to viscera while the central nervous system supply is reduced, and the anomalous findings mentioned must be ascribed to the fact that the experiments were done on healthy anaesthetized subjects. In other words, the problem put to the body as a whole—namely, how it would deal with a full blood volume when the skeletal musculature was generally inactive, and adrenaline, often in high dosage, was introduced was fundamentally an unfair one. As Rothlin (1920) and many since have shown, isolated or perfused mesenteric and several other visceral arteries react to adrenaline by constriction; that on several occasions in our experiments they were irresponsive or dilated must mean that they were nervously inhibited or, though this seems on other evidence less likely in the cases of certain vessels, that their natural tendency to contract was overcome by the raised blood pressure.

Studies of animals after haemorrhage and during shock of various kinds much more closely imitate the conditions met with in patients, and experiments already mentioned, as well as others described in the literature, show that during haemorrhage the arteries, with some exceptions, constrict; certainly the mesenteric ones do so. Further, the liver seems to come more definitely in place in respect of reduction of blood supply; it appears to follow after the renal cortex, in Van Slyke's (1948) view, for he found "that in the dog, the function of the kidney is abolished by haemorrhage before that of the liver." Delorme (1951), however, took the line that the liver may be earlier or more gravely damaged than the kidney in vasoconstrictive states. It is, he wrote, "more susceptible to anoxia than either muscle or kidney (Russell *et al.*, 1944); although less sensitive than the cerebral cortex, it does not possess the protective circulatory adjustments of the latter (Gellhorn, 1943); moreover, the liver is unique among body tissues in that it depends on venous blood for the larger part of its oxygen supply."

[The second lecture, with a list of references, will appear in our next issue.]

The 27th Annual Report of the British Empire Leprosy Relief Association (1950) announces that since January five more lay workers and two nurses have been sent overseas. Five years ago B.E.L.R.A. resolved to recruit six doctors for overseas work, but so far only one of them has been appointed; in more than one area the progress planned has been frustrated for lack of medical supervision. Especially in East Africa the field is open. B.E.L.R.A. is aiming to help to the utmost in the establishment and staffing of leprosaria planned at key points and in the development of those selected for expansion. Lay workers are available, but some of the schemes may well have to be further postponed unless doctors and nurses can be found. The organization has always aimed to save the children before leprosy destroys them, and at the end of 1949 there were 1,500 B.E.L.R.A. adoptees. Dr. Ross Innes (Inter-territorial Leprologist for Kenya, Uganda, and Tanganyika) has completed a massive survey and made recommendations; on the basis of an examination of 362,000 persons throughout the area he estimates an incidence of 80,000 cases in Uganda, 35,000 in Kenya, and 100,000 in Tanganyika, with an average of 10 per 1,000 in Kenya, and 18 in the other two territories. As yet only 6,850 of them are receiving treatment. The report also notes that in Britain the outstanding achievement was the collection of almost £13,000 from the appeal broadcast by a cured leper in January, 1949, and when an anniversary letter was sent the following year to all those who had responded to the appeal, over 25% again contributed.

LOW-SALT DIET IN TREATMENT OF CONGESTIVE HEART FAILURE*

BY

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Up to the turn of the century a diet low in sodium chloride was used most successfully in the treatment of oedema and other symptoms of congestive heart failure (Karell, 1866; Strauss, 1903; Widall and Lemierre, 1903; Finsen, 1904). After the introduction of mercurial diuretics the low-salt diet was used much less because these diuretics were more dramatically effective and easier for the patient than the slow and complicated treatment by dieting. In the English-speaking world in particular (Lewis, 1933; Levine, 1940; White, 1947) it became usual to treat cardiac oedemas with only a moderate or casual salt restriction and with mersalyl ("salyrgan") and a restricted fluid intake. Although many German (e.g., Volhard) and French physicians continued to treat their patients with the low-salt diet, Allen (Allen and Sherill, 1922) seems to be the only American in recent times to have done so on a large scale, and his chief interest was the effect of the salt restriction on hypertension.

During and after the last war, however, the tide again changed, and now great importance is attached to the usefulness of a low-salt diet, and very intensive research is being carried out to investigate the importance of sodium chloride or sodium to the physiology of the organism. This new development began with the publication of a number of clinical reports by Schroeder (1941), Schemm (1942), and Kempner (1944). The results of their investigations have received universal recognition (White, *et al.*, 1946), and have been supported by physiological and pathological investigations on the pathogenesis of congestive heart failure by Warren and Stead (1944) among others.

Schroeder demonstrated in 23 cases of cardiac oedema that a decrease in the consumption of salt to an amount less than the salt content of the urine was always accompanied by a decrease of oedema. Furthermore, he showed clearly that the low-salt diets hitherto used were not sufficiently low in salt, and that a limitation to 1 g. in twenty-four hours was often necessary to achieve a decrease of oedema or merely to prevent its increase. He also demonstrated the very interesting point that a decrease in the amount of the fluid intake on such a low-salt diet had no effect in decreasing the oedema—in fact, in some cases oedema disappeared more readily when the patient was given more water. In 1944 Schemm's second report gave the results of eight years' treatment of 241 patients with oedema, 90% with cardiac oedema. He successfully used a low-salt, acid-ash diet together with a larger intake of water, if possible 3–4 litres a day. Finally, Kempner (1944) in his study of the effect on hypertension of a diet

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consisting chiefly of unsalted rice showed that this low-salt diet was effective in oedematous hypertensives. Heintzelmann (1949), in Denmark, has described the effect of a low-salt diet in the treatment of congestive heart failure.

Recent investigations, done with the aid of radioactive sodium, have demonstrated that patients with congestive heart failure retain much more of the tracer sodium than normal people (Threefoot *et al.*, 1949). The sodium as a diffusible ion accumulates in the blood and extracellular fluid, together with chloride, bicarbonate, and protein, and for osmotic reasons the retained sodium holds water. In the formation of oedema the retention of water is thus secondary to the retention of sodium. If the patient is brought into a state of negative sodium balance the result is the clearing or diminution of oedema. Patients with congestive heart failure are often thirsty, because the oedema fluid is not water but a salt solution, and a relative true dehydration—that is, a deficit of plain water—may be present.

As a result of these facts a rational approach to the treatment of cardiac oedema appears to be: (1) a decrease in the intake of sodium (diet); (2) an increase of sodium excretion (mercurial and acid diuretics); (3) improvement of the action of the heart and circulation of the blood (digitalis, oxygen, rest, etc.); and (4) an *ad lib.* intake of water, which is both permissible and desirable if the first three factors are in force.

The work reported in this paper was designed to determine whether a diet containing less than 1 g. of salt in 24 hours is of such importance and advantage in the treatment of congestive heart failure that it should be made part of the regular routine. The diet used was the same as that used in our previous study of the effect of sodium restriction in patients with hypertension (Bang, Bechgaard, and Levin Nielsen, 1949). It contained about 260 mg. of sodium and about 2,000 calories.

The material consisted of 64 patients (35 males and 29 females) between the ages of 40 and 84; average age 62. All were suffering from congestive heart failure, and manifested oedema and/or cyanosis, pulmonary passive congestion as verified by x-ray film, attacks of dyspnoea, and pulmonary oedema.

The following four cases are presented in some detail. Their severe congestive heart failure showed no improvement with the usual methods of treatment, but responded after they were given a low-salt diet.

Case 1

A housewife aged 44 had had chorea at the age of 12 and rheumatic fever at 17. At 29, during her second pregnancy, cardiac symptoms appeared, and these have troubled her since. At 37 she was admitted to hospital for rheumatic fever, and mitral stenosis was noted; since then she has had increasing palpitation of the heart, dyspnoea on exertion, and oedema, and has constantly received digitalis and mersalyl. She entered hospital for congestive failure six months before the present admission.

When admitted on September 28, 1948, she had shortness of breath, exhaustion, and marked oedema, and a sense of fullness under the right costal margin. She had been up and about, but required several hours of intermittent rest a day. Her weight was 55.8 kg., and height 164 cm. She was weak, cyanotic, and markedly dyspnoeic at rest. The action of the heart was tumultuous (irregular irregularity). The apex beat was in the mid-clavicular line; there was a rough systolic apical murmur, but no palpable thrill; B.P. 110/95. Crepitant rales were heard at bases of both lungs.

The liver edge reached to the umbilicus; there was dullness in the flanks, and marked oedema of the lower extremities and over the lumbar area.

An electrocardiogram revealed auricular fibrillation and negative ST₂ and ST₃ segments. X-ray examination showed mitral configuration, 17/29 cm., and pulmonary passive congestion. Haemoglobin, 110%; sedimentation rate, 36 mm.; urine, albumin +; microscopical examination, normal; blood urea, 32 mg. per 100 ml.

Treatment.—With a full diet containing the usual amount of salt, digitalis, 0.3 g. and, later, 0.1 g. daily, mersalyl, 2 ml. twice a week, and urea, 45 g. daily, the condition of the patient grew gradually worse in the course of five weeks, with increase in her oedema and a weight gain of 7.7 kg. Without other change in the treatment during the preceding month the patient was put on a low-salt diet. Within a few days her weight began to fall and at the same time her dyspnoea and oedema disappeared. After five weeks on the low-salt diet her weight loss amounted to 17 kg., and there was only slight evening oedema of the lower extremities. Since then the patient has done light housework at home for six months on the low-salt diet with digitalis and oral urea daily and mersalyl twice a month. Her weight has increased only 2 kg.

Comment.—This 44-year-old woman with congestive heart failure (rheumatic heart disease with mitral stenosis) became worse in spite of rest in bed for a considerable length of time and in spite of digitalis, mersalyl, and urea. Institution of a low-salt diet, with no other change, brought about immediate decrease of oedema and overall clinical improvement. After discharge on a low-salt diet, digitalis, and mersalyl she has not suffered noticeably from symptoms of congestive heart failure during the past six months.

Case 2

A male window-cleaner aged 39 had been in hospital at the age of 7 and 22 with rheumatic fever and at 32 for pneumonia. During the past three years he had increasing fatigability, dyspnoea on exertion, ankle oedema, and pressure in the epigastrium. He also had several severe and sudden attacks of extreme dyspnoea with a feeling of oppression lasting several hours. At 38 he had severe haematemesis. He was admitted to hospital 18 months previously on account of congestive heart failure; he had been receiving digitalis for many years, and was unfit for work during the last six months.

He was admitted to hospital on February 14, 1949, complaining of shortness of breath and the fear of death. He was not weighed; his height was 168 cm. Marked prostration, dyspnoea, and cyanosis were present. The apex beat was not felt. There were an apical thrill and a pre-systolic murmur, accentuation of P₂, sinus rhythm, and an apex rate of 120 a minute; B.P. was 140/85. The lungs showed: dullness at both bases, and fine moist rales of pulmonary oedema. Signs of ascites were not present; the liver edge was three fingerbreadths below the right costal margin, and there was no peripheral oedema.

An electrocardiogram revealed sinus rhythm, right axis deviation, large P waves in Lead II, and slight depression of ST₂ and ST₃ segments. X-ray examination showed mitral configuration and severe pulmonary passive congestion. The haemoglobin was 95%; sedimentation rate, 8 mm.; urine, albumin +; microscopical examination, normal; blood urea, 42 mg. per 100 ml.

Treatment.—Morphine and oxygen given on admission brought no relief. The expectoration of large amounts of frothy red-tinged material disappeared after a 200-ml. venesection and aminophylline and morphine by vein. He was given penicillin the first few days. After the venesection he remained very weak, with continuous dyspnoea and very little sleep. He received a daily dose of digitalis. After three weeks in bed he still complained of shortness of breath,

fullness in the epigastrium, and thirst. At night he was very confused, had severe attacks of dyspnoea, and suffered much from thirst. Oedema of the legs developed. In spite of mersalyl, diuresis was scant and the oedema increased, so that after 12 days a low-salt diet was begun without otherwise changing the treatment. Two days later the urine output had risen to 1 litre, and mersalyl then brought it up to 3 litres. The patient could not be weighed the first few days, but the oedema disappeared, and so at the same time did the patient's thirst. The dyspnoea, the fullness in the abdomen, and his restlessness at night ceased. The patient was able to leave his bed, but the advanced condition of his heart disease permitted only partial improvement, and continued hospital treatment was necessary. He died three months later.

Comment.—This 39-year-old man with congestive heart failure (rheumatic heart disease with mitral stenosis) had repeated attacks of pulmonary oedema. On his admission to hospital there was no improvement in the severe signs of congestive failure and thirst, in spite of rest in bed, digitalis, and mercurial diuretics. After institution of a low-salt diet with no other changes the more distressing symptoms, including the thirst and the oedema, which had developed under observation, were greatly relieved, though the patient remained unfit for work.

Case 3

An old-age pensioner aged 64 had no history of rheumatic fever or venereal disease. He entered hospital in December, 1947, with bronchitis; swelling of the legs was noted. Digitalis was begun when he was again in hospital in May, 1948.

He was readmitted on June 10, 1948, with congestive heart failure, complaining of increasing shortness of breath on exertion and while at rest. His weight was 71.2 kg.; height 170 cm. He perspired and was dyspnoeic; the veins of his neck were much distended. The apex impulse was in the anterior axillary line, the heart sounds were hammer-like, with persistent irregularly irregular rhythm and an apex rate about 60 a minute. B.P. was 200/130. Lung bases showed dullness, impaired breath sounds, and rales. The liver edge was four fingerbreadths below the right costal margin; there was no detectable ascites, but moderate oedema of the legs.

An electrocardiogram revealed auricular fibrillation, deeply inverted T_1 and T_2 , and a ventricular rate of 80 a minute. X-ray examination showed the heart greatly enlarged, 18/28 cm., and severe passive congestion of both lung bases; no pleural fluid. The haemoglobin was 112%; sedimentation rate, 4 mm.; urine, albumin +, microscopical examination normal; blood urea, 22 mg. per 100 ml.

Treatment.—The day after admission Cheyne-Stokes respirations and his general condition were improved by venesection (400 ml.) and morphine, but in the next few days he grew restless, with dyspnoea and mental confusion. Ammonium chloride had to be stopped after five days on account of nausea. His condition deteriorated, and because of dyspnoea he sat up day and night. On the eighth day treatment with the low-salt diet was started, but to begin with he could eat only half the food. After six days on this diet his condition was noticeably improved. The B.P. fell to 150/100, dyspnoea decreased, diuresis increased (but on account of mental confusion could not be measured exactly), and weight fell 5 kg. After a further 10 days he could sit up, and his mind had cleared. The oedema had disappeared and his weight had fallen another 4 kg. He was sent home on the 67th day.

His condition was then bearable. He continued on a low-salt diet, but still had slight oedema of the legs, and was admitted to the hospital again four months later in order to clear his oedema completely. After this he was

so well that he bought a tricycle. On July 20, 1949, he was admitted on account of hypostatic pneumonia.

Comment.—This 64-year-old man with hypertensive heart disease showed marked congestive heart failure with extreme dyspnoea and cyanosis, which did not improve after eight days in bed, with digitalis, ammonium chloride, and mercurial diuretics. After a low-salt diet was begun there was a fall in blood pressure, a loss of oedema, and marked general improvement. With continuation of the low-salt diet he was later admitted because of slight oedema of the legs, and again with hypostatic pneumonia, but his condition in the intervals between these readmissions was considerably better than before he began the low-salt diet.

Case 4

A grocer aged 56 had had scarlet fever at the age of 8, but no diphtheria, rheumatic fever, or venereal disease. He had always eaten too much, had taken fairly large amounts of alcohol earlier, and was obese, weighing 92–95 kg. Diabetes was known to exist since the age of 42; he had had no insulin. He had urolithiasis at 53. At 54 he entered hospital for arteriosclerotic heart disease with dyspnoea, and had been treated since with an ordinary diet without sugar and with mersalyl, and later theophyllin and digitalis leaf, 0.1 g. a day. He had increasing dyspnoea, especially at night, over the past 12 months and oedema of the legs during the past three weeks.

He was admitted on February 15, 1949, for congestive heart failure, with dyspnoea, pressure in the chest, oedema, and severe suffocative paroxysmal nocturnal dyspnoea, which forced him to sit up in order to sleep. His weight was 83.4 kg.; height 164 cm. He was weak, dyspnoeic, cyanotic, and extremely orthopnoeic. The heart size was indeterminable; heart sounds were of very poor quality; rhythm irregularly irregular; apex rate, 78; B.P. 100/60. The liver edge was palpable a hand's breadth below the costal margin; no ascites was detectable.

An electrocardiogram revealed auricular fibrillation, T waves iso-electric, and low-voltage QRS complexes in standard leads. X-ray examination showed the heart shadow not enlarged, 16/34 cm., and pulmonary passive congestion, without fluid. The haemoglobin was 95%; sedimentation rate, 4 mm.; urine, albumin +; blood urea, 38 mg. per 100 ml.

Treatment.—For one week he was on a diet without sugar but with the ordinary amount of salt, with digitalis, 0.3 g. and later 0.1 g. daily, and mersalyl. There was no change in his general condition or in the oedema, and he continued to complain of severe dyspnoea at night. In the next 10 days, after removing the salt from the diet, diuresis increased, the weight fell by 5 kg., and the dyspnoea, including the nocturnal attacks, disappeared. He was discharged three weeks later in good condition, having lost a total of 14 kg. in weight. He continued on the low-salt diet without sugar, with digitalis daily and a mersalyl suppository twice a week, without any recurrence of signs or symptoms for the next two months, when he died suddenly, presumably of an acute coronary thrombosis.

Comment.—This 56-year-old man with obesity, diabetes, and arteriosclerotic heart disease with severe congestive failure did not improve in spite of rest in bed, digitalis, and mercurial diuretics. After the institution of the low-salt diet without other change, his condition improved, and he was discharged. His improvement was maintained without any return of symptoms until sudden death two months later.

General Comment on Above Cases

The data from these four patients with severe congestive heart failure are presented in some detail. To obtain a true picture of the effect of the low-salt diet

we first tried to ascertain whether the usual methods of treatment (confinement to bed, digitalis, mercurial diuretics) resulted in an improvement in the condition. Only when this method failed were the patients transferred to a low-salt diet. Changes in weight, together with observation of the changes in oedema, were used as objective criteria of the patients' condition. The changes in subjective symptoms—dyspnoea, cyanosis, palpitations, pains, sleeplessness, fatigue, etc.—however, could only be described. There was no doubt about the results: the low-salt diet led to a dramatic change in the condition of these patients in a few days. It was also noted that without salt restriction the diuretics brought about only a temporary decrease in the oedema, but that their effect when used with this diet was greater and more lasting. The latter result is naturally due to the fact that on a low-salt diet there is much less possibility of the oedema re-forming rapidly.

General Observations and Follow-up Studies of the 64 Patients

Having seen the good results of the low-salt diet, we began to use it on patients fairly soon after their admission to hospital as a supplementary therapy in nearly all cases of congestive heart failure. We also used it in some of the cases which could have been treated without the low-salt diet. In these cases the good effects of the treatment can best be judged by examining the after-effects. We have examined these patients to see whether those who kept to the low-salt diet fared better than those who did not.

Attention must be drawn to the fact that all of these patients were suffering from severe heart disease and had a very doubtful prognosis even on a low-salt diet. On account of the relatively small number of cases no great importance can be attached to the difference in the death rate in the two groups, though on analysis it does favour the patients who followed the diet. It is more important to examine the degree of the symptoms of congestive heart failure in the two groups.

Table I shows the results of the treatment of all 64 cases in the hospital period. The low-salt-diet treatment in the hospital resulted in decisive improvement

TABLE I.—Result of Hospital Treatment of 64 Patients with Congestive Heart Failure

Type of Heart Disease	No. Treated	Markedly Improved	Slightly Improved	Unchanged	Worse	Died
Rheumatic ..	11	3	7	0	11	1
Arteriosclerotic	30	21	5 ²	0	4 4 5 6 7	5
Hypertensive	23	12 ³	9	1	18	2
Total ..	64	36	21	1	6	8

Causes of death.—1 Adhesive pericarditis, aortic, and mitral stenosis. 3 Cholecystitis. 9 Cerebral embolus. 4 5 Coronary occlusion. 6 Congestive heart failure, coronary sclerosis. 7 Congestive heart failure, adhesive pericarditis. 8 Uraemic, nephrosclerosis.

in 36 and slight improvement in 21; six of the patients grew worse and died; in one there was no change. Two others died of intercurrent diseases. The causes of death are given at the foot of the table.

After their discharge from the hospital (see Table II) 50 of the 56 survivors from the original 64 were examined from three to eighteen months later. The length of time between discharge and observation is approximately the same for the group that followed the diet and the group that did not. In 27 out of the

TABLE II.—Follow-up Study of 50 of the 56 Survivors After Discharge from the Hospital

Type of Heart Disease	No.	Continued Low-salt Diet				Off Low-salt Diet			
		No.	Condition Maintained	Worse	Died	No.	Condition Maintained	Worse	Died
Rheumatic ..	9	4	3 ⁹	1 ¹⁰	2	5	2	3 ¹² 13 14	3
Arteriosclerotic	22	17	16	1 ¹¹	1	5	0	5	0
Hypertensive	19	10	8	2	0	9	1	8 ¹⁵ 16	2
Total ..	50	31	27	4	3	19	3	16	5

Causes of death.—9 Endocarditis lenta. 10 11 Congestive heart failure. 12 14 Pulmonary oedema. 15 Anasarca (congestive failure). 16 Congestive heart failure. 16 Coronary occlusion.

31 patients who continued the diet their condition remained the same as when they left the hospital; in four the condition had deteriorated, and two of these had died. Nineteen patients did not continue the diet—in three the condition was unchanged, but 16 had deteriorated, and five of these had died, all of congestive heart failure.

Discussion

Time and again at these follow-up examinations we have been able to ascertain a relation between discontinuation of a diet and a deterioration in the patient's condition, an increase in weight, and greater excretion of chloride in the urine. This is not contradicted by the fact that several of the patients have continued in good health on a diet with a greater salt content than prescribed. This even applied to patients who originally were improved only with difficulty with the aid of a low-salt diet in addition to the usual treatment. It can therefore be assumed that the salt tolerance may rise simultaneously with the general clinical improvement in the patient's condition (see Case 1). This means that some patients do not have to continue with the low-salt diet, but are able to live on a more normal diet—for example, simply without table-salt and salted dishes—or often with ordinary bread-and-butter. By watching the patient's general clinical condition, weight, and chloride excretion in the urine one knows whether he is keeping to the diet and, if this is not the case, whether a discontinuation of the more rigid features of the diet is permissible. In cases of incipient congestive heart failure it can be decided on the same basis whether a return to the low-salt diet alone may be expected to improve the condition or whether diuretics are necessary.

Our observations seem to show that if the patient's weight remains the same the sodium chloride content of the urine will correspond more or less to the amount consumed. In cases of increasing weight alone, due to the formation of occult oedema, the excretion is less, and when the oedema decreases again the excretion is greater. Excretion of salt rises violently with mersalyl diuresis; an excretion of 20 g. or more of sodium chloride in 24 hours may be observed. With the aid of mercurial diuretics, temporary relief may be obtained by the excretion of the retained cooking-salt, but if the salt consumption is not decreased, patients will again retain salt, and oedema will again be the result. By keeping to a low-salt diet a more stable condition is achieved than if the oedema is allowed to reproduce. It is possible, that this greater stability and continued freedom from occult oedema is the reason why some patients feel much better on a low-salt diet than on an

ordinary diet with diuretics. But all patients will not or cannot tolerate the low-salt diet often necessary to prevent oedema. In these cases diuretics must be administered with a diet heavier in salt or possibly combined with a weekly saltless day, with unsalted thin oatmeal gruel or boiled rice as the only food.

Formerly one of the methods of treatment of patients with a predisposition to oedema was to reduce the consumption of fluid. When a low-salt diet is used, however, a reduction in the amount of fluid is not necessary; in fact, a fluid consumption which ensures fairly large diuresis (2-3 litres every 24 hours) seems to be the most effective, as an output of 2-3 litres of urine has been shown to give a larger excretion of salt. When allowing the patients plenty of water it must be borne in mind that some water is relatively high in sodium. In Copenhagen, for example, the sodium content of tap-water varies from 9 to 131 mg. per litre, with an average of 50 mg.

Finally, we should like to review the symptoms and signs in which improvement has been observed on a low-salt diet. The effect on the oedema was the easiest to demonstrate. The low-salt diet, and especially the combination of this diet with digitalis, mercurial diuretics, and sometimes ammonium chloride and urea, is the treatment which most readily causes the disappearance of oedema in patients suffering from heart failure. The most striking example of the effect of this treatment was seen in the case of a 64-year-old oedematous woman who lost 27.6 kg. in two months. Both *dyspnoea at rest* and *attacks of dyspnoea at night* often improve or cease altogether, generally after a few days' treatment. We observed good results in many cases, particularly in patients suffering from nocturnal dyspnoea. A number of patients who in spite of morphine had slept badly for a long time soon slept better with this treatment (see Case 4).

A true dehydration from a deficit of plain water, which is often found in oedematous patients, results in the symptoms of an intense thirst and a dry tongue. The symptoms may be present even if the patient's consumption of fluid has not been decreased, and these symptoms are often the first to disappear when the patient is put on a low-salt diet. This is a great relief to patients, but means that they must be encouraged to drink plentifully to ensure a fairly large diuresis.

The *blood pressure* in hypertensive subjects often falls during the treatment (Bang *et al.*, 1949).

With the diet used we did not observe any signs of a lack of salt—that is, fatigue or weakness—but many of the patients complained of the tastelessness or uniformity of the diet. There are, however, two diseases which necessitate care when using a low-salt diet: in cases of advanced kidney disease in which the kidneys are not able to retain sufficient sodium a low-salt diet may mean a definite deterioration of the condition of the patient, with hypochloraemia and acidosis; similarly, in cases of Addison's disease a low-salt diet may bring about a typical crisis.

Summary

Sixty-four patients suffering from congestive heart failure with oedema were, in addition to the usual treatment with confinement to bed, digitalis, and diuretics, treated with a low-salt diet (less than 1 g. of sodium chloride a day).

A detailed case history is given of four of the patients in whom the critical condition was not improved until they were transferred to a low-salt diet.

Follow-up studies were made on 50 of the cases in the series. Thirty-one had kept on with the low-salt diet, and 27 of these continued in as good health as on discharge, one was worse, and three had died.

The results are discussed, and the effects of mercurial diuretics and a low-salt diet are compared.

The restriction of the fluid intake is not necessary or beneficial for the patient with oedema on a low-salt diet.

In some cases of chronic kidney disease with loss of normal ability to conserve sodium, and in Addison's disease, sharp restriction of salt is contraindicated.

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RADIATION, LIGHT, AND ILLUMINATION*

BY

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The visual mechanism is normally stimulated by radiation lying within the comparatively narrow region of from 4,000 Å to 7,000 Å. In nature the source of this radiation is the sun. In the highly complicated structure of modern society, however, man has found it necessary to devise artificial means for the production of light. I shall therefore concern myself to some extent with radiation generally, but more particularly with the limited region in the radiant energy spectrum known as light to which the human eye responds and with artificial light sources.

In trying to present a picture of the evolution of modern light sources in historical perspective it is necessary to consider certain primary matters in connexion with radiation, light, and illumination. Whereas the eye has evolved through many ages under the continuous radiation from the sun, it has from the beginning of time been subjected to a more limited region of the visible spectrum in the perception of colour. It is this fact which led lamp-research physicists to believe that if in the course of the evolution of light sources they had to produce and put before the public lamps giving discontinuous spectra no harmful results would thereby occur.

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