases are generally young men in their thirties or early forties, who are strong, sturdy and very healthy and as such do not care a hang for anything and indulge in extremes of exertion and as a result fall victims. It appears that there is always some neuro-circulatory asthenia. In one such case, dysphagia was a remarkable feature and reduced him to undergo nasal feeding for proper nourishment. The blood pressure, however, when lying in bed showed normal figures (130-85 Hg mm.) in this case. Cases, however, may occur who never recover and remain as invalids for life.

3. Cases of so-called ardent fevers *i.e.* low remittent or intermittent fevers, following attacks of heat-effects, have not come under my observation. In a child, who had an attack of heat-stroke with convulsion, delirium, high temperature, jactations and who, a few days after recovery, began to run irregular temperature, generally not exceeding 101° F., I found a low grade *B. coli* infection. I suppose other cases may similarly be due to an infection from *Streptococci*, making the best of it when the resistance of the body gets lowered as due to heat-effects.

4. Cases of mania, manic depressive, insanity and the lesser forms of dementia, irritability with paraesthesiae may result after attacks of heat-effects. These cases may recover for a time but generally relapse after a repetition of exposure to heat or sun, and may remain so permanently. These cases I suppose are probably due to permanent changes, affecting the nervous system, particularly the cerebrum. Their general health does not usually suffer, but they live as invalids for the rest of their lives. Few cases recover sufficiently to drag on a hibernating existence.

5. Diabetes may follow attacks of heat effects. These are generally mild cases, but very often, become permanent after even any super-imposed mild infection like influenza, chicken-pox, measles etc. How-so-ever, it may be caused, that a general depression of internal glands has much to do with it cannot be doubted.

Aggravation of conditions obtaining anywhere, particularly nervous, circulatory, digestive, genito-urinary and cutaneous systems, are remarkable. Any pyrexial cases may be adversely effective. Cardiac failures and death, uraemic deaths, are quite common in these extremely hot days. The effect upon cases of liver disease did not appear to be so pronounced, though we

should suppose that liver, as the director of the board of metabolism, should have suffered most as a result of perverted conditions of heat-stroke.

Treatment.—As regards treatment of the fully developed cases, I do not propose to repeat the usual routine course. I want only to suggest that besides the usual therapy, the cortical extract may turn out to be a very valuable aid as a therapeutic measure in both conditions of exhaustion and hyperpyrexial and nervous forms.

As regards prophylaxis, I like to stress that besides measures for protection against sun and heat and the heat cottages and wards, (1) the diet should contain plenty of fruits to maintain the alkali reserve and vitamin C content of the body and less of proteins to guard against the intermediate products (Rubner's dynamic action) of metabolism and an extra quantity of salt against its depression. Incidentally, the role of Sodium Chloride and vitamin C may be remembered with regard to the proper functioning of the adrenal cortex. Vitamin B for its regulatory action on the gastrointestinal tract and its action upon the nervous system should be taken in proper quantity in the diets. (2) Exertion should be curtailed as far as possible during the hot part of the day. (3) Proper elimination through the skin, kidneys and bowels should be looked after and helped as occasion arises. Daily tepid baths and plenty of drinks like lemon squash are recommended.

The best prophylactic measure, of course, is to take flight to a cooler country against the hordes of heat Huns.

Summary.—1. Certain physiological reactions to heat are noted and it is suggested that these effects are probably due to a combined effect of both heat and some other debilitating conditions in the patients.

2. Some factors relating to pathogenesis are mentioned and therein a suggestion is made regarding the role of the adrenal cortex, probably working in combination with the hypothalamus, in giving rise to these conditions.

3. A suggestion regarding treatment with adrenal cortical hormone is made.

Heat-Stroke or Sun-Stroke (Sun-traumatism)

BY

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In this paper, I confine myself to the description of heat-stroke alone, leaving aside heat-exhaustion and heat-hyperpyrexia.

General.—The rays of the sun vary according to their wave lengths. The following is the table indicating their nature, visibility, action, and the diseases that are caused.

NATURE.	VISIBILITY.	ACTION.	DISEASES CAUSED.
Heat rays: 1. Infra-Red. 2. Red.	Invisible. Visible.	Heating.	{ Tropical liver. Heat stroke (heat-fever). Tropical anæmia. Tropical neurasthenia.
Luminous rays: 3. Orange. 4. Yellow. 5. Green.	Visible.	Chiefly illuminating.	Glare-headache. Night-blindness.
Chemical rays: 6. Blue. 7. Indigo. 8. Violet.	Visible.	Stimulating and anti-rachitic in moderate doses.	Solar dermatitis, pigmentation.
9. Ultra-violet.	Invisible.	Irritant, destructive of tissues in excessive doses.	Possibly heat-exhaustion.

This classification does not seem to be absolute. The blue and violet rays also play some part in illumination, though to a much lesser extent than the "luminous rays". The latter, in turn, produce heat, but to a much lesser extent than the red and the infra-red rays. The ultra-violet rays are tissue-stimulants when applied in moderation, and tissue-destroyers when applied in excess. They are of great value in preventing and curing rickets and osteomyelitis; they also promote bodily vigour and so are of value in preventing tuberculosis as well as other diseases associated with lowered bodily resistance. They are believed to damage the retina when it is exposed to excessive irradiation. Wearing ordinary glasses cuts out most of the ultra-violet rays, and, to prevent damage by glare, it is necessary to employ tinted glasses.

The prevailing idea that sun-light in the tropics can penetrate through considerable thickness of cloth or any other material is not true. Any substance which cuts the visible rays will also cut out most of the ultra-violet rays and so reduce them to negligible proportions. The real harm that is done by the rays of the tropical sun is that they have a powerful heating effect on the body, so that when a person is exposed to trying atmospheric conditions, the direct heating action of the rays not only add to his discomfort but also make him risk the chance of getting heat-stroke.

Heat-fever or heat-stroke is caused by climatic conditions, in

which the heat-regulating mechanism in man fails to keep the body temperature below the normal upper limit. HILL states that hard muscular work will give rise to heat production and, in a well-trained man, this does not give more than one-third of the energy consumed into work, 2/3 going into heat; whereas, in an ill-trained man, the energy is wasted in the form of heat. Sweat cools the body by evaporation and every gramme of water takes up some 582 calories of heat in turning it into vapour. Strenuous work necessitates the evaporation of approximately 1 pint of water per hour and these must be according to the in-take of water. JONES and MANKIN, in their study of submarine ventilation in tropical countries, showed that a high atmospheric temperature and a relative high humidity did cause discomfort if there was sufficient moisture and ventilation in the submarine. MARTIN, in his work on heat regulation in man, found that the mechanism is essentially an "adjustable insulation". MARSH, in his experiments on man, found that a complete saturation with water vapour at an atmospheric temperature of 90°F., causes an immediate and uncontrollable rise of body temperature. PEMBERTON recorded the case of a man born without sweat glands, but otherwise healthy; owing to abnormal rise in his temperature, he is unable to work in summer. People suffering from ichthyosis are very intolerant to high temperatures. It is evident that a man, when given an injection of Atropin, can easily be reduced to the same condition as a man born without sweat glands.

A few out-standing facts, which have a bearing on the problem of heat-stroke, are the following:—Heat production takes place in the body during life. The generation of heat may be little or more, but the heat-regulating mechanism in the medulla is responsible to maintain a constant temperature. In healthy persons, heat production and loss balance each other, irrespective of the temperature outside. The balance is adjusted by the action of the heat-regulating mechanism, which is controlled by the heat-regulating centre. In cold weather, heat-loss occurs through the cooling effect of the air on the surface of the body and the respiratory tract, while in hot weather, cooling is effected by evaporation from the skin which plays the more important part. In extremely hot weather, the outside air is sometimes hotter than the body. The sweat from the skin on evaporation counteracts the heating effect of the outside air.

HALDANES, in his experiments, showed:—(1) The influence of moisture in the air (it is the wet bulb temperature that counts; the actual height of the dry bulb thermometer does not matter until excessively high temperature is reached). (2) The influence of work; (3) The effect of air movement. ROGERS found that the mortality of the patients with a temperature of 107°F. was 8.3%; with a temperature of 107-109°F. was 29.2%, and over 109°F. was 69.2%. It is a matter of common observation that men, working in deep mines and in stokes of steam-ships without sun-light, get heat-strokes. The condition under which heat-stroke occurs shows that the disease affects human beings exactly in a similar way as in other animals. A frog suffers from heat-stroke when the temperature of the water rises to 104°F. This is due to the Lactic acid concentration in its muscles and blood, which paralyses the thermogenetic centre. Guinea-pigs succumb to heat-stroke when the shade temperature rises to 110°F.; rabbits when it reaches 116°F., and man 130°F. HALLIBURTON says that man succumbs if body temperature is kept at 108°F. for one hour.

Hyperpyrexia due to heat is complicated in man by the effect of

sweating and upon the concentration of the salts in the blood, so that even in a cool atmosphere, heavy manual labour causing profuse perspiration over long periods, when associated with deficient salt intake, produces severe symptoms which can be cured by salt ingestion. It is, therefore, possible that auto-intoxication and not suppression of sweat mechanism is the primary factor in the production of heat-stroke.

The term heat-stroke always conveys that the sun's heat is the leading ætiological factor. There are two other conditions which are to be borne in mind, that give rise to identical symptoms:—(1) Heat-exhaustion, which may occur in any climate, high atmospheric temperature being the chief ætiological factor, with symptoms of shock and collapse is found without fever. (2) Heat hyperpyrexia has a peculiar endemicity, characterised by sudden hyperpyrexia, coma and extreme pulmonary congestion and œdema. New comers to the endemic areas and Europeans are more liable than natives or residents of long-standing. Men over 40 years are more susceptible than those of younger age. (3) Sun-traumatism or sun-stroke results exclusively from exposure to direct sun's rays.

ROGERS analysed the fatal cases of heat-stroke recorded in India during several years. The monthly incidences were found to be: January to April -0; May 7; June 31; July 15; August 4; September 3; and October to December -0. In each locality, 3/4 of the fatal cases occur during the hottest month. HUTCHINSON made an analysis of cases of heat-stroke in soldiers in Allahabad over a series of years. He found that a great majority of cases occurred towards the end of prolonged spells of excessively hot weather. A few days of very hot weather can be tolerated, but if the heat wave continues for some weeks, surely some cases might end fatally towards the end of the period. Attempts have been made to find out the relative effect of the actinic rays and the heat rays. CASTELLANI exposed rabbits to direct sun's rays and found that they died in an hour, while rabbits exposed with protection of red glass survived. A considerable progress of heat rays are thus cut off by the red glass. When the animal bodies are kept cooled, they survive even when their heads are exposed to the sun's rays. Actinic rays themselves are of little importance, but the heat rays of the sun are much more dangerous. All this evidence goes to show that heat hyperpyrexia results from atmospheric conditions under which the heat-regulating mechanism becomes over-taxed and so fails to keep the body temperature low.

The following are conditions that conduce heat fever:—(1) High atmospheric temperature; (2) Plenty of humidity; (3) Stillness of air; (4) Warm and tight clothing; (5) Muscular exertion; (6) Alcohol; (7) Disease and bodily weakness; (8) Condition of the sweat glands; (9) Liquid in-take; (10) Diet; (11) Body build; (12) Age; (13) Acclimatization; (14) Race; (15) Over-crowding; and (16) Bad ventilation.

Heat-hyperpyrexia.—(Heat-stroke or thermic fever). It is an acute condition, developing in the presence of high atmospheric temperature with the sudden onset of hyperpyrexia, coma, pulmonary congestion, and œdema.

Geographical distribution.—Though it has been reported that heat-stroke is occurring in many countries, it is strange to note that careful investigation reveals that it is remarkably restricted in its distribution. A large proportion of cases reported to be due to heat-stroke are really

diseases such as cerebro-spinal fever, apoplexy, tuberculous meningitis, cerebral-malaria, alcoholism or some other phase of an acute disease.

Authenticated cases of heat-stroke are reported from great towns in the United States, the coast of the Gulf of Mexico, the valley of the Nile, the coasts of the Red Sea, Syria, Iraq, the valleys of the Indus and the Ganges, (none from South India) Lower Burma, S. E. China and the Queensland coast in Australia.

Ætiology.—Natives and residents of long-standing do not suffer from heat-stroke. Men of about 40 are more susceptible than those of younger age. Men are more liable to heat-stroke in consequence of their out-door work. There are several theories that have been advanced to account for heat hyperpyrexia. One theory is that super-heating of the blood by high temperature brings about the stroke. Auto-intoxication as evidenced by Indicanuria, Acetone and Diacetic acid in the urine are found to be present in 12% of the cases. (2) HEARNE points out that the onset of serious symptoms of heat-stroke are preceded by 48 hours of suppression of sweat as the sweat glands cease to function, with that the body temperature tends to adjust itself to that of the atmosphere, but in view of the increased respiratory and nitrogenous changes in the body, it is unable to adjust. Coma, convulsions and delirium occur when the body temperature reaches 108°F. The changes in the muscles are attributed to the coagulation of myocin. Marked dehydration raises the hæmoglobin percentage to 110%, with a corresponding rise in R. B. C. and leucocytes. Urea, sugar and Lactic-acid in blood rise while the chlorides and plasma bicarbonates fall. The earliest signs of diminution of the blood chlorides can be demonstrated by a rough test:—To 5 cc. of the urine of the patient, add 5 drops of concentrated Nitric acid and a few drops of 1% solution of Silver nitrate. Normally, a curdy white precipitate forms and, if no change occurs, the chlorides are entirely absent. This is a valuable, though a simple, test to find out if a person is getting a stroke.

Pathology.—A notable feature of a case of fatal heat-stroke is the early appearance of rigor mortis. The blood remains liquid, darker than usual, and feebly clots. The body temperature remains high for some time or it may actually rise for a little time after death. There is hyperæmia of all the viscera and congestion of all the dependent parts of the body. Sometimes, there are petechial hæmorrhages taking place in the skin, the mucous membrane and medulla oblongata, but the brain itself shows no important vascular or naked-eye changes. On microscopic examination, necrotic changes in the ganglion cells, chromatolysis of the nuclei, are found. The cerebrospinal fluid is clear and under pressure.

Symptoms.—Though heat-stroke comes on suddenly, it is very often preceded by a prodromal stage. It frequently develops independently of any direct exposure to the sun; not seldom, the attack comes on during the night. The early stage is ushered by giddiness, frequent passage of small quantities of urine, dryness and heat of the skin, and thirst. There is often a feeling of constriction of chest, with headache. These symptoms can be noticed even a day or two before the full development of the attack. Disinclination for exertion, pains in the limbs, drowsiness, mental confusion, sighing, anorexia, photophobia, nausea and perhaps vomiting, suppression of sweat, sometimes a sense of impending death, an hysterical tendency if exposed to heat and a quickened pulse can be found. The first

indication of something wrong with the person may be a short stage of restlessness, or possibly of wild delirium. This passes on to coma with high temperature, almost reaching 110° F. The reflexes are wholly absent and their return seems to be a favourable indication. WILL-COX describes 3 clinical types of heat-hyperpyrexia. (1) Gastric type (2) choleraic and (3) true hyperpyrexial. It is in the last type that cerebral manifestations are striking, such as coma, delirium and convulsions. A wild impulse to commit suicide, especially by drowning, is frequently met with, and the person may be mistaken as if he has been running "amock". The temperature in these individuals may rise to 113° F. for a short time and yet recover (MARSHAL). Active measures to lower the temperature must be taken very early to prevent immediate death. The immediate cause of death is failure of respiration. Persons may be unconscious for a day or two, and a partial recovery may be followed by relapse. In favourable cases, the disease terminates by crisis and convalescence rapid. Assuming that proper treatment is carried out, the patient is out of danger in a day or two. There may be irregular, low fever for several days. But, if the temperature does not rise above 102° F., there need not be any alarm.

Sequelæ.—In mild cases, there may not be any bad effects; but, if the case is severe, dementia may set in with loss of memory, sometimes irritability and intolerance of heat. In all severe cases, it prevents the person from carrying on responsible work, since the mental changes are often like those which occur in old men. Severe headache and convulsive seizures may persist for some weeks or even months.

Diagnosis.—Hyperpyrexia in an individual is sufficient to differentiate coma caused by cerebral malaria, uraemia, diabetes, alcohol, opium and similar toxic conditions. Carbon-monoxide and Hydrogen-sulphide poisoning may also be thought of. Pontine hæmorrhage, insensibility are accompanied by a febrile condition. Malaria, cerebro-spinal fever, early stages of eruptive fevers in children, cerebro-spinal meningitis, have all to be thought of in differential diagnosis, particularly in the tropics.

Prognosis.—Depends on the duration of the height of fever, previous state of the health of the patient and his habits of life. ROGERS in Calcutta found that patients, who were unconscious for more than 3 hours, never recovered, while those, who have been unconscious for less than 1½ hours, all recovered. The average mortality is about 25 to 40%, but with the early diagnosis and prompt treatment, there need not be any deaths at all. Alcoholism, debility, old age, co-existence of any other disease of heart, kidney or lung, complete loss of knee jerks, are all unfavourable features.

Treatment.—Energetic and prompt treatment must be carried out early. Loosen the clothes, place the patient in a cool place and spray iced-water over his body, and fan him vigorously. Ice water enemata are also recommended. Rectal temperature ought to be the guide in systematically lowering the temperature. The patient may be placed on a large bath towel soaked in cold water. Constant massage of the limbs should be done to maintain the circulation. Some people consider a tepid bath safer when the temperature has been brought under control. When rectal temperature comes down to 102° F., the patient should be placed in bed with an ice-cap on his head. Very close observation of temperature is needed until the patient's condition becomes normal. Any rise of temperature must be dealt with promptly.

OTHER LINES OF TREATMENT.—Stimulants, like Ether and Camphor are given if the pulse is feeble. Caffeine as a stimulant to the heart is also recommended. Free blood-letting is extremely useful in cases of cyanosis associated with convulsions. ROGERS suggests administration of intra-venous or rectal alkaline Saline in choleraic forms. As soon as the patient begins to swallow, frequent sips of water are to be given freely. Artificial respiration will be necessary in cases where breathing is laboured. Lumbar puncture is needed in cerebral cases to relieve intracranial pressure. The sweating might take place after 3 weeks.

TREATMENT DURING CONVALESCENCE.—The patient ought to be treated like a case of concussion of the brain (cerebral-concussion). Bowels should be moved by mild laxatives. Diet should be light and nutritious. All excitement and fatigue must be avoided for several weeks. Such cases may be sent to a cool place, and old people should be advised not to return to the tropics. RAWLING advocates sub-temporal decompression in all cases in which headache with convulsions persists for 3 months. These symptoms are due to cerebral oedema. Alcohol should be forbidden for a long time after recovery.

Prevention.—The most practical method of preventing heat-stroke is to watch the patient for symptoms of urinary irritability, restlessness and insomnia. A special room with a refrigerant apparatus ought to be an addition to hospital equipment in the tropics. Air-conditioned houses and dwelling rooms are commonly found in New York and other places, but they are solely an expense. During hot weather, exercises should be forbidden, plenty of water should be taken, good ventilation, wearing of light clothing, and protection of the head with an umbrella or a pith-hat or a big white turban, as you see in the Marwadies and Sikhs, are essential to prevent heat-stroke.

Child mortality is very high in Ellore, Bezwada, Hyderabad etc., during summer months. Probably, it is due to heat stroke. Prevention by such of the methods mentioned above is feasible.

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Sun-Stroke and Heat-Fever of the Summer Season

BY

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Introduction.—The summer is a dreadful season to the live world in the tropics. Animals as well as vegetations are hard put to it to withstand the scorching heat of the glazing mid-day sun of the summer. Animals often take cover and pant with open mouths to keep cool, by evaporation and ventilation. Plant life is maintained by a rapid circulation of its sap to prevent over-heating of the exposed surfaces. Luckily, the heat produces fall in barometric pressure in various centres, and consequent circulation of the wind. Had this been denied, life would, perhaps, have been impossible in the tropics. For days together, during the weary season, the maximum shade temperature rises fairly above 100°F. (110°F. to 120°F. even being reached), the minimum keeping above 80°F. The white-skinned races have to live very cautiously; but the natives with their coloured skins are inured to a great extent. They work with impunity and their cattle graze in the open field, or work with their masters, unmindful of the glare and heat. Nevertheless, cases do occur when man or animal, on strenuous exertion, sometimes, topples down to die in no time. A stroke it has been aptly called.

Definition.—*Sun-stroke, Heat-stroke, Siriasis, Heat-apoplexy, Insolation, Heat-asphyxia*, etc., are terms somewhat loosely applied to two main types of acute illness that follow undue exposure to the hot summer sun; the one is a hyperpyrexia and the other is a syncope, both of which very often rapidly end fatally. Heat-exhaustion is the syncopal form; and heat hyperpyrexia the febrile form. MANSON has made the following distinction between sun-stroke and heat-stroke. When the subject of the stroke was under cover before the attack, the affection is called heat-stroke; when the subject underwent exposure to the sun before the attack, it is called sun-stroke. There are endemic areas throughout the tropical belt of the world. Sometimes, strokes are so numerous that an epidemic may be declared.

Ætiology.—What is it that counts? Is it only the heat? Perhaps not; for, even the white man has been known to work strenuously in heated atmospheres, under modern conditions of industry, for hours without any ill effect. The engine rooms of steamships, trains, and factories, welding, smelting, glass-blowing etc., all kick up temperatures, in comparison to which the maximum shade temperature of the tropics wanes into insignificance. So, heat is not the only determining factor in an attack; otherwise, cases would have been much more frequent.

In addition to exposure to the sun, the body must be deprived of the power of its adaptability to the emergent condition in some way or other. Thus, it may be heavily and oppressively clad, as in the case of marching soldiers; or it may be dissipated by Alcohol, Hemp, or excessive strong tobacco, or other excesses; or it may be already weighed down by pre-existing disease conditions, like malaria, kala-azar, dysentery, tuberculosis, liver, kidney, or heart trouble.

Yet, the healthy, young, robust man is sometimes struck down for indiscretion which, at the time, the victim considered negligible. To cite a case,—a young medical student of the sixth year class, travelled the previous day by a mid-day train, finished his business for which he had to undergo exposure to the sun, and returned home at night. The next morning, at quarter to ten, he had to partake of his meal hastily and run for catching a train again, as he had business in another village. On alighting, he had to walk in the sun through open fields for some four miles. On reaching his destination, he felt very hot and oppressed, doused his head with cold water and drank freely of it. Within two hours, he started back in the sun to catch the returning afternoon train. Of course, he had no umbrella to protect his head, as the modern youth has ruled umbrella out of fashion. He felt gradually worse, and, on reaching the village station, he drank a lot of water and again washed his head and face. In the train, he felt almost overpowered by a sense of oppression, headache and restlessness. His eyes became red. He had two companions who took off his shirt, fanned him as best they could, and requisitioned water for him to drink and cool his head at every stoppage. When he alighted at 5 p.m., he was staggering and was helped to the nearest bench, where he lay down and immediately lost his senses. His physician found the temperature to be 108°F.; presently it rose to 110°F. Active measures to bring down the temperature were resorted to by a host of medical men, who did team work to revive him. But, within five hours of his arrival, he died. The temperature, when it came down at last, rapidly reached the lowest point in the thermometer and with it heart and respiration failed.

Pathogenesis.—Besides the heat rays of the sun, there are a host of other rays that probably act on the body banefully. These are some infra-red rays, the actinic and other ultra-violet rays. As has been shown by RONTGEN, rays of the ultra-violet series have penetrative action, while actinic rays produce chemical changes within tissues. In the Medical Annual 1934, it has been shown that infra-red rays have the power of producing sensations of discomfort and a feeling of dryness and stuffiness of the nose due to a reflex congestion of the nasal mucous membrane.

Much depends on the power of the body to perspire freely. When a condition known as heat stagnation occurs, the air immediately next to the skin and within the clothing and just outside it, fails to evaporate the perspired water, giving rise to a sensation of discomfort. But the discomfort produced by the direct rays of the sun is widely different. It is of a penetrating, burning, distracting and oppressive nature.

When a stroke of the hyperpyrexial type occurs, the sweating mechanism fails before-hand and perspiration is suppressed. The main source of heat adjustment is thus thrown out of gear, the body temperature rises by retention of the body heat and also by external heat, perhaps also by imbalanced heat production. Damage to internal organs is caused by the excessive heat and presently a condition of vasomotor paresis and a process of auto-intoxication set in.

But the syncopal form, which is marked by collapse and heart failure may, sometimes, be preceded by excessive sweating. Thus, just after exhausting toil in the sun, if a person drinks profusely of cold water, there is a risk of his perspiring profusely, which may lead on to collapse and prompt failure of the heart.

Notable features of morbid anatomy in heat-hyperpyrexia are early rigor mortis, engorgement of the venous system with the dark fluid blood; the lungs and other viscera are also engorged. The blood and muscles all yield an acid reaction. When death occurs at the height of fever, the temperature may still go on rising.

Nomenclature.—Under definition, the difference of sun-stroke from heat-stroke has been shown; and the two chief types of sun-stroke *e.g.*, the syncopal form and the hyperpyrexial form have been differentiated. Besides these, there is a subacute, distinct and frequent form of fever produced by the summer sun, that may be recognised as a disease entity under the caption "Heat fever of the summer season" or "Heat-fever" simply, or "Sun-stroke fever". MANSON BAHR has recognised a condition produced by the action of the direct rays of the sun on the human body that is characterised by fever, covering a wide range between the above two acute extremes *e.g.*, hyperpyrexia and syncope. He has styled it "Sun-traumatism", meaning evidently an injury produced by the sun. The title, when applied to fever, cannot be considered to be so very apt, when traumatism is actually produced by the sun, in the form of burns, freckles, cramps, etc.

Hence it is suggested that sun-stroke as a whole may be tabulated thus:—

I. Sun-stroke—acute:—(1) Syncopal form; (2) Hyperpyrexial form.

II. Sun-stroke—subacute:—(1) Heat-fever; (2) Sun-traumatism.

It would now be convenient to treat each of the above items separately.

I. **Sun-stroke—acute.**—(1) *Syncopal form*:—SYMPTOMS. It is ushered in by a sensation of sudden faintness after exposure to the hot summer sun. The patient feels giddy, staggers and falls down unconscious. This happens also to the toiling animal like the horse, the buffalo or the bullock. It sometimes also falls to the lot of the marching or fighting soldier to be thus afflicted. Death is not rare in such cases. The unconsciousness may not be deep or long-standing. The patient is pale and cold, the temperature being subnormal. The pulse is rapid, soft, small and flickering. The breathing is rapid and shallow. Cases that recover gradually come round,—being left in a condition of extreme prostration, depression, and with a headache. Cases that do not recover rapidly pass on to a condition of heart-failure or die immediately.

TREATMENT:—Stimulation should be the watch word in these cases. Patient should be laid on his back with loosened dresses in a cool shed; smelling salt held to his nose. If the patient can swallow, a dose of stimulant mixture may be given by mouth; if not, stimulant may be injected.

2. *The hyperpyrexial type*:—SYMPTOMS. In this type, the mortality is much higher. The actual rise of temperature is, in the majority of cases, preceded by suppression of sweat and prodromal feelings. A sense of oppression, burning, dryness of the mouth and throat, of some undefinable apprehension, headache, nausea, fatigue, etc., occupies the patient. Presently, the temperature begins to rise, the skin becomes dry and hot; the headache increases to giddiness and mental confusion; thirst becomes unquenchable; and there may be irritability of bladder. The eyes become red and suffused; the gait becomes staggering; there may be muscular twitchings

or spasms; and with the rise of temperature, the patient falls unconscious, with a low fluttering pulse and respiratory difficulty. Rapidly, the hyperpyrexia advances, the coma becomes complete, and the lungs show signs of congestion and œdema. The reflexes are lost, including the knee-jerk. Death may occur within a few hours or minutes at the height of temperature or with its fall.

The above is called the nervous type. A gastro-intestinal type is recognised, in which the axillary temperature may not rise at first while vomiting and purging set in. The resemblance to cholera proper is great. The rectal temperature rises secretly from the beginning and the axillary temperature follows suit, tending towards hyperpyrexia.

DIAGNOSIS:—In these cases, as well as in those of the nervous type, a difficulty often arises in differentiating the disease from malignant malaria. Immediate microscopical examination of the blood may be considered imperative. In the absence of facilities, reliance must be placed on detailed case history and other data.

TREATMENT:—Early, prompt and active treatment is essential. Steps should be taken to reduce the temperature not so much by direct ice action as by promoting evaporation. It should be remembered that failure of the sweating mechanism is the prime defect. The injection of Atropine is absolutely contra-indicated as it tends to check sweating. So also is Strychnine contra-indicated, as the disease tends to produce convulsions and spasms. The patient should be laid on his back in a cool, shady and airy place, stripped of all clothings, and vigorously fanned, while he is liberally bathed in iced water, and ice caps are put on his head. The backbone may be rubbed with ice to keep the spine cool. At other places, rubbing with ice does no good, as the skin is unnecessarily blanched thereby and evaporation is hindered.

However, in reducing the temperature, one word of caution is necessary. When the temperature begins to come down, readings should be taken from the rectum every few minutes and cold applications stopped, when the rectal temperature reaches between 103° and 104°F.; for, the downward progress continues, and in order to save the patient from dangerous collapse, stimulants should be employed at this stage. The patient should now be wiped dry and wrapped in a thick sheet or blanket. If sweating appears, it is a good sign. Impending respiratory failure can, sometimes, be checked by artificial respiration. In persisting very high temperature, ice enemata may be tried in addition to the above methods.

II. **Sun-stroke—sub-acute.**—1. *Heat-fever of the summer season*:—This is a sure disease entity that ought to receive definite recognition. In endemic areas, this is a regular annual disease produced by the hot summer sun. It differs from the previous extreme conditions in that it is devoid of the lightning-like emergency. The atmospheric heat and the direct rays of the sun are both to blame. Children suffer most; adults are also affected. But, from the habits of the adults, males suffer more often than females. And from the nature of their avocations, the peasants and their children suffer most. In fact, sun-stroke-fever or heat-fever is a recognised disease entity in the vocabulary of the peasants, though it has not yet been dealt with methodically in treatises on Tropical Medicine.

SYMPTOMS:—The onset is not sudden; or when it begins with

faintness or convulsions, these stop promptly and the child goes on suffering from the fever that was ushered in with these. The healthy child that ran about in the sun, or the healthy adult who over-worked in the sun, suffers from anorexia, langour, headache, and incidentally the skin is found to be hot. Sweating is often suppressed. A pungent burning sensation and restlessness supervene. There is thirst. The eyes get red; the headache increases, and convulsions may take place, or delirium may set in, or gradual stupor may supervene. The temperature rises; variously; in grave cases, it may rise up to 106° or above. Other systems show nothing extra-ordinary except some congestion at the bases of the lungs or some tympanites. Extreme cases occur at the years of sun-stroke epidemics. These may end fatally in about a week; otherwise, recovery is the rule. Most prominent symptoms are: the burning sensation and restlessness, headache and delirium and pungent dry skin. The patient may be semi-comatose. There may be photophobia and intolerance of sounds. Knee jerk is lost in cases that are unconscious. The pulse is full and rapid and in grave conditions it becomes small, soft and flickering. Vomiting or purging may or may not be present.

For morbid anatomy, congestion and irritation of the meninges are surmised.

TREATMENT:—The patient must be kept in a quiet, cool and darkened room. The head should be shaved and doused with cold water or cooled with ice application. The bowels, if not open, should be kept acting. Food should be bland and soothing. Diaphoretics, diuretics, Bromides, Urotropine, Sodii-benzoas, etc., are indicated. Later on, at the stage of convalescence, Iodides may be required or a tonic with Iron and Glycero-phosphates may be indicated.

But the key-note of the treatment is to induce sweating. For this purpose, hot vapour bath is effective. (In village practice, vapour bath is improvised thus: In an earthenware pot, water is boiled with a cover on its mouth. A hole is dug in a suitable place, or a receptacle prepared so

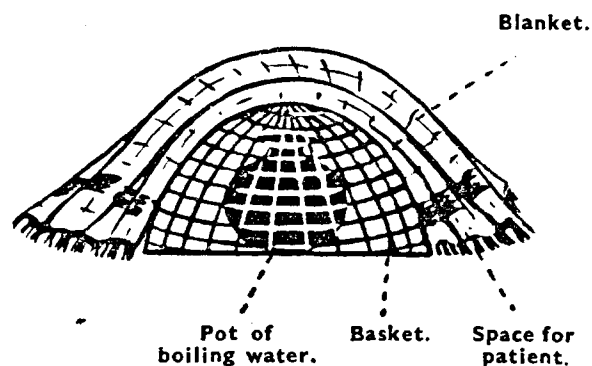


Diagram showing the improvised vapour bath apparatus.

as to hold the pot tightly. The pot with the boiling water, tightly covered, is placed in this place. A bamboo-work basket is placed on it so as to act as a fender. The whole apparatus together with the patient is now covered with a blanket or two, and then the mouth of the pot is uncovered. (*Vide illustration*). Vapour is emitted and sweating promptly follows, and

defervescence ensues. If sweating can be established, the patient feels great relief and the disease is easily controlled.

2. *Sun-traumatism*:—(a) *Sun-burns*:—The heat-rays and the light-rays of the sun act separately. Sun-burns are produced by the light rays and are, therefore, seen even in the coldest of countries. All stages of inflammation are met with, from erythema to blisters and deep ulceration. Europeans under the tropical sun are always affected by repeated and prolonged exposure to the rays. In the natives, burns may occasionally occur from excessive exposure.

The effect of the heat-rays is immediate, and act by dilatation of the cutaneous vessels producing erythema, (capillary congestion). The effect of the light-rays may not be immediate as is seen in the case of X-ray workers.

(b) *Freckles*:—These are discrete pigmented spots, occurring on those parts of the surface that are exposed to the sun, therefore, on the face and arms of the Europeans in the tropics. This is in fact an attempt of the body to protect itself from the actinic rays of the sun. It is this attempt of Nature that has produced coloured races in the tropics.

(c) *Heat cramps*:—Persons working in the heat may, sometimes, develop cramps of muscles, produced by exertion. Calf muscles, arm muscles and muscles of the abdomen may be affected. Fibrillary twitchings of muscles are also seen. These happen more prominently to workers under artificial heat in modern industries. Stokers of steamers are often affected.

Summary.—1. The summer sun is borne with difficulty in the tropics, both by the animal and the vegetable world. The main contrivance in all is to keep cool by rapid evaporation.

2. In respect of cooling, nature helps by producing fall of barometric pressure in various centres, which induces circulation of the wind. Rain storms and hail storms are also thus produced.

3. Sometimes, heat waves persist for days together, when the maximum shade temperature may reach even between 110° and 120° F. It is during these spells that sun-stroke cases generally occur.

4. Natives with their coloured skins, and their cattle and other animals, generally carry on their work in the open field, unmindful of the heat.

5. Plain and simple heat perhaps does not matter much, as even the European often works in very hot atmospheres under modern conditions of industry without any ill effect.

6. Cases generally occur amongst those whose health is already affected by disease, dissipation or deprived of its adaptability by unsuitable clothing, etc.

7. The indiscreet, healthy youth, who exerts much in the open sun with uncovered head, may be easily affected.

8. Sun-stroke is known by various names. MANSON makes the difference between sun-stroke and heat-stroke by stating that the one is

produced directly by the sun, while the other is produced by heat in the shade.

9. The various forms of the sun-stroke are tabulated.

10. Besides the heat-rays of the sun, some infra-red rays, actinic rays, and ultra-violet rays—all take part in producing the peculiar morbid conditions.

11. In the hyperpyrexial type, the primary and main defect is the suppression of perspiration. This is followed by a process of auto-intoxication, and vasomotor failure together with failure of respiration. Often respiration fails first.

12. Notable features of morbid anatomy are early rigor mortis, early decomposition, engorgement of venous system and viscera, acid reaction of blood and muscles.

13. Of the sub-acute type, heat fever has so far received scarce attention as a separate disease entity. An attempt has been made to give this type a definite shape and description under the caption "Heat Fever of the Summer Season".

14. MANSON calls an undefined group, as sun-traumatism. But that title is not considered suitable and actual forms of sun-traumatism have been grouped together.

Thanks are due to DR. SATYA KUMAR MULLICK, a village practitioner, for having frankly described his experience with heat-fever cases. The improvised vapour bath arrangement is his. Mention should also be made of the name of Master KALI DAS BHATTACHARYYA, the author's ten-year-old son, who has made the drawing of the vapour bath contrivance.

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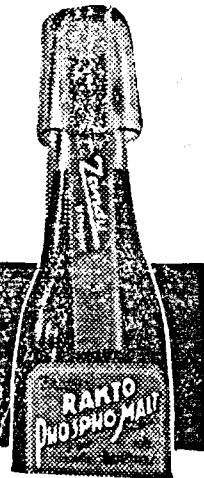
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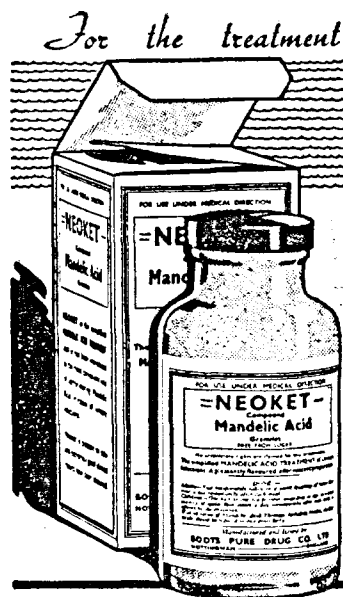
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Summer Diarrhoea in Children

BY

P. KUTUMBIAH, B.A., M.D., M.R.C.P. (LOND.),

Vizagapatam.

AMONGST the diseases which afflict infants and children during the summer months, the most dreaded is diarrhoea. The prevalence of this disease is not strictly confined to the summer months, as sporadic cases occur throughout the year. Summer is a long drawn affair in the plains of South India, and its duration depends on the success or failure of the monsoon. Under ordinary climatic conditions, the curve of incidence of this disease rises rapidly at the beginning of the second quarter of the year and reaches its highest point towards its close with a slight drop, if at all, in the third quarter, and then it begins to drop steeply in the fourth quarter of the year. When the monsoon fails, this drop is delayed and takes place at the close of the fourth quarter. In some years, a good number of cases occur in January also. February and March are the only two months which are comparatively free from this scourge of the hot summer.

Ætiology.—In its simplest form, diarrhoea is probably the result of a mild catarrh due to direct irritation of the intestinal mucous membrane. In most cases, this is brought about by indiscreet feeding of hand-fed babies; the common errors being either too large feeds or too strong feeds. Another common source of trouble is the preparation of feeds in bulk, and utilising them in small quantities during the course of the day. Fermentative changes take place in the mixture, and these give rise to irritation of the intestines. Another cause of simple diarrhoea is the chilling of the body surface. Children are particularly susceptible to changes produced by alteration in temperature. This is most likely to occur during the third quarter of the year, when the temperature is fairly high, the humidity very great, and sweating is very common. Sweating is a common symptom in children, suffering from rickets, and this disease is more likely to occur in hand-fed babies during the first two years of life, when diarrhoea also is very common. The high temperature prevailing during the summer months may itself initiate diarrhoea probably by inducing septic changes in the contents of the bowels. Heat seems to influence the bowel secretions so that they favour the multiplication of noxious organisms and increasing their capacity for mischief. It is now generally conceded that the severer forms of summer diarrhoea are due to infection as is shown by the constitutional symptoms like fever, toxæmia, restlessness, thirst and dehydration which are rarely absent in these forms. But in spite of much research, the bacteriology of summer diarrhoea is by no means clear. It would appear that all cases are not due to the same organism and amongst those, which have been isolated from the stools in different epidemics, are: (1) the *Proteus vulgaris*, (2) the *Bacillus coli* (3) the *Streptococci* (4) the *Bacillus enteritidis* (5) *Morgan's bacillus* (6) members of the *Para-typhoid* group (7) various strains of *Dysentery bacilli*. Any of these, or several of them in

combination, seem to be capable of inducing the disease, provided the predisposing conditions exist. The most important of the latter is a sustained high temperature (60° F.) of the air and particularly of the soil. BALLARD pointed out long ago that it is when the temperature recorded by the four-feet earth thermometer reaches 56° F. that cases begin to occur. Such a temperature probably provides the conditions necessary for the growth of the infecting organism. It is clear from the above observations that conditions favourable for the occurrence of summer diarrhoea are prevalent throughout the year in the plains of South India, where temperature below 60° F. is a rarity. The decline in the incidence of this disease during the first quarter of the year must be attributed to the rains, which lower the soil temperature below 56° F. and thus prevent the necessary growth of the infecting organism. Summer diarrhoea is more prevalent in large towns than in villages. This may be due to the unhygienic surroundings such as dirt, overcrowding, bad drainage and lack of ventilation. All these factors may play a certain part in lowering the resistance of the child and making it more susceptible to infection. The infection is most probably conveyed by dust, and there is no doubt that flies play a very important role as carriers of infection. In the worst cases, in addition to the toxins produced by the microbes, there is also a decomposition of the protein constituents of the milk and the formation of noxious chemical substances, which, when absorbed, aggravate the toxic symptoms. The relationship of the milder forms to the serious ones appears to be that the one predisposes to the other. The disease is more common in hand-fed infants. Children under five years of age are more susceptible to the disease and it is most fatal in the first year of life. Boys are more commonly affected than girls.

Very little work has been done on the bacteriology of summer diarrhoea in India. De. S. P. (1936) published a bacteriological study of 37 cases of diarrhoea, occurring during the months of June, July, August and September. He found that in 26 out of the 37 cases, some non-lactose fermentor could be isolated. Of the remaining 11 cases, in six some protozoa, either *Entamoeba histolytica* or *Giardia intestinalis* was present. In the remaining five, no possible aetiological agent could be found. In 73% of the cases, some bacteriological agent was responsible for the condition. *B. salmonella* group was present in 9 cases; Morgan's bacilli in five cases; *B. asiaticus* in five cases; bacilli of Flexner in 2 cases; *B. shigæ* in one case and *B. fæcalis* alkaligenes in 3 cases. He came to the conclusion that summer diarrhoea is a disease caused by bacterial infection and not purely due to dietetic irregularities. No one organism could be isolated and considered as the causative organism.

In the year 1939 and 1940, 591 cases were admitted into the Children's ward. Of these, 69 were admitted for diarrhoea, of which 69, 43 were boys and 26 girls. There were 12 deaths among these and 8 cases were discharged as moribund.

The following table gives the distribution of the cases according to the months for the years 1939 and 1940 :—

MONTH.	Total Number of admissions.		Total Number of cases of diarrhoea.		Deaths.	
	1939	1940	1939	1940	1939	1940
January ...	19	17	6	1	2	0
February ...	13	15	1	1	0	0
March ...	21	17	2	1	0	0
April ...	21	24	3	1	1	0
May ...	15	30	3	5	3	0
June ...	21	32	3	4	0	1
July ...	25	37	3	2	1	0
August ...	26	39	6	2	1	0
September ...	30	36	2	2	0	0
October ...	26	31	6	2	1	0
November ...	26	31	2	3	1	0
December ...	30	19	3	5	1	0
	273	328	40	29	11	1

It is clear from the above table that the incidence of diarrhoea in different years varies markedly. Whether the incidence is great or not is dependent on the prevailing range of temperature and the amount of rainfall during the year. In 1939, there was a complete failure of the monsoon and the temperatures were higher than normal throughout most of the months of the year. Accordingly, the incidence was great and the death rate was high. In 1940, there was a fairly good amount of rains and so, the incidence was very much less and the death rate low.

Morbid anatomy.—Very few lesions are seen in the intestines after death. Inflammatory redness and swelling, patches of false membrane, hæmorrhagic extravasations and more or less deep ulceration of the mucous membrane may all be present. Ulceration of the bowels is more common in sub-acute and chronic cases than in those which run a shorter course. In the "choleraic" form, the most marked changes are seen not in the intestines but in distant parts of the body. Anæmia of the brain and other organs is common, but occasionally, there is hyperaemia of the meninges and the cerebral cortex due probably to the action of microbes or their toxins. In the chronic forms, meningeal hæmorrhages or thrombosis of the dural sinuses may be present. Kidneys are often the seat of parenchymatous nephritis. The liver is often fatty. Patches of bronchopneumonia may be found in the lung.

Varieties.—For purposes of description, it is convenient to describe three varieties of acute diarrhoea in infants and children; (1) simple

non-inflammatory diarrhoea; (2) inflammatory diarrhoea—acute enterocolitis; (3) choleraic diarrhoea—infantile cholera.

1. *Simple non-inflammatory diarrhoea*.—This is a disorder of function due to mild intestinal catarrh, which generally yields readily to treatment. Symptoms may start suddenly or may be preceded by signs of indigestion and abdominal pain. The tongue is furred; there may be nausea or vomiting. The child is peevish and restless. There may be a rise of temperature which generally comes to normal, if the bowels are moved. Motion consists of faecal matter, containing small lumps of undigested fat; sometimes, motions may be sour-smelling or frothy from fermentation. When the lower portion of the bowels are involved, motions may contain visible mucus and this is accompanied by straining and occasionally with a streak of blood. Motions are often coloured green. The number of motions may vary from 5 to 12 in 24 hours. After the irritating matter has been discharged, the motions tend to subside. Generally, the whole condition lasts for a day or two. But, in hot weather, it may persist for a longer time and occasionally the condition may pass on into the more serious forms.

2. *Inflammatory diarrhoea (entero-colitis)*.—This is much more serious than the simple non-inflammatory diarrhoea. It may begin suddenly or gradually and may follow a mild attack of the non-inflammatory variety. Its chief features are a rise of temperature with frequent watery discharges from the bowels. There may be some abdominal pain. If there is any gastric catarrh, then the child vomits. The motions vary from 10 to 20 times a day and become thinner and thinner. They are very offensive and may vary in consistency and may be thin and pasty or frothy and dark, or consist of a greenish watery fluid. Accompanying the motions, there is fever from the first. The child wastes quickly and grows very weak. His face is pale, eyes get hollow, tongue may be a little furred but it is usually clean and red and inclined to be dry. The skin is dry and there may be reddening of the buttocks and back of the thighs from the irritation of the discharges from the bowels. The urinary output is scanty and anuria may supervene. Occasionally, the bladder is found to be full of urine but the urine is not discharged either due to atony of the bladder or spasm of the sphincter. In a few days, the depression of the child becomes extreme; the anterior fontanelle sinks deeper; pulse becomes quick and feeble; breathing hard and shallow; eyes are hollow and the lids are purple and close incompletely; face becomes livid; great thirst is noticed; tongue becomes dry and brown and may be covered with thrush; extremities get cold, though the rectal temperature may be high; when the larger bowel is involved, griping and straining become noticeable. There may be prolapse of rectum. Stools become gelatinous or contain mucus and may be streaked or stained with blood. In fatal cases, death is almost always preceded by the signs of general collapse; in some cases, drowsiness, deepening into coma, may intervene in the final stages and the infant passes into a stage of spurious hydrocephalus. Sometimes, convulsions precede death. The duration of the disease is usually short and comes to an end on the third or the fourth day. Of the patients who survive, some may improve quickly; others suffer for months and die at last of chronic intestinal derangement, complicated with intestinal ulceration.

3. *Choleraic diarrhoea (cholera infantum)*.—This is an extremely severe form of the above-described variety. It is more often met with in infancy

particularly below one year of age. It is characterised by its rapid course, grave symptoms and its too commonly fatal ending. It may attack apparently healthy and well nourished babies. The symptoms set in suddenly. Vomiting is early and persistent. The motions are thin, flocculent, and very offensive in the beginning and later, become serous in character, non-offensive, staining the clothes with a yellowish stain, with no trace of faecal matter. There is no griping or tenesmus. Dehydration is severe and rapid, and may set in during the course of a few hours. The patient is very restless in the beginning, but very soon sinks into a state of collapse. Finally, death is ushered in with either convulsions or hyperpyrexia.

Complications.—These are seen in more protracted cases and are of a bad prognostic significance. Simple and parasitic stomatitis are not rare and increase the difficulty of feeding the patient. Inflammation of the lung is often noticed; as the child becomes exhausted, collapse of the lung may occur. Paresis of the bowels with distension of the abdomen may supervene. Convulsions and coma may occur at any time during the course of the disease.

Diagnosis.—One has to exclude other fevers which start with digestive disturbances. Pneumonia and some of the eruptive fevers, particularly measles, may begin in this way. Careful examination of the child with a view to exclude these diseases must be made. Acute entero-colitis is not likely to be confused with enteric fever, as enteric fever is a rarity during infancy, while inflammatory diarrhoea is most common, and when enteric fever occurs in childhood, constipation and not diarrhoea is the rule. The seat of inflammation in the bowels may be often inferred from the appearance of the stools. The higher in the intestinal tract the inflammation is situated, the more intimate is the admixture of the mucus with the faecal contents of the bowels. If the catarrh is limited to the upper part of the bowel, no mucus is visible to the naked eye; but it can be detected by the microscope. If, under the microscope, the mucus is seen to be stained with bile, then, the jejunum and the ileum are also implicated. The presence of bile indicates also that there is increased peristalsis. If microscopic mucus is constantly present, then the lower bowel is involved. Pulpy stools, containing dabs and drops of free mucus, point to the colon and pure mucus in large quantities to the sigmoid flexure. Scybala embedded in mucus show catarrh of the rectum. In such cases, painful tenesmus is always present.

Prognosis.—Prognosis in all forms of inflammatory diarrhoea is always serious in infants under one year of age, especially in those who suffer from previous digestive disorders. The prospect is less bright in infancy than in later life, and is gloomy in proportion to the degree to which the nutrition of the patient is impaired. The rectal temperature is a good guide to the gravity of the child's condition. If it remains at or above 105° F. or shoots up suddenly from a low to a high level, the prognosis is bad. On the other hand, a fall in the temperature is a hopeful sign, and if the rectal temperature becomes normal, recovery is very probable. In addition to high temperature, other bad prognostic signs are persistence of vomiting, tendency to collapse, noisy breathing, convulsions, loss of elasticity in the skin.

Prevention of summer diarrhoea—Breast feeding of infants offers the most certain protection against all forms of diarrhoea. So, it should be

insisted that all infants should be breast-fed as long as possible and only under exceptional circumstances, infants should be allowed to be hand-fed. Breast-fed infants should not be weaned in the hot months of the year. Particular attention must be paid to the health of the infants during the summer months. Any complaint, particularly any bowel disorder, should be promptly attended to. The management of infants, during the summer and the monsoon months, calls for particular attention, especially that of the hand-fed babies. Infants require smaller amount of nourishment during the hot months than in the cold months. This fact is worthy of remembering in the feeding of hand-fed babies. No attempt to increase the quantity or quality of the feeds during the summer months is to be made as this is likely to upset digestion. Indiscreet feeding is the starting point of all bowel complaints in infants, and this should be avoided. Milk and water are the vehicles of infection in all cases of diarrhoea. So particular attention must be paid to keep these important articles of food free from contamination. Feeding bottles, teats and utensils used in preparing the feeds must be kept scrupulously clean to avoid any possibility of infection. Flies play a very important role in the spread of infection and the menace of flies is greatest in the hot and early rainy months. Every precaution must be taken to protect all articles of diet from flies, so as to avoid any contamination. All feeds must be prepared fresh and should be immediately given to the child. Under no circumstance, feeds should be prepared in bulk for the whole day and stored, as fermentation and infection are more likely to occur under those circumstances. In elderly children, particular attention should be paid to other articles of food, particularly sweatmeats etc., during the hot months.

Treatment.—Treatment of diarrhoeas in children is very important as it is so often mismanaged with tragic results. For the purpose of treatment, it is important to realise that all diarrhoeas in children, occurring during summer months, are potentially dangerous. It is very difficult to draw a line between the non-inflammatory types and inflammatory types. It is a safe rule to treat all diarrhoeas occurring in summer months in children as of the inflammatory variety.

SPECIFIC TREATMENT.—The bacteriological study of summer diarrhoeas has proved very disappointing from the point of view of treatment. Since no particular organism has been incriminated, it is not possible to follow any specific line of treatment. But bacteriology has revealed a few important facts (1) Diarrhoea in children under one year of age is very rarely due to amœbic infection. This fact is very important to remember, as even now, a good number of cases receive injection of Emetine. Emetine is very badly tolerated by infants and it is very dangerous to give it when there is no indication for it. It is a matter of common experience that in a good percentage of cases, Polyvalent Dysenteric Serum produces a dramatic change in the condition of the patient. It is permissible to infer from it that a certain percentage of these cases of infantile diarrhoea owe their origin to Dysenteric organisms, most probably *B. shigæ*. 2 c. c. of the concentrated Polyvalent Serum should be tried in all severe cases. Occasionally, a few cases respond to Anti-streptococcal serum. As was shown in the bacteriology of diarrhoea, the Bacilli of dysentery, and Streptococci have been isolated from the motion, but there are a good number of cases in which no indication for a specific line of treatment is forthcoming. Some cases respond to Bacteriophage treatment.

SYMPTOMATIC TREATMENT.—There are two essentials to be kept in mind right through the treatment. (1) The first is the elimination of toxins from the system. (2) The second is to circumvent the effects of dehydration.

1. *Elimination* is best obtained by giving a mixture containing Sodium Sulphas 20 grains; Sodium Bicarbonate 5 grains; Sodium Citrate 5 grains. Glycerine 50 minims; Aqua anici 1 dr; every half hour or one hour until the stool becomes watery and brown. If there is vomiting, wash out the stomach with 1% Sodium Bicarbonate solution, using a catheter and funnel. A rectal wash with the same solution is also of great use in certain cases. A great deal of benefit is also obtained if along with the above mixture, 5 grains of Crooke's Collosal Kaolin or Lactokaolin with vitamin B is given after every dose of the salt mixture. This promotes absorption of the toxins and helps a great deal in controlling toxæmia.

2. *Dehydration* :—One has to be on the look out for it from the commencement and, when suspected, it has to be energetically combated. Indication that there is dehydration is obtained by the sunken anterior fontanelle, by the loose inelastic skin, diminution of the quantity of urine passed and huskiness of the voice. The only way to combat dehydration is to give the child immediate subcutaneous Saline. Saline per rectum is useless in these cases, for it is never retained and only adds to the difficulties of nursing. 4—6 ounces of normal Saline under the skin of the abdomen every 6 or 8 hours will save the lives of many of these children. In urgent cases, Saline up to 10 ounces should be given intraperitoneally. In all cases, dehydration can be prevented by prescribing a continuous drink—boiled water to which $\frac{1}{4}$ teaspoonful of Sodium Chloride, $\frac{1}{4}$ teaspoonful of Soda Bicarb and $\frac{1}{2}$ a tabloid of Saccharine are added for every pint. This not only allays the thirst and restlessness of the child but also prevents acidosis which, in many cases, complicates dehydration. Acidosis is easily recognised by the deep gasping air hunger, the grey sunken face and cherry red lips. Occasionally, the above treatment results in the production of acute œdema as a result of Sodium Chloride retention. If this occurs, the Sodium Chloride has to be dropped.

The question of diet during the treatment is all important. It demands much care and thought. In the non-inflammatory variety, all that is required would be to stop milk for a day and replace it by barley water or, if the child is old enough, by arrowroot kanji. In other cases, the diet must be very strictly supervised. Milk and milk-products must be absolutely stopped for 48 hours and, for that duration, rice water is prescribed. This is made by adding 1 tablespoonful of rice to $1\frac{1}{2}$ pints of water; boiling, straining and adding a pinch of salt and 1 teaspoonful of brandy. Children like this. In the great majority of cases, it is better to start with this than to give albumin water; for, 90 per cent of these cases are infections with putrefactive organisms. Some German authorities prescribe weak tea.

After 48 hours on a diet which is free from milk and sugar, the question of further feeding arises. Indications as to the nature of the diet to be given can be obtained from the nature of the motions. If the stools are foamy, very frequent, sour smelling, acid and actually scalding the buttocks, then it would be found these infants receive large amounts of sugar and other carbohydrates before developing any symptom. If the stools are firmer, less frequent, foul smelling, alkaline and not irritating to the skin,

then it would be found that these children were on an almost pure protein diet before the diarrhoea began. In the first or the fermentative type, it is advisable to put the child on either albumin water or milk, free from fat. There are various forms of de-fatted milk which are very useful. Peptolac or Spruelac, where it can be afforded, in appropriate dilution, are suitable for this variety. In the putrefactive type, carbohydrates in some form or other are indicated, like arrowroot kanji, or rice water kanji, in the earlier stages. After that, milk, de-fatted and diluted, must be prescribed. It is advisable not to add sugar in the earlier stages but as the child improves, the strength of the feed can gradually be increased and Dextro-maltose (Meads) or Glucose in small quantities may be added to it. If any symptoms reappear at any time, then the earlier treatment must be reverted to and the rigorous diet of the first 48 hours must be enforced.

Treatment of certain complications.—1. *Vomiting*:—This is best dealt with by washing out the stomach with Soda bicarb solution and also by subcutaneous Saline. Vomiting is an expression of infection of the stomach and toxæmia. So, the eliminative treatment must be carefully carried out.

2. *Hyperpyrexia*:—Is a very serious complication and is often attended by convulsions. Cold packs or cold sponging and cold enemata may have to be resorted to. GREEN ARMYTAGE recommends one grain each of Dover's powder and Aspirin for these cases.

3. *Collapse*:—This frequently precedes death. Hot mustard packs, Brandy by mouth or injections of Camphor or Ether-in-oil have to be tried. Intra-peritoneal Saline may, sometimes, be necessary to overcome this collapse.

4. *Rectal tenesmus*:—This is, sometimes, seen in cases in which the ileo-cæcal junction is involved, and it occurs more often in cases of some days' duration. The motions in these cases contain macroscopic mucus and streaks of blood. Washing out of the rectum, with normal Saline solution will control this condition.

5. *Convulsions*:—This may either initiate an attack or may be a terminal event. Bromides and Chloral Hydras in appropriate doses should be given to control this complication.

Reference:

1. De. S. P.—Ind. Jour. of Ped. III, 2. p. 183—190.



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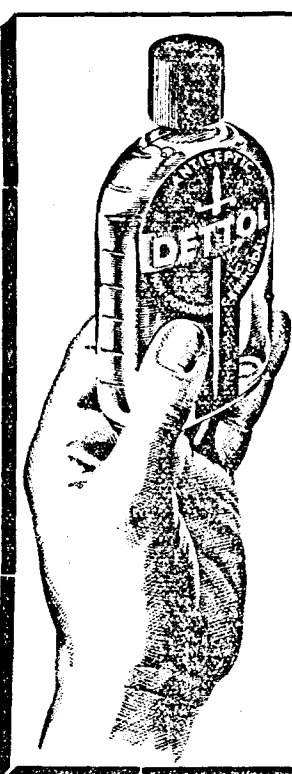
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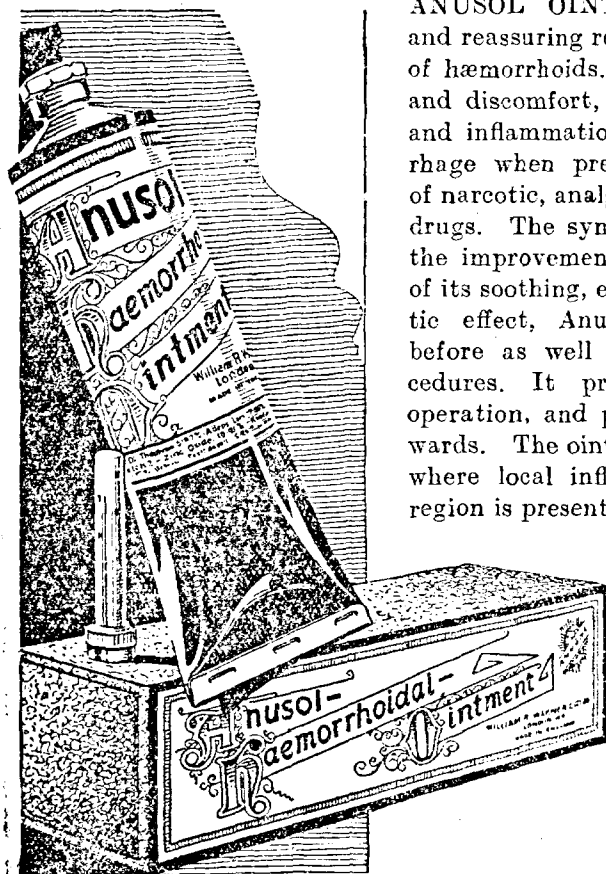
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Infantile Diarrhœa in Summer

BY

L. K. MITRA, M.B. (CAL.), D.C.H., M.M. (LOND.), L.M. (DUB.),

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THE common cause of diarrhœa in infants in the summer months is the infection of the intestinal tract by pathogenic organisms, such as, Flexner, Shiga or Dysentery, Sonne, Morgan or Gaertner bacillus infection. The incidence of this variety of diarrhœa becomes more common with the growth of the infant and during artificial feeding.

The source of infection is contaminated food, the most common of which is milk. Contamination of milk may be due to handling of it by a carrier or a mild ambulant case. Often, the infected water used for washing utensils or for fraudulent dilution of milk is the cause of it. Dust carried by strong wind in the summer is also a cause of contamination. The temperature of milk in the summer season becomes more suitable for the growth of organism in it, so that when it is taken, it is enough to cause diarrhœa.

In the summer season, not only the true infective diarrhœa (summer diarrhœa) but also the dietetic diarrhœa becomes more prevalent. In winter or moderately cold weather, infants can digest food well or stand better any parenteral infection without any symptoms of gastro-enteritis. When the weather becomes warmer, the food requirements rapidly fall because, the loss of heat by the infant is much less. General overfeeding does, of course, occur at this time of the year in both breast-fed and bottle-fed infants because, ordinarily, the food of an infant is not cut down in summer, and the diarrhœa sets in due to indigestion. Moreover, when an infant is overfed, he taxes his digestive and excretory organs to the utmost and is also in an unstable metabolic state, which is completely upset by slight enteral or parenteral infection, resulting in diarrhœa and vomiting. The infant properly fed or slightly underfed withstands a mild nasopharyngitis or a furunculosis etc., without any gastro-intestinal symptoms. Therefore, the coming of summer months may cause diarrhœa in an infant, having a diet suitable for winter or cold weather.

Symptoms.—In infective diarrhœa, the onset is sudden with a rise of temperature and sometimes with vomiting. The stool soon ceases to be fæcal, consisting mainly of mucus and blood, and is small and frequent. The onset in dietetic diarrhœa is slow and insidious; and if the diet is not diminished and the weather is very hot, the condition becomes progressively worse with a slight rise of temperature. The stool contains more faecal matter than mucus and is profuse.

Dehydration is the early symptom in all cases of diarrhœa in infants, which is aggravated, when it is associated with vomiting. Dehydration should not be regarded as a simple water deficiency, but also deficiency of minerals (chiefly Na and Cl) of which the plasma and interstitial fluids are composed. Both water and salts maintain the tonicity of the body fluids, and the loss of one leads to a prompt excretion of the other. The minerals of plasma and interstitial tissue fluid are chiefly Sodium and Chlorine while those of intracellular fluid are chiefly Potassium and Phosphorus. In the attempt to conserve fluid, the volume of urine is diminished as dehydration advances; often, only a small amount is secreted which is practically free from Na and Cl. There may be complete suppression of urine. The symptoms of dehydration are depressed

fontanelle, sunken eyes, inelastic skin and scaphoid abdomen. After this, the infant passes into the stage of intoxication, when all the symptoms are accentuated. The face becomes pinched and ashen, with an anxious look, the nose, fingers and toes cold, the pulse small, and the tongue dry. If the vomiting has been violent, a state of alkalosis may exist, otherwise acidosis.

The next stage is the stage of decomposition and this is the terminal stage. Now the eyes are dull; the infant is unable to recognise its parents; the temperature tends to run up higher. There may be such apathy that it is impossible to get sufficient fluid intake. Complications, such as terminal broncho-pneumonia, purpura, sinus, thrombosis etc., may develop.

Prognosis and complications.—The older the infant, the better is the prognosis. In infective diarrhoea, under the age of six months, the mortality is about 50%. When death takes place, it may be due to dehydration and inanition or commonly to a terminal broncho-pneumonia. Other complications are meningitis, peritonitis, sinus, thrombosis, convulsion, nephritis, purpura, thrush and sclerema.

Treatment.—In mild cases, when the duration is short, little or no treatment is required, as the best treatment is to stop all food except water, and the infant automatically refuses food, taking drinks of bland fluid only. In the more severe cases of dietetic diarrhoea, if seen at the start (as in private practice), the same treatment as mentioned above should be undertaken. More often, a case is not seen until things have become well established, such as—the temperature is high, the dehydration is marked and the diagnosis is not clear whether it is of the truly infective or dietetic type; after all, it does not matter much, as the initial treatment is the same in either case.

The treatment of dehydration can be done by administration of fluid through the following channels:—

(1) *By mouth*:—This is the method of choice in the absence of vomiting. This is best done by giving Saline in half its normal strength with 5 per cent Glucose, very often for the first twelve hours. The fluid from the stomach and intestines is absorbed very quickly. The disturbance in acid base equilibrium is quickly overcome by Saline, as a rule, provided the kidney function is present or re-established, the excess of acid or base being excreted by the kidney. After twelve hours, Saline should be given intermittently, if necessary; otherwise, plain water or green cocoanut water is given; because cases have been reported, where half the normal strength Saline was persisted for longer periods, with oedema of the bases of the lungs and accumulation of fluid in the various body cavities.

(2) *Subcutaneously*:—When the vomiting is excessive, normal Saline with 2% Glucose at a temperature of about 100° F. should be given subcutaneously. The best sites for injection are the outer sides of the thighs under the fascia lata. Saline given under the axillæ makes the patient more liable to contract a terminal pneumonia. It should be given better by drip method and not more than 4 oz. in one spot. The slower it is given, the less violent is the tissue reaction. Infants almost invariably run a mild temperature after subcutaneous Saline and Glucose are given.

(3) *Intraperitoneally*:—Intraperitoneal Saline with 2 per cent Glucose has been given occasionally, but the writer does not advocate this method at all, because there is some amount of infection of the peritoneal cavity along with the risk of injuring the bowels.

(4) *Intravenously*:—This is the most satisfactory method of treating dehydration, especially in extreme dehydration, when the circulation is so

poor that the fluid given subcutaneously or intraperitoneally is not rapidly absorbed. Either blood transfusion or normal Saline with 10% Glucose can be given. Amount of blood should never exceed 10 c.c. per pound of body weight, and it must be citrated and be given by drip method, about 30 drops a minute. The best sites for intravenous injections are the arms and ankles. The part should be fixed by the help of a splint and after fixing the needle to the vein, the rubber tubing should be fixed to the part by adhesive plaster, so that a little movement of the infant will not affect it in any way. One to three ounces of Glucose Saline can be injected at one sitting, the rate of injection should not exceed 10 c.c. per minute. The longitudinal sinus is no longer used for giving Saline as it is very risky.

Drugs.—An initial dose of Castor oil is given to clear out the intestines in all cases, except in true infective diarrhoea, and also when the infant is much dehydrated and the condition is low. Castor oil in minute doses is given for anti-diarrhoeal effect. Bismuth or Kaolin should also be given in large doses (20 to 30 grains). Tr. Belladonna m. l is given with Castor oil to prevent griping. The following prescription is often found to be very useful:

R	Ol-Recine	...	℥	v.
	Mucilage	...	qs.	
	Bismuth Carbonas	...	gr.	xx.
	Tr. Belladonna	...	℥	i.
	Liq. Hydrarg per chlor	...	℥	x.
	Aqua ad	...	3	ii.

Opium in any form is contra-indicated in infants, but if the general condition of the infant is good and the diarrhoea cannot be checked, then DOVER's powder gr. $\frac{1}{8}$ or Tr. Opi. ℥ $\frac{1}{8}$ per every 3 months of age up to one year, can be given once or twice a day.

Specific treatment should be done in infective diarrhoea. In British Medical Journal (Feb. 21, 1941), cases have been reported where, M. and B. 693 has been tried in bacillary infection with good results.

Stimulants.—When the infant is collapsed and cold, Brandy 5 to 10 drops in water is given every 4 hours. Coramine liq. 2 to 5 minims is given with water twice or thrice daily. Intranasal Oxygen should be used when air-hunger is present.

Diet.—Rest to the intestines is a part of the treatment. All foods are stopped; only bland fluids are given as described before. The most controversial question is—how long the infant should be starved? Some continental clinics claim excellent results with many days' starvation. In others' opinion, long starvation is a mistake as the infant has little or no reserve store of carbohydrate. In milder cases, 12 hours' starvation is enough; and in severe cases, the infant may be starved as long as 48 hours. It is needless to mention that, during this period, bland fluid should be given as described before. In severe cases, the food should be given at 2 hours' interval and in small quantities. After diarrhoea, an infant generally cannot digest fat well, so the milk should be skimmed or should have low fat contents. In severe cases, whey or Sherry whey is given at the start. As the condition improves, diet should be slowly worked up to a more nourishing one. When cow's milk is given, Lactic acid should be added to it (2 to 3 drops per every ounce of milk). Among the proprietary foods, Eledon (plain or with carbohydrate) Secway, skimmed dried milk, or Plasmon is very useful. Benger's Food and Mellin's Food are often added to cows' milk to make it more digestible and to increase the food value respectively, without upsetting digestion.

Summer Complaints with Reference to Skin Lesions

BY

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Prickly Heat.—(Miliaria, Strophulus, Lichen tropicus). Beng.—*Ghamachi*; Urdu—*Pith*; Telugu—*Vadagullah*.

DEFINITION.—The disease is characterised by sudden appearance of discrete, pin-head-sized (acuminate) papules and vesicles, usually whitish, situated at the orifices of sweat ducts and attended by itching and burning.

SYMPTOMS.—The affection is inflammatory in nature. A reddish areola is seen around a vesicle. In sundaminas, no such areola is visible. When the vesicle is ruptured by pressure, a fine crackling sound is heard. Some patients and even their friends find pleasure in rupturing the vesicles with an oyster shell. When vesicles dry up, fine desquamation occurs. Sometimes the disease is complicated by boils on account of excessive itching. The face, neck, trunk and upper arms are commonly affected. But the disease may be generalised excluding only the palms, soles and mucous surfaces.

AETIOLOGY AND PATHOLOGY.—The disease is especially common in the tropics and is essentially a summer complaint. Those who sweat profusely and possess delicate or seborrhœic skins are predisposed to it. Hence excessive clothing and prolonged covering of the body are often the exciting causes. All ages are affected but the affection is rare in old age. There are several theories regarding the aetiology of the condition. These are as follows:—

(1) ROBINSON thinks that it is an inflammatory affection of the epidermis in the neighbourhood of sweat outlets.

(2) POLITZER regards it as a cystic dilatation of sweat ducts, resulting from occlusion of the orifices. The horny layer of the epidermis absorbs moisture from sweat, becomes swollen and thus blocks the outlets of the ducts of the sudoriferous glands.

(3) TOROK believes that it is only a superficial dermatitis, resulting from sweat irritation. Normal sweat is acid, but when there is excessive flow, associated with abnormal metabolism, it is alkaline. An alkaline sweat sets up the dermatitis.

(4) ACTON proves that there is an infection of sweat gland mouths by *Staphylococcus Aureus* and *Albus*. He lays down seborrhœa *i.e.*, excessive greasiness or oiliness of the skin and scalp as the predisposing cause.

(5) SMITH in his excellent paper ascribes *Monilia* as the cause of prickly heat.

Whatever the theories may be, the facts are that there is excessive perspiration which certainly irritates and macerates the skin; there are vesicles which might have been caused by blockage of the sweat outlets and consequently cystic dilatation of sweat ducts; there is dermatitis caused by infection of the suitable soil with *Staphylococci* and *Monilia*.

I think the soil and the seed are both necessary for production of this condition. The microbial causation gets further support from the fact that the disease is readily amenable to treatment by antiseptics and disinfectants such as Alcohol, perchloride of Mercury, Sulphur, etc. As regards *Monilia*, I have rarely found it in large numbers in the scales from the affected skin.

DIAGNOSIS.—The disease is easily diagnosed but may have to be differentiated from allergic dermatitis or eczema and sudamina. In allergic dermatitis or eczema, the distribution may not be confined to the face, neck and trunk, no typical superficial whitish vesicles are seen, and there may be a previous history of such an occurrence, irrespective of the summer season. Itching is generally intense and paroxysmal. Sudamina is non-inflammatory.

TREATMENT.—Good cold baths with soap and water or cocoanut oil. Bland diet not liable to produce excessive perspiration. No excessive and constant clothing. The following lotion, followed by the dusting powder, is invaluable:—

Resorcin	... gr. xv	Acid Boric	... 1 part
Hyd. perchlor	... gr. 4	Camphor	... 1 "
Acid Salicylic	... gr. v	Sulphur Precipit	... 1 "
Rect. Spt.	... 3 p	Zinc Oxide	... 2 "
Aqua	... 3 p	Starch or Talcum	... 3 "

Furunculus (Boil).—Strictly speaking, a boil or furunculus is clinically different from an abscess. A boil is generally follicular, conical, tough, small, more superficial, exceedingly tender and painful, generally occurs during the hot weather and possesses a central core, whereas an abscess may be either small or big, is more deeply situated, rounded or convex in outline and fluctuating and possesses no core. A pustule is still more superficial, smaller, can be broken easily and may be either follicular or non-follicular. Whatever the differences may be, microbial cause is the same—*Staphylococcus aureus* and sometimes *Albus*—in all of them. Boil and abscess may be considered together as the terms are often loosely applied.

DEFINITION.—A boil is a circumscribed lesion usually in a hair follicle, sweat gland or duct or ceruminous gland, either single or multiple, hard and tender to touch and generally conical, having a central yellowish white plug called the "core".

SIGNS AND SYMPTOMS.—Boils occur commonly on the face, scalp, axillae and buttocks. When they come out in crops for several weeks, the condition is called *furunculosis*. Although inflammatory signs such as redness, oedema and tenderness are generally present in boils, these may be absent in some cases. It is rare to have one boil at a time. Pilo-sebaceous follicles are commonly affected. *Staphylococci* get entrance into the follicles from outside but sometimes through lymphatics or blood from a septic focus. It is rare to find a boil on the palms and soles and the lip, as pilo-sebaceous follicles are absent there. Neighbouring glands may enlarge and suppurate. The so-called boil on the lips is really a cellulitis which may be complicated by serious symptoms of thrombophlebitis and meningitis. A boil in the external auditory meatus by an infection of the ceruminous glands is exceedingly painful. A "blind" boil is one which is slow in pointing and exists for weeks.

ÆTIOLOGY AND PATHOLOGY.—Boils generally occur in the summer months. Friction, pressure by clothes, excessive perspiration and sebaceous secretion etc., play an important part in the causation. Some are predisposed to boils every summer on the scalp or axillae. Diabetes is a potent predisposing cause. Even those who are potentially diabetic, are liable to get boils. Nephritis, anæmia, constipation and presence of skin diseases like allergic eczema, scabies, folliculitis, dermatitis etc., are important predisposing factors. Focal sepsis in the gums and tonsils may also serve as depot from which micro-organisms are conveyed to the sites where boils develop.

There is always an acute inflammation, resulting in thrombosis of vessels and necrosis of tissue. Extensive round cell infiltration is also present. The core in a boil consists of a slough, representing the affected hair-follicle and surrounding tissues.

DIAGNOSIS:—A boil has sometimes to be distinguished from a carbuncle. The latter is larger, flatter, shows typical induration around and several sinuses discharging pus. It occurs usually on the nape of the neck and back, is more painful and the slough is larger.

An abscess is more deeply seated, flatter and convex and does not contain any core. Fluctuation is present at some stage and superficial inflammatory signs may be absent. It is always easy to show the presence of pus in an abscess by aspiration with a syringe.

TREATMENT.—*Preventive*:—1. Examine the urine for sugar and albumen and casts and treat accordingly.

2. Give low carbohydrate and high fat diet.
3. Keep the skin clean with spt. lotion and dusting powder.
4. Avoid excessive perspiration, mechanical friction with clothes, collars, handkerchiefs, hats etc.
5. Give tonics in case of debility.
6. Give ultra-violet baths to those susceptible to boils and administer Yeast to raise the opsonic index.

Abortive:—Abortive treatment is of immense value and therefore must be given whenever there is an incipient boil; otherwise, the boil is bound to enlarge and go on for weeks, causing great suffering. Unfortunately, many patients do not come for the abortive treatment. Abortive methods help quick resolution. The exposure of an erythema dose of ultra-violet light from a mercury vapour lamp is generally followed by excellent results. The light is not only externally bactericidal but also increases phagocytosis and blood supply to the parts. Hence the involution rapidly takes place. A weak mercurical ointment (Hyd. Amm. gr. v to one ounce of white Vaseline) if massaged for 1 minute, once or twice a day, is highly beneficial. In case of coarse skins, the strength should be increased to 2 to 3%. Infra-red rays are also helpful. $\frac{1}{2}$ E.D. (erythema dose) of X-rays filtered through 3 mm. Aluminium filter is very efficacious, especially on boils on the face. When I myself start getting a boil, I apply a dose of ultra-violet light and rub Mercurial ointment and do not disturb it otherwise. I get excellent results. Boring with Phenol is not often possible and is terrifying to patients.

Curative:—1. Do not press a boil and do not incise it till there is

frank pus. Do not apply hot linseed poultices, as fresh ones may crop up in the neighbourhood.

2. Sodi. citras internally in big doses to hasten necrosis and liquefaction.

3. Acid Sulphuric Dil m. xx. internally twice a day after meals.

4. Tonics and Yeast Vite tablets or Brewer's Yeasts to raise the defence mechanism.

5. Calcium Sulphide $\frac{1}{4}$ gr. three times a day is said to be efficacious but has been found to be valueless by me. Hence, I have given up such a prescription.

6. Stanoxyl tablets and Sulphanilamide group of drugs are seldom useful.

7. Staphylococcal toxoids and autogenous vaccines are sometimes useful but fail on many occasions.

8. Colloid Manganese injected intramuscularly yields good results in some cases and hence is worth a trial.

9. Auto-hæmotherapy should be practised in recurrent boils.

10. Injections of Insulin should be given in obese persons and in diabetics.

11. Externally, I shall paint either with Gentian Violet 5% in 20% Alcohol or Tincture of Iodine or massage with 1% Mercurial ointment and then apply a plaster with central hole containing either 10% Ichthyol and Basilicon ointment or Diachylon ointment with 4% Phenol or boil Elastoplast.

12. If the boil is much inflamed, cold Boro-starch poultice or Camphor with sandal wood paste or Morison's cream consisting of 2 parts of exiccated Mag. Sulph. and 1 part of Glycerine Carbolic (1-10) or Antiphlogistine are valuable.

13. Staphylococcal Antivirus applied with absorbent lint is of doubtful utility.

14. In recurrent boils, filtered X-ray exposures, either fractional ($\frac{1}{4}$ E. D. weekly) or one $\frac{3}{4}$ E. D. or ultra-violet light baths, are the treatment of choice.

15. In axillary boils—rest in bed with arms away from the body and X-ray exposures are strongly recommended.

16. When there is frank pus, the boil should be incised and ultra-violet exposures should be given to hasten cure.

17. In a boil of the external auditory meatus, pack the meatus with 25 % Ichthyol in Glycerine and then apply a hot water bag.

18. Lastly, a change to a cooler and healthy climate should always be advised if it is practicable for the sufferer.

Intertrigo.—**DEFINITION**:—This is a special form of circumscribed dermatitis, located in regions of the body where the skin surfaces are opposed, and is characterised by erythema, maceration of the skin, fissures and erosions.

SIGNS AND SYMPTOMS:—Delicate skins in the regions of axillae, groins, anus and beneath pendulous breasts are usually affected. Due to moisture, warmth and friction between the opposed surfaces of the skin, especially in

children, the skin gets inflamed, macerated, fissured and sometimes eroded. Burning and itching are prominent symptoms.

ÆTIOLOGY, PATHOLOGY AND DIAGNOSIS:—The disease is common during the hot months but may also occur at other times. The condition may either be erythrasma caused by *Microsporon minutissimum* or neurodermatitis or superficial actinomycosis or moniliasis or epidermophytosis or rarely seborrhoeic dermatitis. If no specific micro-organisms such as above are found in the sodden epidermal scales, it must be regarded either as one of external mechanical origin (true intertrigo) or internal sympathetic nerve disturbance (neurodermatitis). I am inclined to regard the condition as a form of neurodermatitis as the disease is associated with paroxysmal itching, sometimes unilateral and often rebellious to treatment.

TREATMENT:—1. Keep off the friction between the opposed surfaces of the skin.

2. Hot compress with weak Lysol (10 drops to 1 pint) is highly soothing and curative.

3. Lotio argenti nitras gr. xx to one ounce of Spt. aetheris nitrosi or Tr. Benzoini Co. to paint.

4. Dusting powder as in prickly heat to dust.

5. Olive oil, cocoanut oil or cold cream is sometimes more useful than paints and dusting powders which produce an uncomfortable drying effect.

6. Fractional doses of X-rays are extremely beneficial. I had to give X-rays to a unilateral lesion under a pendulous breast, which resisted all forms of treatment.

Erythrasma.—**DEFINITION:**—The disease is characterised by reddish or brownish patches, especially located in the axillae, genito-crural regions, gluteal clefts and in folds of abdomen and under breasts.

SIGNS AND SYMPTOMS:—The skin not only gets inflamed but is also sodden and whitish. The sodden skin can easily be scraped off. It smells badly. Painful cracks may be present. Itching and soreness are marked.

ÆTIOLOGY AND PATHOLOGY:—Warmth and moisture play an important role. Hence the disease is prevalent in summer. An organism, named *Microsporon minutissimum* (*Nocardia minutissima*) showing delicate thread-like hyphae and granules but no branches, piled into heaps in the sodden epidermis, has been incriminated as the aetiologic agent. Yeasts and *Actinomyces* have been also sometimes found in such lesions. The micro-organisms can best be examined in a wet preparation of scales treated with McGuire's stain (Toluidin Blue 1 gm., Glacial Acetic acid 2 c.c. absolute Alcohol 4 c.c. and distilled water 94 c.c.). Excess stains must be wiped off after pressing the coverslips. Mycelia, granules, yeasts are all well seen by this stain.

TREATMENT:—Hot compress with weak Lysol and weak Sulphur and Salicylic ointment according to tolerance of the skin. Cocoanut oil before bath is a good preventive. Painting with 3% Alcoholic solution of Mercurchrome or 5% Gentian Violet is also useful.

Tinea cruris.—(Eczema marginatum of Hehra, Dhobie's itch).

DEFINITION:—It is an affection of the skin and not of hairs, characterised by sharply defined patches with papulo-squamous, festooned margins

distributed chiefly in the groins and buttocks, and caused by a fungus named *Epidermophyton inguinale*.

SIGNS AND SYMPTOMS:—The primary lesion is said to occur on the toes, but in many cases no toe lesions are detectable. The disease begins as an erythematous papule, which spreads peripherally, and eventually, patches—either small, circular or like a big sheet covering a large area—are formed. As the name indicates, definite, raised, papular margins are visible. Later, scales form which can be scraped off with a Volkman's spoon. The colour of the patches is either reddish or, more commonly, reddish brown. There is no complete healing in the centre of a patch. Induration is present. No hairs are affected, but nails may be affected. Nails become discoloured, rough and elevated at the tip due to heaped-up fungi and products of tissue reaction. Secondary infection with *Staphylococci* may occur, but is not common. There is never any primary suppuration. Itching is present, but may be absent. The lesion is, sometimes, papulo-vesicular when it affects the palms and soles. It then simulates vesicular eczema. Though text books ascribe the *Epidermophyton* as the cause of some form of vesicular eczema, I have rarely found the fungi in such lesions. The patches, in such lesions, are circumscribed and papulo-vesiculo-squamous, and appear like ringworm lesions, but no history or presence of ring worm lesions in other parts of the body is found. Moreover, the scales are very adherent and one has got to scoop them out with a needle or a pair of forceps. Microscopically, no scales or scabs or vesicles show any mycelia. On the face of these facts, such an eczematous lesion can neither be called a tinea or trichophytide. Tinea cruris may occur on any part of the body, but it is rare on the face and scalp. When it affects the toes, secondary infection may occur and the condition is then popularly called "Mangoe toe", owing probably to its resemblance to ripe mangoes or prevalence in the mangoe season or summer. Tinea cruris, sometimes, causes marked thickening of palms and soles, and may be unilateral. There may be areas of depigmentation and hyperpigmentation also, in the hyperkeratotic areas.

ÆTIOLOGY AND PATHOLOGY:—The disease is prevalent during the hot months. The parasite is a microscopic plant, like a creeper, living on the human epidermic soil and so, like other plants, is bound to germinate and flourish during the hot weather and the rains. Men and women are both affected. The disease is rare amongst children, though cases have been found. *Ooriyas*, in this country, appear to be especially susceptible to the affection. I have seen girls coming after a change from Puri, extensively affected. The disease is not an animal ring worm, hence animals cannot be incriminated as intermediate hosts. *Dhobies* (Washermen) are blamed for the transmission of the disease. If that were so, the majority in a family would have been affected. If the disease were so contagious, then a suffering husband would have easily transmitted the disease to his wife, but many cases are known where husbands or wives, suffering from the disease for years and living together and begetting children, have not been able to transmit the disease to their partners. Personally, I would have been infected on my fingers and nails long ago. When scales or scraped nails are soaked in Liq. Potassae, for 15-30 minutes under a coverslip, and then examined microscopically after pressing on the slip, numerous tortuous mycelial filaments, showing quadrangular cells or oidia inside, are seen. No spores are found. For diagnosis, microscopical examination alone is sufficient and no cultural examination is necessary. It is difficult to find

mycelia only when the treatment has been started or scales have been collected not from the margins of a lesion. Mycelia can also be more readily demonstrated by examining the scales, treated with equal parts of saturated solution of Sodium Sulphide and 90% Alcohol. McGuire's stain can also be used for staining the mycelia, but the method is inferior to the former.

When small bits of scales are treated with Alcohol for 5-10 minutes and then inoculated on the surface of Sabouraud's maltose agar and incubated at room temperature for 3-7 days, small, circular, feathery, lemon-coloured colonies develop, and these become purplish later. Microscopically, well-developed characteristic spindle spores are seen, and no conidia are found. The spindles may be pleuriseptate. It is difficult to reproduce the disease in animals. So far, the disease has been found in man only.

DIAGNOSIS:—Tinea cruris has to be differentiated from the following:—

1. Erythrasma and intertrigo—No typical fungi (*E. inguinale*) are found.
2. Macular leprosy—Diminution of sensation will be present.
3. Neuro-dermatitis, on the scrotum, pubis, groins and buttocks are liable to be confused with tinea cruris. In the former, itching is marked and paroxysmal, no festooned margin is visible, scales are few and less easily separable, papules are harder and shiny, no fungi are found and there is no relation with the summer season. I have seen many such cases wrongly diagnosed as tinea.
4. Pompholyx and vesicular eczema—No fungi are found; there are no ringworm lesions elsewhere on the body and the disease is not amenable to remedies for tinea.
5. Syphilitic hyperkeratosis of palms and soles—Scales are not easily separable, no mycelia are found, itching is usually absent, W. R. is positive and the disease is amenable to anti-syphilitic treatment. It may be stated here that I have seen syphilitic hyperkeratosis and tinea co-existent.

TREATMENT.—The following paint is very valuable and can be used at night and in the morning:—

$\mathcal{R}$ Acid Salicylic	...	1 dr.
Resorcin	...	1 dr.
Phenol	...	$\frac{1}{2}$ fl. dr.
Glycerine	...	$\frac{1}{2}$ fl. dr.
Tr. Iodine	...	2 fl. drs.
Glacial Acetic Acid	...	1 fl. dr.
Tr. Benzoini Co.	ad	1 oz.

If the skin is much inflamed, the paint should not be applied until hot compress and soothing applications like Olive oil, liniment of Calamine or Acriflavine lotion have lessened the angry look. WHITFIELD's ointment should be used after bath. The following is its formula:—

$\mathcal{R}$ Acid Salicylic	...	15 grs. (3%)
Acid Benzoic	...	25 grs. (5%)
Adipis Lanae	...	$\frac{1}{2}$ oz.
Paraffinum Molle ad	...	1 oz.

Ung. Chrysarobin $\frac{1}{2}$ —1 dr. to an ounce can also be used, but its stain is objectionable. Moreover, the drug may irritate the skin and the eyes and may produce inflammation of the kidneys, if its use is prolonged for a

long time. Cignolin ointment is also efficacious but expensive. It is difficult to obtain a radical cure. The fault lies mainly with the patient. He should use a preventive oil (cocoanut oil with Resorcin and Eucalyptus) before bath every day and, whenever tiny lesions appear, he must use the above paint. That cocoanut oil is a good preventive for tinea cruris is further proved by my inability to grow the fungus well on cocoanut oil Sabouraud's agar medium. The fungus is markedly inhibited by the cocoanut oil but not by Olive oil. If nails be affected, these must be soaked, scraped and painted with the above paint, alternately with an Alcoholic solution of Mercurochrome ($\frac{1}{2}$ dr. to 2 oz. of 95% Alcohol). Until and unless the nail lesions are cured, there is always a danger of relapse due to auto-inoculation. If any primary focus be lurking in between the toes, this must be eradicated. In the hyperkeratotic type, the above treatment may not be sufficient, and X-rays may have to be applied. In the intertriginous type, affecting the toes and groins, washing with Potassium Permanganate solution (1-5,000), painting with 2% solution of Mercurochrome in 95% Alcohol, and dusting with prickly heat powder, are recommended. The above Salicylic paint or Gentian Violet lotion can also be applied with success. In cases of big hydroceles or scrotal tumours, it is better to have an operation in order to reduce the area of the opposed skin surfaces.

Tinea Versicolor (Pityriasis versicolor).—Beng.—*Chhuli*; Hindi—*Shiula*.

DEFINITION:—It is an affection of the skin, located chiefly on the neck and the trunk, most prevalent in the summer months, caused by a vegetable parasite, *Malassezia furfur*, and characterised by brownish macules covered with scales.

SIGNS AND SYMPTOMS.—Patches are usually small, but when neighbouring lesions join together, big patches are formed. The colour may be reddish, brownish, blackish or there may be no colour, showing whitish, hypopigmented spots only. It is for these reasons that the following clinical varieties have been recognised:—

- (1) Tinea rosea—reddish.
- (2) Tinea versicolor—brownish.
- (3) Tinea nigra—blackish.
- (4) Tinea flava—whitish.

All these types of lesions are probably the same as these are caused by the same group of organisms, showing the same type of mycelia and spores and sometimes one type is followed by another type, and one or more types are sometimes present in the same case. In a case of tinea versicolor, when desquamation occurs, whitish patches as in tinea flava have been seen to follow.

Distribution of the lesions shows that there may be guttate lesions at orifices of lanugo hairs. Patches are usually present on the neck, chest, upper part of back, axillae and shoulders and rare on the face, but patches have been seen on the cheeks, around the eyes, on the eye lids, around the mouth, on the scalp rising from the neck, the ears, arms, thighs, knees only, penis, scrotum and palms. In the last situation, the lesion becomes noticeable as raised, sodden, scaly patches, when the skin is soaked in water for a little while. Scales can then be easily scraped off and the fungi demonstrated. Patches are finely corrugated. Scales in the lesions are fine, branny (fufuraceous) and may not be noticeable until scraped.

Itching is slight or absent. Adults are commonly affected and men more often than women. The lesion may persist in winter in a mild form.

AETIOLOGY AND PATHOLOGY:—The disease is caused by *Malassezia furfur*. The fungus was called *Microsporon furfur*, but I have renamed it as *Malassezia furfur*, based on my cultural experiments and the nomenclature has been accepted by DODGE (1936) in his book on Medical Mycology. If a few scales from an active lesion are examined with MCGUIRE's stain, numerous septate, short mycelia and clusters of spherical or oval conidia are found. The fungus invades the corneous layer only, but there is some change in the underlying dermis also e.g. dilatation of vessels. The organism was discovered by EICHSTEDT in 1846 and since then, repeated attempts have been made by various workers to cultivate it.

It was successfully cultivated and reported by me in 1927, and my finding has been corroborated by OTA and HUANG in 1933. Tuberculous subjects are popularly regarded as susceptible to the affection, but there is no authenticity in it in all cases. Scalp dandruff is regarded by some as the primary source for the body infection, but as there is no evident dandruff in the majority of cases of tinea versicolor, this theory cannot be accepted.

DIAGNOSIS:—The disease may be confused with the following:—

1. Leucoderma or vitiligo—Patches are ivory white and may be present on any part of the body and there is no seasonal prevalence.
2. Macular leprosy.—Diminution of sensation is present.
3. Dermal leishmaniasis—There is usually a history of kala-azar or typhoid-like fever, distribution of the lesion is characteristic, patches are not sharply defined, erythematous lesions are generally present on the nose and the chin, and L. D. bodies may be demonstrable.
4. Chloasma—No scales are found and the lesions are commonly present on the cheeks and forehead.
5. Seborrhoea corporis—The disease is rare here; scales are soft and greasy and Bottle bacilli are present in them.

In all the above infections, no typical mycelia and spores of *M. furfur* are found.

PROGNOSIS:—The disease is highly liable to relapses, if not adequately treated.

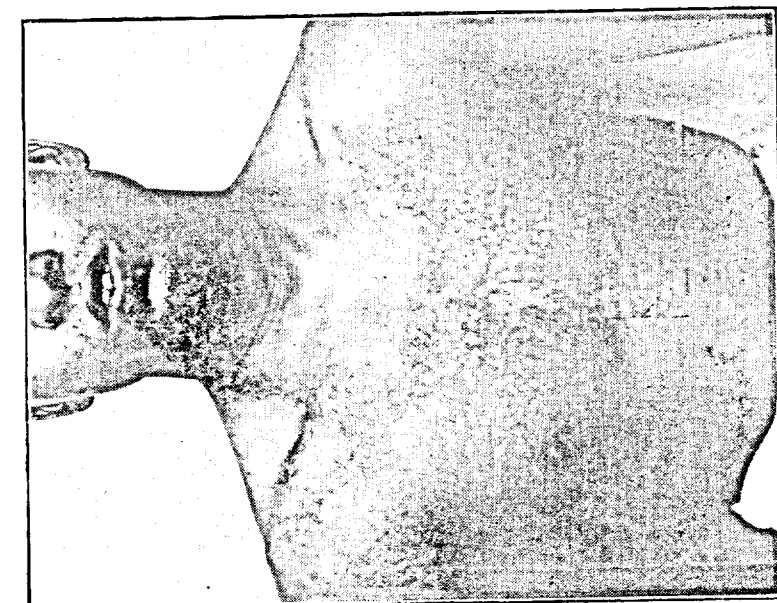
TREATMENT:—1. Sulphur soap to be used for wash.

2. R Resorcin	... gr. 15		
Acid Salicylic	... gr. 10		
Hyd. Perchlor	... gr. ½	Or	Sulphur Precipitate ... 3 ½
Rect. spt.	... 3 ½		Acid Salicylic ... gr. 20
Aqua	... 3 ½		Vaseline white ... 3 1
	To apply.		To apply.

3. Ol. Psoralea—to apply for removing the depigmentation.
4. Coconut oil before bath and prickly heat dusting powder as a preventive. If there is dandruff on the scalp, treat by Resorcin lotion (30 gr. to an ounce).
5. Underwears should be disinfected.



Tinea cruris.



Tinea Versicolor et Flava.

Summer Complaints with Reference to Skin Lesions—By G. Panja.



Pustular Folliculitis.

6. Crocker's plan—wash; paint with 5-40% solution of Sodium Hypo-sulphite and follow it by 3% solution of Tartaric acid or 5% solution of Hydrochloric acid. Nascent Sulphur and So_2 are liberated. A few applications are necessary.

7. Excellent results have been reported by SCHOLTZ from treatment by ultra-violet rays without the use of any other remedy. But, it is difficult to understand how bare-bodied Indians still get the disease, though they get a lot of exposures to the sun.

Pustular folliculitis.—This disease is not essentially a summer complaint. In summer, it gets highly aggravated. So, a few points may be mentioned about this important Indian affection.

The disease is characterised by papulo-pustular lesions in hair follicles, situated on the legs, pubis, forearms, beard and moustache. Each pustule is pierced by a hair which can be easily pulled out, letting out a bead of pus. The disease is highly resistant to treatment. ACTON and MCGUIRE demonstrated *Tricophyton violaceum* in the lesions and regarded the fungus as the primary aetiologic agent and *Staphylococci* as secondary invaders. I could not find any fungus either in smears of pus or hairs, microscopically and culturally, and also in sections of tissues. Agglutinins were demonstrable in blood against *Staphylococci* in significant titres. I have been able to reproduce the lesions in man by inoculation with the *Staphylococcus aureus*. Hence, I regard the condition as staphylococcal, pustular folliculitis in which the hair acts as a foreign body and so long as the hair persists, it is extremely difficult to cure the condition.

TREATMENT:—1. Painting with a mixture of equal parts of Gentian Violet 5% and Brilliant Green 2% in 20% Alcohol, is highly useful and may cure some cases.

(2) Depilatory doses of X-ray, applied to all the affected follicles, have cured all my cases. To prevent a relapse, when the hairs regrow, Spt. Perchlor lotion and dusting powder should be used and friction with clothes is to be avoided.

Carbuncle and Summer Boils

BY

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THESE are ranked among the minor maladies that afflict mankind and boils are particularly so common that they are apt to be overlooked, so that they, sometimes, form the initial lesion in a train of events ultimately having the most serious and even fatal consequences.

Carbuncle is a localised infective gangrene of subcutaneous tissues caused by *Staphylococcus pyogenes aureus*. When the infection of a boil penetrates into the deeper layers of the skin and the subcutaneous tissue, a carbuncle is formed. This consists of a series of communicating abscesses which discharge by separate openings on the surface. The condition is essentially an infective and suppurative one. The gangrene which ensues is secondary to infecting agent. The infection may be blood-borne or surface one. It is definitely not a blood-borne infection. The theory is that organisms are rubbed into the surface and, having entered the superficial lymphatics, are carried into the lymph field of cutis vera and subcutaneous area. Then they concentrate in the connective tissue which surrounds the fat lobules and it is in this part that infection begins. As the tension increases, the fat undergoes necrosis and liquefaction, and a stage is reached which is particularly favourable to an extension of infection. The deep fascia presents a barrier to the downward spread but columnæ adiposæ encourage an upward infiltration and it is along those deposits that infection travels to the superficial layers of the skin and thence to the surface. The important part played by the arrangement of the fatty tissue in the origin and extension of the carbuncle is well demonstrated in the microscopic section of the lesion.

The disease begins as a painful infiltration of the subcutaneous tissue and deeper layer of the skin, resulting into a brawny swelling which is generally circular in shape and reasonably well defined at the periphery. The overlying skin has a characteristic dusky-red hue. There are often patches of lymph exudation upon the surface. The subcutaneous infection remains superficial to deep fascia, but extends slowly and insistently. Later, points of necrosis become evident on the skin. Through these, pus and fragments of necrotic tissue make their appearance. Skin destruction continues to extend and as the surface separates, a mass of suppurating and sloughing tissue is revealed. The periphery is indicated as a zone of increased swelling.

Carbuncle can occur in any part of the body, but specially in areas with thick skin of comparative low vascularity which are subject to surface irritation. Skin of the back of the neck is a common site. When carbuncle develops, the intensity of local infection is apt to be related to some general factor of a debilitating character, such as diabetes and nephritis. In other cases, it is an indication of inherent want of resistance to staphylococcal infection. While local lesion is in the process of developing, there are various evidences of toxæmia, such as pyrexia, want of appetite and a sense of debility. Pain is so intense that it interferes with sleep.

Varieties of treatment have been employed but the general tendency, in recent years, favours conservatism. Possibility of aborting carbuncles

is small. If the condition is treated at a sufficiently early stage, before suppuration has occurred, or while it is still localised, some advantages may be gained by attempting to increase the reaction within and around the part infected. Injection of patient's own blood 10 to 20 c.c. into the subcutaneous space, at various points in the immediate neighbourhood of infection, has proved to be the way in which response is most likely to be achieved.

The patient should be put to bed, encouraged to drink increased quantities of fluid and the bowels should be made to act regularly, avoiding over-purging. Specially, in underfed patients, it is important to give them an adequate amount of nourishing food and to aid this, bitters or Alcohol may be necessary. It is essential to test the urine for sugar. Diabetics are more likely to suffer badly from carbuncle. When carbuncle is present, the amount of glycosuria increases until this is treated by diet and Insulin therapy.

In early stages, application of heat—and dry heat in preference to moist—is essential. Hot Antiphlogistine pad is best. Some prefer hot wet dressing such as Mag. Sulph. in Glycerine 20%, but this has the disadvantage of infecting skin glands and hair follicles. JOHN HESFORD suggests application of 12% Sodium Sulphate solution, in preference to Mag. Sulph. cream, as this is less painful and more easily applied; and it is also less painful and more efficient than hypertonic Saline, which is sometimes used. The solution of Sodium Sulphate, according to him, is warmed to body temperature and several pieces of gauze (fluffed up, not folded) are soaked in it and put on the carbuncle. They are covered with a piece of Jaconet, over which is put ample amount of cotton wool, and are then fixed firmly but not tightly with bandage. This warm Sodium Sulphate pack should be changed every 2 hours throughout the day and once or twice in the night, if the patient is awake. In the early stages of carbuncle, there is often much pain and this must be controlled adequately with drugs, as sleep is essential. If Morphia is necessary, it should be given. With this treatment, nearly all carbuncles will remain local, and pus and slough will be discharged and healing will be as quick as, if not quicker than, if an excision had been made, and scar tissue will be less. I have found Mag. Sulph. cream excellent in the early stages of carbuncle.

As soon as suppuration is evidenced by bogging of the skin, or escape of pus on surface, incision should be practised, and tension relieved. Anæsthesia is avoided because anæsthetics are often unsuitable for carbuncular subjects. Two clean crucial incisions are made into it. They should not extend into the surrounding unaffected tissues. Flaps are dissected from the underlying necrotic tissue, and held back in everted position by tissue forceps. The apices of these flaps should be cut so that the infected area is completely thrown open. The necrotic tissue should be excised thoroughly with scalpel and scissors or endothermy done. After this, I always carbolise the part and wash with rectified spirit. Following the operation, the wound is lightly packed with Mag. Sulph. cream. According to others, Sodium Sulphate packs are given. The infected necrotic tissues will soon begin to come away freely. The dressings are changed twice daily at least, and at each time, some necrotic material should be removed. The disadvantage of Mag. Sulph. cream is that it produces burning sensation and irritation. Hence, some surgeons pack the space lightly with sterile gauze, soaked in 1—1000 Acriflavine in liquid Paraffin, which is very soothing.

An outer dressing of sterile gauze and wool is applied and changed as often as required. The Paraffin Flavine pack should be left for 48 hours. After a few days, some prefer to dress the surface with Cod-liver-oil. I have found Eusol to be very good in dressing the wound after a week. The inside of the wound is packed lightly with Eusol gauze and the outside is dressed with Mag. Sulph. cream compresses. Four hourly hot Boracic fomentations encourage healing. After a good amount of granulation tissue is formed, a stimulating ointment like Scarlet Red may be applied as the latter encourages epithelialisation. Ancillary means may be adopted with advantage. The general condition of the patient should be improved. Diet of ample vitamin content is provided. Fluids are given in abundance. Dressing with sterile Vaseline, after the wound is clean, has often been used, it being changed once in every 3 days.

Another and entirely different treatment which has met with a considerable amount of success in the last few years is by *short wave diathermy*. The disadvantage is that the necessary apparatus is not available everywhere. The carbuncle is covered with a layer of gauze, and one of the electrodes from the diathermy machine is put over this, but is separated from it by a layer of felt. If the carbuncle is on the back of the neck, the other electrode is put either on the forehead or on the front of the neck. When the current is turned on, the patient gets a comfortable feeling of warmth in the neck. This treatment is continued for 10 to 15 minutes. Great relief is often experienced even after the first application, the effect of which seems to be to make the carbuncle clear up more quickly. The diathermy should be given daily until discharge of dead tissue occurs freely. During the days when this treatment is being given, and afterwards, Sodium Sulphate packs may be put on with advantage.

A single erythema dose of X-ray has been found valuable in many cases.

COUREY WHEELER mentions an interesting and novel method of treatment of carbuncle by means of *suction*. By application of suction, pus, blood and debris are painlessly withdrawn. It floods the tissues with blood and is a simple substitute for the injection treatment of the whole blood beyond the margins of induration as suggested by CARP. No anaesthetic is required and the procedure is free from risk. To apply suction, 2 or 3 BIER's cupping glasses of different sizes should be at hand. The glass is connected by pressure tubing to an electric suction apparatus regulated so that the glass is held firmly in position without causing pain. The strength of the suction is gradually increased until thick pus and blood well slowly into the glass. The process is continued for about 5 minutes and repeated daily for a few days. The pain is a signal to reduce the amount of negative pressure. After each sitting, patient usually acknowledges considerable relief and asks for a repetition. The slough finally appears in the glass. If suction apparatus is not at hand, the common suction cups, such as a breast pump, will suffice but the latter cause pain. In widespread induration, a smaller size cup is placed centrally over the involved area. The best type of a case, responding to suction, is that in which there is widespread induration; toxæmia and high temperature are present.

DR. LYTH, the originator of Sodium Sulphate treatment, recommended Elastoplast for the treatment of carbuncles. BERTWISTLE uses a modification of DR. J. C. LYTH's treatment. It consists of application of small

pad of gauze, soaked in Sodium Sulphate, on the carbuncle and then hermetically sealing the whole area and for some distance beyond, with Elastoplast and leaving for 3 days when it is found that the site is elevated by a collection of blood and pus. The slough is then in most cases seen lying free in the base, ready for removal with forceps. The surprising feature is the complete absence of pain.

In humid high temperatures of India, it is very uncomfortable if the dressings are not changed for once in 24 hours. I have had good results from 4 hourly compresses of S. S. Mag. Sulph. In sensitive patients, the application of Mag. Sulph. directly on the wound is painful and irritating. In these cases, some surgeons cover the actual wound with fine gauze, soaked in Hexyl Resorcinol and secure this by cross strips of adhesive plaster. This is changed every 24 hours, while Mag. Sulph. cream compress is applied over it. This softens necrotic tissue which can be easily removed by forceps and scissors. Along with this treatment, some people have found Collosol Manganese, either orally or by injection, beneficial.

The use of vaccines and sera in the treatment of carbuncle has not found much favour, but OVENS reports good results following injections of Anti-streptococcal Serum intramuscularly.

Certain carbuncles do not localize, but continue to spread, sometimes at an alarming rate. This occurs in patients who are specially debilitated either due to diabetes or some other cause. When adequate general measures and the local treatments described above are carried out, a carbuncle usually ceases to spread, but if this is not the case, further steps may be required. There are two alternatives, one being injection of blood into the surrounding tissue and the other the excision of carbuncle.

About $\frac{1}{2}$ c.c. of a strong solution of Sodium Citrate 25% is drawn into a 20 c.c. syringe which is then filled with blood from the patient. This is injected at a number of points about an inch apart around the carbuncle outside the indurated area. About 5 c.c. are injected at each place, deep down near the core of the lesion. 50 to 60 c.c. of blood may be required. If this method does not help, the carbuncle must be excised.

A special method of treatment of carbuncle with pure Carbolic acid is useful in some cases. The carbuncle is touched with Carbolic acid on a swab stick at various points, a few cms. apart, in the circumference concentrically. Thus, a few holes are made through which pus and necrotic tissue come out.

Carbuncle has a worse prognosis in diabetics. In some cases, it may have an accompanying glycosuria not due to true diabetes. It is better to get the diabetes under control as soon as possible. In diabetics, owing to the grave prognosis and their poor powers of resistance, it is usually inadvisable to try conservative treatment with Sod. Sulph. packs. In these cases, the patients should be given double the amount of Insulin with sufficient amount of Glucose intravenously (to avoid hypoglycæmia) before the operation. The presence of acetone is a very bad sign in the cases of diabetic carbuncles and Glucose has to be given in sufficient amounts. The usual amount of Insulin may be continued from the next day. The patients who have traces of sugar and no acetone in urine, have the best prognosis.

In the end, I want to refer specially to the carbuncle of the face. This requires special attention due to its grave prognosis, as the carbuncle on

the face is likely to be followed by cavernous sinus thrombosis, which is invariably fatal, particularly if it is squeezed or scraped or cut. This is due to the communication of the superficial veins and the cranial venous sinuses. Even a small facial carbuncle must be treated with great caution from the very beginning, and the patient should be put to bed.

The lesion should be touched, and handled as little as possible. The part of the face in which it is situated should be immobilised by covering a wide area around it with Elastoplast, the carbuncle itself being left exposed. Further to obviate the movement of the face, the patient should not be allowed to talk, and only fluids should be given. If the carbuncle is near the lips, all drinking should be through a straw. It should be bathed every 2 hours with hot 12% Sod. Sulphate solution for $\frac{1}{2}$ hr. at a time. This is best carried out by holding the face over a basin of very hot solution and keeping the part really hot by holding a large pad of absorbent cotton wool, soaked in the solution, gently against the carbuncle for a minute or two at a time. All sloughs should be allowed to separate by themselves or, at the most, should be gently bathed away. Incision or excision on facial carbuncle, even if it does not produce cavernous sinus thrombosis, leaves an unsightly scar, or such deformities as ectropion or a distorted mouth.

Facial carbuncle has also been treated by *Infra-red rays* with success by ROBERT LEAVER. He exposed the facial carbuncle to infra-red rays at 10 a.m., 2 p.m., then at 3 a.m. and 2 p.m. next day, when a small amount of pus leaked from the central part; finally, on the 3rd day, pus was pouring out freely and the patient completely recovered. Facial carbuncles have been treated with Prontosil Album with success by DR. BARBER. Its action can be explained by its direct action on the bacteria at the site of infection with consequent liberation of a large amount of toxin into the blood stream. The result is an acute toxæmia, shown by shivering and temperature, and muscular pains. This sudden liberation of the toxin from the action of Prontosil on the bacteria, effects the stimulation of adrenals and thyroid to secrete, thus bringing about the initial pallor of the skin and chillness due to intense action of the sympathetic and, later on, febrile reactions. The subsequent feeling of exhaustion and subnormal temperature are due to temporary exhaustion of adrenal-thyroid sympathetic system.

Before coming to general treatment, I want to say that some people prefer to dress the carbuncles, other than those on the face, with Camphenol (equal parts of Carbolic acid and Camphor) for 24 to 48 hours. This renders the wound and subsequent dressings quite painless and is followed by great relief of local and general symptoms.

In general treatment of carbuncle, in addition to the general tonics and plenty of fluids, I have found Sulphonamide tablets a great help. I generally give 6 tablets a day for 3 days, and repeat it after a week if necessary.

Furunculosis and boils are very common ailments. They are favoured by humid atmosphere, water impurity during rainy season, and unclean skin. Gastro-intestinal diseases, which are so common during these months, lower the resistance of the body. Boils, so characteristic manifestation of this season, are again caused by lowered vitality and ill-health. Mango-boils, seen remarkably in children, are said to be caused by eating raw mangoes. Their ætiology is uncertain.

Boil arises as a result of some irritating pressure on an unclean skin area in a person who for some time is run down. It is the result of Staphylococcus infection of hair follicle, sweat gland or sebaceous gland. The infecting agent is Staphylococcus pyogenes aureus. BURNET, in his recent work, has shown that pigment characteristics do not necessarily indicate different strains, for Staphylococcus aureus may have Albus variants. Various toxins are produced by Staphylococcus. Two have been isolated, one *Alpha* and the other *Beta*. *Alpha* plays the important part in human skin infections. It is hæmolytic as it contains leucocidin. In skin and subcutaneous tissues it has the effect of producing localised necrosis. Recent methods of immunising Antigen treatment are based upon the knowledge of Staphylococcus anti-toxins.

Staphylococci are constantly present on the skin surface, specially Albus. Infection may enter the shallow pit of hair follicle which has, around the hair, a pass into sebaceous gland which opens on one side, but sebaceous material has a certain degree of resistance to infections. It is when the hair is shed that the infection is most liable to occur as the epithelium, covering the papilla, has not proliferated and the infection is liable to enter at that place. True boils are due to staphylococcal infection of sebaceous and sweat glands. The outer sheath of the hair follicle being relatively avascular, infection advances unchecked, and reaches the perifollicular space by destroying the outer sheath. It does not spread to the papilla due to its vascularity. The pathological picture of the boil shows that the follicular cavity is filled with pus and inflammatory debris. The walls have yellow slough; and around the core, there is a zone of reactionary tissues which, by exerting their phagocytic action, establish a line of demarcation, thus securing the complete separation of central slough easily. After the slough separates, the cavity is covered with granulation tissue and, in a short time, the space is filled in, and epithelium completes the closure.

The attention is drawn by the sense of itching and discomfort at a certain point of the skin; a small, highly sensitive pimple-like projection occurs. This is umbilicated in the centre, from which serum oozes out. Pain is present and is of a throbbing nature, and is felt due to increased tension. Absorption of toxin occurs which causes rise of temperature, loss of appetite and malaise. In acute stages, lymph-glands may be enlarged.

The early treatment consists in removing the irritating pressure, such as from collar or braces, and the boil should never be incised as against the common adage "When pus is present, let it out". The papilla, being vascular, contains venous sinuses which are opened, and thus there is a danger of septicæmia and pyæmia, and the boil, which was a local infection, becomes generalised.

The most important treatment is the application of heat to the part as it comforts the patient, increases reactions in the part and hastens separation of slough. Linseed poultice is not used now, as it has no antiseptic value, and it is a moist dressing. Hot Boracic fomentation is also avoided due to its moist nature. Same disadvantage is with Mag. Sulph. in Glycerine 20%. The best is a pad of Antiphlogistine $\frac{1}{4}$ " thick, and of proper size, as it retains heat for quite a long time. The skin, surrounding the boil, is washed with Rectified Spirit, and a small amount of Ammoniated Mercury ointment is rubbed round about to avoid surface infection. The

heated Antiphlogistine is placed between two layers of sterile gauze and laid over the boil. A layer of cotton wool helps to retain the heat and crepe bandage retains this in position. Dressings are repeated 4 hourly and continued till the slough separates. Desiccated Mag. Sulph. in Glycerine ($2\frac{1}{2}:1\frac{1}{2}$) may be used. After the separation of slough, the granulating cavity is dressed with an antiseptic lotion as Eusol or Acriflavine, or a solution of Aluminium Acetate 10%. In later stages, Scarlet Red ointment stimulates epithelialization.

In early stages, I have found that by covering the boil with 2 to 3 layers of Elastoplast only shows a wonderful result. The latter acts by protecting the boil from being rubbed, and by splinting the inflamed area and providing rest to the particular areas. Thus, complete rest to the inflamed area is one of the elementary principles of therapy. Colloidin, painted on to a boil and allowed to harden, is recommended, but it is not as good as Elastoplast treatment. A certain piece of Elastoplast, a little larger than the boil, is stretched out and stuck on so that it covers the whole boil. A circle of Elastoplast, a little larger than the former size is covered over it. A striking and immediate reduction of pain follows, and in many cases, swelling subsides quickly and the boil disappears in an almost miraculous manner. If the boil begins to discharge, a small hole in the centre through which the pus may escape, may be substituted for the first piece. This may be changed if it gets soiled. Thus treated, the boil may heal without leaving a scar. This treatment is most successful in small boils and at an early stage. Boils of subacute type respond to an antiseptic ointment such as Phenol 1%; Camphor 3%; Salicylic Acid 2%; Lanoline-Vaseline $\frac{1}{2}$ oz. A thick layer on a lint is taken and applied. It should be changed as often as required. It is soothing, antiseptic and hastens separation of slough.

There are various methods of arresting boil formation. The focus is sterilized with pure Carbolic acid by means of a pointed sterile match stick dipped in the acid and inserted into the centre of the boil by rotatory movement, gently and steadily. A sterile dressing is applied. No fluid should touch that part. Ultra-violet and X-rays have been used to arrest infection with varying degree of success. The rays are concentrated upon the focus by means of a lead shield perforated in the centre to expose the boil. Ultra-violet rays are given 3 minutes each day, lamp being set up at a distance of 4" from the skin. Following these applications, the area is dressed with Ichthyol ointment 20%.

It is possible to abort a boil by injection in the contiguous tissues with small quantities of patients' immunised blood. The normal serum contains rather less than one unit of Staphylococcal Anti-toxin per c.c., but in the presence of Staphylococcus infection, this may be raised to 4 or 5 units. 10 c.c. of blood is taken, and this is injected at 4 or 5 points around the periphery of the boil at a distance of $\frac{3}{4}$ " from its centre.

Vaccine treatment is useful specially in cases in which successive crops of infection or recurrent boils arise. The results are difficult to assess. An Autogenous Vaccine appears to be most reliable. Recently, Staphylococcal Toxoid, *i.e.*, Staphylococcal Toxin, to which Formalin is added, and Vaccoid, a combination of Toxoid and Vaccine, have also been prepared. They are supplied in weak and strong solutions, the latter 3 times stronger. Dosage is from .1 c.c. to 1 c.c. I think that it is better to begin with .02 c.c. and

increase it to .5 c.c. diluting the original Vaccine by normal Saline. Injection is repeated every third day. Weak solution is used in children and in adults who show marked re-action. It proves valuable in most difficult type of cases, or the recurrent boils. In recurrent boils, general artificial sun-light baths, twice a week may be tried, and in resistant cases, a Glucose tolerance test should be preferred. This may reveal decreased Glucose tolerance test or a latent diabetic tendency, which may be treated by restriction of carbohydrate intake or reduction of weight.

Coming to the drug treatment, salts of Manganese and Tin, inhibit growth of Staphylococci probably, by stimulating antibody formation. Stannic Oxide 10 gms. OD. can be given orally. Stannoxyl—2 milli litre ampoules—can be used for intra-muscular injections. Intra-muscular injection is the most effectual method of administering this drug.

Among the salts of Manganese, orally Manganese Chloride 5 gm. daily can be given. Manganese Butyrate is used in intra-muscular injections. 20 m. at intervals of 5 days are given. Colloidal Manganese, .5 c.c. every 3rd day, is also useful. Collosol Manganese can be given orally in drachm doses.

Yeast (*Cerevisia fermentum*), if injected, increases the proportion of leucocytes, after a transient leucopenia. Compressed distiller's Yeast is the best preparation. It is given before meals, one table spoonful once a day. General tonics as Iron and Arsenic are indicated. Concentrated preparations of vitamin D are often beneficial. Sulphur in various forms has been found useful. Calcium Sulphide gr $\frac{1}{4}$ to gr $\frac{1}{2}$ T. D. S. after meals. Colloidal Sulphur 3i to 3ii B. D. are used.

With the above treatment, urine should be examined regularly for albumin and sugar. Any derangement in general health should be treated. Constipation should be corrected. Diet should be light and easily digestible. Alcohol and condiments are forbidden, and sugar should be taken sparingly. Internally, Alkalies such as Pot. Citras and Sodium Bicarb 3i of each may be taken three times a day, and are found quite useful.

In case of large boils, when softening occurs at the apex of the boil, the area is carefully incised and pus allowed to well out. Boric fomentations and Mag. Sulph. cream should be continued. Application of Antivirus ointments and Jellies are more beneficial at this stage than in early stages. When the core is separated, dressings of 2% Hydrarg Ammoniata in Zinc ointment or Lassar's paste will hasten the healing process.

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Multiple Summer Boils, Furunculosis and Abscesses-Interesting Experimental Findings

BY

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DURING the summer months, many people suffer from multiple boils, furunculosis, or abscesses, continuing for a long time and showing very little improvement by treatment. The discomfort is extreme. Cases were known where more than a dozen of these boils had to be opened within a period of three weeks. Children and adults of both sexes are equally affected.

Bacteriologically, these are mostly due to *Staphylococcus aureus*, seldom *Staphylococcus albus*. Sometimes, secondary infection by *Streptococcus* takes place. Vaccine, either stock or autogenous, does not seem to have any effect. Some believe it to be due to eating too many mangoes, others to excessive consumption of sweets and the consequent rise of blood sugar. Lay people believe it to be due to some kind of poisoning of the blood.

Scientific analysis.—Though it is not a fatal infection, yet it would be a matter of great regret if the skill of the medical man fails to cure such a simple infection, which causes extreme suffering. The following problems regarding these infections are required to be solved, *viz.*:—(1) Why one or two boils or furunculosis, appearing first, do not act as an autovaccine and stop further appearance? (2) Why stock or autovaccine fails to stop this recurrence? (3) Why multiple boils, furunculosis or abscesses appear generally during the summer months?

The answer to the first two questions was obtained by us a few years ago from a case of impetigo in a boy of 7. In our article "Inhibitory Influence of Chronic Amoebiasis on Immunity against Bacterial Infection"—*Journal of the Indian Medical Association*, August '40, it was suggested that amoebic infection inhibits the formation of antibodies in the system against pathogenic micro-organisms. Since then, a large number of cases of multiple boils, multiple furunculosis, abscesses, chronic staphylococcal eczema and impetigo were studied. Laboratory investigations revealed amoebic infection in about 95% of these cases. In all the cases of multiple boils, vegetative forms of *Entamoeba histolytica* were present in the faeces. Autovaccine or stock vaccine had little effect, but simple specific treatment with injection of Emetine, oral administration of Stovarsol or Carbarsone, alternating with Ext. Kurchi Liq., sometimes together with autovaccine, stopped any further appearance. Recurrence after a few months coincided exactly with the reappearance of amoeba. On the face of these experimental data, there cannot be any doubt that a distinct relationship exists between amoebic infection and multiple pyogenic infection. The pre-existence of amoebic infection exerts an inhibitory influence on the formation of antibodies in the system against pyogenic bacteria. When amoebic infection was cured, the other disappeared in no time. In about 5% of cases when amoeba could not be discerned in the stool—the presence of one of the following causes was observed, *viz.*:—(1) Syphilis. (2) Diabetes. (3) Albuminuria. (4) Chronic Bac. Dysentery (Flexner) infection. (5) Leprosy.

Sometimes, both amoebiasis and syphilis existed together and even after curing amoebiasis, the particular pathogenic condition did not

disappear. But as soon as syphilis was cured, the chronic infection disappeared in no time.

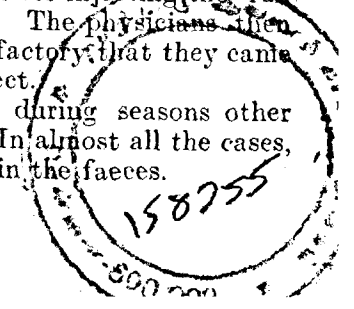
Having found out the solution of the first two problems, there still remains the third, *i.e.* why these occur during the summer months.

For a long time, we were engaged in trying to find out the cause of the prevalence of multiple boils and furunculosis during the summer months. During a study of soil bacteriology by us, it was noticed that about 5% of the total bacterial population of the soil from a shaded place in this country during the summer months consisted of amoeba, when temperature, reaction, humidity and moisture of the soil were favourable to its growth. Food and water can easily be contaminated by such increase in the number of soil amoeba. It is, therefore, a very sound presumption that the increased incidence of multiple abscesses, boils and furunculosis during the summer months, is due to the increase of amoebic infection. During the summer months, generally, the body resistance is lowered and the pyogenic bacteria, entering into the circulation, multiply unhindered if the formation of a specific antibody against the particular pyogenic micro-organism is inhibited by the already existing amoebic infection in the system. Due to this inhibitory influence, neither the pyogenic bacteria nor an autovaccine can stimulate antibody formation. Thus, the boils or furunculosis appear one after another. This is our explanation of multiple summer boils, furunculosis or abscesses confirmed by laboratory investigation.

Treatment.—In every case of multiple pyogenic infection, the stool should be examined for amoeba. If it is not possible to examine the stool, there is no harm in giving at least three consecutive injections of $\frac{1}{2}$ or 1 grain Emetine according to age and constitution, and a course of *Staphylococcus* stock or autogenous vaccine. In all cases, the injection of Emetine should be followed by oral administration of Stovarsol, Carbarsone or Liq. Ext. of Kurchi. After one or two injections of Emetine, the appearance of new boils or furunculosis will be stopped and, if anti-amoebic treatment and vaccine are continued, a complete cure will be obtained within a week or 10 days. As soon as amoeba is made to disappear from the stool, the natural antibody formation starts with surprising rapidity.

Just when we were writing this article, two more cases were sent to us for investigation. Both patients—European females of middle age, suffering from multiple boils—were sent to the laboratory for the preparation of autovaccine from the pus. Permission was asked from the respective physicians for the examination of the faeces of their patients, explaining our hypothesis. It was evident that they did not accept our hypothesis regarding the significance of the presence of amoeba. In both cases, the vegetative form of *Entamoeba histolytica* was present, though neither of the patients complained of any irregularity of bowels, and there was no history of previous dysentery. This result was communicated to the respective physicians with our suggestion of anti-amoebic treatment. The physicians, however, gave only the autovaccine. After injecting the full course of autovaccine, the crop continued to appear. The physicians then gave injections of Emetine. The result was so satisfactory that they came to the conclusion that our theory was probably correct.

Multiple abscesses or boils were also noticed during seasons other than summer months, particularly among children. In almost all the cases, vegetative form of amoeba was found to be present in the faeces.



Some Aural Complaints Common in the Summer

BY

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OWING to the peculiarities of temperature, atmospheric humidity and changed habits, certain diseases of the ear are more commonly met with during the summer months. The diseases may vary from such trivial conditions as impaction of wax, staphylococcal infection of the pinna or the external auditory canal, to the more serious inflammatory conditions of the middle-ear with its possible complications.

I. Impacted wax.—Wax of the ear or cerumen is secreted by the ceruminous glands situated in the outer part of the membrano-cartilaginous meatus. It is formed of two components, one is oily which holds in solution, the second is the solid waxy portion. Under ordinary circumstances, the secretion as a whole is fluid enough to escape from the external auditory canal, aided by the movements of the jaw. In some persons, however, either due to the lack of the oily substance or because of a narrow meatus, presence of dermatitis or an osteoma, the wax keeps on collecting, and the one last to be formed further pushes in the mass already present.

SYMPTOMS:—So long as even a small passage is left for the sound to reach the ear, the presence of the mass of wax remains un-noticed. Usually, what happens is that, owing to introduction of some water, especially during swimming or diving, the mass swells up, giving rise to deafness which is of a sudden onset. This may or may not be accompanied with pain, depending upon whether dermatitis of the meatus is produced or not. Some persons might be very sensitive to this, and they may complain of tinnitus, and even vertigo.

DIAGNOSIS:—In most of the cases, the history of sudden onset of deafness without any other alarming symptom would lead one to suspect this condition. This is confirmed by examining the meatus which is entirely filled with brownish, black or yellowish white mass.

PROGNOSIS:—It is a common saying in otology that the deafness easiest to cure is the deafness due to wax. In a majority of cases, on removing the impacted wax, hearing returns to normal. However, in some cases, a diseased condition of the middle-ear may also be present, and this simple treatment may prove futile in restoring the hearing to normal.

TREATMENT:—This is simple. In some cases, the mass can be removed by means of a hook or a foreign body remover with a loop at one end, while in a large number of cases, the wax is quite hard, and it must be sufficiently softened before it can be removed either by an instrument or by syringing. For this purpose, a few drops of a solution of Sodii Bicarb. 3ss. in 3i of distilled water, are instilled into the meatus every 2—3 hours, i.e., about 6 and 8 times in a day. The ear is then syringed with warm Boric lotion. The Soda Bicarb. drops will have made the mass soft enough to enable its removal in one sitting. The meatus is carefully dried with

wicks of cotton wool, and then the meatus and the tympanic membrane are carefully examined. The skin of the meatus may be only sodden, in which case a little Spt. Vini. rect. is applied to it on a wick of wool, and repeated once a day, for two or three days. In other instances, it may be ulcerated and painful, when the following drops are instilled twice a day.

R	Acid. Carbolic	...	m	iv
	Spt. Rect.	...	3	ss
	Glycerine	...	3	ii
	Mft. Guttae.			

The ear is lightly plugged with cotton-wool, and dry fomentations applied externally, every four hours, if the pain is at all severe. The superficial epithelium, covering the tympanic membrane may become damaged, owing to the continued pressure of the wax or inflammation. This usually clears up by the use of the Carbolic-glycerine drops mentioned above. It sometimes happens that the tympanic membrane is pushed inwards, and presents the appearance of a 'retracted drum' with a dull appearance, prominent hammer-handle and absence or shifting of the 'cone of light.' This retraction of the drum may be due to the pressure of the wax, maintained over a long time. Such cases need inflation of the middle-ear by means of the Politzer's bag or eustachian catheterization. Any accompanying diseases of the middle-ear is to be treated accordingly.

II. Keratosis obturans—This condition, although pathologically different, will also give rise to the same signs and symptoms as impacted cerumen. Some regard it as being composed of masses of epithelium intermixed with wax, while others are of the opinion that it is a fungoid condition, produced by a mass of hyphæ, mixed with a certain amount of wax.

SYMPTOMS:—In general, they are identical with those of impacted wax. Pain, however, is usually more marked, because the superficial epithelium of the meatus is almost always in an inflamed state.

DIAGNOSIS:—Examination of the meatus shows the presence of a plug of this substance which is usually hard and is much more adherent to the meatal wall than ordinary impacted wax.

PROGNOSIS:—As mentioned above, the presence of dermatitis of the meatus calls for more prolonged treatment. The tympanic membrane is also liable to greater injury than in an ordinary impacted wax.

TREATMENT:—Removal of the mass without previous softening may be very difficult, and, in any case, is extremely painful. The mass is at first softened with the Sodii. Bicarb. solution. It might be necessary to soak the mass for about two days before an attempt at removal is made by syringing it out. It often happens that only a superficial layer is removed at the first syringing; therefore, the process may have to be repeated. In children, it might be necessary to administer a general anæsthetic before it can safely be removed. To prevent recurrence, the meatus is treated for sometime as for the fungus disease of the ear, to be mentioned hereafter.

III. Furunculosis and eczematous condition.—One of the most painful affections of the ear is a furuncle. It always involves the outer or fibro-cartilaginous portion of the meatus. It may be single or multiple. Sometimes, they come up in crops.

ÆTIOLOGY:—Certain constitutional diseases are the predisposing causes;

among these, diabetes is very important. Patients with this disease often suffer from recurrence of furunculosis of the meatus until proper attention is directed towards the underlying general disease. Depressed bodily state is also a pre-disposing factor. In common with the greater frequency of boils and other staphylococcal, cutaneous infections of the other parts of the body during the summer months, aural furunculosis is much more commonly met with during this time of the year. The exciting cause is always a local infection by *Staphylococcus aureus*.

SYMPTOMS:—The initial symptoms begin with a feeling of tightness or fulness in the meatus which is followed by pain, increasing in severity till in many cases it becomes excruciating. Tinnitus aurium is complained of at times. If due to swelling the meatus becomes occluded, there will be varying degrees of deafness. On manipulation of the pinna, pain is elicited, and any examination of the ear is usually resented. Therefore, it is of utmost importance to make any physical examination with great gentleness. Pressure on the tragus is extremely painful, as a rule, but movement at other points of the pinna will also be tender according to the site of the furuncle. If it is situated on the anterior wall of the meatus, the maximum tenderness will be on pressing the tragus; if on the posterior wall, gently pulling the pinna backwards will be painful; pulling it upwards would be painful if the boil is situated on the superior wall; and its presence on the floor of the meatus will elicit tenderness if the lobe of the ear be pulled downwards. In the same way, the enlargement of the pre-auricular, post-auricular and the glands situated below the lobule depends chiefly upon the site of the furuncle on the anterior, posterior or inferior wall respectively. Mastication is very painful due to the anatomical relationship of the cartilage of the temporo-mandibular joint and the fibro-cartilaginous meatus. There is a certain amount of trismus accompanied by slight rise of temperature, about 99°F., and loss of appetite and general malaise. The pain may radiate over the side of the head and towards the eye.

DIAGNOSIS:—The presence of tenderness on moving the pinna is an essential diagnostic feature. If this sign is absent, it is almost certain that the fibro-cartilaginous portion of the external auditory canal is free from any boil. On examining with a speculum in the early stages, a circumscribed area of redness and swelling is noticed. The swelling may be very slight, and may be merely suspected. In such circumstances, the use of a probe will be helpful, the area of inflammation being tender to slight pressure. Later on, the swelling and redness increase, and ultimately when it comes to a head, a yellow point surrounded by a zone of inflammation is in evidence. There may be more than one of these boils, and the meatus may be diffusely inflamed, narrowing or even blocking the canal completely. If the furuncle has spontaneously discharged itself, the meatus will be filled with a scanty amount of thickish pus mixed with epithelial debris. The membrana tympani is usually normal in appearance, unless, of course, due to neglect of the boil, and owing to contact with the pus or epithelial debris, it also becomes superficially inflamed. If the furuncle is incidentally or ætiologically related with a suppurative otitis media, signs of this condition will be present. The pus will be more copious, muco-purulent, and there will be inflammation and perforation of the tympanic membrane with loss of hearing.

It is of the utmost importance from the point of view of treatment that the condition be distinguished from acute inflammatory disease of the middle-ear complicated with mastoiditis. While in most of the cases, the characteristic signs and symptoms related above are sufficient to settle a diagnosis in favour of furunculosis, there will be some cases who will not present such unequivocal symptoms as to enable one to clinch the diagnosis. Confusion is worse confounded if the two diseases co-exist. The following points may be of help in distinguishing the one from the other. Unfortunately, in a small number of patients, we will still be left in doubt, and under such circumstances, it is safer and wiser to treat the case as of middle-ear disease. No great harm is done if a mastoidectomy is performed, where no mastoiditis exists, considerable harm may be done, if neglected:—

	Signs and Symptoms.	Furunculosis.	Mastoiditis.
1	Age	Not so common in child-hood	Common in children.
2	History	Sometimes, boils elsewhere, glycosuria in adults	Presence of acute otitis media, coryza, bathing, teething adenoids and nasal infection.
3	Fever and malaise	Seldom severe	May be so.
4	Early tenderness	Usually greatest on tragus	Usually post-aural.
5	Meatal swelling	Anywhere in the canal, may be completely occluded	Sagging of the postero-superior wall.
6	Membrana tympani	Usually normal	Inflamed or perforated.
7	Discharge	Scanty, thickish	Copious, mucopurulent or mixed with a little blood at times.
8	Retro-auricular groove	Absent	Present.

Apart from the above differential diagnosis, it is said that, if a hollow needle is introduced in the post-aural swelling, it will strike pus immediately beneath the skin in furunculosis, and pus will only be drawn out after striking against the bone if it is due to acute mastoiditis. This is not true of all cases; we are familiar with cases of acute mastoiditis, who come to us in such an advanced stage that a big freely fluctuating abscess is present post-aurally, and the merest prick of the needle will bring out pus. Skiagraphy of the mastoids is of help, unless the mastoid itself be the seat of a chronic lesion.

PROGNOSIS:—In the normal course, a furuncle takes about five days to heal. Recurrences may occur, especially if the meatus is not properly cleaned, or if the patient suffers from some general disease, *e.g.* diabetes. In some cases, extension may result in perichondritis, or a permanent narrowing of the meatus may result. In the mastoid region, there might be subcutaneous œdema or an abscess. Pre-auricular and post-auricular gland get inflamed and may suppurate.

TREATMENT:—In the initial stage, when there is a circumscribed area

of inflammation, the meatus should be cleaned and drops containing acid Carbolic m iv, Spt. Rect. m xxx, Glycerine Zii be instilled three times a day. Fomentations externally are soothing. Applications of infra-red rays are very beneficial and tend to abort the disease. When the inflammatory process has progressed, a pledget of cotton wool, soaked in the Carbolic-Glycerine mixture, is gently introduced in the meatus, and left in situ for about six hours, when it is changed again. If the patient is seen when the furuncle has come to a head, it should be incised with a special furuncle knife or a fine curved bistoury, whose blade is wrapped up with a thin layer of cotton-wool, leaving its point exposed. If the patient is seen with a distinct fluctuating mass, immediate incision is the only treatment.

Prolonged use of Carbolic acid itself, may cause collection of much sodden epithelium; therefore, after the pain subsides, a little cotton wool soaked in Ichthyol Z ss., Glycerine Z iv is to be introduced into the ear and changed after about twelve hours. The patient is instructed to prevent water from getting into the ear while washing or bathing. Perichondritis or abscess-formation is treated with suitably placed incisions and drainage. In order to prevent recurrence, bi-weekly intramuscular injections of 1 c.c. Collosol Manganese or subcutaneous injections of a stock Staphylococcal Vaccine are given. The patient is advised not to scratch the ear. Any itching can be relieved by the application of a little absolute Alcohol on a wick of cotton wool. This removes the mild type of dermatitis often left behind as a sequel.

True eczema of the external ear is not a common condition. It is usually associated with urticaria, vasomotor rhinitis or eczema elsewhere in the body. The so-called eczema, much more commonly met with, is really an infective dermatitis, secondary to some other source of infection, e.g., suppurative otitis media. This condition is very commonly met with in children and especially during the hot and rainy seasons. In mild cases, all that is seen is a slightly rough surface of the skin of the meatus, with a scanty collection of flakes of epithelium and a few tiny scratches. If itching is intense, application of Ichthyol ointment gr. xx in Z iv of Eucerine will remove it. This is then followed by application of absolute Alcohol once a day for about a week.

In more advanced cases, a typical eczematous condition is seen, which may be dry or weeping. Secondary infection with pyogenic organisms may have taken place. When the weeping and irritation are troublesome, a pad of gauze soaked in the following lotion is applied:

$\mathcal{R}$ Zinci Oxide	...	grs. xx.
Calamine	...	grs. xl.
Tr. Lavandulae	...	Z ss.
Aqua distil	...	Z ii.

As soon as the 'weeping' has subsided, the eczematous area is liberally smeared with Ichthyol ointment. Any accompanying condition of the middle-ear is treated accordingly. In chronic cases, exposure to the ultra-violet rays and foreign protein therapy are beneficial. Applications of absolute Alcohol afterwards, will help to restore the skin to its normal tone.

IV. Parasitic diseases.—1. *Fungus infection or otomycosis*:—Moisture and warmth favour the growth of the fungus which finds lodgement in the external auditory canal. It is possible that the presence of

wax or other oily substance in the meatus forms a suitable nidus for its growth.

ÆTIOLOGY:—The disease is usually caused by the mould known as *Aspergillus*, although several other kinds have been described, namely, *Monilia*, *Saccharomyces* and *Verticillium graphii*. *Aspergillus niger*, *A. flavus* and *A. nidulans* are the varieties commonly met with.

SYMPTOMS:—The fungus may remain inside the ear as an innocuous mass for any length of time. When impaction occurs, as in the case of cerumen, deafness of abrupt onset may be complained of. The hyphae of the fungus may invade the stratum corneum of the skin of the meatus or even the superficial epithelium of the tympanic membrane. This will occasion a condition of external otitis when pain and tenderness become prominent symptoms. Tinnitus aurium or vertigo may be complained of.

DIAGNOSIS:—The fungus may form only a scanty mass in the canal adherent to the walls, and still allowing a clear view of the tympanic membrane. In other instances, the meatus may be found wholly or partially occluded by it. In still others, it forms a coating on the tympanic membrane. The colour of the mass varies with the type of mould present. It is blackish white if the fungus is *A. niger*, yellowish if *A. flavus*, and dark green if it be *Verticillium graphii*. *A. niger* is the commonest to see, and the appearance is often described as that of a piece of wet newspaper—the blackish areas are due to the spores of the fungus. Sometimes, the fungus is attached to the tympanic membrane, and presents a very characteristic appearance. It looks like a whitish mass of some fluffy substance like cotton-wool, interspersed with blackish dots. If viewed through a magnifying auriscope, it is easy to see the hyphae which compose the whitish portion, and the blackish spores are seen to grow out of it on slender stalks. On removal, the meatus and membrane might present an inflamed appearance, or the latter is only thickened and reddish.

PROGNOSIS:—Although removal is not difficult, the disease is rather intractable, and recurrences are quite common. Any damage to the tympanic membrane may also cause some amount of deafness.

TREATMENT:—The mass is removed by syringing after first softening with Soda Bicarb solution. The meatus is dried with wicks of cotton wool, and a 10 per cent solution of Silver Nitrate is applied for about a week. If this fails to check the growth of the fungus, application of a 2 per cent Alcoholic solution of Salicylic acid or $\frac{1}{2}$ per cent Alcoholic solution of corrosive sublimate should be applied well all round the meatus. Any drops containing Glycerine are, as a rule, avoided, although sometimes the pain is so severe, owing to the external otitis, that we have to prescribe sedative ear drops, containing Morphine and Cocaine in Glycerine. This is, however, discontinued as soon as pain subsides.

2. *Animal parasites*:—During the rainy months, insects in very large numbers crowd round a light, and sometimes it so happens that an insect finds its way into the ear. Its movements and fluttering give an uncomfortable feeling in the ear, and its sting is painful. Furthermore, any clumsy attempt at its removal may cause injury to the meatus.

TREATMENT:—The simplest thing to do is to fill the meatus with some oily substance. A little cocoanut-oil or mustard oil available in every

household will do. This will suffocate the insect and it will flow out with the oil on tilting the head, or afterwards removed with a cotton-wool swab under direct vision. One of my patients once tried an ingenious way of getting out an insect which got into his ear. He argued that these insects are attracted by light; why not then hold a light against the ear? He did it but failed to coax the intruder to come out. We laughed at the story. However, sometime later, I was able to test not only the logic but the truth of his argument. A colleague came to me one night, complaining of an insect having got into his left ear about a few minutes ago. I started examining the ear with the reflected light and had hardly introduced the speculum in the meatus, when the insect—a greenish little object—came into view just near the tympanic membrane. As soon as the beam of the light fell on him, he at once turned towards it, and quietly crawled out into the speculum towards the source of the light.

The other variety of insect that might get into the ear is the ordinary house-fly. Usually, it is found dead in the pus in a case of profusely suppurating otitis media, and is removed easily; however, the house-fly may cause a much more serious state of affairs, if it lays its eggs in the ear, producing small caterpillars. This condition is called myiasis, like the myiasis in the nose—a commonest occurrence.

SYMPTOMS:—The patient presents himself for examination, complaining of a severe boring pain in the ear, and a sensation as if something is crawling and biting inside. Owing to the severity of the pain, there is sleeplessness, general malaise, and loss of appetite. There is varying degree of deafness. The condition may go on to acute otitis media and its complications.

DIAGNOSIS:—Sometimes, it happens that the patient gives a history that worms came out of the ear. Several times, my patients have brought a few maggots, carefully kept in a small phial. On examination, a blackish, wriggling, shimmering mass is seen in the meatus. Sometimes, a worm or two might be seen crawling in the meatus. There is some sero-sanguinous discharge in the canal. On removal of the maggots after killing them, it is commonly found that they have already damaged the tympanic membrane and acute otitis media is present.

TREATMENT:—Syringing the ear with a lotion of Chloroform and Turpentine is advocated. It may not be advisable to syringe the ear, especially in the presence of acute middle-ear infection. Therefore, the maggots are first killed by filling the meatus with Olive oil, which chokes their respiratory organs. They can then be removed by gently irrigating the meatus with warm Boric lotion. Their removal from the middle ear cavity is more difficult, as happened in a case of mine—a child—aged two years. The maggots had destroyed the entire membrane, and it took three days before the last of the worms was washed out of the cavity. After cleaning, a seadive ear drop is prescribed. The following formula is good enough:—

R Acid Carbolic	...	℥ iv.
Morphine Hydro.	...	
Cocaine Hydro.	...	aa gr. ii ss.
Spt. Rect.	...	℥ xxx.
Glycerine	...	3 ii.
Mft. Guttae	...	

V. Aural diseases due to bathing.—During summer months, bathing is freely indulged in—especially in tanks, baths, rivers and the sea. Not infrequently, certain aural diseases are caused by it. These are either exacerbation of old lesions or are produced for the first time. The presence of water may swell up a mass of wax or fungus, and make its presence felt by giving rise to pain and deafness. The most important, however, is the increased incidence of middle ear suppuration. During the hot rainy days, when the water of the rivers is dirty and muddy, I have often heard the history that middle ear trouble started after a dip in the river or tank. A large number of such cases have had otitis media previously, and their trouble is an exacerbation of the old lesion. It has also been observed that the incidence is less in those who bathe in sea-water or clean river-water than those who bathe in stagnant tanks and common baths. The reason is obvious. As a prophylaxis, persons, specially those who have had otitis media once, should be warned against bathing and diving. They should be advised to plug their ears well with cotton wool. When the disease has already set in, it is treated according to whether it is just swollen wax, external otitis or otitis media.

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Summer Dyspepsia

BY

PARMANAND AHUJA, M.B., B.S.,

Karachi.

THE title of this article may appear to be a 'terminological inexactitude'. It may sound an unorthodox caption. But, my apology for using this expression is my inability to group together certain symptoms which a subjectively large majority of people of a tropical country like India feel on the advent of the summer season till about its middle. They do not complain of these symptoms in winter during which period their health is good. But the close, sultry, hot, dry weather, preceding the monsoon rains, brings in its train, certain changes in the system, consequent on the atmospheric conditions, which disturb the delicate heat-regulating mechanism in the human body, resulting in the symptom-complex which, for want of a better name, I have ventured to group under the term 'Summer Dyspepsia.'

Symptomatology.—The patient complains of lassitude, anorexia, loss of appetite, desire to take more of fluids than solid food, preference for certain articles of diet and abhorrence for other kinds. There are occasional attacks of headache, nausea and vomiting, especially in the early morning hours, with epigastric uneasiness or actual pain. There is a tendency to costiveness, but rarely diarrhoea may supervene, alternating with constipation. At times, there is a feeling of heaviness after meals, distension in the epigastrium and round about the umbilicus, relieved by passing of flatus. There is disinclination for close application to any work or study, feeling of drowsiness after meals, sense of ennui or fatigue after light exertion, mental or physical. Physical examination reveals flabby toneless condition of abdominal muscles. Liver is enlarged and hard to feel; stomach and intestines are distended, and packed faecal masses may be felt in the left iliac fossa near the pelvic rim. Tongue is dry and coated with flaky fur; breath may be heavy and foul. Abdominal reflexes are slow and sluggish. General outlook is apathetic, listless and asthenic. Heart and lungs do not reveal any abnormality except when there is accumulation of gas in the stomach, when general uneasiness and palpitation may be complained of by the patient in the cardiac region. If the constitution gets under-mined, mental disturbances in the form of irritability, loss of memory and even neurasthenia or hysteria may supervene.

Ætiology.—During hot weather in the tropics, there is always reduced bodily activity and the digestive powers are not so strong or vigorous as during cold weather. General musculature and tone of the intestinal muscles get loose and lax, resulting in constipation and intestinal stasis. High atmospheric temperature, ranging from 110° to 125°, coupled with high humidity of the air, has a very depressing effect on the organism as there is impediment in the transmission of the body-heat to the external air, in accordance with the law of radiation and conduction. This leads to 'locking up'—if one may use that expression—of the body-heat with congestion of internal organs like liver, pancreas etc. with deleterious effects to their delicate parenchyma and consequential impairment of their functional activity and digestive power. The only means of getting rid of heat during summer is by evaporation of sweat, which is in reverse proportion to the humidity of the air. If in addition, despite lowered physical

activity necessitated by hot weather, large carbohydrate intake, which puts strain on the digestive organs, and specially on the internal secretion of the pancreas, primarily responsible for the carbohydrate metabolism, is persisted in, it only adds insult to injury. Proteins have a high specific dynamic action, and a diet which includes proteins in excess is naturally a 'heating diet'. In hot weather, the production of excessive heat inside the body may be not only uncomfortable but may be actually injurious by taxing the peripheral circulation and sweat glands on account of the necessity of transferring the body-heat to the hot humid air. Such 'heating diet' may be indulged in not only in the form of proteins but highly spiced condiments, Alcoholic beverages etc. It has been rightly observed "that whenever the proportion of Alcohol circulating in the blood becomes greater than what the cells can readily deal with, it acts as a protoplasmic poison. The presence of undecomposed Alcohol leads to a diminution of the chemical energy of the cells which interferes with the ordinary course of metabolism."

Again, in summer season, there is great craving for iced drinks, sherbats and mineral waters. They not only give rise to congestion of the throat and distension of the stomach with gas, but, if taken with meals in excess, they dilute the digestive juices and impair the digestive powers.

All these factors combined bring about an asthenic dyspeptic condition with the train of symptoms narrated above.

Prognosis is hopeful and favourable—the mild and moderate cases recovering spontaneously as the intensity of the hot weather abates and the barometer registers a fall in the temperature. By the advent of winter, musculature of the body in general, and of the intestines in particular, regains its tone and vigour, keeping pace with the increased physical activity experienced all round, and the digestive organs, relieved of their congestive hyperaemia and the diluent effect of excessive fluid intake on their fermentative action, assume their normal metabolic equilibrium, resulting in improved relish for food, relief from constipation and a general sense of well-being and wholesome health.

On the other hand, if the person is a subject of chronic dyspepsia and indigestion, that condition is very much aggravated during summer months and the change of weather will not make any material difference and, in the very nature of things, there can be no expectation of his coming to the normal unless and until the underlying cause or the organic disorder, responsible for the chronicity of the disease, is treated.

Treatment.—It is mainly on symptomatic and general lines. With the change in the season, there should be corresponding change in the diet and habits of the individual. All 'heating diet' viz., proteins, especially animal proteins, rich fatty foods, spices and highly-seasoned articles of diet should, as far as possible, be taken sparingly. Alcoholic beverages should be eschewed altogether. Meals should be moderate in quantity, and overfeeding with bulky carbohydrate food should be avoided. Those predisposed to digestive disorders should be on their guard not to be betrayed into any dietary indiscretion during this season of the year. Use of fruits like banana, mangoes, dates, melons etc., should be restricted. Iced drink and aerated waters, with or immediately before or after meals, should be avoided. Instead, butter milk (lassi) and cocoanut water are cooling and refreshing substitutes and should be encouraged. Apart from iced-drinks, water may be taken freely and liberally between meals and in smaller

quantity with meals. Regular hours of eating, rest after meals—particularly after the midday meal—are helpful, and avoidance of exposure during hot hours of the day is desirable. Regular open air exercise, short of fatigue, and walking in cool hours of the morning are helpful to improve bodily activity, to tone up the muscles and to activate and oxygenate the blood and mobilize the sluggish liver etc.

By way of drugs, occasional use of a Saline aperient is serviceable to relieve constipation and milk out the bile passages.

A supporting abdominal binder should be worn if enteroptosis is evident. A digestive such as Takadiastase or Pepsin, Pancreatin and Papain with or without Strychnine or Nux Vomica is indicated for treatment of atonic dyspepsia to which the majority of the cases belong. The following prescription is recommended.

R Sod. Bicarb	...	gr. xv
Tr. Nucis Vom.	...	℥ x
Tr. Calumbae or Gentian	...	℥ xv
Spt. Ammon. Arom.	...	℥ xv
Spt. Chlorof.	...	℥ x
Aq	ad	℥ i

to be taken half an hour or an hour before food. If symptoms point to hypo-acidity, Sodii. Bicarb. may be replaced with acid Hydrochlor dil. ℥ x.

Iron, Arsenic and Strychnine or Nux Vomica are of use in cases of marked anæmia or debility.

Flatulent distension of the stomach will require appropriate remedies like Tr. Card. Zingiberia, acid Hydrocyanic, dil. Spt. Ammonia Arom. and Spt. Chlorof. in Peppermint water. A pill containing Creosote, Menthol or Carbolic acid given immediately after food is praised by some, in such cases. Exhibition of vitamin B as Bemax, Marmite or in pill form, exerts favourable influence. Cases of organic disease of the stomach or intestine giving rise to dyspeptic manifestations are clearly out of the scope of this article.

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Calcii Hypophosphites	...	" 10
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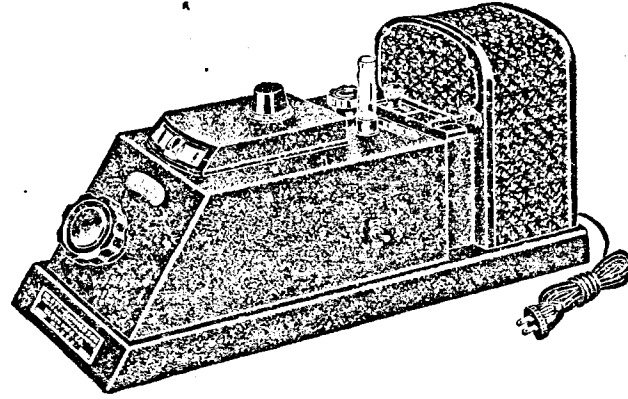
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Summer Complaints of the Eyes

BY

S. SANYAL, M.B.,

H. O. R. Institute, Calcutta.

SUMMER complaints of the eye may be divided into the following groups:—

- I. Those associated with direct injuries to the eye by heat and light rays.
- II. Those associated with systemic reaction of the whole body in response to low humidity and high atmospheric temperature.
- III. Those associated with allergic conditions which are manifested more in this season than any other.
- IV. Those associated with the local infection of the eye which is prevalent during this season.
- V. Those associated with general infections in which the eye sometimes takes part.

I. Those associated with direct exposure of the eye to heat and light rays.—Mostly, the conjunctiva is affected, specially in those leading an out-door life without taking proper care to protect their eyes from heat rays.

It is true, that a great majority of people in our country become accustomed to such exposures, but a certain percentage, specially fair-skinned ones, are the worst sufferers.

The symptoms are those of hyperæmia of the conjunctiva *viz.*, redness of the bulbar and tarsal conjunctiva, lachrymation, photophobia and gritty sensation. If the congestion is persistent, there may be slight discharge, collecting at the angles of the inner canthus.

Too much exposure to heat and light may cause a certain amount of retinal irritation, which is chiefly expressed in the eye, as photophobia, lachrymation, inability to see anything for any length of time and close work soon blurs the vision, and if there is associated choroidal hyperæmia, there may be actual pain in the eye, which is variously expressed as deep boring, or hypersensitiveness to touch, indrawing of the eyes; ophthalmoscopically, nothing except signs of retinal irritation are found. Correction of error of refraction does not materially help. Dark adaptation is altered, and the light minimum is raised. Blue field is markedly diminished. In some of these cases, there may be partial blue blindness.

II. Those associated with systemic reaction of the heat and light rays.—In this group, the manifestations are:—

(1) A hyperæmia of the tarsal and lower palpebral conjunctiva, which generally extends slightly to bulbar conjunctiva with oedema of the lids, which is, in some cases, out of proportion to the hyperæmia of the conjunctiva.

They may remain for a few hours or persist for some days, specially if the patient exposes himself to the sun. A periodicity in the increasing of symptoms is sometimes observed, the symptom increasing or coming every three or four hours, remains for a few minutes to an hour or so, and

then gradually diminishes till the next period. In others, a definite increase of symptoms begins either at 12 O'clock in the noon or at 3 P.M., and persists till the evening. Throughout the rest of the day, the patient is comparatively free from any symptom. The chief complaints are: sensation of foreign body, a hot feeling in the eye, and itching. The latter symptom may be either generalised and so intense as to simulate spring catarrh, or it may be localised, generally to the inner canthus. Lachrymation of varying degrees is a constant feature in association with gritty sensation. With burning sensation, it may not be so constant.

In some of the cases, the symptoms and congestion may be so acute as to simulate an infective conjunctivitis as is shown by the following case. —H. F. came complaining of lachrymation, gritty sensation and slight photophobia, specially in the left eye. Discharge was very slight, just a small amount at the inner canthus. Examination showed: L. E.—oedema of the lids. Both the bulbar and the palpebral conjunctiva were hyperæmic, other parts being normal.

The symptoms increased every fourth hour throughout the day. At night after 9 P.M., she was comparatively free from any trouble. R. E.—was comparatively free.

The present complaint has begun only about 3 days back, and the onset was sudden. The conjunctival scrapings did not show any bacteria or any other infective agent. No eosinophils were seen. In these cases, the conjunctiva is always bacteria-free.

(2) *Another type* is that of localised congestion and oedema of the bulbar conjunctiva, generally appearing in the inter-palpebral region, on the external side, about 2 or 3 millimeters in diameter, shading off gradually into the surrounding area. It is not painful to touch or pressure. Episcleral tissue is not generally much involved.

Histologically, it is something like urticaria with oedema of the conjunctiva, with scattered cells between them; no eosinophilic cells have been found. The symptoms are a sense of discomfort and gritty sensation with sudden desire to rub the eyes thoroughly at times. Slight photophobia and lachrymation are also present. 'Lunge' scleral transilluminator helps to show the nature of the nodule.

(3) Slight ciliary injection of one eye without any pain, slight mistiness of vision, lachrymation, photophobia and foreign body sensation. Biomicroscopy shows only slight oedema, chiefly confined to the anterior layers of the cornea. Scrapings of the cornea do not reveal any bacteria.

The associated symptoms are those of imbalance and irritability of the autonomic nervous system of the whole body; and periodicity in the aggravation of the symptoms is a noteworthy feature. Smallest impulses which, in ordinary cases, do not produce any disorder in the normal nervous function, produce an abnormal irregular and exaggerated reaction in these cases.

In the digestive system, this irritability is manifested by the spasm generally at the sphincters, but in some cases the whole organ may participate in the reaction.

Irritability of the œsophagus causes difficulty in swallowing any solid articles of food, the general complaint being pain during the passage of

the bolus. In the stomach, the smallest amount of food, specially solid, causes a sense of fullness of the stomach nausea, which, in some cases, causes an actual regurgitation of the whole food. This specially occurs if the patient is compelled to forced feeding. Spasm of both the cardiac ends of the stomach causes pain just under the xyphoid cartilage; that of pyloric end causes an acid eructation hyperacidity due to inability of the alkaline fluid of the intestine, thus regurgitated into the stomach, and the patient complains of acid eructation shortly after the food. There is loss of proper taste for the food, and metallic acid or bitter taste in the mouth is complained of. Salt and acid food and chillies are generally liked by the patient. Few patients like sweets or milk.

Gums may be suddenly swollen and painful. In extreme cases, neuralgia of the trigeminal nerve may be caused due to spasm of the pyloric end and of ileocecal valve. It goes away as suddenly as it comes. Small intestines may be bloated and pressure on the navel region may cause some pain. In the large intestine, the cæcum is painful to touch and the junction of the transverse colon with the ascending and descending portions may be thrown into spasm, sometimes, simulating gall bladder. Some tumour cord-like spasm of the ascending colon is a frequent phenomenon, and when this occurs, the patient is thrown into agony and the pain may be so intense as to shake the whole body. Closure of the transverse colon at both ends and the dilatation of this portion of the gut, gives pressure on the stomach, giving rise to a sense of fullness of the stomach. Constipation is a marked feature and adds to the discomfort. In the circulatory system, palpitation of the heart—sometimes, the rates rising as high as 140—may occur. The volume of the pulse is large, and pressure low. Blood pressure may rise. This rise may come on suddenly, remain for an hour or two, and then subside. Or, there may be recurrences every three or four hours, so that the symptoms may almost be anticipated. In still further heightened excitability, the pressure may be continuous for hours together.

Nervous system:—Burning of the soles and palms, scalp or of the whole body; irritability of temper, loss of memory, talking too much or preferring to remain alone; inability to do mental work for a long time. Any attempt at reading causes pain in the vertex, even the slightest physical exertion may cause annoyance; may be mentally depressed; thinks that life is not worth living or may be planning very high work without any attempt to execute it. Vertigo.

The symptoms that have been described are those of an imbalanced nervous system. Probably, the summer season is not directly responsible for it, but the large number of patients that crop up during this season and the amelioration of the systems, if there is a shower of rain any day, seems to have some connection with the heightening of the nervous irritability in an already neurotic or in those that are just maintaining the nervous balance.

The heat and light rays in this season, affecting the capillaries of the cutaneous blood vessels, do some injury to the skin, as is shown by the increase in pigmentation and sweating, which is Nature's effort to protect the organism, so that the whole circulatory apparatus is indirectly affected and along with it the autonomic system. If the nerves are strong,

this stimulus increases the tonicity; otherwise, the opposite reaction occurs. Instances of this are also frequently seen in ultraviolet ray exposures where much damage may be done to the system by over exposures.

The conjunctiva, cornea and the retina, being of ectodermal origin, react to the same stimulus to a certain extent, and it is no wonder that the eyes are also affected.

The treatment of this condition is briefly, frequent cold baths, Ephedrine, Calcium, Liq. Auri. et Arsenic Bromides, Penta Bromides, cold fruits, milk and milk-preparations, sometimes salt-free diet, fruit-juice and small and guarded doses of ultra-violet rays. Absolute rest in a cool, dark-room may be necessary.

The eye symptoms generally do not require any separate treatment; cold douching of the eyes, frequently with cold Saline, Liquid Paraffin with Cocaine ($\frac{1}{2}$ %) will be beneficial.

Iced compress to the eyes, forehead, vertex, nape of the neck and abdomen, generally relieves the symptom. Dark goggles are soothing.

III. Allergic conditions.—Besides those, associated directly with the effect of heat on the body, allergic conjunctivitis and keratitis are common in this country, of which spring catarrh is well known. This is a disease of which, although no definite cause can yet be found, the evidences point out it to be of an allergic nature.

The various theories that have been held from time to time are: (1) It is an expression of endocrinopathy (2) vagotonic conditions associated with insufficiency of the suprarenals (3) allergic reaction of the conjunctiva, due to hypersensitivity and intoxications from specific allergens.

The evidences adduced in favour of this theory are: (1) Local hypersensitivity to specific antigen may be produced in the conjunctiva, either as a part of a general hypersensitiveness or, if the allergen is directly introduced into the conjunctiva, there is only local reaction. (2) The existence of specific hypersensitiveness has been investigated by various authors and, in a certain percentage of cases, they have been found to exist. (3) Familial tendency, although not common, is not rare. (4) Histologic study (5) Clinical symptoms are similar to other allergic manifestations. Clinically, the disease is divided into palpebral and bulbar forms. When the palpebral form—occasionally an abortive form—occurs the whole eye may be blood shot with little or no discharge, the disease subsiding spontaneously in a few days; but more commonly, the flat papillæ of various sizes appear on the tarsal conjunctiva, most numerous on the proximal border of the tarsus, the distribution being irregular. In the later stages, the whole of the tarsal conjunctiva may be studded with flat-topped papillæ and the conjunctiva takes on a milky appearance. In the lower lid, however, the papillæ are numerous and more rounded. Occasionally, in the upper lid, the papillæ may grow enormously, and cauliflower-like excrescences with deep furrows between them may appear. The type of vascularisation is peculiar, and differs from that of trachoma. In the affected area, a deep capillary net-work sends up vertical branches, appearing to the naked eye as small petechial hæmorrhages which, in their turn, send out a second regular fine net-work, giving a reticular appearance, best visible in biomicroscopy. In each flat-topped papillæ,

there may be several such vascular centres. Another characteristic is 'Trantas' Points' which are small, white, chalky concretions appearing on the conjunctiva. The discharge is characteristic. It appears as a thin film over the tarsal conjunctiva, is dirty white in colour, can be readily peeled off from the surface of the conjunctiva without any bleeding, microscopically contains epithelial debris and eosinophils in large numbers and is characteristic of this disease.

In the bulbar type, discrete lesions may appear on the limbal conjunctiva or in confluence appearing as a ring of gelatinous mass 2 to 3 mm. in diameter round the cornea, and it generally persists throughout the year in India. The most common site is the palpebral fissure. In the raised mass, Trantas' Spots may appear. In a case seen by the author, there was a big tumour-like formation in the palpebral fissure, occupying nearly half of the lateral side, extending to the cornea for about 2 millimeters. There was no pain. Scraping of the tumour gave a gritty sensation.

The bulbar type has got to be differentiated from similar gelatinous masses seen in the kerato-conjunctivitis (SANYAL), in which there are no eosinophilic cells; the gelatinous appearance is due to the swelling and degeneration of the epithelial cells without any significant hyperplasia of the mesodermal tissues or their hyaline degeneration. The virus is found in the cornea.

Corneal involvement:—(1) Direct extension from the limbal lesions. (2) Secondary dystrophies.

Direct extension is the more common type and has long been recognised. It takes the following forms: (1) Vegetations identical with those of limbus. (2) Large vegetations destroying the Bowman's membrane. (3) Gelatinous pannus-like membrane. (4) Small epithelial lesions like O-cells of trachoma. (5) Interstitial lesions like sclerosing keratitis from the limbus. (6) New capillaries may spread from the conjunctiva without any dicrotonous branching, being surrounded by a zone of infiltration which never ulcerates.

Similar vascularisations have been described in allergic interstitial keratitis with eosinophilic cells in the cornea by the writer.

Corneal dystrophies:—The most common is the keratitis epithelialis vernalis of TOGBY, in which cornea appears as if finely dusted with flour, with œdema and formation of fine cystic spaces in the cornea. Discrete opaque points in the epithelium, due to complete degeneration of all cells and their replacement by fibrous tissues.

(2). Peripheral degeneration in the interstitial tissues resembling the arcus senilis.

PATHOLOGY:—In the palpebral form, there is an enormous overgrowth of the connective tissues, accompanied by proliferation of epithelial cells which become several layered deep and send off-shoots down into the connective tissue below. Numerous new blood vessels appear; cells consist chiefly of lymphocytes, plasma cells and eosinophils.

In the limbal type, the epithelial hyperplasia is more evident than the connective tissue proliferation—the former sometimes reaching 30 to 40 layers. They also send down epithelial plugs and are so numerous as to resemble epithelioma.

SYMPTOMS:—The most prominent symptoms are burning and itching of a most intolerable nature. Other symptoms are a mild photophobia and lachrymation. Discharge is slight.

PROGNOSIS:—This disease tends to cure spontaneously after some years, but sometimes it may persist as long as 24 years.

TREATMENT:—There is no specific treatment for this disease. The most efficient treatment is to change the patient from a hot to a cold place. In the alternative, rest in bed, avoidance of sun's rays, frequent cold bath or washing the head, arms and feet with graduated cold water upto ice-cold temperature, milk diet, avoidance of animal proteins and irritating and hot spices or food, the moderate use of general ultraviolet rays, injection of Colloidal Calcium, Arsenic in the form of Liq. Arsenicalis or Liq. Auri et Arsenic Bromide in 2 to 3 minim doses and Ephedrine are useful. The following prescription in association with a salt free diet has also been found useful.

R Hydrarg cum creta	...	gr. i
Sublimated Sulphur	...	gr. $\frac{1}{4}$ to i
Ephedrine Hydrochlor	...	gr. $\frac{1}{4}$
Atropine Sulph.	...	gr. $\frac{1}{400}$
Calcium Lactas	...	gr. x

Morning and evening, followed by a cup of hot milk and Penta Bromides dram 1 doses with Tr. Nux. Vomica $\mathfrak{m}$. ii per dose.

LOCALLY, frequent washing the eyes with ice cold Saline or Boric lotion relieves the patient.

The following prescription will be found effective:—

R Acid Acetic dil	...	$\mathfrak{m}$ i
Sol. of Adrenaline Chlor (1:1000 P. D. & Co.)	...	$\mathfrak{m}$ v
Cocaine Hydrochlor.	...	gr. ii
Aqua Rose	...	$\mathfrak{z}$ iv

Another drug which has been recently found very effective is a proprietary medicine 'Pescin' which is a combination of several metallic ions in colloidal state; other drugs used are monohydrated Sodium (gr. v to ounce i), Lactic acid $\frac{1}{4}\%$, Chaulmoogra oil etc.

If vegetations are large, surgical methods may be resorted to. Radium and Carbon dioxide snow have also been used for the same purpose.

Irradiation of the forehead and the skin of the eye with ultraviolet rays, one or two minutes, at a distance of one foot, at intervals of 3 days is effective. Irradiation of the root of the nose between the eyebrows for an area about the size of a four anna bit, the dose rising slightly above the erythema dose, gives relief in many cases.

In my opinion, the treatment of this disease is a treatment of the autonomic imbalance; so that each individual case requires separate consideration. Attention to this will shorten the course of the disease and prolonged suffering for so many years.

Hay fever conjunctivitis:—It is an allergic conjunctivitis, associated with hypersensitivity of the upper respiratory tract, occurring during summer in young individuals, about the age of puberty due to pollen grains.

Summer Complaints of the Eye—S. Sangal.

Fig. I.
Section of the tarsal conjunctiva, showing papillae formation. The epithelium is many-layered.

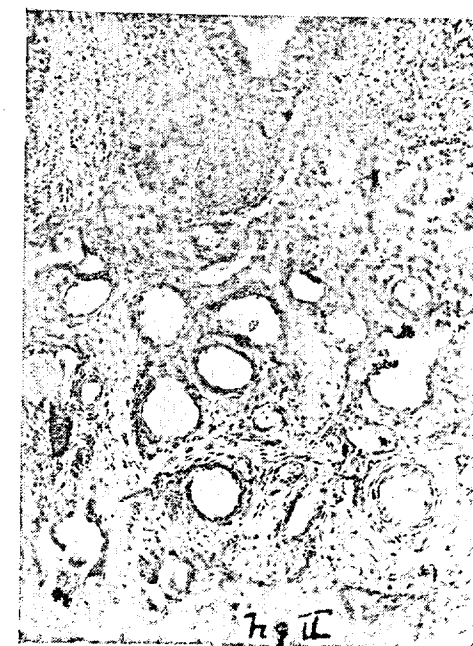
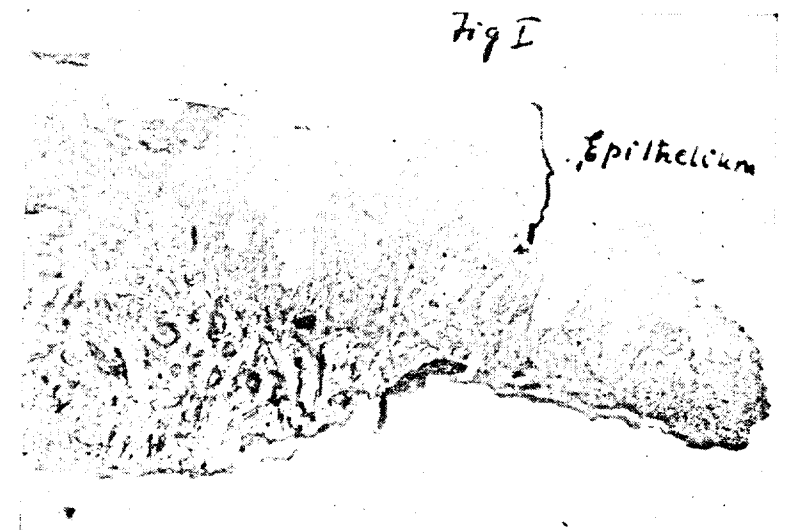


Fig. II.
Section of the tarsal conjunctiva, showing hyperactivity of the sub-epithelial layers.



Fig.
Section of the tarsal conjunctiva, showing hyperplasia of the epithelial cells, and secondary phlycten.

Summer Complaints of the Eye—S. Sanayal.

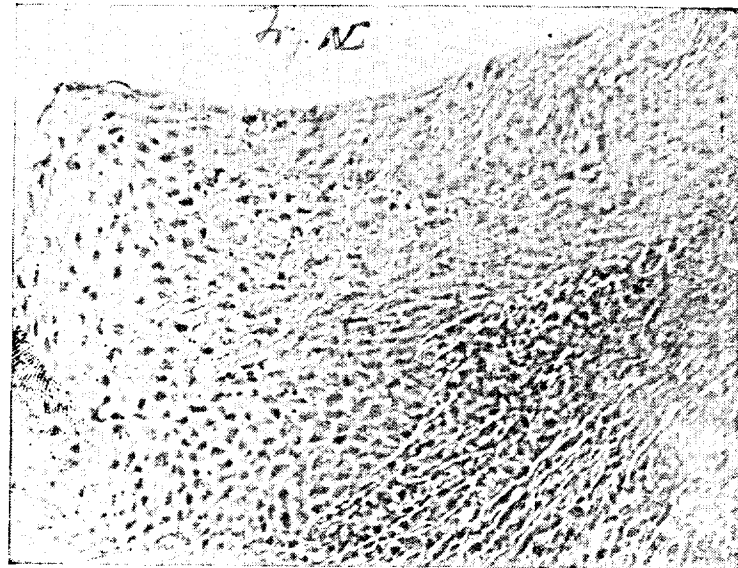


Fig. IV.

Section of the bulbar conjunctiva, showing hillock-like hyperplasia of the epithelial cells. They look like secondary phlyctens. Highly magnified.

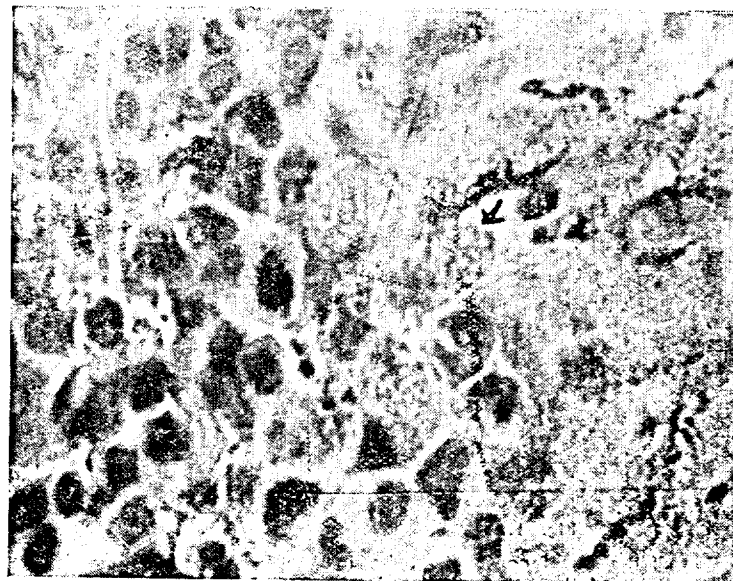


Fig. V.

Eosinophils in the sub-epithelial layer.

SYMPTOMS:—It comes on suddenly, accompanied by intense redness, lachrymation, itching and burning sensations. Sometimes, lids become oedematous. If the allergen is withdrawn, the symptoms subside within a few minutes. If, however, repeated attacks occur, a slight chemotic condition may persist throughout the year.

A type of allergic conjunctivitis occurs in Bengal, during the summer season amongst those working in the fields. It affects all ages, but the young are specially prone to it. The onset is generally sudden and there is intense redness of the conjunctiva, with oedema of the lids. There is some amount of mucoid or mucopurulent discharge. The symptoms of itching, photophobia and lachrymation are present. The tarsal conjunctiva is intensely red and velvety, no haemorrhages are seen. The bulbar conjunctiva is also brick red and may be slightly chemotic.

In some of these cases, which persist for some days, yellow areas like the early trachoma follicles or secondary phlyctens may be seen in the bulbar conjunctiva.

Cornea may also be affected as small infiltration near the limbus or as keratitis, extending from the limbus. In the latter type, a single blood vessel may be seen to be proceeding from the conjunctiva to the cornea. These blood vessels are tortuous but do not branch or form loops. The discharge shows leucocytes, 80% of which are eosinophils and a large amount of epithelial debris. There may be blood eosinophilia.

Histological section of the tarsal conjunctiva shows epithelium to be several layered. In some places, the normal arrangement of the epithelium is replaced by cuboidal cells of irregular size.

The sub-epithelial connective tissues also grow with the formation of papillae. There is intense dilatation of the subepithelial capillaries with proliferation of endothelial cells which, in some places, aggregate like a lymphatic follicle without any capsule. Plasma cells and histiocytes are also seen; in between these may be seen many eosinophilic cells, some of which may be seen escaping through the epithelium to the outside. The epithelium may become degenerated at places and lead to cystic formation, but no degenerative changes are found in the subepithelial connective tissues.

In the bulbar conjunctiva, instead of papillae, the epithelium becomes many layered and at places, the growths may be so much that they project above the surface of the epithelial layer like a hillock and clinically look like secondary phlyctens. There is intense reaction in the subepithelial tissue. Similar pictures are seen in the cornea also.

The histological changes are in short like those of acute spring catarrh and other allergic conditions. In these cases, the allergen has not always been detected, but few cases gave reaction to the pollens prevalent during the season at the particular locality.

The prognosis is good as the disease rapidly subsides if the patient can be removed from the site of allergens; otherwise the patient is liable to suffer periodically, unless the allergen can be found and desensitised.

Treatment should be simple and soothing. Iced normal Saline drops. $\frac{1}{2}$ % Cocaine alkaloid in sterilised liquid Paraffin; Iced compress and desensitisation, if possible. Treatment should also be directed towards the

restoration of the imbalance of the autonomic nervous system. Cold bath, milk and fruit diet, avoidance of heat and sedatives to the nervous system such as Bromides, Arsenic, Gold, Atropine, Ephedrine, Arnica, Calcium are useful. 'Charnosal', a proprietary medicine which contains these drugs, in appropriate doses, with addition of extracts of some Indian vegetable drugs, is useful.

IV. Infective group.—The conjunctiva and cornea may either be primarily infected or may participate in the general infective conditions.

(1) **KOCH-WEEK'S INFECTION** affects people of all ages except the very young. After incubation period of about 36 hours, a sensation of burning occurs, soon followed by intense redness of the eyes, sometimes associated with small hæmorrhages. The disease reaches its height in about 3 to 4 days, when it begins to subside and, in about 10 to 14 days, usually disappears. As infection does not confer any immunity, relapses may occur or the disease may drag on to a chronic stage. The discharge is muco-purulent or frankly purulent, and is highly contagious. Cornea may be affected and perforation may occur. Secondary phlyctens are common.

Bacteriology:—These are hæmophilic gram-negative bacilli, measuring about 0.5 to 2 μ , somewhat similar in appearance to Influenza bacillus.

Koch-week's bacillus as found in Bengal, differs from those of European and American countries in that subcultures on ordinary nutrient agar may be done.

Treatment:—Gentian Violet lotion (1-5000) as wash, Argyrol (5%), Yellow Oxide of Mercury (0.5%) are useful. Care should be taken to isolate the patient.

(2) **INFLUENZA:**—Apart from mild catarrhal conjunctivitis, occurring during the epidemics of influenza, isolated cases, characterised by a thin, watery or flocculent discharge, containing large amounts of Influenza bacilli in the epithelium, occur and run a typical course of about 10 days and disappear without any complication.

Besides the above type, another type which is found occasionally in Bengal during routine bacteriological examination of the eye, is characterised by the formation of thin membrane over the tarsal conjunctiva. This membrane can be peeled off in a sheet without any hæmorrhage. It contains epithelial debris having numerous Influenza bacilli in the cells and few leucocytes. This discharge is slight. Photophobia, lachrymation and intense itching are the prominent symptoms. No eosinophil can be found. Cultures from epithelial scrapings show typical Pfeiffer's bacillus.

(3) **KERATO-CONJUNCTIVITIS (SANYAL):**—This is a disease with protean manifestations, affecting both the cornea and the conjunctiva. Discharge is slight. The chief characteristics are the areas of yellowish spots, generally of the inner portion of the tarsal conjunctiva, which are histologically places where subepithelial degeneration has taken place. Cornea is also affected as keratitis or ulcers. Pannus is, sometimes, found simulating trachoma. Inclusion bodies are found in the epithelial cells. It is very difficult to eradicate the disease by ordinary treatment. Sulphanilamide in the form of an ointment, is effective.

V. Those associated with general infections.—(a) *Small-pox* in this season, probably, is the most potent factor in the destruction of eye-sight

in India. The conjunctiva and the cornea may both be affected. (1) In the conjunctiva, about the 5th day of illness, an exanthematous catarrhal conjunctivitis appears which clears up under ordinary treatment. (2) In hæmorrhagic types, a subconjunctival hæmorrhage may appear. (3) Purulent types also occur. (4) Pustules may appear generally on the bulbar conjunctiva, either on the outer or medial side of the bulbar conjunctiva. Clinically, they look like phlyctens, but excite great reaction, and there may be great pain and oedema of the conjunctiva. If they appear on the limbus, corneal ulcerations may occur. Less commonly, tarsal conjunctiva is affected. In the cornea, the pustules may appear anywhere on the cornea, and may be either single or multiple. They rapidly go on to necrosis, and may involve the whole of the cornea. Generally, however, it is limited to one portion. Perforations may occur. Sometimes, there is a total necrosis of the cornea. Hypopyon and panophthalmitis may occur.

Treatment should be non-irritant and soothing. Liquid Paraffin or sterilised Castor oil with $\frac{1}{4}$ % Pantocaine or similar drugs, Eserine Sulph. $\frac{1}{8}$ % as lotion and Sulphanilamide ointment are the best drugs. Cold compress is generally soothing and may be given in the form of several layers of small pieces of lint soaked in iced water and placed over the closed eye for 5 to 10 minutes, changing it as soon as the lint becomes hot.

(b) *Varicella:*—Generally, a catarrhal type of conjunctivitis occurs; occasionally, however, phlycten-like pustules occur on the conjunctiva, which soon become necrotic and form excavated ulcers. If they occur on limbus, there may be corneal ulcers. The pustules are generally accompanied by great reaction and pain. Treatment is the same as for variola.

(c) *Cholera:*—Low tension, ciliary injection, dullness of the cornea, ulceration and keratitis lagophthalmos of cornea. Treatment consists of some sterile oily substance as drops till the general condition improves. Honey as drops is effective in many cases.

(d) *In summer diarrhoeas of children:*—Kerato-malacia may occur. The treatment depends on the improvement of the general condition. Locally, Cod-liver oil or 'Adexolin' as drops may be used. Injections of vitamin A and B are helpful. Small doses of ultraviolet rays over the closed eyes and abdomen—not more than one or two minutes' exposure—at distance of 3 ft. guarding the over-reaction by Ephedrine, Calcium, Glucose and Penta Bromides in small doses, save the eye in many cases; Calcium should be given preferably as injections in the form of Colloidal Calcium.

(e) *Furunculosis* of the eye lids and stytes are common complaints of children in this season. Treatment is as for these conditions in other parts of the body. Local ultraviolet rays, at 4 inches' distance, for 2 to 5 minutes, relieve the pain and help to localise it and burst. Prontosil group of drugs may be given internally. Animal proteins should be prohibited.

(f) *Streptococcus infection of the conjunctiva:*—During the reaping of paddy harvest, cases of infection by this organism may occur. Generally, it occurs after some slight injury by the ear of the paddy crop, either during thrashing or reaping.

SYMPTOMS are those of intense chemosis of the conjunctiva and discharge which is copious. Lids are very much swollen and the cornea may show ulceration. If the organism spreads internally, panophthalmitis,

thrombosis of the cavernous sinus and meningitis may occur. Peri-auricular glands may be enlarged and painful. Pain over the eye is common and the eye is sensitive to touch. Head-ache, general malaise and slight rise of temperature occur from the beginning.

PROGNOSIS should be guarded.

TREATMENT:—Cold compress, ultraviolet rays over the closed lids; Sulphanilamide (0.8% sol.) 2 c.c., may be injected subconjunctivally every 12 hours and as ointment (5%) locally; oral administration and parental injections may be necessary.

86, Dharmatolla Street,
Calcutta.

"Zalavani" for Slight Temperature in Summer

BY

D. V. CHAFKAR, B.A., M.B.B.S.,
Gadag.

A few interesting facts, which I have noted in my humble practice at my place, are recorded here. For a private practitioner, research of a scientific nature is not possible, in the absence of a well-equipped laboratory and hospital, where cases can be kept under observation. However, my article may be a stimulus to some better-placed professional brother to start an investigation in the matter, and thus help our art of healing.

In the early days of summer—that is, in "*Phalgun and Chaitra*—children very often get a low temperature, of 99° or 100°, without any apparent cause. Drugs do not seem to help a cure. At least I often met with a failure. Then, I tried the following method and found it useful.

I know of a custom amongst us, in which babies and young children are given a bath with water heated in the sun in a copper vessel in the early days of summer, for the relief of low temperature. They call it *zalavani* in Marathi.

A "Ghada-full" of water is kept in the sun, with the mouth covered with a thin piece of cloth. With this water, children are given a bath in the evening. If the water is too hot, it is allowed to cool down to comfortable warmth and then used. No cold water is added to it. Boiled raw mango is used for smearing the body with its pulp like soap. After the bath, the child is allowed to go to bed.

I treated a few cases in this way, and, to my surprise, I found it useful. I do not know how it works. As my experience is too small, I simply wish to make a note of it.

I cannot account for the rise of temperature in children in summer, except that the heat-centre in children is not capable of adjusting the body-temperature with sudden and rapid rise of the surrounding atmospheric temperature. It takes a little time for this adjustment, and probably the bath helps it, as the bath is given more than once during the course of a week.

Corrigendum.

On page 486 of this issue, line 3, from bottom, substitute '*Tinea Cruris*' for '*Tinea Cruris*'.

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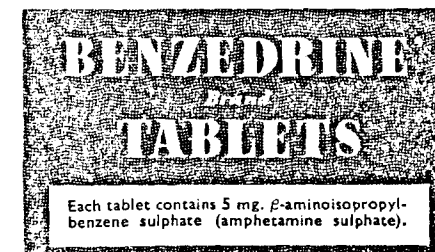
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British Medical Journal,
Nov. 9, 1940, p. 628.



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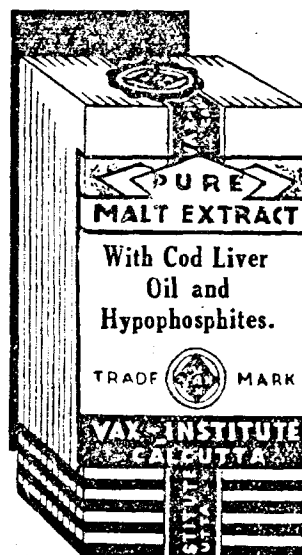
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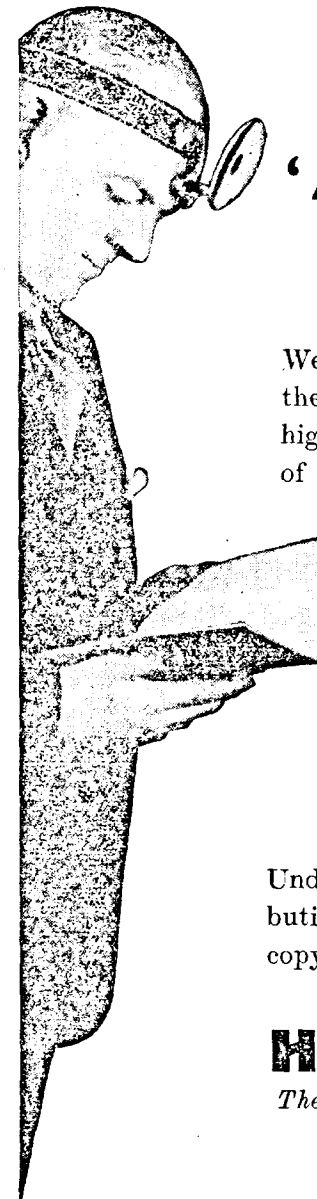
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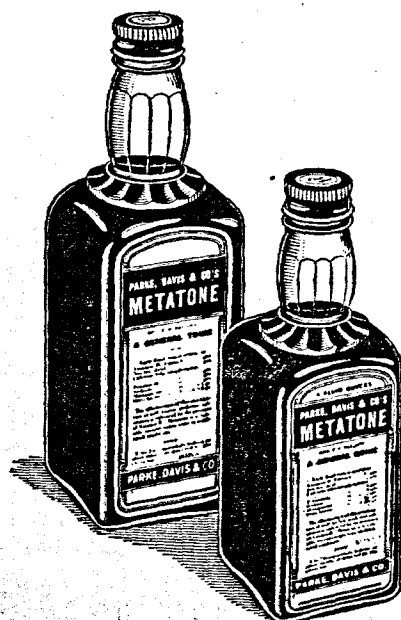
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