CHAPTER 9

SALT DEFICIENCY AND HEAT EXHAUSTION STATES

It is probable that the inducement to add salt to food occurred as man developed from a hunter living on an exclusively animal diet, to a shepherd and a tiller of the soil eating mainly vegetables. Vegetables contain a great deal less salt than meat, and so man began to suffer from a shortage of salt and to develop a craving for it; the influence this had on his way of living can be seen from the history of his customs and from his writings.

For more than 2,000 years it has been recognised that high external temperatures may have an injurious effect on the human body, and the effects of heat have been described in medical literature for some 200 years; little was known, however, about the pathological processes involved in the effects of heat until the beginning of the present century when they were noticed in workers in the iron and steel industries. One of the pioneers in the systematic investigation of these states was Edsall, who recorded in 1904 the significant finding of a diminished excretion of chlorides in the urine of patients showing clinical signs of cramps and prostration after exposure to intense heat. During the war of 1939-45 the importance of disorders resulting from high temperatures increased greatly, especially on ships operating in tropical waters. The need to replace the sodium chloride lost in sweat assumed great significance.

This chapter records experience gained in Australian naval vessels during the war period in tropical climates, including the Gulf of Aden and the Red Sea, the East and West African coasts, and the equatorial zones of the Indian and Pacific Oceans. Although the climate of a tropical zone could not be controlled, much could be done to mitigate its ill-effects, and in this the medical officer contributed greatly to maintaining the health and contentment of a ship's crew.

Physiological Factors. The use of salt in food is neither regular nor reliable, and varies greatly according to individual taste. The natural intake of salt in an ordinary mixed diet is probably less than 3 grammes a day, but with that added during cooking and at the table, the daily average would be about 10 grammes. The approximate sodium content of common articles of diet is given in the accompanying table.

<table>
<thead>
<tr>
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<th>Milligrammes per 100 grammes</th>
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<tbody>
<tr>
<td>Meat</td>
<td>65-80</td>
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<tr>
<td>Milk</td>
<td>43</td>
</tr>
<tr>
<td>Eggs</td>
<td>185</td>
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<tr>
<td>Offal (liver, kidney, ham)</td>
<td>110-160</td>
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<tr>
<td>Fish</td>
<td>120-190</td>
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1 In writing this chapter the author has leaned heavily on Surgeon Lt-Cdr M. J. L. Stening's article "Salt Deficiency States in Tropical Climates", Journal of the Royal Naval Medical Services, Vol. 31, pp. 129 and 195 (1945).
SALT DEFICIENCY AND HEAT EXHAUSTION STATES

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<thead>
<tr>
<th></th>
<th>Milligrammes per 100 grammes</th>
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<tbody>
<tr>
<td>Cereals</td>
<td>5-30</td>
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<tr>
<td>Potatoes</td>
<td>3-4</td>
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<tr>
<td>Vegetables:</td>
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<tr>
<td>green</td>
<td>3-15</td>
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<tr>
<td>root</td>
<td>10-60</td>
</tr>
<tr>
<td>Nuts</td>
<td>2-4</td>
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In temperate climates an ordinary mixed diet, palatably salted, supplies the normal mineral needs of the body. In the tropics, however, especially if there are also factors of bad ventilation, unsuitable clothing and muscular work which cause profuse sweating, the great amount of salt so lost calls for an increased intake which is varied according to the need. In a series studied by Surgeon Lieut-Commander M. J. L. Stening, in Australia, the ration of salt varied from 4 grammes a day in more favourable conditions to 8 grammes a day in adverse conditions. Some men who felt the need for increased amounts of salt took voluntarily as much as 30 grammes a day with benefit. The ration was best absorbed, and interfered least with the appetite, if taken independently of food—say before breakfast and before going to bed. If taken in water in a concentration of about 2 grammes per cent, the flavour of the water was pleasant. Saline solution, however, was not the most popular medium, and salt was more freely taken as salt, although Surgeon Lieutenant C. K. Churches in Warrego, who had to supply salt tablets for the boiler-room staff, found that men would take salt in tablet form where they disliked other methods of administration. A convenient salt tablet contained ½ gramme with an additional equivalent amount of dextrose, taken in one to three doses daily. Curiously, a taste seemed to grow for it, due perhaps to the allaying of thirst which should follow a correct distribution of sodium chloride in the body fluids. The benefits of taking extra salt were immediately apparent, which is surprising when it is remembered that the absorption of sodium chloride occurs mainly in the colon.

The fluid structures of the body can be divided into three parts: blood, interstitial fluid, and intracellular fluid, which represent 5, 15 and 50 per cent of the body weight respectively. The volume of the interstitial fluid is the most labile and expands or contracts with the changing physiological needs of the body. When the fluid balance of the body is disturbed this compartment acts as a buffer to the other two and may undergo profound alteration that is not reflected in the volume of blood or of intracellular fluid.

The body contains about 100 grammes of sodium, of which about half is present in the blood and tissue fluids. Whereas sodium is regarded as the main extracellular base, a similar alkali metal, potassium, is the chief cellular component. All the common salts of sodium and potassium are freely soluble, and therefore readily absorbed from the alimentary tract, but there follows the disadvantage of ineffective storage. There is no way in which the body can store reserves of these elements in an
insoluble and inactive form, and it is therefore essential to supply them constantly from the food.

Water and salt metabolism are intimately related because of the constancy with which the body maintains the osmotic pressure of the extracellular circulating fluid. Movements of water about the body are almost invariably accompanied by a simultaneous movement of salts. Sodium chloride is lost from the body in the sweat, urine and gastro-intestinal discharges. The loss from sweating is considerable: in a hot environment as much as 10 to 15 litres of sweat may be lost in eight hours, and each litre may contain 3 or 4 grammes of sodium chloride, though this amount is not constant and depends on many factors, including degree of acclimatisation and personal idiosyncrasy, as well as the rate of secretion of sweat. Normally, sodium chloride is eliminated by the kidneys, and the quantity ingested is balanced by the amount excreted in the urine, so that the body is physiologically in chloride equilibrium. The loss of sodium is even more significant for the total electrolyte content of the body fluids than is the loss of chloride. When chloride is lost, the total concentration of acid radicals can be maintained by lessened elimination of carbon dioxide in the expired air, with the result that the concentration of bicarbonate rises to the extent that the chloride falls. However, when sodium is lost the sum of the acid radicals in the blood must decrease correspondingly in order to maintain the balance of acid and base. The result is that the electrolyte content of the blood and tissues falls and dehydration is the consequence. The only significant indication then can be the replacement of the depleted sodium chloride.

That human beings have a remarkable power of adapting themselves to climate is shown by the fact that the body temperature remains the same in the arctic zones as in the tropics. It is controlled mainly by variations in the cutaneous vascular tone and in sweat-gland activity, by evaporation from the lungs and by the activity of the skeletal musculature, which are under the control of the nervous system. Very few animals have such a well-adjusted system as man, and this is one of the fundamental reasons for his exceptional ability to withstand a wide range of climatic conditions.

The body temperature is kept constant by maintaining a balance between two opposing factors—heat production and heat loss. Heat production takes place mainly in the skeletal muscles. The metabolic activities of the body at rest are so adjusted as to maintain the appropriate internal temperature. When the muscles are performing hard work, they may generate up to five or six times as much heat as they do when at rest. In cold and temperate climates heat is lost from the body mainly by contact with the cool air and by radiation. In tropical zones, where the ambient air is hot, these means are not available and heat is therefore lost by evaporation—that is, mainly by sweat-gland activity. A secondary means is by increased pulmonary ventilation: the expired air is saturated with water vapour and heat is thus lost from the lungs.
A distinction must be drawn between imperceptible perspiration, which is probably a transudate from dilated cutaneous capillaries, and sweat proper, which is an actual secretion from the sweat glands. In general, the response of the sweat glands is greatest when there is interference with heat loss. Sweating on the body surface is produced by a nervous reflex governed by the sweat centre, which is closely allied with the heat-regulating centre in the hypothalamus. Cooling by evaporation is most effective when the air is dry and in movement, and the value of electric fans and other air ventilation systems which disturb stagnant air is therefore considerable.

The importance of estimating and recording the air temperature (dry-bulb and wet-bulb recordings) and of assessing the velocity of the air current and the relative humidity, was recognised as fundamental. The conditions under which a patient suffering from clinical salt-deficiency had been working were checked by taking wet-bulb and dry-bulb recordings with a sling psychrometer. The difference between the wet and dry readings gave the degree of evaporation. The sling psychrometer consisted of a pair of thermometers attached to a handle by means of which they could be whirled rapidly; the bulbs were thus affected by the temperature and moisture of the air. A large number of readings were taken, at various stations on the ship, and revealed a wide range of conditions: from the comfort of the shaded upper deck where the readings were 90°F. (dry bulb) and 80°F. (wet bulb), to the oppressive 140° and 92° of the boiler room.

In order to determine the degree of salt deficiency, it was found unnecessary to resort to elaborate plasma-chloride estimations; the information could be obtained accurately enough by quantitative assessments of the concentration of chlorides in the urine. In all cases of salt deficiency diagnosed in Stening's series, urinary chloride was shown to be low. The method used was to add 5 drops of pure concentrated nitric acid to 5 cubic centimetres of the patient's urine. The test was completed by adding 5 drops of a 1 per cent solution of silver nitrate. It was found by experience that the diminution of urinary chlorides could be classified as follows:

| Thick curdy precipitate produced instantly. | Normal reaction. |
| Curdy precipitate slow to form. Loss of curd on movement of the test tube with formation of white deposit. | First-degree diminution. |
| Absence of curdy precipitate but appearance of a white haziness without deposit. | Second-degree diminution. |
| No precipitation, no haziness, no change. | Third-degree diminution. |

In association with this test a quantitative estimation of urinary chloride was introduced, and served as a confirmatory check of the qualitative assessments. This estimation was of great value and reliability in determining prognosis and response to treatment. It was found that the danger-
ously low level of sodium chloride excretion was 3 grammes a day; readings of a urinary output of 5 grammes or less a day indicated urgent salt replacement therapy. When it was not practicable to make daily estimations of the urinary output, importance was attached to readings less than 0.5 gramme sodium chloride per cent in a urinary specimen. Three solutions, A, B, and C, were prepared for making these tests:

A: 29.075 grammes of fused silver nitrate per 1,000 c.cm.
   1 c.cm. = 0.01 gramme sodium chloride.
B: 40 c.cm. of a saturated aqueous solution of iron alum mixed with 600 c.cm. concentrated nitric acid.
C: Potassium thiocyanate.
   1 c.cm. = 1 c.cm. of solution A.

The estimation was performed by taking 1 cubic centimetre of the patient's urine and 3 cubic centimetres of solution A and 1 of solution B. These solutions were mixed and titrated with solution C until the red end-point. The number of cubic centimetres of solution C used, subtracted from 3.0, gave the urinary sodium chloride in grammes sodium chloride per cent.

The routine examination and treatment of patients suspected of salt deficiency in Stening's series took the following course. The patient was asked how long he had been in the tropics, to determine his degree of acclimatisation; he was given a physical examination, which included sphygmomanometer, pulse, respiration and mouth-temperature readings, and estimations were made of his urinary chloride diminution and of the sodium chloride excreted in the urine. Temperature and humidity readings were then taken of his place of duty, and from this and his clinical condition the method and strength of the salt dosage were decided. His response to treatment was observed, and he was given advice for keeping himself fit in the future.

In this series, approximately 120 patients were diagnosed, investigated and treated for clinical states of salt deficiency, although a considerably greater number derived great benefit from salt consumption for sub-clinical states. In fact it is doubtful whether any single member of a ship's company who served at sea in the tropics for more than six months was not affected to some extent by uncompensated salt loss.

Symptoms of salt deficiency in the sub-clinical states included: faintness, dizziness, headache, lack of concentration, mental apathy, and irritability, anorexia, weakness, postprandial nausea, undue tiredness and lethargy, disinclination for effort, sleeplessness, mild nocturnal cramps, frequency of micturation without polyuria, and insatiable thirst.

It was commonplace in areas of high heat and humidity to find whole ships' companies, particularly troops in transports who were not acclimatised, complaining of lack of energy and concentration, undue tiredness, and other symptoms which were lightly ascribed to "tropical neurasthenia". With the lowering of basal metabolism that results from exposure to the heat of the tropics, the rate of oxidation in the tissues is adjusted to maintain the appropriate internal temperature. To keep heat production
at a low level it is therefore necessary to reduce the amount of muscular work and to conserve energy. In the tropics heat production at rest is only about 30 large calories per square metre, compared with 40 in temperate zones. However, this grading of muscular activity and lowering of the metabolic rate do not of themselves impair a man’s efficiency or rob him of enthusiasm. The symptoms already listed were at first subliminal, and were dangerous in a member of a fighting ship. The gradual and cumulative effect of salt loss is insidious in its onset, aggravates existing symptoms, and usually brings others in its train. The improvement in general well-being and strength which follows an adequate, routine dosage of salt, can only be experienced satisfactorily by contrast with the former lethargy and asthenia. Sodium chloride deficiency then reveals itself as a real phenomenon.

R. A. McCance produced experimentally a severe salt deficiency in patients who sweated profusely after exposure to a radiant heat bath. He found great differences in the individual losses of water and of sodium chloride; the amount of sodium chloride lost ranged from 0.14 to 3.65 grammes, under the same conditions of heat and time. Such differences explain why some people escape the major symptoms of salt deficiency.

Another variable to be considered is the speed with which some individuals show signs of deficiency compared with others who have lost about the same amount of salt. It seems likely that the critical sodium chloride level is a function of individual susceptibility and, especially, of acclimatisation to heat. The well-known resistance of the inhabitants of India, tropical Africa, Arabia, Melanesia and other zones of the “brown tropics” to hot environments, is an example of perfect acclimatisation. The degree of acclimatisation necessary for men who are serving in ships in tropical zones, where they may be forced to eat, sleep, rest and take recreation in sweltering heat for many months without respite, is built up slowly but surely and depends on many factors in addition to habituation to intense heat. Stening recorded that new ratings without previous service in the tropics were exposed gradually to the sun with beneficent effects. There was a marked difference between the good health and relative freedom from heat-exhaustion states of seamen whose duty was on the upper deck and the condition of stokers and other ratings between decks.

The induced state of acclimatisation is akin to the condition of an athlete in training; physical fitness or endurance can only be acquired by degrees, and are quickly lost if the subject is affected by intercurrent disease or if he disobeys the primary rules laid down for those in training. Training on a ship in the tropics includes careful personal hygiene, improved living and working conditions, freedom from intercurrent disease, and a knowledge of the occurrence and prevention of salt-deficiency states.

The clinical states of salt deficiency can be listed for descriptive purposes as:
Heat exhaustion proper, or heat shock;
Heat exhaustion with abdominal pain;
Heat cramps: acute severe, or chronic mild;
Salt-deficiency state with gastric symptoms:
  dyspeptic type;
  vomiting or emetic type.
Salt-deficiency state with alimentary symptoms:
  apyrexial diarrhoeic type;
  vomiting-diarrhoea-pyrexial type.
Salt deficiency state with low-grade pyrexia;
Heat-stroke proper, or hyperpyrexia.

Heat shock. When an individual is subjected to intense external heat, distinction must be drawn between the clinical state, resulting from the profuse sweating secondary to the exposure, and that following the effect of heat itself on the body. It is in circumstances of extremely high heat and humidity that the danger of inadequate heat loss from insufficient evaporation may occur. The natural powers of cooling may be overwhelmed and the body temperature may rise, causing not only retention of heat but actual increased production. Treated symptomatically in a cool atmosphere, these patients will recover readily if the exposure to extreme heat has not been too prolonged. However, when individuals are exposed to environmental conditions favouring profuse sweating, the uncompensated loss of salt from the body may manifest itself clinically in states closely allied to “effects of heat” cases. The predominant sign in the clinical picture of heat exhaustion is shock associated with demonstrable diminution of sodium chloride excretion in the urine.

The safeguard of prophylactic salt administration against the occurrence of heat exhaustion does not seem to have been the subject of concern in many ships in tropical climates, and the morbidity and even mortality in ships’ companies, particularly in closely-packed troop transports, was not inconsiderable. It was in handling problems of this nature that ships’ medical officers felt their isolation from their fellows so keenly. Surgeon Commander J. M. Flattery tells how he treated cases of heat exhaustion, two of them severe, in H.M.A.S. Australia in 1941. Beyond a knowledge that salt was lost from the body by excessive sweating, and a vague statement in one of his medical text books that taking sodium chloride and water freely should be helpful in preventing heat cramps, he had no guidance on how to handle such cases or how to determine the appropriate dose of salt. He decided, however, to persuade every member of the ship’s complement to take extra salt daily as a prophylactic measure. After this regimen had been introduced only one case of heat exhaustion occurred, and that was in a stoker who had not been taking the salt in case it made him sick. The problem in Australia was aggravated by the ship’s travelling for 15 months continually backwards and forwards from the tropics to very cold climates; this made acclimatisation impossible, and the men were inevitably more susceptible to the effects of heat.

At every possible opportunity Flattery discussed the problem with medical officers of other ships, including hospital ships, to discover...
whether the simple precaution of taking extra salt had been adopted, and whether it was regarded as a commonplace. In only one instance did he find it to be rigidly enforced, but the majority of officers said frankly that the practice of taking extra salt as a strict routine was completely unknown. The majority of cases of heat exhaustion, and all those of severe degree seen in Stening’s series, occurred before the daily ration of salt was introduced. Once the men became “salt-minded” the number of cases fell sharply, and those that did occur were less severe; there were no deaths in this series.

Stokers, who were exposed to unusually hot conditions in the boiler and engine-rooms, were affected most commonly. The patient frequently fainted or collapsed on duty and was carried semi-conscious to the sick bay. Questioning usually revealed a prodromal phase for some days previously, of lassitude, dizziness, weakness, nausea and headache, or in short the symptomatology of the sub-clinical states of salt deficiency. Because the state of shock precludes the other signs of heat prostration, the more expressive term “heat shock” is preferred. The clinical manifestations of this shock were similar to those encountered in the shock of trauma, operation or haemorrhage; they included cold extremities, a pallid and moist skin, feeble or impalpable pulse, and collapsed superficial veins. In heat shock thirst was a common complaint and the patient frequently vomited if fluids were given by mouth. The mouth temperature of patients in this collapsed condition was lowered to an average reading of 97.4°F. In addition to the marked pallor of the skin there was pronounced evidence of dehydration in the eyeballs (intra-ocular tension), tongue and sunken cheeks and temples. The pulse was rapid in rate and low in tension to the degree of impalpability, and the blood pressure was always depressed, affecting both the systolic and diastolic values more or less proportionately (average readings 100 and 60 millimetres of mercury).

Abdominal symptoms, varying from the discomfort of minor gastric disturbances to severe pain, diarrhoea, repeated vomiting and mild or severe abdominal cramps, may complicate the clinical features of the collapse of heat exhaustion. In the stage of recovery after the initial collapse, the pulse, although still rapid, is of better volume and the blood pressure rises to normal readings. The mouth temperature rises usually to a level slightly above normal. A mild pyrexia of 99 to 100.4°F. may last for 36 hours, and particular regard should be paid to these readings. The skin becomes warmer and the reactionary sweating may be profuse. This fact, like the fever, is carefully noted in heat-exhaustion cases, and is a good prognostic sign. When a rise in temperature occurs in patients with a dry pungent skin, incipient heat-stroke is indicated.

If treatment is prompt, the prognosis is good and recovery is the rule. The readiness with which heat-stroke can occur if the patient is not immediately and effectively treated, makes the diagnosis of heat shock of paramount importance. Theoretically heat-stroke should not occur if

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prophylactic treatment is carefully planned and observed, and if heat shock is diagnosed accurately and early and treated correctly.

A considerable loss of water and salt from the body has already occurred in patients before the onset of heat shock, with the result (particularly if the fixed base—sodium—of the extracellular fluid is depleted) that the body becomes dehydrated and the circulating blood volume diminishes as part of the general dehydration. The oligaemia, in turn, lessens the venous return to the heart, with consequent extra-cardiac circulatory failure and shock. The loss of salt plays the essential part in the causation of the dehydration underlying shock. It is assumed that the water content of the body is largely a function of the quantity of dissolved electrolytes; unless the electrolytes are replaced, therefore, dehydration cannot be alleviated by the supply of water.

There are thus two essentials in the treatment of heat shock: a suitably cool environment and a ready correction of the deficiency of sodium chloride in the body fluids. In most forms of peripheral circulatory failure with shock, the administration of sodium chloride solution is a valuable measure. This is especially true when dehydration has occurred. Shocked patients can rarely take fluids by mouth. Intravenous infusion is the method of choice, usually in the form of an isotonic (0.9 gramme per cent) solution of sodium chloride. Hypertonic saline (1 litre of 2 per cent, or ½ litre of 5 per cent solution) may be infused slowly if a more rapid replacement of the depleted electrolytes is desired. Normal saline was used in all the cases in Stening's series when intravenous infusion was indicated.

The following case report is typical of patients seen by Stening. A stoker aged 22 years collapsed while on watch in the engine-room and was carried into the sick bay semi-conscious. He was extremely pale, with a cold sweaty skin. Sphygmomanometer readings showed a systolic pressure of 100 millimetres and a diastolic pressure of 70 millimetres of mercury. His radial pulse was palpable (120 per minute) and mouth temperature 97.4°F. He did not complain of abdominal pain, but felt faint and dizzy. His urine showed a second-degree diminution of sodium chloride and a quantitative estimation of 0.2 gramme per cent.

It was learned that he had had a prodromal period of two or three weeks complaining of undue tiredness, occasional attacks of giddiness and faintness, and nausea at meal times with no appetite for food. During the night he had been restless and did not sleep well, but he ascribed this to the fact that he had only been at sea for a few months and was not yet accustomed to disturbed hours of sleep. When he was off watch he spent most of his time on the stokers' mess-deck (lower deck), where the atmospheric conditions were particularly bad—indeed, not a great deal better than those in the engine-room itself. He was not acclimatised to tropical conditions and although instructed had not placed much importance on the regular intake of extra salt.

The response to treatment in this case was rapid. One litre of normal saline was infused intravenously, and then salted fluids (½ dram of salt
in water each half hour for six doses) were readily taken without nausea. His mouth temperature rose to 101° on the first day. The qualitative urinary assessment showed a first-degree diminution after the ingestion of a little more than 36 grammes of salt. On the second day the patient was afebrile and felt much better. By the third day there was no diminution of urinary chloride, and from that stage a routine minimum dosage of 1 dram of sodium chloride was taken twice a day and increased when he had additional muscular work or when conditions were unusually hot, or when he experienced the subjective feelings associated with the subclinical state of salt loss.

*Heat exhaustion with abdominal pain* was treated as a separate clinical state because of the relative frequency of abdominal pain of varying degrees of severity and in different parts of the abdomen, associated with but precluding the collapsed condition of heat shock. Because the state simulated surgical abdominal conditions, particularly acute appendicitis and a perforated peptic ulcer, a standard method of treatment was adopted which allowed hypochloraemia to be dealt with but did not interfere with the conservative observation of the abdominal condition. The response of each patient was rapid and the diagnosis established well within a prescribed six-hour period. Medical officers who were unfamiliar with the diagnosis of heat exhaustion with abdominal pain found it difficult to decide whether to treat these as emergency surgical cases or to wait until the condition had declared itself. Of the eight patients diagnosed with and treated for this condition in Stening's series, all were working between decks, and the majority were stokers in the engine and boiler rooms. Early in the series one patient had a laparotomy performed because of an incorrectly diagnosed perforation of a duodenal ulcer.

Three distinct types were seen, simulating clinically the symptoms and features of acute appendicitis, perforated peptic ulcer, and a non-specific intra-abdominal catastrophe respectively.

In the first group—four appendiceal cases—pain of a colicky, cramp-like nature began in the region of the umbilicus, but moved in two or three hours to the lower right abdomen and became a dull, persistent pain. The onset of pain was associated with nausea, and in some cases vomiting. The shock in these four cases was not as pronounced as in the other types, but the patient looked pale and had a moist skin. The mouth temperature was slightly raised and the pulse disproportionally rapid (average 110 per minute) and of good volume. The rectum was not tender. There was as a rule no true muscular guarding, but deep tenderness in the right lower abdomen.

The provisional diagnosis of perforated peptic ulcer was understandable when the patient had complained of excruciating upper abdominal pain which occurred suddenly and caused him to fall to the deck writhing in agony. Associated with this pain was shock of a moderately severe degree, and a rigid abdomen caused by generalised and persistent cramp-like contractions of the abdominal musculature. A prodromal phase might
include nausea, occasional vomiting, anorexia, and postprandial dyspepsia, for weeks beforehand. Physical examination revealed deep tenderness of the abdomen but localised to a most tender area in the epigastrium. The greatest confusion in the differential diagnosis was caused by the reaction of the abdominal muscles to palpation: very gentle palpation of the muscles, or sudden pressure, produced a violent contraction of the muscles, after which they became board-like and painful. In severe paroxysms the stony hardness of the affected muscles was characteristic. The spasms did not yield to deep pressure and the more prolonged the period of palpation, the more severe the pain and the more board-like the rigidity.

In the third type, of which there were three cases, the sudden occurrence of severe abdominal pain associated with faintness and collapse suggested an intra-abdominal catastrophe of indefinite origin, such as the rupture of a hollow viscus or intraperitoneal spontaneous haemorrhage. The clinical signs supported this view incompletely, but the appearance of marked pallor in a patient with a cold and clammy skin, rapid pulse of low tension, increased respiratory rate and non-localised deep abdominal tenderness without muscular guarding, was disturbing until the diagnosis of salt deficiency was confirmed.

In all types the qualitative and quantitative estimation of urinary sodium chloride showed a diminution in the quantity excreted. The response to salt therapy was so rapid and definite that any anxiety about the actual or potential occurrence of an organic abdominal lesion was soon allayed.

The routine treatment adopted for patients of all three types was to put them to bed in Fowler's position (except those who were severely shocked), and record pulse, temperature, respiration and vomiting at two-hourly intervals. The degree of diminution of urinary chloride was assessed and checked quantitatively. A continuous intravenous drip of normal saline was started; the rate of flow depended on the degree of deficiency of salt and on the clinical state of the patient. If more than 1 litre of saline was indicated, the rate of flow for the second litre was maintained at 40 drops a minute. When the diagnosis was established Fowler's position was relaxed and salted fluids in calculated amounts were allowed by mouth until the quantitative estimation of a urinary specimen was more than 1 gramme sodium chloride per cent, or of the 24-hour specimen more than 10 grammes of sodium chloride a day.

During a period of excessively hot weather when Australia was closed up for three days in the action state, it was necessary for most of the ship's company to remain at their action stations continuously, except for a few short breaks. After the action a leading steward, 30 years old, who had been on duty in the after-control position on the lower deck, complained of nausea, anorexia, and dyspeptic symptoms. His work required him to lift heavy boxes from the storeroom on the platform deck to the main deck. The exertion made him sweat profusely. His symptoms had been present for about three months, but had become worse as a
result of excessive sweating when the ship was closed up. While carrying a tray the man suddenly collapsed with excruciating pain.

When examined in sick bay it was found that his abdomen was board-like and tender, particularly in the epigastrium in the midline. He had vomited ineffectively once since the onset of the pain, and the attack was accompanied by a moderate degree of shock. His mouth temperature was 98°, pulse rate 120 per minute but of good volume. He lay curled up on his side in bed and was immobile rather than restless. He resisted any movement or palpation. A provisional diagnosis of perforated peptic ulcer was made. Routine examination of the urinary specimen showed no abnormal constituents, and an assessment of urinary chloride showed a second-degree diminution. Preparations were rapidly made for operation and as a pre-operative measure an intravenous saline drip was begun. About 200 cubic centimetres of normal saline were given before the patient was anaesthetised. It is worth noting that an injection of 1/4 grain morphine sulphate had given him no relief from pain; and his abdomen was still rigid and board-like. An incision was made under general anaesthetic but revealed no free fluid in the peritoneal cavity and no abnormality in the anterior gastric and duodenal surfaces or of the lesser and greater curvatures of the stomach, or of the pancreas, the gall bladder or the appendix. The abdomen was closed with a drainage tube into the lesser sac.

The post-operative period was uneventful. A continuous intravenous drip of glucose saline was given, augmented by rectal salines. The atmospheric conditions even in the sick bay were trying, and the patient’s temperature rose to 102.8°F. on the second day after the operation. After 48 hours the tube, which had not drained, was removed and a graduated milk diet was started by mouth. The skin surface was kept sponged and cool, and in addition to the intravenous administration of saline, mildly salted fluid was allowed by mouth. In all, 5 litres of glucose saline were infused intravenously in the first two days. Improvement was then noticeable, but the qualitative assessment of urinary chloride still showed a first-degree diminution. It was not until the fourth day that a normal reaction was observed to the precipitation test. On the sixteenth day the patient was fit and well. He was then sent ashore for full investigation. A fractional test meal showed a mild hyperchlorhydria, but there was no radiological evidence of ulcer. Serum and cerebrospinal Wassermann reactions were negative, although the examination of the nervous system did not at any time suggest the diagnosis of a gastric crisis.

The case was most instructive, and as it occurred early in the series it emphasised the necessity for careful investigation and pre-operative salt therapy in cases of abdominal pain. There can be little doubt that the correct diagnosis was heat exhaustion with abdominal pain. The lifting of heavy weights vertically from the deck called for great strength of the abdominal muscles, and was responsible for the severe cramp-like contractions. The extreme irritability of muscles in cramp to palpation, and the persistence of the contraction, were very similar to the muscle rigidity
of a perforation. The urinary chloride assessment was instructive, but the improvement with such a small dose of saline (about 2 grammes) was not rapid enough to make diagnosis certain and so delay operation. In the light of subsequent cases at least 10 grammes of saline should have been given intravenously as a pre-operative measure.

There can be no doubt that cases have occurred in ships in the tropics where laparotomy has been performed and no perforation found. It is also known that in these cases salt was not exhibited, and attacks of similar pain occurred after the operation. Assessment of the urinary chloride both before and after operation is necessary in the tropics; when a surgical operation is performed under general anaesthesia, the sweating in the excitement stage, during and after the operation, is considerable, and the degree of salt depletion should always be checked.

Heat cramp is perhaps the most dramatic clinical manifestation of salt deficiency. The term is applied to the painful muscle spasms which may affect any striated muscle in the body. The muscles in most active use about the time of the attack are the ones most usually affected. Experience gained in Stening's series showed that the calf muscles (gastrocnemii), the ventral abdominal muscles, and the small muscles of the hands and fingers were most commonly affected by cramps. In Australia the incidence of heat cramp was relatively higher among stokers, who contended with a high working temperature in the boiler and engine-rooms as well as with high atmospheric temperatures. Intercurrent diseases appeared to play little part in increasing susceptibility to heat cramps, but gastrointestinal disturbance (three cases in the series), causing increased loss of chlorides in vomiting and diarrhoea, may be a determining factor.

The severity of the cramps may vary within wide limits. Some patients may have only the intrinsic muscles of the hand and fingers involved. An intermittent painful cramp of the muscles of the calf of the leg may occur at night or after exercise. However, the picture presented by a patient in the throes of severe cramps of the abdominal muscles is striking. Severe paroxysms of intense pain lasting for one or two minutes recur every five to ten minutes. The contractions cannot be relaxed voluntarily and may spread from one muscle group to another or appear in many groups simultaneously. During the cramp the muscles are hard, and if the abdominal muscles are affected there is board-like rigidity of the abdomen. For some hours after a severe contraction there is a residual tenderness or dull ache in the muscle. The severity of the attack appears to depend on the degree of hypochloraemia, and not on the rate of loss of salt. The clinical and biochemical changes do not provide a full explanation for the occurrence of cramps but suggest that the lowering of the sodium and chloride in the body fluids is the principal causation mechanism.

The prevention of heat cramps entailed certain general measures and one specific prophylactic. A state of general good health, well-directed diet, facilities for work and restful sleep in a suitably cool environment, were the best general protection. In addition, during the first few days
of unduly hot weather, or in a sudden change from cool to hotter working conditions, muscular exertion was restricted and only gradually increased. The specific prophylactic was salt, which was taken regularly each day; the dosage was increased depending on activity, working conditions or degree of acclimatisation. In Australia all stokers were given 1 dram of salt when starting their watch, which meant that they took a minimum of 2 drams a day apart from what they had with meals. This was taken readily once the men realised how great an improvement it made in their well-being.

Ten patients were treated in the series for heat cramps, and they represented several aspects of the clinical state of salt deficiency: the classical form of "stoker's cramp" or severe abdominal cramp occurring characteristically in a stoker in the engine-room; the cramp of moderate degree developing in insufficiently acclimatised individuals or in patients in whom the loss of salt had occurred from other sources in addition to the skin; and lastly, the chronic form of mild isolated cramps affecting the muscles more frequently used during work and so simulating "occupational cramps", but actually arising from the cumulative loss of sodium chloride in small amounts over a long period. The following is the history of a patient suffering from "stoker's cramp".

A stoker petty officer aged 35 years complained while on duty in the forward engine-room of severe abdominal pains which made him double up and writhe on the deck. When examined in the sick bay he complained of intensely painful cramps over the whole of the anterior abdominal wall. He was sweating freely. Palpation of the abdomen showed that the whole of the ventral abdominal musculature, particularly the rectus abdominis muscles, felt as hard as stone. There was superficial tenderness associated with the contraction but no exact localisation of the tenderness. Mouth temperature was 97.4°F., and pulse rate 90 per minute and of good volume (systolic blood pressure 110 millimetres and diastolic 80 millimetres mercury). The urine showed no abnormal constituents, but assessment of the chloride indicated a second-degree diminution. Quantitative estimation was 0.3 gramme sodium chloride per cent. Atmospheric conditions in the forward engine-room were extremely oppressive. Moreover, the patient strongly opposed the consumption of extra salt in his engine-room, and had not himself taken the daily ration.

The response to treatment was dramatic. One litre of normal saline was infused intravenously, but complete relief from pain was noted after about ½ litre (5 grammes salt) had been given. Two drams of salt were then taken in 10 ounces of cold water without difficulty. The skin was dried and sponged, and electric fans were brought in to cool the atmosphere. After 24 hours the urine showed a second-degree diminution, and 5 grammes of sodium chloride had been excreted. The patient felt very well and was free from abdominal pain but had some residual tenderness of the muscles of the recti. After 48 hours' treatment the urine was assessed as normal with a daily excretion of 12 grammes sodium chloride.
After the attack the patient was strongly convinced of the need for salt in the engine-room.

_Gastric symptoms_ rarely predominated in the clinical picture of salt-deficiency states, although symptoms of a minor gastric disturbance—impairment of appetite, nausea either before or after meals, and occasional postprandial vomiting—were frequent in the sub-clinical states. Occasionally, however, symptoms of a more pronounced gastric upset loomed so largely as the clinical feature that a gastric type was included in the classification of salt-deficiency states. This group fell into two types—the dyspeptic, and the vomiting or emetic type.

In the first of these the most constant complaint was a vague abdominal discomfort, without pain. The discomfort was aggravated by meals, even small quantities of food, particularly meat, producing a sensation of undue fullness. Nausea was usually felt after a meal, but vomiting was unusual. There was always a loss of appetite. The gastric symptoms were usually associated with other symptoms of the sub-clinical states of salt deficiency—headache, giddiness, weakness, lassitude and general lack of physical energy and mental alertness. At no time was the typical peptic ulcer syndrome closely simulated, and epigastric pain and tenderness were not features of this type. Investigation always showed a diminution in urinary chloride excretion, both qualitatively and quantitatively, and an absence of occult blood in the stools. The immediate improvement in wellbeing and the subsidence of the gastric symptoms with alkali-salt treatment was marked, and served in conjunction with the increased excretion of sodium chloride in the urine as a therapeutic aid to diagnosis.

It was common to find an increase in appetite for food during the early days of service at sea in the tropics, followed by a definite decline and eventually a loss of weight. The impairment of appetite, which probably resulted from decreased alimentary function, tended to improve with the development of acclimatisation, but at no time was the appetite as good in the tropics as in temperate or cold climates. A modification of appetite may mostly be regarded as an adaptive phenomenon; proteins and fats have less appeal in the hot climates, which is not surprising in view of the high calorific value of fat and the specific dynamic action of protein metabolism.

The vomiting or emetic type of gastric disturbance was less common and found mostly in men not yet acclimatised, who had recently joined their ship and were subjected to unusually trying conditions of heat, particularly if they were not fully alive to the importance of replacing adequately the salt lost in the sweat. Vomiting was a common symptom in other states of salt deficiency, but not as a rule the predominating one. Sudden and severe vomiting was the outstanding symptom of this clinical state; the attacks were not attended with nausea, were not always associated with meals, and frequently were repeated. They were accompanied by a feeling of faintness, weakness and impending collapse, sometimes by mild colicky pains, and also by moderately severe diarrhoea.
The abdomen was not usually tender; occasionally there was a slight fever with a disproportionately rapid pulse rate. In all cases urinary chloride estimations confirmed the diminished excretion of sodium chloride.

*Apyrexial diarrhoeic type.* After *Australia* had come from cold wintry weather in the North Sea to the sweltering heat of the Gulf of Aden, and before the importance of taking extra salt had been realised by the ship's company, there were 45 cases of diarrhoea due to salt deficiency. Almost all had very frequent watery motions unaccompanied by fever or pain. The stools were unformed and for the most part fluid, not offensive or abnormal in colour, and containing neither mucus nor blood, nor any other abnormality. The diarrhoea was often of a lincteric or postprandial nature, and the certainty of a bowel motion after a meal greatly lessened the desire for food, although the appetite was not impaired by nausea. The bowel appeared to be in an irritable phase with an excessive reflex response to food. Abdominal discomfort was often present but did not amount to pain. Mild tenesmus occurred in some patients, and the passage of frequent watery stools was sometimes followed by a feeling of exhaustion and faintness. Pyrexia was not seen in this series, and its absence was a notable feature. The subjective feelings of the sub-clinical states were usually present and included weakness, lassitude, headache, attacks of faintness and giddiness, and undue fatigue. The urine showed a first-degree or second-degree diminution in chlorides.

When salt was taken it passed almost unabsorbed through the stomach and small intestines, the absorption being mainly in the colon. Adequate therapeutic salt administration was effective. Patients in whom vomiting was associated with diarrhoea were given dilute hydrochloric acid in small doses of 15 minims in water three times a day, in consideration of the possibility of the diarrhoea being gastrogenous in origin from a temporary hypochlorhydria or achlorhydria from excessive loss of sodium chloride from the body.

Stening suggested that the diarrhoea (and in some cases the vomiting) might be caused by irritation of the smooth muscle of the gastro-intestinal tract by a process similar to that causing cramps in striated muscles. The treatment for this type was almost exclusively salt replacement. Salt was given in large doses by mouth either as a powder or in a tablet. If it was not tolerated by mouth, an intravenous infusion of 1 litre of 5 per cent glucose in normal saline was given and repeated if necessary. The response was ready, and a forming of the stool and slowing of the frequency of bowel actions was noticed within 12 hours.

The vomiting-diarrhoea-pyrexial type was instructive, and included most of the symptoms and signs of the salt-deficiency states. The overlapping of symptoms and the merging of the clinical picture were most pronounced.
The prodromal phase included the symptoms of the sub-clinical state of salt deficiency, more particularly weakness and lethargy, headaches and giddiness. Vomiting usually occurred before the onset of diarrhoea and made its appearance suddenly and unexpectedly. Both vomiting and diarrhoea developed rapidly and caused a considerable loss of fluid and further loss of sodium chloride. A condition of mild shock was usually present. There were also abdominal pains, vaguely localised and of a mild cramp-like or colicky nature. There were no cases with well-developed muscular cramps. In association with the vomiting and diarrhoea, the outstanding aspect of this subgroup was the rise in body temperature, to a limit of 103.5°. This was accompanied by a dry, pungent skin. The stimulation of a skin reaction with the onset of sweating caused a lowering of temperature, relief of headache, and a general improvement.

In all cases the excretion of chloride in the urine was low and the response to supplementary salt therapy ready. Local measures to keep the body temperature below 102 degrees were taken in addition to the administration of salt.

*Incipient heat-stroke.* The pyrexial type of salt deficiency, which was regarded as a precursor of heat-stroke proper, was not commonly encountered; only five patients in Stening’s series recorded a temperature higher than 104°F. A raised body temperature was justifiably regarded as a danger sign in all salt-deficiency states. The factor of impairment or absence of normal sweating occurred in the pyrexial type and in heat-stroke, in addition to the salt depletion of the body which is the pathological basis of all the clinical states of salt deficiency.

There appears to be no distinct line between severe heat exhaustion and heat-stroke. In fact the term hyperpyrexia would be a much more accurate one than heat-stroke. Heat exhaustion cases will, if untreated, pass on to hyperpyrexia, and once the temperature reaches very high levels—107° to 108°—with accompanying coma or convulsions, the clinical picture is that of hyperpyrexia, although the essential pathology is the same.

How hypochloraemia interferes with sweating was not known. It was suggested that the sweat glands were unable to secrete sweat when the concentration of sodium chloride in the blood was low; or alternatively that a low sodium level induced a secondary reduction in blood volume, as it was generally accepted that shrinkage of blood volume depressed sweating. However, it was evident that hypochloraemia gravely embarrassed all the normal metabolic functions of the body, apart from the fact that it interfered with sweating. Adaptation to high temperature involves an increased capacity to produce sweat, a greater sensitivity of the temperature-regulating apparatus, and an economy of salt excretion. The effectiveness of adaptation varies from one person to another.

In a state of shock, a hot-water bag applied to a cold part of the body surface warms more rapidly and to a greater degree than in health. This observation was of the greatest importance in interpreting the
phenomenon of hyperpyrexia, and demonstrated clearly how severely the
temperature-regulating function of the skin was impaired as a result of
peripheral circulatory failure. The body of a severely dechlorinated patient
in a state of oligaemic circulatory failure characteristic of severe heat
shock, would probably react in a similar manner to high environmental
temperature if the protective cooling of the sweat secretion was interfered
with, the metabolic functions of the body upset, and the delicate adjust-
ment and coordination of the temperature-regulating mechanism disturbed.

The onset of heat hyperpyrexia may be sudden with a rapid, unheralded
rise of temperature. In most cases it is preceded by various sub-clinical
signs of salt deficiency. Important prodromal signs of the development of
hyperpyrexia in a patient with a low-grade pyrexia are dryness of the
skin, photophobia associated with sunken and tender eyeballs, tinglings
and numbness in the hands and feet, and mild cramp-like feelings in the
muscles of the limbs. The pulse is quickened and may become irregular in
time and amplitude; the headache becomes worse and the patient may be
mildly confused and very restless. When the sudden rise in temperature
occurs the skin becomes hot and pungent and the face flushed, with bright
eyes and moderate congestion of the conjunctiva. The patient who has
developed a fever exceeding 108° should be treated without more than
a minute’s delay. It is important to reduce the temperature quickly to
a safe level and maintain it there, and to replace the salt deficiency.

The most efficacious way of reducing body temperature was found
to be by spraying the patient with cold water and using fans to supply
a free current of air. The patient was stripped of clothing and laid on
a sheeted bed so that as much air as possible could flow round the
body; cold water was then sprayed or sponged on and the air was kept
circulating. When the temperature had fallen below 102° this treatment
was suspended, and when a relapse seemed unlikely the patient was
given salty fluids to drink: half a dram of salt in half a pint of water
every half hour. If sweating occurred the prognosis was good. Severely
dechlorinated patients required large amounts of salt. Relapse was guarded
against by prescribing a routine daily dose of common salt.

**Heat-stroke proper, or hyperpyrexia.** The predisposing factors in heat-
stroke are both internal and external: deficient fluid, or more particu-
larly deficient salted fluid intake, and very high atmospheric temperatures
with high relative humidity and stagnant air. Contributing factors are
the fever of an intercurrent disease, unsuitable clothing, and unhygienic
living conditions. The classical type of heat-stroke occurred when muscu-
lar exertion was performed in a high environmental temperature beyond
the stage of fatigue or even of exhaustion. The patient collapsed, apparently
moribund, with fixed stare, contracted pupils, shallow breathing, imper-
ceptible pulse, and deep cyanosis. Cessation of sweating was the danger
sign in these cases, and then came the sudden rise in temperature.
Coma, convulsions, vomiting and diarrhoea sometimes accompanied the
high temperature. More rarely encountered was the psychopathic form,
in which the mental balance was upset and the patient became confused, excited, and even violent. Death could usually be avoided if the patient was treated promptly and effectively. The depth of coma, the rapidity of regaining consciousness, and the response to temperature-reduction measures were guides to prognosis.

The mortality from hyperpyrexia was high in the ships of Allied navies serving in the tropics. In the Australian Navy the occurrence of hyperpyrexia was rare but two cases were recorded, both of them fatal. In June 1940 a stoker in Hobart, which was then in the Red Sea area, was brought to the sick bay, breathing stertorously and in a comatose condition; his complexion was florid, but there was no cyanosis. He was diagnosed as suffering from heat-stroke, and was treated with an ice pack, followed by an iced enema. He did not react favourably, and artificial respiration was applied and preparations made for intracardiac injection of adrenalin, but before this could be administered the heart stopped beating. In the next month while Hobart was still in the Red Sea area a telegraphist on whom a dental operation had been performed was put in the sick bay and kept under observation by the sick-berth staff. The sick bay was extremely hot, as it had been closed down for “Action Stations”, but the patient’s condition appeared satisfactory, and he began to regain consciousness. An hour later, however, he was cyanotic and no respiration was apparent. His axillary temperature was too high to be recorded on the clinical thermometer, and although treatment was begun at once, he did not recover. As a dental operation had been performed, an X-ray examination of the chest of the body was carried out. It showed no evidence of a foreign body or of pulmonary collapse and the cause of death was given as hyperpyrexia.