ipsilateral side was fixed or its excursion very much reduced, while on the contralateral side movement was free; yet the only case in his series to develop pulmonary complications produced a lower-lobe atelectasis on the contralateral side.

Summary

The incidence of pulmonary complications following upper abdominal operations remains high.

After an upper abdominal operation the vital capacity is regularly decreased to the region of 35% of its pre-operative volume.

Intravenous drip procaine is an excellent method of relieving post-operative pain in cases in which it is important not to decrease pulmonary ventilation.

There is evidence that some increase of vital capacity follows the relief of post-operative pain by this method in abdominal cases. This increase, however, is so small that it is suggested that other theoretical causes for the lowered vital capacity need investigation.

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LOW-SALT DIET IN TREATMENT OF HYPERTENSION AND HYPERTENSIVE HEART DISEASE*

BY

H. O. BANG, M.D.

P. BECHGAARD, M.D.

AND

A. LEVIN NIELSEN, M.D.

(From Medical Departments 2 and 7 of the Municipal Hospital, Copenhagen)

The treatment of heart disease and hypertension has of late attracted much attention, thanks especially to papers by Allen and Sherrill (1922), Schroeder (1941), Schemm (1942), and White *et al.* (1946). Kempner (1944) claimed to have seen an exceptionally favourable response of hypertension to his salt-poor "rice diet." The favourable effect on these diseases was, however, observed far earlier by Karell (1866), Achard and Loeper (1901), and Widai and Lemierre (1903). In Denmark, Finsen (1904) proposed a low-salt diet in diseases associated with oedema. According to recent American experiences a rigorous restriction of the salt content of food (under 0.75 to 1 g. of NaCl daily) is necessary, at least in the case of hypertension.

Present Investigation

Our object has been to test the above American results. In particular we have tried to prove that sodium chloride alone is the deciding agent and that the possible favourable response to the dietary treatment is not due to other factors, such as confinement to bed and rest in hospital.

Diet.—Originally we used a diet containing about 2,500 calories, but we soon had to reduce it to about 2,000 calories, the patients being unable to eat it all. The solid food consists of:

Morning: 45 g. white bread (salt-free), 10 g. butter (salt-free), 5 g. sugar, 1 g. tea.

Either simultaneously or later in the morning: 25 g. oatmeal (as porridge, cooked without salt), 5 g. sugar, 125 g. stewed fruit.

Supper: 100 g. white bread (salt-free), 30 g. butter (salt-free), half an egg, 50 g. tomato (or 100 g. apple or 10 g. cucumber), 25 g. fresh meat (or 20 g. fish), 10 g. lemon juice with 10 g. sugar (or 50 g. juice in water).

Dinner consists of fruit jelly as the first dish (without milk), and boiled or fried (in salt-free butter) fish or meat, with vegetables and potatoes afterwards. The composition of the diet per day, calculated after Sherman's tables, appears to be: sodium 262 mg., chlorine 385 mg., calcium 254 mg., iron 7.9 mg., with protein 50 g., fat 88 g., and carbohydrate 258 g., giving 2,019 calories. The diet is insufficient with regard to calcium, and perhaps vitamin B and iron. During the experimental periods we added daily doses of vitamins A and D and gave a weekly injection of a vitamin B preparation.

Material.—The series consisted of 26 patients, all admitted to the Municipal Hospital, Copenhagen. They presented such a degree of hypertension that after a fortnight's confinement to bed they still had a systolic blood pressure above 180 mm. Hg and/or a diastolic pressure above 120 mm. Hg. Seven of the patients were men and 19 women, with ages ranging between 45 and 71 years (average 59). Their hypertension had been recognized from well over 1 year up to 14 years previously. None of the patients seemed to suffer from the rapidly progressive malignant form of hypertension. The hypertension was in most cases complicated by heart disease (possibly with insufficiency), retinopathy, or nephropathy. In the accompanying table the patients have been grouped according to diagnosis, with the uncomplicated cases at the top, and then the different complications in order of increasing severity. Patients whose urea clearance was less than 50% of the normal value have been entered under a diagnosis of nephropathy. None of the patients had had syphilis or rheumatic fever.

Experimental Period.—During the preliminary fourteen days the blood pressure was measured frequently, and patients in whom it fell below the above limits were not submitted to treatment. A number of the less severe cases were therefore excluded. Four days before starting treatment with the low-salt diet we began to take the blood pressure of each patient in the morning; preferably this was done by the same physician, at least three times. The blood-pressure values are based on the lowest recorded for each day. The values stated in the table are means for the last four days. Column I gives means for the preliminary period, column II for the low-salt-diet period, and column III for the control period of high salt intake. During the preliminary period we measured the chloride and nitrogen excretion daily for two or three days. Ophthalmoscopy was carried out, as well as radiography of the heart, electrocardiography with precordial deflection,

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estimations of serum chloride, serum bicarbonate, and blood urea, and in most cases urea clearance. The urine was examined microscopically in all cases, and finally the patients were submitted to a general clinical examination. During the low-salt-diet period the daily chloride and nitrogen determinations were continued, while the remaining examinations were performed once a week. The patients were weighed not less than twice weekly, often daily. This period lasted from 10 to 51 days (average 15.8 days).

Control Period.—After the expected fall in the blood pressure had occurred and stable conditions seemed to have been attained, we—without making other changes, and unknown to the patient—added 4.5 g. of sodium chloride to the diet in order to see the effect on blood pressure, weight, and the subjective symptoms. To avoid any suggestive effect of the salt addition the patients had previously been given "dummy tablets" every day. Only two of the patients discovered the change, and then only just before the end of the control period. The control period lasted from 5 to 15 days (average 8.4 days). In three cases with a blood-pressure fall during the low-salt period

in a single case as much as 17.4 kg., or 20% of the body weight. During the following period with the addition of salt all the patients except two gained weight, but only from 0.1 to 1.9 kg., the patients with oedema not having been submitted to this control treatment. The gain in weight, like the preceding weight loss, occurred in the first few days after the change. The four patients whose blood pressure did not rise during the salt treatment put on 0.6, 0.7, 0.9, and 1.9 kg. respectively—in other words, the same as the others.

Prior to the low-salt-diet period the urine contained 2 to 6 g. of chloride daily. During this period it fell to 0.3 to 0.5 g. daily, to rise again a few days after the addition of sodium chloride to 1 to 3 g., in conformity with a chloride content of about 2.7 g. in the salt added. The nitrogen excreted in the urine during the entire course of treatment varied from 3 to 7 g. daily, dependent on the patient's appetite, the diet not being poor in protein. The serum chloride concentration generally showed a slight fall to low normal or slightly subnormal values. The bicarbonate concentration in serum remained normal, as also did the blood urea.

Blood Pressure of 26 Hypertonic Patients during Low-salt-diet Treatment, and During a Control Period with Addition of Salt

No.	Sex	Age	Morbus cordis	Nephropathy	Encephalopathy	Blood Pressure			Weight (kg.)			Response
						Preliminary Period	Low-salt Period	Control Period with Salt Intake	I	II	III	
						I	II	III				
1	F	44				206/124	125/75	145/91	63.5	-1.8	+0.5	+
2	F	56				210/129	206/115	216/115	60.0	+1.1	+0.5	—
3	F	64				190/111	136/82	156/87	60.5	-2.8	+2.3	+
4	F	50				250/165	147/101	182/104	99.8	-2.8	-0.5	+
5	F	58	+			224/103	173/84	209/81	65.4	-1.5	-0.8	+
6	M	65	+	+		186/99	163/71	184/84	82.8	-2.2	+0.6	+
7	M	62	+	+		168/134	162/110	—	84.3	-17.4	—	—
8	F	59				209/109	169/101	194/102	61.0	0.0	+0.7	+
9	F	70	+			245/124	194/110	210/122	77.4	-0.7	+0.8	+
10	F	60	+			195/109	162/102	179/102	74.5	-1.2	+0.2	+
11	F	57	+			179/127	150/106	179/120	87.8	-3.1	+1.1	+
12	M	53	+			178/135	157/116	182/134	96.1	-1.3	+0.9	+
13	F	66	+			189/93	159/70	163/65	68.0	-1.7	+0.9	—
14	M	71	+	+		236/120	246/120	—	82.5	-1.6	—	—
15	F	54	+		+	215/120	157/102	189/110	53.7	-0.3	+1.6	+
16	F	68	+		+	200/110	130/87	170/97	61.9	-0.7	+1.8	+
17	F	63	+			248/122	163/78	—	66.6	-6.6	—	?
18	F	48	+	+		191/110	165/92	180/88	76.5	-1.8	+1.9	+
19	M	45	+	+		246/122	218/110	—	77.0	-1.6	—	?
20	F	54	+	+		216/115	188/106	192/112	76.8	-1.5	+0.7	—
21	M	53	+	+		216/157	164/100	162/92	73.6	-0.1	+0.6	—
22	F	60	+	+		214/160	135/94	187/106	68.3	-1.5	+0.8	+
23	F	57	+			202/100	155/84	175/85	71.9	+0.1	+1.4	+
24	F	59	+			178/108	157/73	155/91	42.6	-2.7	+1.9	—
25	M	66	+	+		255/122	212/91	—	72.4	-4.9	—	?
26	F	61	+			210/163	135/102	161/122	66.1	-0.6	+1.8	+

we did not carry through a control period. Therefore these results must be regarded as uncertain.

Results

Of the 26 patients treated 23 showed a fall in blood pressure during the low-salt-diet period. In four of these the addition of salt produced no rise, and in three this control test was not performed. In the remaining 16 with blood-pressure rises during the control period the fall occurred after from 4 to 16 days (average 8.5 days) on a low-salt diet. In patients with oedema the fall occurred later, after the considerable fluid and salt output had ceased. The low-salt diet was discontinued if no response was seen within three weeks. In the 16 cases with a rise during the control period the fall averaged 51 (21 to 103) mm. Hg systolic, while in the 4 with a negative result of the salt addition it averaged 33 (21 to 53) mm. Hg.

During the treatment with a low-salt diet we generally observed a slight loss of weight in the first few days. Two patients, however, put on 0.1 and 1.1 kg. respectively. In patients with oedema the weight losses were much greater—

Eye examinations revealed normal fundi in five patients and from moderate to pronounced changes in the rest (after Keith and Wagener: group I, 3 patients; group II, 15 patients; group III, 2 patients; and group IV, 1 patient). The findings were generally the same before and after treatment. In two cases only did we observe diminishing exudate and haemorrhage during the course of treatment.

Seven patients had normal electrocardiograms. In six patients T_1 was iso-electric and in eight negative. In three, iso-electric T_1 waves became definitely positive, and in one a negative T_1 wave became iso-electric.

X-ray examination often showed the heart to be enlarged. In two cases a decrease of 1 cm. was observed, which, however, is insignificant. Radiographic signs of pulmonary stasis disappeared regularly during the treatment.

The subjective symptoms of hypertension—i.e., headache, sensation of pressure in the head, hot flushes, dizziness, tiredness, increasing to regular encephalopathy—disappeared in all cases except one even in those with no

objective effect on the blood pressure. On the other hand, the patient who experienced no subjective relief showed a marked fall in blood pressure. Sensations of oppression and dyspnoea subsided or disappeared completely. The subjective effect on the patients was on the whole so striking that the majority wanted to continue the low-salt diet after their discharge. Within the control period the symptoms began to recur in several cases, and in these we stopped the addition of salt after a short while.

The following are three typical cases.

Illustrative Cases

Case 22 (Fig. 1 and Table).—Extraordinarily, this patient was given an addition of 4.5 g. of sodium chloride daily the last six days before receiving the low-salt diet. Addition of salt to the low-salt diet caused a rise in the blood pressure, and discontinuation of the salt addition a renewed fall. Transition to an ordinary mixed diet again produced a rise.

Case 16 (See Fig. 2 and Table).

Case 17 (Fig. 3 and Table).—Patient suffering from hypertensive congestive heart failure. The considerable chloride excretion parallels the decrease of weight (loss of oedema), after which followed the fall in blood pressure.

FIG. 1.—Chart of Case 22.

FIG. 2.—Chart of Case 16.

FIG. 3.—Chart of Case 17.

Discussion

The material here presented, consisting mainly of elderly hypertonics with complicating heart failure, shows that a low-salt diet may produce a fall in blood pressure in many hypertonics, but not in all. The fact that the addition, without the patient's knowledge, of sodium chloride during a low-salt diet period makes the blood pressure rise again seems to show that sodium chloride as such plays a decisive part in some cases of hypertension. We do not yet know how the salt acts. But this very question is of the greatest theoretical interest and requires further investigations, among others into plasma volume and renal function. The oedema-clearing effect of the low-salt diet is conspicuous. One should, however, be careful in using this treatment in cases of certain chronic renal lesions where the kidneys are unable to reduce the sodium excretion in the normal way. Here, as seen in one case, we may soon produce an extrarenal uraemia "par manque du sel."

Besides the effect on blood pressure, oedema, and dyspnoea, and the relief of the subjective troubles, we saw no favourable response to the diet except in some cases electrocardiographic changes and, rarely indeed, reduction of exudate in the fundus. We know, however, that electrocardiograms from untreated hypertonics very seldom "improve" (Bechgaard, 1946).

It is our opinion that the results achieved should stimulate workers to further experiments with the low-salt diet.

Summary

Twenty-six patients with hypertension, in some associated with heart failure and/or nephropathy, were treated with a diet containing less than 1 g. of sodium chloride. Sixteen of these experienced a fall in the blood pressure, which rose to some extent again after a daily addition of 4.5 g. of sodium chloride. In addition the low-salt diet proved to have a favourable effect on cardiac oedema. Owing to the continuous loss of sodium the treatment is contraindicated in certain cases of chronic nephritis.

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SYMPTOMATIC TREATMENT OF MYOTONIA ATROPHICA

BY

L. BUSSELL, D.M., B.Sc., M.R.C.P., D.C.H.

Clinical Assistant, Royal Infirmary, Sheffield

The value of the antihistamine drugs in increasing mobility in Parkinsonism has been noted by various clinicians. This spasmolytic action of the antihistamines is probably due to lengthening of the refractory period of muscle, which then becomes unable to maintain a tetanus. Such an action has been demonstrated on isolated rabbit auricle for quinidine, pethidine, procaine, and other local analgesics (Dawes, 1946). It was later shown that "anthisan" ("neoantergan") was twice as potent as quinidine in this respect (Dews and Graham, 1946).

In the case described below this action of the antihistamine drugs was applied to the treatment of myotonia atrophica.

Case Report

The patient, a Derbyshire farmer aged 49, presented the classical features of myotonia. The facial expression was woebegone, with bilateral cataract and receded frontal hairline. The neck was so wasted that his collars had become unserviceable, and on special occasions he resorted to a neckerchief. There were weakness and wasting in the arms, legs, and small hand muscles, with a delay of several seconds in release of the handgrasp. He was unable to cut up his own food at meals or to do heavy work. He managed, however, to run a small poultry farm, clumping around with a noisy stamping gait, and carrying buckets of water or poultry meal on the crook of his best forearm. The testicles were atrophied and bronchiectasis was present. He had had four attacks of pneumonia in recent years. Although friendly enough with his doctor, others found him moody and difficult.

Symptoms began in 1943, but the condition escaped diagnosis until 1946, when quinine was given with benefit. Treatment lapsed after the first few weeks and had not been resumed.

He was the only member of the family to show the full picture of the disease. His grandfather and father both suffered from senile cataract. His grandfather was a powerful man of great physical stature, who narrowly missed recruitment into the Life Guards. Both his grandfather and his father had been exceptionally heavy beer-drinkers. The patient's brother, a middle-aged man, and the brother's two daughters were said to suffer from short sight. All three wore glasses with lenses of startling thickness. The patient himself had two healthy sons in their teens.

Treatment was started in June, 1949, with 50 mg. of "benadryl" three times a day. After one week the patient was able to use a knife and fork and to cut up his own food for the first time for two years, and he began to carry buckets of water in the normal way by hand. There was objective improvement in the myotonia of the hands, and the small hand muscles became firmer to palpation. He was then given 200 mg. of anthisan three times a day for a further two weeks. Improvement continued, and he was able to fork dung and "sweep out" with greater effect than before. The myotonia of the hands was not, however, altogether abolished.

Discussion

The patient was not a good subject for assessment of a therapeutic remedy. The severe wasting placed a limit on the value of spasmolytic drugs, while the mental condition introduced a big subjective factor. If the value of antihistamines in myotonia is confirmed, they might be employed in two ways: the antihistamine drug could be given with quinine to obtain a summation effect, or the two drugs could be given alternately, say for a week each, to avoid the unpleasantness of cinchonism.

In considering the treatment of myotonia atrophica it is well to take account of the relationship between this disease and myasthenia gravis. A pharmacological antithesis is recognized between these two diseases, drugs which benefit the one causing an exacerbation in the other (Goodman and Gilman, 1941). It was thought possible that this antithesis might operate in the case of the thymus gland. Tumours of this gland are sometimes found in myasthenia, and a remission in the disease sometimes follows thymectomy. Accordingly, after three weeks' antihistamine treatment in the above case it was decided to try the effect of thymus-gland administration. For a fair trial it would be necessary to give fresh gland extract, by injection. As fresh extract is not readily available, dried thymus tablets were given by mouth for a month—10 gr. (0.65 g.) thrice daily. There was no objective change. The patient, however, reported continued subjective improvement, with increase of the myotonia in the hands after withdrawal of the tablets. This may have been a psychological effect, and owing to the preceding antihistamine treatment the experiment was considered inconclusive. It did, however, succeed in showing that thymus gland by mouth does not provide a replacement therapy for the disease.

Further work on the antihistamines is required to establish whether antihistamine and spasmolytic action run parallel. If this is so, the newer and more potent antihistamines should repay trial in myotonia. It is also possible that some of the local analgesic substances possessing a quinidine-like action on rabbit auricle (Dawes, 1946) may prove suitable for clinical use in myotonia.

In general it is to be expected that drugs such as quinine which benefit myotonia would also be beneficial in the rigid type of Parkinsonism.

Summary

A pharmacological basis for the action of spasmolytic drugs in Parkinsonism and in myotonia atrophica is described.

On this basis a spasmolytic agent which is active in one of these diseases should also be active in the other.

A case of myotonia atrophica is reported. Benadryl and anthisan both produced striking symptomatic improvement.

The possible role of the thymus gland in myotonia atrophica is discussed.

I wish to express my thanks to Professor Wayne for helpful criticism and for permission to treat the case.

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Mayfield and Hedrick present in the United States *Journal of Nutrition* (1949, **37**, 487) a study of the nutritive value of protein in foods and how it may be affected by the various processing procedures. The effect of varying the type of cattle feed and subsequent changes of roasting and corning on the digestive and biological value of beef protein were studied. Canning caused a lowering in the biological value and digestibility of the beef, but roasting and corning produced no change.