

CLINICAL MANAGEMENT

Potassium and Blood Pressure: How to Test the Effects of DASH Diet in your Patient with Hypertension?

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ABSTRACT

This article reviews the 90+ year history of increasing potassium intake (K) and lowering sodium (Na) intake in the treatment of hypertension (HTN). It then reviews the DASH Diet eating plan as an intervention to lower blood pressure (BP) by both increasing K intake and lowering Na intake. The term DASH Diet Sensitive (DDS) HTN is used to describe those whose BP decreases significantly when consuming the DASH Diet. A method to determine your patient's BP is outlined that has been found effective even in the most extreme form of salt-sensitive HTN—classic primary aldosteronism. This requires a series of home BP measurements before starting and during the 14 days of the DASH eating plan and checking a spot urine for Na/K/creatinine to monitor adherence. The beauty of this method is that if the patient follows the recommendations exactly, the maximum systolic BP effect is apparent by 1 week and the diastolic effect in 2 weeks. Thus, only 3 weeks is required to see if this is an effective intervention in your patient's HTN. If so, the next task is to determine if DASH is an eating plan that your patient (and family) can live with.

Keywords: Adherence, Blood pressure, Compliance, DASH diet, Home blood pressure, Nocturia, Nutrition, Potassium, Sodium, Urine Na/K ratio.

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A BRIEF HISTORY OF POTASSIUM, SODIUM, AND BLOOD PRESSURE

The role of dietary K in blood pressure (BP) regulation has a long history in humans and animals. The exact mechanism of the effect of K on BP is not known, but in general it seems to counteract the toxic effects of excess dietary Na on BP without affecting BP itself, yet increasing longevity in animals and humans. In 1928, Addison¹ reported that adding potassium chloride (KCl) to symptomatic hypertension patients (BP 170-262/84-152) lowered BP by

$\geq 30/\geq 12$ and improved not only BP but also the symptoms of severe HTN, especially edema. In 1931, Priddle² reported 11 patients with a systolic BP > 200 mm Hg whose BP fell an average of 64/24 over 1 to 18 weeks, which "was considered quite noteworthy." More pronounced was the improvement in the patient's clinical condition: insomnia, nervousness, dizziness, frequent headaches and shooting pains in the head have disappeared. A decrease of nycturia (nocturia) was constantly experienced by the patients as they improved. In cardiac failure with dyspnea and edema, compensation was quickly restored.... Patients who were missed from the clinic for some time, upon investigation stated that they felt so well they could see no need for returning.... It is believed it will be necessary for all cases with well established HTN to continue indefinitely their diet regulations (low sodium diet), and in many, the high intake of potassium as well.

It should be noted that although HTN is considered an asymptomatic condition, many patients today with drug-resistant HTN have many of these same symptoms which in my experience are relieved by the DASH eating plan. The most dramatic results of very low sodium (10 mM/day) and high-potassium diet were those published by Kempner³ using the rice-fruit diet in 1940s. In a series of patients with overt congestive heart failure (CHF) and malignant HTN, this regimen rapidly controlled BP and produced dramatic regression of left ventricular hypertrophy (by chest X-ray and electrocardiogram) and clearing of Grade IV retinopathy (retinal photograms) in only a few months. Even today, viewing this article's dramatic illustrations or improvement in patient status is amazing to view for students and fellows in HTN training.

Extensive studies in hypertensive rat models in 1957 by Meneely et al,⁴ in 1958 by Dahl⁵ and in 1985 by Tobian et al⁶ showed that increasing K intake to animals with salt-induced HTN reduced the damaging effects of a high-salt diet with marked reduction in mortality from stroke, CHF, and renal disease despite not much of an effect on the attained BP in many rat models of salt-induced HTN. The introduction of thiazide diuretics in 1957 seemed to lower interest in using modifications of diet in the management of HTN.

In 1962, Priddle⁷ reported on his team's 30-year experiences in using low Na, high K intake recently combined with the addition of a thiazide diuretic, which

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had revolutionized their ability to slow the progression of hypertensive cardiovascular disease due to HTN and then to control BP using chlorothiazide 250 mg 5 days a week, a 50 mM Na with an added 2 gm KCl. Many of their patients who had previously been difficult to control now had improved BP and their feeling of well-being returned. He noted that it was useful to monitor the urine Na/K ratio as a method of checking compliance with the regimen. The ratio of Na/K decreased from ~2.5 on the ordinary diet to ~2 on the Na-restricted diet and by adding a thiazide diuretic and K it decreased to 0.8. At the same time, a series of elegant balance studies published in JAMA in Fallis and Ford⁸ (Fig. 1) demonstrated that after equilibration on the 50 mM Na, 50 mM K diet, the addition of 50 mg hydrochlorothiazide (HCTZ) produced a significant fall in BP in only 7 days, likely related to the impressive natriuresis as BP was not affected when the Na intake was increased to 100 mM/day.

Figure 1 shows the effects of this regimen on BP, Na, and K balance from the run in control diet by the addition of HCTZ. Note the sudden jump in sodium excretion, no change in K balance, and the fall in mean arterial BP. Not shown are similar plots showing that increasing diet K to 100 mM enabled HCTZ to be increased to 100 mg/day

without significantly affecting serum K. In 1968, a similar program used by Priddle's team for up to 12 years reported a 50% reduction in mortality in elderly⁹ patients from the Metropolitan Toronto Homes for the Aged attending the Cardiovascular Clinic of the Geriatric Center for 4 years (LoNa, HiK, HTZ protocol) whose intake was validated by 24-hour urine collections. For unknown reasons, this information did not make it into general medical practice nor management of HTN guidelines. The association of a retirement population's Na/K intake and stroke was reported in a Southern California retirement community in 1987.¹⁰ The higher the diet Na/K ratio, the greater the stroke rate. Two more recent studies on K supplementation emphasize the ability of increasing K intake to lower BP. In 1991, a Kenyan double-blind randomized trial added 64 mM K/day K to 10 mg bendroflumethiazide¹¹ in 84 black patients with untreated HTN. K supplementation for 28 weeks led to a decrease in diastolic BP from 108 ± 3 to 88 ± 4 mm Hg, which was similar to those taking the thiazide. The authors concluded that this supports the notion that K supplementation may be an effective approach in mildly hypertensive blacks. Today we would not consider those with an entry BP average of 108 to be "mildly HTN." He et al¹² added supplemental KCl or citrate

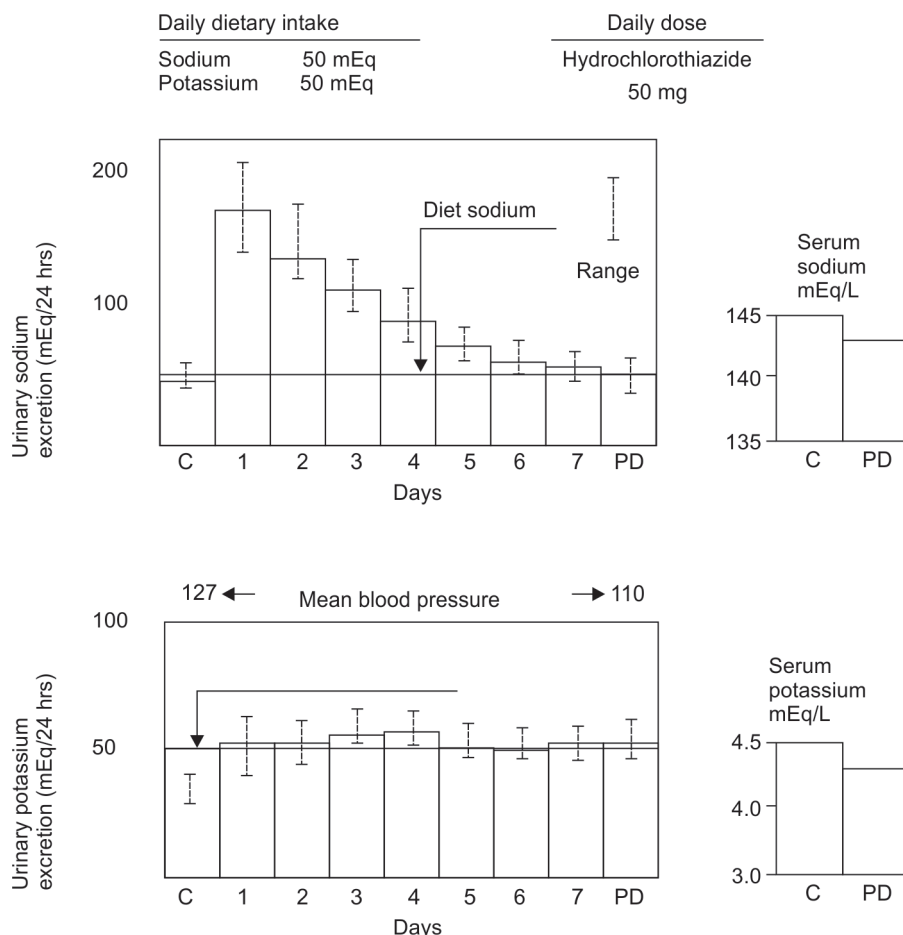


Fig. 1: Most desirable regimen, accompanied by least negative potassium balance

to increase urinary potassium to an average of ~160 mM/day and reduced BP by ~11/5 in only 1 week. Thus, increasing K intake and lowering Na is an effective and rapid nondrug method to try in patients who are interested in this approach.

A major advance in nutritional management of HTN was the publication of the DASH Diet studies which have included investigation of the effects of DASH on BP control systems. The best review of all aspects of the initial DASH program are contained in the supplement to the Journal of the American Dietetic Association in 1999.¹³ It is recommended reading for all who wish to gain a detailed understanding of DASH Diet Program. Most relevant to our discussion here is the observation that the components of the DASH Diet shift the “pressure natriuresis curve” so that BP is less affected by changes in sodium intake than when ingesting a standard American diet.¹⁴ Furthermore, the DASH eating plan improves the patient’s sense of well-being,¹⁵ which is unusual when one uses pharmacological interventions. Thus, the BP effects of increasing K intake (and lowering Na) in most with high BP are worth testing in all patients before implementing drug therapy. Those with drug-resistant HTN are also good candidates as one of the most common causes of drug resistance is due to high-salt and low-potassium intake. The next question is how to do this in one’s practice? I have used urinary monitoring of Na/K as discussed below to monitor compliance with dietary changes so you and the patient can better understand how they are doing in their new eating program.

Physiology of the Urine Na/K Ratio and its use in Monitoring a Low Na and High K Intake as on the DASH Diet

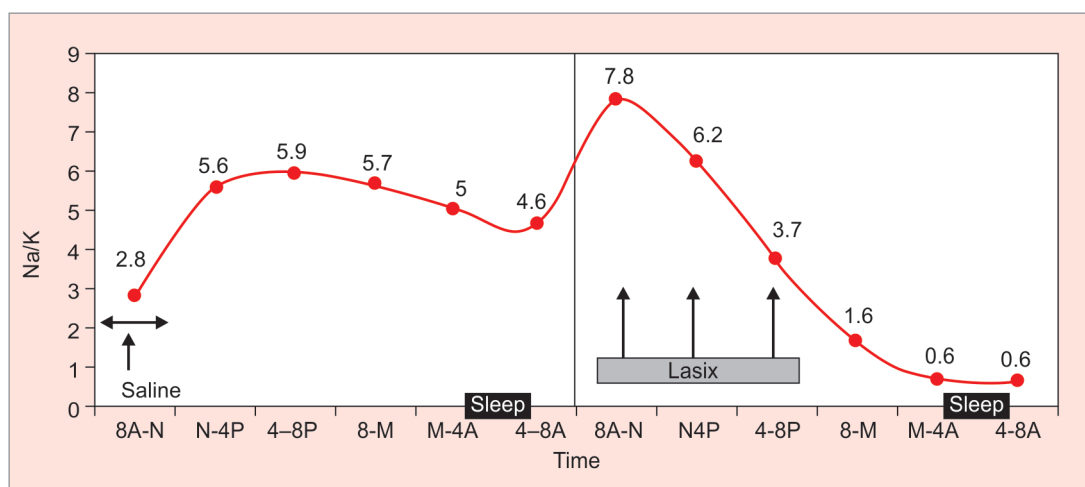
As noted earlier, the monitoring of Na/K in patients has been recommended since 1962⁷ to document compliance with high K, low Na diet.

The ability of the Na/K ratio to reflect Na and K balance under ever acute changes in sodium balance is shown in Graph 1.¹⁶ In normals, acute changes in sodium balance only lower the Na/K to <1 under severe sodium depletion. Note that the Na/K ratio in urine collected every 4 hours only reached <1 when sodium retention in the urine became extreme after sodium depletion with the Lasix. More extensive information on Na and K changes in normotensive and hypertensives with this protocol originally designed to quantitate the renin–angiotensin–aldosterone system and sodium metabolism is detailed elsewhere.¹⁷

As can be seen in Graph 1, the Na/K ratio in urine monitored every 4 hours increased acutely with saline (sodium) loading and only became <1 at the extreme Na depletion after 20 hours of Na loss due to the low Na intake (10 mM on “Lasix Day”) and the three doses of Lasix. Thus, an Na/K <1 is unlikely to occur in subjects even under acute changes in Na and K intake and output. Longer balance studies described below show the power of measuring the Na/K to assess daily Na and K intake in HTN.¹⁸

Using the “Sleep Urine” to Monitor Dietary Sodium Compliance

Luft et al¹⁹ have shown that a night-time collection, which I call the “sleep urine,” is the best to document dietary compliance for sodium intake. The “sleep urine” is that passed from the time of retiring to sleep until the first voiding in the morning on arising. Placing the collection bottle on the toilet before going to bed has been a useful way to improve the complete collection of this sleep sample. Using controlled metabolic conditions, Luft et al²⁰ demonstrated that only one sleep urine was needed to document (>85% probability) that a patient was on low-sodium diet (65 mM/day). Studies using the chloride test dipstick in hypertensives required two sleep urines to classify as being on a low Na intake (<60 mM/day).



Graph 1: Changes in urine Na/K ratio with acute changes in sodium balance in normals

Use of this feedback improved dietary compliance²⁰ and in hypertensives improved BP control and lowered need for medications.²¹ This can also be done by lab assay for Na or using a newer chloride dipstick available for home use.²² However, taking KCl as a supplement will affect the accuracy of this method²³ and thus needs to be included in the evaluation of the dipstick results. A spot urine is also acceptable at least for the Na/K ratio. If the Na/K in a urine is not <1 , a patient is not DASHing.

How to do the DASH in your Practice

All guidelines recommend “healthy lifestyle interventions” before starting pharmaceutical methods, but most physicians tell me their patients never seem to get any effect. I believe this is because most dietary Na and K interventions are not properly monitored with urine Na/K measurement to document adherence to the regimen. Thus, when patients state that they are following a prescribed low-sodium diet (<100 mg Na), 80% of those who provided a 24-hour urine were not on the prescribed intake.²⁴ My response to patients who fail to get a BP lowering effect from the DASH eating plan is to only accept failure if compliance is documented by checking the urine Na/K or as I say “Show me the pee!”

Taking Home Blood Pressure

As a baseline BP I recommend taking for at least 1 week before beginning the DASH. If on meds this should be taken in the AM before meds. After sitting for 5 minutes rest, the BP should be taken three times. Averaging the last two gives a more stable BP reading. This should be continued during the 2 weeks of the DASH testing. Daily readings are needed as some will get a very quick fall in BP on the DASH and may need to step down their other BP meds, if any. Plotting the daily average BP will quickly enable the patient and your staff to determine if the BP has fallen from the baseline over the next 2 weeks.

The DASH Diet for Hypertension Book

Since the paperback DASH diet book by Thomas Moore et al²⁵ came out in 1991 based on the low-sodium version of the DASH²⁶ Diet, I have used this to guide patients from all walks of life who are interested in trying to manage their BP by “nondrug” means or who were referred to me for drug-resistant HTN. Before retiring, I had the book stocked in the pharmacy or gift shop of the hospital where I was practicing. Now it is available as an eBook as noted in the citation. The results in those who adhere to the plan have been dramatic not only in BP reduction. The most striking effect has been seen in patients with classic hypokalemic primary aldosteronism with drug-resistant

HTN. The BP almost always falls to goal in 1 week (or markedly improves), the serum K has normalized in days, and the profound symptoms of hypokalemia mentioned earlier disappear by 2 weeks. Some who have been in a near bed-ridden state have quickly returned to their normal state of health and once again are able to exercise and resume their job responsibilities. For examples of the effect in these drug-resistant patients I recommend that one go to our Yahoo Group “Hyperaldosteronism” and read these patients’ stories going back over 10 years. Many of my patients have told me this book saved their lives and their jobs as their BP becomes controlled and their feeling of well-being returns – perhaps because they are able to stop many of their other BP meds that may be making them not feeling well. Adding the measurement of Na/K even in a spot urine helps give you and the patient feedback on their Na/K intake. Adding a urine Cr to the spot urine²⁰ enables one to use the formula developed by Mann to estimate the 24 hour intake as well. Sampling of the mid-day urine improved the prediction.

This DASH book first reviews the science behind the DASH Diet in easy to understand terms for most patients and then challenges them to a 14-day trial. It next reviews concisely the problem of HTN and then discusses how the DASH was tested. After reviewing the keys to moving to the DASH eating plan it provides the exact menu one must follow for 14 days to test the DASH’s effect on their BP (Chap 9) with the goal of Na $<1,500$ mg and $>4,700$ mg K/day. There are also a number of recipes that can be used once the DASH has been shown to lower their BP to maintain the program. As reported in the original DASH studies, many patients report that they feel better than they did before the DASH and many with HTN or primary aldosteronism can lower/stop their other BP meds under physician guidance. They can also observe that when they stray from the DASH the BP quickly rises again. Thus measuring BP at home gives quick feedback that they need to review what they are eating. Using an App that enables them to track diet Na and K intake (myfitnesspal) is also useful as a learning tool to alert them to indiscretions. Some worry about BP rising too during testing the DASH before starting BP meds. During the controlled low Na DASH studies a systolic BP of more than 170 mm Hg or a diastolic BP of more than 105 mm Hg was used to halt the DASH study if it occurred during the 6 weeks of the original studies. No patients had such an increase in BP during the low-sodium version of the DASH studies.

SUMMARY

- Adding potassium by pill or diet and lowering sodium by diet or doing both with the DASH Diet are useful interventions to improve BP.

- Compliance is easy to monitor to document a goal of <1,500 mg Na and >4,700 mg K by testing spot or the sleep urine for Na, K, and creatinine.
- Blood pressure lowering is quick and nearly maximum by 2 weeks.
- A method to test this in only 3 weeks is presented.

REFERENCES

1. Addison WL. The use of sodium chloride, potassium chloride, sodium bromide, and potassium bromide in cases of arterial hypertension which are amenable to potassium chloride. *Can Med Assoc J* 1928 Mar;18(3):281-285.
2. Priddle WW. Observations on the management of hypertension. *Can Med Assoc J* 1931 Jul;25(1):5-8.
3. Kempner W. Some effects of the rice diet treatment of kidney disease and hypertension. *Bull N Y Acad Med* 1946 Jul;22:358-370.
4. Meneely GR, Ball CO, Youmans JB. Chronic sodium chloride toxicity: the protective effect of added potassium chloride. *Ann Intern Med* 1957 Aug;47(2):263-273.
5. Dahl LK. Sodium intake of the American male: implications on the etiology of essential hypertension. *Am J Clin Nutr* 1958 Jan-Feb;6(1):1-7.
6. Tobian L, Lange J, Ulm K, Wold L, Iwai J. Potassium reduces cerebral hemorrhage and death rate in hypertensive rats, even when blood pressure is not lowered. *Hypertension* 1985 May-Jun;7(3 Pt 2):I110-I114.
7. Priddle WW. Hypertension – sodium and potassium studies. *Can Med Assoc J* 1962 Jan;86:1-9.
8. Fallis N, Ford RV. Electrolyte excretion and hypotensive response. *JAMA* 1961 May;176:581-584.
9. Priddle WW, Liu SF, Breithaupt DJ, Grant PG. Amelioration of high blood pressure in the elderly. *J Am Geriatr Soc* 1968 Aug;16(8):887-892.
10. Khaw KT, Barrett-Connor E. Dietary potassium and stroke-associated mortality. A 12-year prospective population study. *N Engl J Med* 1987 Jan;316(5):235-240.
11. Obel AO, Koech DK. Potassium supplementation versus bendrofluazide in mildly to moderately hypertensive Kenyans. *J Cardiovasc Pharmacol*. 1991;17:504-507.
12. He FJ, Markandu ND, Coltart R, Barron J, MacGregor GA. Effect of short-term supplementation of potassium chloride and potassium citrate on blood pressure in hypertensives. *Hypertension* 2005 Apr;45(4):571-574.
13. Harsha DW, Lin PH, Obarzanek E, Karanja NM, Moore TJ, Caballero B. Dietary approaches to stop hypertension: a summary of study results. DASH collaborative research group. *J Am Diet Assoc* 1999 Aug;99(8 Suppl):S35-S39.
14. Akita S, Sacks FM, Svetkey LP, Conlin PR, Kimura G; DASH-Sodium Trial Collaborative Research Group. Effects of the Dietary Approaches to Stop Hypertension (DASH) diet on the pressure-natriuresis relationship. *Hypertension*. 2003 Jul;42(1):8-13.
15. Plaisted CS, Lin PH, Ard JD, McClure ML, Svetkey LP. The effects of dietary patterns on quality of life: a substudy of the Dietary Approaches to Stop Hypertension trial. *J Am Diet Assoc* 1999 Aug;99(8 Suppl):S84-S89.
16. Grim CE, Robinson MR. Heritability of sodium and potassium excretion during the Saline-Lasix protocol in African American twins. Presented at Am Soc Hypertension May, 2011.
17. Luft FC, Weinberger MH, Grim CE, Fineberg NS. Effects of volume expansion and contraction on potassium homeostasis in normal and hypertensive humans. *J Am Coll Nutr* 1986;5(4):357-369.
18. Luft FC, Sloan RS, Fineberg NS, Free AH. The utility of overnight urine collections in assessing compliance with a low sodium intake diet. *JAMA* 1983 Apr;249(13):1764-1768.
19. Luft FC, Fineberg NS, Sloan RS. Overnight urine collections to estimate sodium intake. *Hypertension* 1982 Jul-Aug;4(4):494-498.
20. Luft FC, Sloan RS, Lang CL, Cohen SJ, Fineberg NS, Miller JZ, Weinberger MH. Influence of home monitoring on compliance with a reduced sodium intake diet. *Arch Intern Med* 1984 Oct;144(10):1963-1965.
21. Weinberger MH, Cohen SJ, Miller JZ, Luft FC, Grim CE, Fineberg NS. Dietary sodium restriction as adjunctive treatment of hypertension. *JAMA* 1988 May;259(17):2561-2565.
22. Mann SJ, Gerber LM. Estimation of 24-h sodium excretion from a spot urine sample using chloride and creatinine dipsticks. *Am J Hypertens* 2010 Jul;23(7):743-748.
23. Luft FC, Sloan RS, Fineberg NS. The effect of chloride-containing potassium supplements on chloride titrator estimates of dietary sodium intake. *J Am Coll Nutr* 1985;4(2):233-239.
24. Gerber LM, Mann SJ. Inaccuracy of self-reported low sodium diet. *Am J Hum Biol* 2012 Mar-Apr;24(2):189-191.
25. Moore, T.; Jenkins, M.; Svetkey, L.; Karanja, N.; Lin, PH. The DASH diet for hypertension. New York: Pocket Books; 2003.
26. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER 3rd, Simons-Morton DG, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med* 2001 Jan;344(1):3-10.