【Original Article】

Effective low salt diet for a salt - sensitive hypertensive patient Different reduction rates between the morning SBP and the night SBP

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Summary

Hypertensive patients must reduce their salt intake.[1-5] However, little is known about the quantitative relationship between personal salt intake and home blood pressure.[6] The author experienced brain infarctions upon hypertension three times. After his first hospitalization, a 7 g/day salt diet recommended by the hospital was performed at home. Nonetheless, such a diet could not prevent further reoccurrences. Hence, a lower salt diet was required. To know the proper salt intake, the fluctuations of data during a 7 g/day salt diet were utilized. The *r* between the night SBP and daily salt intake was 0.662**. By the regression equation, suitable salt intake of 3.5 g/day corresponding to optimal SBP of 120 mmHg[7,17] was gained. After 19 months of this salt diet, the night SBP decreased to the expected level of 120 mmHg. The morning SBP took only 5 months to reach a homeostatic level of 132 mmHg. Different reduction rates between both were characteristic. Moreover, the morning SBP was influenced by salt of the previous day's breakfast and lunch, and hardly by the previous day's dinner. Salty food was suggested to ingest at dinner. The 3.5 g/day salt diet was effective in preventing reoccurrences and met recent recommendations. [8]
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Key words: increased resting blood pressure, SBP reduction rate, 3.5 g/day salt diet, correlation coefficient (*r*) between salt intake and SBP, regression equation between SBP and salt intake

Introduction

As a salt-sensitive hypertensive patient under medication of nifedipine (20 mg/day), the author suffered a brain infarction (Wallenberg syndrome with abasia) in Oct. 1994. After leaving hospital for the first occurrence, he began a 7 g/day salt diet following the recommendation of the hospital. However, there were the second reoccurrence in Feb. 1998 and the third in Oct. 1998. Immediately after hospitalization for each brain infarction, SBP values were raised as high as 220 mmHg.

There is little in the literature on the relationship between personal salt intake and home blood pressure. Therefore, as a scientist, the author examined its relationship and estimated the suitable salt intake to control his hypertension in order to avoid a lethal reoccurrence.

After the third infarction, he became aware of the importance of the 56 days' data of fluctuations from 3 to 10 g/day under a 7 g/day salt intake. During relatively healthy period of these days (n=56) just two weeks before the second infarction, actual salt content of each food was happened to be carefully measured by a salt meter. The distribution was checked by probability paper, but out-of-range values were not excluded.

The correlation coefficient (r) between daily salt intake and the night SBP and the regression equation were calculable due to such fluctuations.

In fact, the proper salt intake of 3.29 g/day corresponding to optimal SBP 120 mmHg was easily calculated by equation (1) later-mentioned. Hence, daily salt intake should be controlled within 3.0~3.5 g/day, and a 3.5 g/day salt diet was adopted considering long time feasibility. The gradual change in salt diet from 7 g/day to 3.5 g/day via 5.5 g/day was executed on the safe side.

Unexpectedly, under the 5.5 g/day salt diet, the morning SBP increased as high as 180-190 mmHg/week, which indicated increased resting blood pressure.[9-11] Such resting high SBP was overcome by a 3.5 g/day salt intake.

Even though attending Japanese doctors strongly opposed such a low 3.5 g/day salt diet in fear of dehydration, the author intuitively felt that the 3.5 g/day salt diet was a final measure to prevent further reoccurrences. Indeed, immediately after such a low salt diet, he felt free from serious malaise.

The 3.5 g/day salt diet has been successfully performed to prevent further reoccurrences of brain infarction for more than one decade, and he has been living with a good quality of life (QOL).

Methods

Measurement of systolic blood pressure (SBP)

There was no standard methodological theory of how to utilize sequentially measured values of SBP. The measured SBP of salt sensitive hypertensive patients was highly unstable and the sequential effect was statistically significant. So, in this research initially measured values of SBP were consistently adopted. From the viewpoint of small sample theory considering a standard deviation of measured SBP (13 ± 5 mmHg), a monthly average of SBP ($n \fallingdotseq 30$) was generally used for evaluation of increasing or decreasing SBP.

Moreover, concerning SBP change during the day, the selection as to the measuring time of SBP has not been established. Considering circadian rhythm[13,14], SBP was measured every night just before sleeping (at 24 ± 1 hr) and every morning immediately after awakening (at 8 ± 1 hr) in bed. SBP was determined in the lateral position utilizing a cuff type instrument (Omron) on the left brachial at the same level as the heart. In order to avoid missing SBP measurement, each SBP would be better measured in bed.

Measurement of salt in food and urine

In order to measure the salt content in food, each item of food was weighed on an electric balance (Shimazu) and its salt content was simply measured by a salt meter. At the same time, salt content and daily calories (ca.1,600 kcal/day) were calculated by the Standard Table of Food Composition in Japan.[12] The excess calories were adjusted by daily walking (conversion rate: 400 kcal per 10,000 steps).

When the salt content of the food was not clarified, the water extracted from food was measured at 25 ± 3℃ by a salt meter of Horiba c-121 (sensibility, 0.01%) or of Sato (sensibility, 0.01%). If necessary, food was ground in a mortar and salt was extracted by appropriate amount of 5 to 25 times water or hot water. The oily substances were removed through filter paper before measuring. When oily matter was difficult to filter, it was easily excluded after solidifying it in a freezer at ca.1℃.

The ingesting time of each meal was at $8{:}30 \pm 1$ hr for breakfast, $12{:}30 \pm 1$ hr for lunch, and $19{:}00 \pm 1$ hr for dinner, respectively.

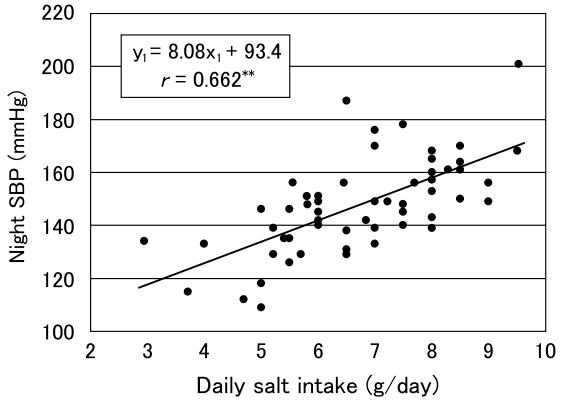
In order to clarify the relation between daily salt intake and its secretion into urine for 24 hours, the amount of salt in urine was measured as mentioned above.

Results

Relationship between daily salt intake and the night SBP under a 7 g/day salt diet

When a salt diet of 7 g/day was followed, daily salt intake fluctuated from ca.3 to ca.10 g/day and the average of actual salt intake for 56 days was 6.7 ± 1.5 g/day (from 1st Dec. 1997 to 25th Jan. 1998). The relationship between daily salt intake and the night SBP is shown in Fig. 1.

Fig.1 Correlation between daily salt intake and the night SBP under a 7 g/day salt diet is significant (r = 0.662, p <0.01).**



The fluctuations under a 7 g/day salt diet were useful for calculating the correlation coefficient and the regression equation. Hence, r = 0.662** was statistically 1 % significant and its contribution rate was 0.438. The residual contribution rate 0.562 implies the existence of other factors as spiritual conditions, and environmental factors such as temperature, air pressure, and humidity under the salt diet.

The regression equation between the night SBP and daily salt intake was predictable for proper lower-salt diet and its equation was as follows:

$$y_1 = 8.08x_1 + 93.4 \cdots (1)$$

x_1 = daily salt intake (g/day)

y_1 = night SBP (mmHg)

Although the average of night SBP and its standard deviation were 148 mmHg and 18 mmHg respectively, as shown in Fig. 1, the residual standard deviation of the night SBP (above equation (1)) was only 8 mmHg. The coefficient value of x_1 might be the quantitative measure of salt sensitivity for a hypertensive patient in a 7 g/day salt diet under medication of nifedipine.

The r between the night SBP and daily salt intake of breakfast (av. salt intake; 1.5 ± 0.6 g), lunch (av. salt intake; 2.2 ± 0.9 g) and dinner (av. salt intake; 3.0 ± 0.8 g) for 56 days were respectively calculated. As a result, the r were all significant, 0.404** (p <0.01) for breakfast, 0.486** (p <0.01) for lunch and 0.347** (p <0.01) for dinner, respectively.

In order to evaluate salt influence from the compensated values (r divided by g of each meal's salt intake), the r/g were 0.269 (breakfast), 0.220 (lunch), and 0.115 (dinner), respectively. The effect of each meal's salt on the night SBP decreased in such an order. The aftermath of dinner's salt on the night SBP was least among the three meals in spite of the smallest elapsed time after ingesting.

Moreover, a well known fact of salt retention for 3-4 days had been common in Japan,[21, 22] but the author remained in doubt. In order to examine how many days the salt intake influenced on SBP, the correlation (r) between the night SBP and daily salt intake (three meals) during previous 4 days were 0.007 (non significant), 0.176 (n.s.), 0.020 (n.s.) and 0.148 (n.s.), respectively, and none of them were significant. These results indicate that the effect of daily salt intake was within ca.24 hours.

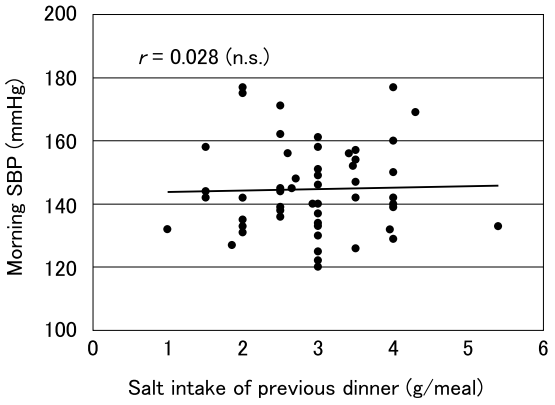
Effect of the previous day's salt intake on the morning SBP under a 7g/day salt diet

The morning SBP was measured before breakfast. Some influence from the previous day's salt on the morning SBP was expected because there was no relation between the morning SBP and the same day's salt intake. As a result of calculation, the r between the morning SBP and the salt intake of each meal of the previous day were 0.219 (p = ca.0.10) for breakfast, 0.242 (p <0.08) for lunch, and 0.028 (n.s.) for dinner,

respectively.

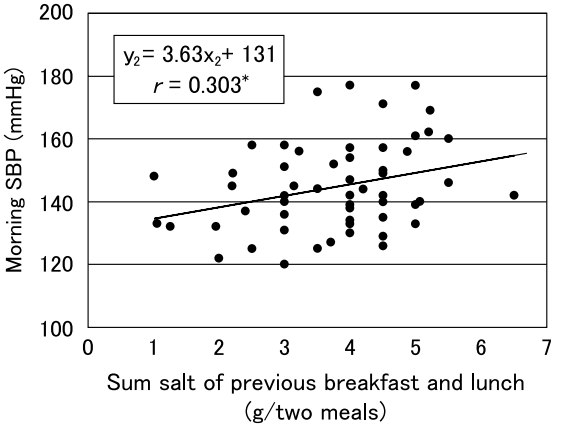
The morning SBP was hardly influenced by salt of the previous day's dinner as shown in Fig. 2.

Fig. 2 Correlation between salt intake of previous day's dinner and the morning SBP under a 7 g/day salt diet is not statistically significant.



The morning SBP was rather influenced by the sum salt of the previous day's lunch and breakfast, as shown in Fig. 3.

Fig. 3 Correlation between sum salt intake of previous day's breakfast and lunch and the morning SBP under a 7 g/day salt diet is significant (r = 0.303*, p <0.025).



The regression equation between the morning SBP and the sum of the salt intake of the previous day's breakfast and lunch was as follows:

$y_2 = 3.63x_2 + 131$ ・・・(2)

x_2 = sum of salt intake of the previous day's breakfast and lunch (g/two meals)

y_2 = morning SBP (mmHg)

The *r* between the morning SBP and the sum of the salt intake of the previous day's breakfast and lunch except dinner was 0.303*, which was statistically 2.5% significant. And it was smaller than the *r* 0.662** between the night SBP and daily salt intake. The regression equation (2) was used for prediction of a homeostatic level of the morning SBP under a 3.5 g/day salt diet.

As far as salt influence of previous days on the morning SBP was concerned, the *r* between the morning SBP and salt (two meals) of the previous 3 days were 0.303* (p <0.025), 0.014 (n.s.), and 0.054 (n.s.), respectively. Moreover, for further confirmation, the *r* between the morning SBP and daily salt (three meals) of the previous 3 days were respectively calculated, 0.254 (p <0.06), 0.083 (n.s.), and 0.002 (n.s.). Thus, the influence of salt intake was not continued over 24 hours.

Decreasing rate of the night SBP under a 3.5 g/day salt diet

As mentioned above, a 7 g/day salt intake was not enough to prevent reoccurrences of the second and the third brain infarction. So, according to the regression equation (1), a 3.5 g/day salt diet was calculated as appropriate salt intake corresponding to optimal SBP 120 mmHg[7] which was expected to prevent the fourth reoccurrence. Prior to the practice of the 3.5 g/day salt diet, the gradual reduction of the salt intake from 7 g/day via 5.5 g/day was performed as a precaution. Although a 5.5 g/day salt diet was executed after the third reoccurrence, the monthly average of night SBP was not smoothly reduced.

Under the 5.5 g/day salt diet from January to July in 1999, the monthly averages of daily salt intake, the monthly averages of the night SBP and the expected monthly average values of the night SBP calculated by the regression equation (1) are shown in Table 1, respectively.

Table 1. Monthly average of the night SBP under a 5.5 g/day salt diet. Significance probability concerning the difference between the night SBP and the expected value is shown in parenthesis.

Time	Daily salt Intake	Night SBP	Expected value
	Monthly av.	Monthly av.	Calculated by eq.(1)
	g/day	mmHg	mmHg
Jan-99	5.99 ± 0.95	148 ± 16 >	142 ± 8 (p <0.01)
Feb-99	5.56 ± 1.02	147 ± 13 >	138 ± 8 (p <0.01)
Mar-99	5.58 ± 0.75	144 ± 14 >	139 ± 8 (p <0.06)
Apr-99	4.88 ± 1.13	135 ± 14	133 ± 8 (p <0.18)
May-99	5.41 ± 1.13	132 ± 13 <	137 ± 8 (p <0.07)
Jun-99	5.27 ± 0.96	153 ± 14 >	136 ± 8 (p <0.001)
Jul-99	5.47 ± 0.83	149 ± 10 >	138 ± 8 (p <0.001)
av.	5.45 ± 0.97	144 ± 13	138 ± 8

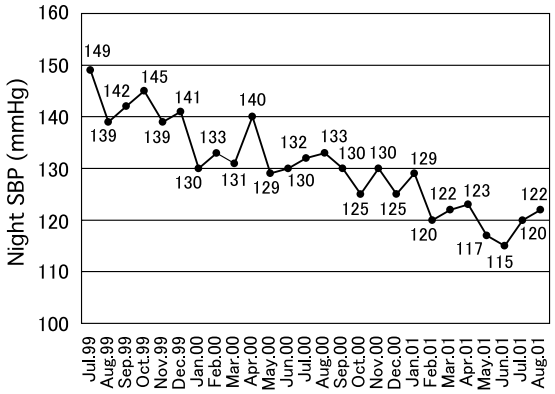
In spite of the salt reduction from 7 g/day to 5.5 g/day, the monthly averages of the night SBP were not always lowered. Compared with the expected monthly values calculated by regression equation (1), the monthly averages of the night SBP were unexpectedly higher. Though the monthly averages of April and May showed relatively lower, their averages were not statistically 5.0 % significant.

Hence, a 3.5 g/day salt diet was actually begun from August (exactly, 28th July, 1999), after irregular high blood pressure under 5.5 g/day salt diet appeared. Such increased resting high blood pressure[9-11] more dominantly emerged, in the case of the morning SBP as shown in the next section.

The reduction effect of a 3.5 g/day salt diet on the night SBP for 2 years is shown in Fig. 4.

The monthly averages of the night SBP under a 3.5g/day salt diet decreased rather slowly with a zigzag and, after 19 months, finally reached the homeostatic level of 120 mmHg (average of 7 months from Feb. to Aug. 2001), which met the expected SBP value calculated by the regression equation (1). As a result, the increased resting high SBP of the night was overcome under a sequential 3.5 g/day salt diet, even though it took longer than that of the morning.

Fig. 4 Reduction of the night SBP under a 3.5 g/day salt diet for two years. Numbers are the monthly averages of the night SBP. Monthly average of 7 months from Feb. to Aug. 2001 is 120 mmHg which is recognized as the homeostatic level.



Decreasing rate of the morning SBP under a 3.5 g/day salt diet

In order to avoid unexpected disorder prior to a 3.5 g/day salt diet, a 5.5 g/day salt diet from a 7 g/day was practiced as a cushion period. In case of the morning SBP, this diet was especially in vain due to the appearance of increased resting high SBP.

Each monthly average of the morning SBP under a 5.5 g/day salt diet became significantly higher (p <0.001), compared with the expected SBP values. The monthly averages of the sum salt of the previous day's breakfast and lunch, the monthly averages of the morning SBP and the expected monthly average values of the morning SBP under a 5.5 g/day salt diet calculated by the regression equation (2) are shown in Table 2.

In July, the monthly average of the morning SBP increased as high as 172 mmHg and, during a week (from 13th to 19th July, 1999), stayed at high as 180-190 mmHg. After the increased resting high SBP was unexpectedly appeared under a 5.5 g/day salt diet, three days (from 28th July) of a 3.5 g/day salt diet relieved serious malaise. And, the 3.5 g/day salt diet rapidly overcame the resting high blood pressure within 5 months (from Aug. to Dec. 1999).

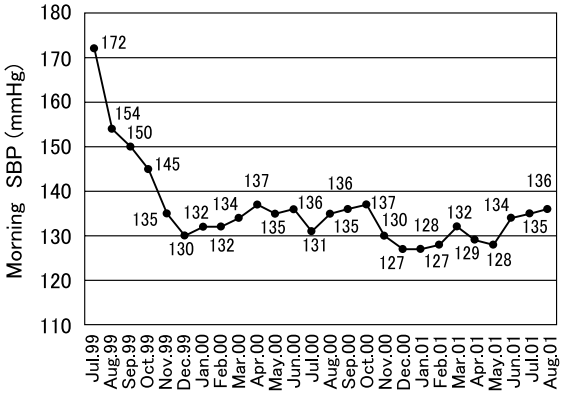
Table 2. Monthly average of the morning SBP under a 5.5 g/day salt diet. Difference between the morning SBP and the expected value is statistically significant as shown in parenthesis.

Time	Salt intake	Morning SBP	Expected value
	Monthly av.	Monthly av.	C alculated by eq.(2)
	g/previous day's two meals	mmHg	mmHg
Jan-99	3.57 ± 0.80	157 ± 14>	144 ± 14 (p <0.001)
Feb-99	3.16 ± 0.73	157 ± 13>	142 ± 14 (p <0.001)
Mar-99	3.33 ± 0.69	155 ± 11>	143 ± 14 (p <0.001)
Apr-99	2.85 ± 0.96	154 ± 11>	141 ± 14 (p <0.001)
May-99	3.31 ± 0.85	153 ± 14>	143 ± 14 (p <0.001)
Jun-99	3.43 ± 0.79	164 ± 14>	142 ± 14 (p <0.001)
Jul-99	3.46 ± 0.90	172 ± 12>	142 ± 14 (p <0.001)
av.	3.29 ± 0.82	159 ± 13	142 ± 14

The monthly averages of morning SBP were reduced faster month by month under a 3.5 g/day salt diet, as shown in Fig. 5.

After smooth decreasing of 5 months, the morning SBP reached a final homeostatic level of 132 mmHg (average of 21 months from Dec. 1999 to Aug. 2001), which was a little lower than the expected value 137 mmHg calculated by the regression equation (2). Although the difference between them was 5 mmHg, it was not statistically 5% significant. Moreover, the average of the sum of the salt intake of breakfast and lunch for 2 years under a 3.5 g/day salt diet was 1.92 g/day and it was used for calculation of the above expected blood pressure.

Fig. 5 Reduction of the morning SBP under a 3.5 g/day salt diet for two years. Numbers are monthly averages of the morning SBP. Monthly average of 21 months from Dec. 1999 to Aug. 2001 is 132 mmHg which is recognized as the homeostatic level.



Daily salt intake and salt secretion into urine during 24 hours

The salt excreted into urine in 24 hours was measured. The correlation between daily salt intake and salt in urine of 16 samples (selected from Mar. to July 2002 under a 3.5 g/day salt diet continued after the above 2-year experiment) was calculated as follows.

$y_3 = 1.07x_3 - 0.81$ ・・・ (3)

x_3 = daily salt intake (g/day)

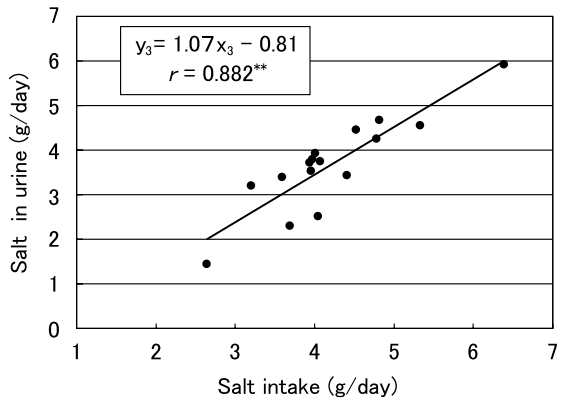
y_3 = excreted salt in urine (g/day)

The experimental result of the relationship between daily salt intake and excreted salt in urine is shown in Fig. 6.

The r was as high as 0.882**. When the salt intake was 3.5 g/day, the amount of salt in urine was 2.39 g/day (calculated by the regression equation (3)) which was 84% of daily salt intake.

His creatinine value was 0.98 ± 0.14 mg/dl (10 years' average, n=10). Renal activity was not dysfunctional.

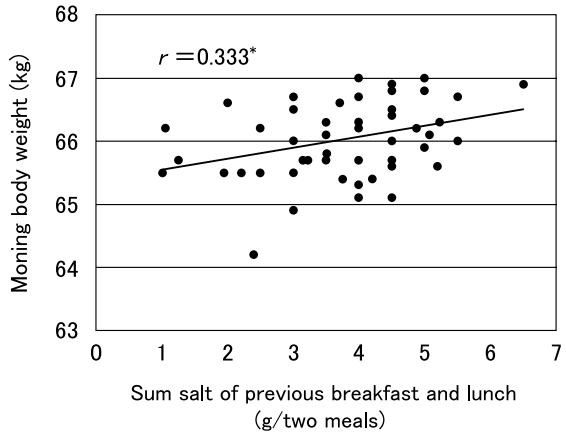
Fig. 6 Correlation between daily salt intake and salt into urine in 24 hours is highly significant (r = 0.882, p <0.01).**



Effect of the sum salt of the previous day's two meals (breakfast and lunch) on the morning body weight under salt diets of 7, 5.5, 3.5 g/day

Under a 7 g/day salt diet, the *r* between the sum salt of the previous day's two meals (breakfast and lunch) and the morning body weight (av. 66.0 kg, n = 56) was 0.333* (p <0.02), as shown in Fig.7.

Fig. 7 Correlation between sum salt of previous two meals (breakfast and lunch) and the morning body weight under 7g/day salt diet is significant (r = 0.333*, p <0.02).



Body weight corresponds to the volume of body fluid. The effect of the previous day's salt on the morning body weight supported the fact that the previous day's salt had an influence on the morning SBP, as shown in Fig. 3.

Under a 5.5 g/day salt diet (during Jan.-July 1999) shifted from 7 g/day salt diet, the monthly averages of the morning body weights unexpectedly did not decrease and rather increased by 0.2~0.5 kg (whose values were the subtraction of 66.0 kg from 66.2-66.5 kg). Daily drinking water increased by 1~3 cups of 200cc which were equivalent to ca.200 to 600 g/day, whose values corresponded to the above mentioned increase of body weights.

On the contrary, immediately after reducing salt diet to 3.5 g/day from 5.5 g/day, the monthly average of the morning body weight was 65.1 kg (in Aug. 1999) shifted from 66.2 kg (in July 1999). The difference of the morning body weight before and after 2 g salt reduction was 1.1 kg which was equivalent to 1.1 liter body fluid. At the same time, drinking water rapidly decreased ca.1,100 g/day (equivalent to ca.5~6 cups of 200cc), which corresponded to the decrease of the above mentioned body weight of 1.1 kg. Under a 3.5 g/day diet, night body weight was ca. 700 g/day heavier than morning body weight. Naturally, daily fluid volume in the morning might be the lowest.

Discussion

Even though there exists true physical blood pressure, instantly before and during measurement, the values of measured blood pressure have been already affected within seconds by baroreceptors, chemoreceptors, and central nerve system, as shown in the famous Guyton's Fig.1 diagram[17]. Hence, in this study, initially measured values were prospectively adopted.

Traditionally, in medical or epidemiological study, the salt in urine for 24 hours has been estimated as real salt intake. But in this research, the bias between both was ca.16%. The integrated salt is better measured from each food by using salt meter.

The relationship between daily salt intake and the night SBP was analyzed from the data of fluctuations under a 7 g/day salt diet for 56

days. The standard deviations of measured SBP generally ranged at ca.13 ± 5 mmHg so that the evaluation by a monthly average (n ≒ 30) was a necessary and sufficient condition according to small sample theory. The *r* 0.662** was significant and the regression equation was calculable to obtain the target level of salt diet corresponding to optimal SBP 120 mmHg.[7, 17] Though the statistically calculated value was 3.29 g/day, salt intake of 3.5 g/day was adopted considering feasibility.

In particular, a 3.5 g/day salt diet could overcome the increased resting high blood pressure that appeared during a 5.5 g/day salt diet. The underlying mechanism was not clear but a big difference in amount of drinking water between 5.5 and 3.5 g/day salt diet of 1.1 kg was remarkable. The 3.5 g/day salt diet showed such a rapid decrease in drinking water (ca.1.1 kg/day) accompanying 1.1 kg loss of body weight. This loss value agrees with the recommendation of 1 liter water supply for 2 g salt loss of sweating in sports medicine. The gain of fluid seemed to be a cause of the increased resting blood pressure. According to miscontrol of fluid volume and autoregulation of Guyton,[17, 18] the SBP increases by a rise in peripheral resistance.

Strangely, 5.5 g/day salt diet increased more body weight than 7 g/day salt diet. During this period, the phenomena of increased resting BP were observed and this unexpected increase of fluid volume seemed to be caused by misjudgement of BP control system.

In order to evaluate the effect of a 3.5 g/day salt diet, the monthly averages of the night and the morning SBP were compared. Under a 3.5 g/day salt diet, the monthly averages of the night SBP decreased with a zigzag change and, after 19 months, reached the homeostatic level of 120 mmHg which was accorded with the expected value calculated by the equation (1), as shown in Fig. 4.

On the contrary, the monthly averages of the morning SBP under a 3.5 g/day salt diet decreased rather smoothly month by month. After 5 months, it reached a homeostatic level of 132 mmHg whose reduction was less than the expected homeostatic level of 137 mmHg (n.s.) calculated by the equation (2), as shown in Fig. 5. The homeostatic level of the morning SBP was higher than that of the night SBP. The reason might be attributed to a rise in the activity of angiotensin Ⅱ[13] and aldosterone[13, 14] in the morning.

And, the faster reduction rate of the morning SBP, under a 3.5 g/day salt diet might be attributed to alleviation of many hormones in plasma to increase the blood pressure, for example, noradorenarine[5], angiotensin II[13], aldosterone[13], vasopressin[15], and ouabain like substances.[5, 16] The action of the nervous system to increase the blood pressure in the morning might be weakened by such ultra-low salt intake as 3.5 g/day.

The night SBP was influenced by the fluctuation due to the amount of water and salt through three meals and after the effect of nervous action during the daytime. These factors might explain zigzag and slower reduction of the night SBP.

Meanwhile, the morning SBP was influenced by the sum salt of the previous day's breakfast and lunch. Rather smooth reduction of the morning SBP with less zigzag could be explained by the lower influence (r = 0.303*) of previous day's salt with more time elapsed than the night SBP. And, the lowest fluid volume of morning might accelerate reduction rate of the morning SBP, since the morning body weight was ca. 700 g/day lighter than the night body weight.

As the above mentioned, different reduction rates and homeostatic levels concerning the mornig SBP and the night SBP were characteristic. These conspicuous differences suggest that the standard values of the morning SBP and the night SBP should be distinctively defined. Further, if daily salt intake was strictly controlled at 3.5 ± 0

g/day and each meal was ingested at punctual time, homeostatic levels of morning and night SBP could be more quickly reached.

It was clear that a 3.5 g/day salt diet could decrease the increased resting high SBP of morning and night, and both SBP finally reached different homeostatic levels. This could be explained by the fluid volume control and the autoregulation system, with what might be called daily settlement of morning and night SBP following each circadian rhythm.

Moreover, the morning SBP was hardly affected by the salt intake of the previous day's dinner as shown in Fig. 2. This seemed to be attributed to the lower activity of aldosterone at night. The recovery of salt by aldosterone in kidneys at night was weakened and salt intake at dinner hardly affected the morning SBP. Considering this situation, salty food should be ingested at dinner. Such recommendations have been independently reported.[19, 20)]

This research of statistical results indicated that the salt influence on the morning SBP and the night SBP respectively appears within 24 hours with significance, not within a few days as conventional thinking[21)] as widely known. In extreme case, the experimental data of salt reduction were reported to require 3-4 days to reach equilibrium level under constant salt intake.[22)]

Daily salt apparently settlemented within 24 hours. This fact was assumed to correspond to daily circadian rhythm of angiotensin II[13)] and aldosterone, etc.[13,14)] Under coexistence of various nonequilibrium reactions of living body, the salt metabolism seems to perform daily settlement following circadian rhythm, not within a few days.

The phenomena of salt influence of previous day's breakfast and lunch on the next morning SBP might be explained as the mechanism like subsurface current. That is to say, it is postulated that Na supplied from storage such as bones and water shifted from cells might control the blood volume in the morning or whenever necessary, since bone itself has a mechanism of sodium storage.[23)]

Incidentally, in order to perform the effective 3.5 g/day salt diet for long-term, further research might be required to pursue creating cooking design with consideration for both physical and chemical taste, if possible, with some alcoholic beverage.

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食塩感受性高血圧患者に対する低塩分ダイエットの効果
朝と夜の収縮期血圧の降下速度

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要　旨

降圧剤治療中の患者が、血圧を下げるため、減塩する時、減塩目標を合理的に決めたい。ここに、個人の、血圧と塩分摂取量との相関を知る必要がある。著者は、1994年10月に初回の脳梗塞で入院した。退院後、病院指示の 7 g/day の減塩を行った。しかし、7 g/day のダイエットでは、再発を防げず、1998 年 2 月と 10 月に脳梗塞を再発した。偶々、第二回の発病の 2 週間前までの、比較的平穏な 56 日間、毎食の塩分を実測し、記録した。発病を避けて、塩分と血圧との対応データが得られた。その相関係数は、0.662** で、回帰式は予測に使える。再発防止のため、理想血圧 120 mmHg に血圧を低下するには、3.5 g/day の減塩が必要と計算された。この塩分ダイエットで、血圧は予期レベル迄下がり、10年以上再発を防止出来た。朝血圧は、前日の朝と昼の食事の塩分の影響を受け、夕食の塩の影響を受けないので、夕食で塩分の多いものをとることが薦められる。朝、夜の血圧の低下速度の大差など興味ある知見を得た。

キーワード：高止り血圧、血圧降下速度、3.5 g/day塩分ダイエット、塩分と血圧の相関係数、血圧と塩分の回帰式

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