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SODIUM-RESTRICTED DIETS AND THE USE OF DIURETICS

Rationale, Complications, and Practical Aspects of Their Use

Committee on Sodium-Restricted Diets
Food and Nutrition Board
National Research Council

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INTRODUCTION

The popularity of sodium-restricted diets in managing patients with disorders of sodium and fluid balance, hypertension, or both has decreased substantially in the past two decades, largely because potent diuretic agents have become available. However, as the complications of diuretic therapy have become recognized and better understood, the use of sodium restriction in concert with the judicious use of diuretics has received increased attention. It now appears that combined use of sodium restriction and of an appropriate diuretic agent is usually the safest and most efficacious approach for treating diseases whose manifestations are related to retention of sodium.

This report, which is designed primarily for the clinical dietitian and public health nutritionist, provides fundamental information on the pharmacological and physiological principles that are involved in use of diuretics and sodium-restricted diets. It also contains a brief presentation on management of selected diseases. To achieve the appropriate balance between diuretic drugs and sodium restriction requires close cooperation between the physician and the dietitian or nutrition counselor. The physician and the dietitian must reach a compromise between judgments regarding the therapeutic necessity of a particular intake of dietary sodium and the reality of the patient's lifestyle. They must also recognize that recommending a sudden and drastic reduction in sodium intake, or an unrealistically low sodium intake, often results in self-deception on the part of both the physician and dietitian, in that the patient fails to follow the advice. Furthermore, a too drastic restriction of sodium intake concomitant with use of potent diuretics may lead to derangements of body fluid volume and composition and to other complications.

Rather than following the traditional diet manual approach, i.e., listing 200, 500, 1,000, and 2,000 mg sodium diets, emphasis in this report centers on planning the diet *with* the patient, taking into account the nature of the patient's health problem, nutritional status, food preferences, and various factors that influence food habits and knowledge of nutrition and health. The patient's understanding of such a mutually planned diet should be evaluated in terms of his willingness and ability to comply.

EXPLANATION OF TERMS

Sodium is an element that exists in food only in association with other elements. For example, in table salt, sodium is combined with chlorine as sodium chloride. Sodium also appears in food as sodium bicarbonate (baking soda) and sodium benzoate (a preservative).

Equal weights of different sodium compounds do not, of course, contain equal amounts of sodium. *Therefore, all references in this text pertain to sodium expressed in milligrams (mg) or milliequivalents (mEq).* One mEq of sodium is equal to its gram-atomic weight (in mg) divided by the valence (one). Therefore, 1 mEq of sodium is 23 mg. Thus, the sodium content of 5 g of sodium chloride is 1,965 mg, or 85.4 mEq, and the sodium content of cow's milk is approximately 500 mg/l, or 22 mEq/l.

Similarly, 1 mEq of potassium is 39 mg, and the potassium content of 5 g of potassium chloride is 2,615 mg, or 67.1 mEq. The potassium content of cow's milk is 1,365 mg, or 35 mEq/l.

NORMAL PHYSIOLOGY OF SODIUM AND POTASSIUM METABOLISM

Life evolved in the ocean, in an external medium so vast that concentrations of its dissolved constituents changed only slowly over millions of years. The ocean contained dissolved sodium, potassium, calcium, magnesium, chloride, bicarbonate, and phosphate, as well as other ions and substrates in concentrations that were compatible with living systems. These simple systems have little tolerance for fluctuations in osmolality or electrolyte concentrations, an intolerance that is evident in the complex species that subsequently evolved from primitive forms. The cells of multicellular organisms are protected from drastic changes in osmotic pressure by being bathed in a relatively small volume of fluid that has properties resembling those of the external environment that is provided by the primitive ocean. Control systems in mammals maintain the internal medium within tolerable limits despite wide fluctuations in external conditions and dietary intake. At the functional center of the system is the kidney.

FUNCTION OF THE KIDNEY

Each of the two kidneys is composed of over a million minute tubules called nephrons (Guyton, 1976). Each nephron begins at the glomerulus, a tuft of capillaries that is highly permeable to the nonprotein component of the plasma. The pressure of the blood within the capillaries causes fluid to leak from plasma into the tubule. In adult humans, approximately 125 ml of this fluid, which contains the same concentrations of electrolytes as plasma, is filtered from blood each minute (about 180 l/day). As the filtrate passes down the tubules, nearly all of it is reabsorbed back into the blood, so that ordinarily only about 1 ml

of the 125 ml of filtrate that is formed each minute leaves the kidneys as urine. Depending on the body's needs, the daily volume of urine can be reduced to as little as a few hundred milliliters or increased to as much as several liters. The capacity of the kidney to vary the rate of loss of water and electrolytes and to vary the rate of reabsorption from the tubules allows for control of the concentration and volume of the internal fluid medium.

Several hormones, e.g., aldosterone (secreted by the adrenal glands), angiotensin II (a peptide created by the action of the enzyme, renin, which is released from the kidney), and antidiuretic hormone (ADH, which is secreted by the posterior lobe of the pituitary gland), as well as alterations in arterial blood pressure, modulate the function of the kidney.

MODULATION OF RENAL FUNCTION

- *Angiotensin II* is a small peptide that is formed by the action of renin on one of the plasma proteins (Laragh and Sealey, 1973). Renin is formed by the juxtaglomerular apparatus of the kidney and is released into blood by sympathetic stimulation, by decreased renal arterial perfusion pressure, or by a change in delivery of tubular fluid to the distal nephron when there is a decrease in blood volume or in blood pressure. Once in the circulating blood, renin immediately acts on its protein substrate to form angiotensin I, which is then converted to angiotensin II. Angiotensin II may alter sodium excretion by affecting renal blood vessels and by stimulating the adrenal cortex to release aldosterone.
- *Aldosterone* is an adrenal cortical hormone that travels through the bloodstream from the adrenal zona glomerulosa to the kidneys. Its effect is to stimulate reabsorption of sodium from tubular fluid back into blood and to stimulate secretion of potassium into tubular fluid (Laragh and Sealey, 1973). Therefore, aldosterone simultaneously acts to conserve sodium and to eliminate potassium from the body. Aldosterone release is stimulated by an increase in body potassium as well as by angiotensin II.
- *Antidiuretic hormone (ADH)* is a peptide hormone that is released from the posterior lobe of the pituitary gland in response to an increase in plasma osmolality or to a decrease in blood pressure or body fluid volume (Hays and Levine, 1976). Its effect on the kidneys is to promote reabsorption of water into the blood. As the level of ADH

increases, the concentration of the urine also increases. Consequently, more water will be conserved by the body.

- *Arterial blood pressure* and *intrarenal distribution* of blood flow may also be important determinants of renal excretion of fluids and electrolytes (Stein, 1976). Small increases or decreases in pressure in renal arteries and capillaries produce similar increases or decreases in rates of excretion of electrolytes and water. Renal effects of pressure change result from alterations in the volume of fluid that is filtered by the glomeruli into the tubules and in tubular reabsorption of filtered fluid.

REGULATION OF WATER AND ELECTROLYTE INTAKE

In addition to controlling rates of loss of water and salt from the body, the fluid and electrolyte control system, under some circumstances, also regulates intake of these substances. Certain areas of the brain, especially of the hypothalamus, exert some control over the desire to drink fluids and to consume salt (Hays and Levine, 1976). At the present time, knowledge of mechanisms is incomplete, although it has been shown that an increase in body fluid osmolality or decreased body fluid or blood volume are associated with increased thirst. In addition, salt appetite and drinking may be stimulated by angiotensin II.

CONTROL OF SODIUM, POTASSIUM, AND FLUID VOLUME BY INTERACTIONS OF THE COMPONENTS OF THE SYSTEM

Acting alone or in combination, components of the control system maintain the internal fluid environment at the level of constancy that is required for efficient cellular function. A better understanding of the operation of the system can be obtained by considering its responses to several types of challenges.

- *An increase in sodium intake*, such as that which results from the ingestion of a large quantity of salted food (potato chips, pretzels, ham, etc.), tends to increase sodium chloride concentration and the osmolality of extracellular fluids. As the ingested sodium chloride enters blood, the system responds through corrective changes. Initially, a slight increase in osmolality stimulates the thirst center of the brain, thereby evoking a sensation of thirst. Water intake increases and continues until enough

water has entered the body to dilute body fluid to a desirable level, or set-point, of the control system. At that time, extracellular fluid sodium concentration and osmolality will return to normal, although the total fluid volume may be greater than normal. As a response to this deviation from the fluid volume set point, a number of renal readjustments--such as a decrease in renin release, in angiotensin II formation, and in aldosterone secretion--occur that increase the filtered load of sodium chloride and water and decrease tubular reabsorption. These changes in renal function will lead to increased loss of sodium and water into urine. This continues until fluid volume returns to normal.

If available drinking water is insufficient to dilute ingested sodium chloride, the persisting high osmolality of the body fluid will be corrected as much as possible by the effects of a high level of ADH. ADH will stimulate tubular reabsorption of sodium-free water and allow ingested sodium to be excreted, with only minimal water loss, until body fluid osmolality returns to the desired level.

- *A prolonged low-sodium diet* that is properly planned to meet other nutritional needs can be tolerated with few deleterious effects because the system is capable of limiting sodium loss. If daily sodium intake suddenly decreases, urinary sodium loss will exceed intake for a day or two, until negative sodium and fluid balances increase renal reabsorption and curtail loss. Mechanisms that are responsible for the decreased renal excretion include an increase in renin release, angiotensin II generation, and stimulation of aldosterone secretion. These sodium-retaining measures will continue as long as the fluid volume remains contracted. In the event of extreme sodium deprivation, sodium loss may decrease to a few milligrams per day. Throughout a period of low intake, plasma sodium concentration and osmolality will be controlled near the set-point by ADH and thirst.

- *The system's response to a large increase in potassium intake* begins as soon as the body and adrenal content of potassium is elevated by dietary intake. As a consequence, the rate of secretion of aldosterone is stimulated, and secretion of potassium into tubular fluid and loss in urine is increased. Aldosterone secretion continues at above normal levels for as long as the potassium content of the body is elevated above the set-point.

SODIUM AND POTASSIUM INTAKE AND EXCRETION

Virtually all of the sodium and potassium that is ingested by a healthy person is absorbed from the gut. In adults, the amount of sodium and potassium that is excreted daily (through urine, sweat, feces, etc.) normally equals intake, so that a state of sodium and potassium balance exists. Infants, growing children, and pregnant women are in slightly positive balance with respect to these two ions because of gain in tissue, which reflects increments in both extracellular (sodium) and intracellular (potassium) volumes.

Human milk contains 161 mg (7 mEq) of sodium and 507 mg (13 mEq) of potassium per liter. These levels are low compared to milk from other sources and formulas but are appropriate for the healthy growing infant. Average daily intakes of sodium by infants in the United States vary from 690 mg (30 mEq) at age 2 months to about 1,380 mg (60 mEq) at age 12 months (Committee on Nutrition, American Academy of Pediatrics, 1974). Intakes of potassium vary from 780 mg (20 mEq) at age 2 months to about 1,560 mg (40 mEq) at the end of the first year. These figures reflect the use of formulas and cow's milk, plus a variety of solid foods. For adults in the United States, customary daily intakes of sodium are 2,300-6,900 mg (100-300 mEq), which is equivalent to 6-18 g of sodium chloride. Intakes of potassium vary from 1,950 to 5,850 mg (50-150 mEq). These intakes are considerably in excess of minimum needs.

DISTRIBUTION OF SODIUM AND POTASSIUM

Body water comprises about 60 percent of body mass. Approximately one-third of this water is extracellular and two-thirds is intracellular. Sodium is the principal cation in extracellular fluid and is involved primarily with maintenance of osmotic equilibrium and body fluid volume. Potassium is the principal cation in intracellular water. Indeed, nearly 80 percent of total body potassium is in intracellular fluid.

FACTORS BEARING ON SALT PREFERENCE

Under ordinary circumstances, modern man will consume large amounts of table salt whenever it is freely available. There has been considerable debate over whether appetite for salt is innate or learned. Certainly, ability to *taste* salt is innate to all mammals. In free-living animals, the bulk of evidence suggests that salt hunger is more characteristic of herbivores than of carnivores (Denton, 1969). The generally accepted view is that carnivores obtain adequate sodium from eating the flesh of animals, whereas herbivores subsist solely on plants that are low in sodium. However, it has been proposed recently that the high ratio of potassium to sodium in plants may be as important as the low sodium content *per se* (Mitchell, 1978), suggesting that high potassium intake may trigger a physiological need for sodium.

There are many accounts of salt hunger in wild animals (Bott *et al.*, 1967). For example, wild herbivores (such as rabbits, kangaroos, elk, and deer) often display what is described as "appetitive behavior" (random exploratory movements), which ceases when they encounter a source of salt. This behavior is well known to hunters, who use salt licks as a means of luring animals from the forest. Blair-West *et al.* (1968) have suggested that salt hunger in wild animals is due to sodium deficiency, on the basis that avid salt appetite of animals in alpine regions of Australia correlates with high blood levels of aldosterone and low urinary excretion of sodium.

Laboratory animals also show a preference for salt. Young rats nourished exclusively on mother's milk consistently demonstrated an active appetite for salt when first given access to salt solutions (Richter, 1956). The most preferred salt solution was 0.9 to 1.0 percent, which is isotonic to blood. Similarly, Pfaffmann (1963) observed

that normal rats that have been well supplied with salt show a preference for isotonic and hypotonic solutions over distilled water, but an aversion to hypertonic solutions. Pike and Yao (1971) have demonstrated an increased appetite for salt in pregnant rats. Rats fed low sodium diets and given access to distilled water and saline solution drank sufficient amounts of saline to prevent the biochemical, histological, and histochemical effects that were observed previously in sodium-deficient pregnant rats.

The literature abounds with reference to salt craving in man. Man has fought wars for the possession of salt (Eskew, 1948; Richter, 1956), and cultures have been organized about salt. Gandhi (1930) and more recently Multhauf (1978) have written accounts of the historical role played of salt.

There is some evidence that salt hunger derives from an innate neural organization that generates a behavioral drive in the presence of sodium deficiency (Denton, 1965). Moreover, neural pathways for taste are connected to pathways associated with feeding (Morgane, 1969; Pfaffmann *et al.*, 1977). As an example, feral men, such as the Chimbu, whose vegetarian diet is very low in sodium, have mean peripheral blood levels of aldosterone that are four times greater than those of New Guineans and Australians on a Western diet (Denton *et al.*, 1969). In addition, patients with adrenal deficiency experience acute craving for salt and salty foods (Richter, 1956).

However, most individuals consume salt well in excess of metabolic need. It is not known whether individual consumption of salt is due to the innate neural organization, to learned eating behavior, or to both.

Maller and Desor (1973) found that the volumes of salt solutions (concentrations from 0.003 *M* to 0.2 *M*) consumed by newborns during 3-minute periods did not differ significantly from the volume of water consumed. On the basis of studies of differential sucking response, Jensen (1932) concluded that within the first 2 weeks of life, infants could differentiate between salt concentrations of 0.30 and 0.45 percent. However, differential sucking occurred 95 percent of the time without marked facial expressions, and facial expressions of disgust seldom appeared. Steiner (1973) has also studied the gustofacial response in newborns and concludes that the application of salty substances does not elicit typical or easily classifiable responses.

A study of normal 4-month-old and 7-month-old infants failed to demonstrate that consumption of strained foods

was influenced by presence or absence of added sodium chloride (Foman *et al.*, 1970). Filer (1972) has suggested that studies of this kind mean little with respect to food preference because the infant is the passive recipient in the interaction between mother and infant, i.e., the mother's perception of adequate salt flavor may override any infant taste preferences. However, Reynolds and Filer (1965) have observed that when preschool children were given a choice of beef stew with or without salt, they selected the salted stew more frequently.

Does expsoure of the infant and the young child to salty foods bring about some degree of behavioral imprinting, so that in adolescence and adult life sodium intake will persist at higher levels? Although the answer to this question is not known, available evidence suggests that between 1930 and 1970 sodium intake of preschool children increased some 50 percent, whereas potassium intake remained virtually constant. This conclusion derives from data on urinary sodium and potassium (mEq/g creatinine) reported by Macy (1951) and found in the Preschool Nutrition Survey (PNS), which was conducted in 1968-70 (Owen *et al.*, 1974).

Urinary Constituent	mEq/g Creatinine	
	Macy Study	PNS
Sodium	180	260
Potassium	116	116

It is not clear to what extent a change in amount of salt that is incorporated into the food supply is responsible for the apparently greater intake of sodium by preschool children in late 1960's as compared with those in the 1930's, nor is it clear that the higher intakes are a reflection of greater preference of salt.

In a study by Desor *et al.* (1975), children 9-15 years of age and adults were given salt solutions of 0.29, 0.58, 1.17, and 2.34 percent. The majority of the subjects preferred the 0.29 percent salt solution. However, approximately 20 percent selected the saltiest (2.34 percent) solution. When data on the age-groups were evaluated according to race, it became apparent that significantly more blacks than whites in the 9-15 group preferred stronger concentrations of salt. Fully 30 percent of the black children preferred the 2.34 percent solution. Black adults tended to select stronger solutions than did white adults, although the results were not statistically significant. The basis for this preference by blacks is unknown.

Salt is often used by the elderly to compensate for sensory losses. The senses of taste and smell tend to decline with increasing age (Schiffman, 1977). The elderly complain significantly more frequently of weakness, sourness, bitterness, and dryness of food than do young persons (Cohen and Gitman, 1959; Schiffman, 1977). Complaints of bitterness often concern foods (such as green pepper) that have a pleasant odor but a bitter taste. As olfactory sensitivity becomes relatively impaired with age, the bitter taste becomes comparatively more pronounced. Salt is often used to compensate for these losses of taste and smell acuity. Thus, salt restriction may be more troublesome for aging persons for whom sensory losses can lead to loss of appetite.

To a limited extent, simulated flavors of various individual foods are now available on the commercial market. These flavors have been tested by Schiffman (in press) and have been shown to improve taste and olfactory acuity in the elderly. It is doubtful that at this time the use of simulated flavors is practicable for elderly individuals living alone or in any free-living situation. However, the enhancement of food flavor by the simulated flavors could have application in institutional feeding (such as in nursing homes), where poor appetite may contribute to deterioration of physical health. Moreover, the potential of simulated food flavors for use by the elderly who are on sodium-restricted diets appears to be a fruitful area for further investigation.

MANAGEMENT OF DISEASE STATES WITH DIET AND DIURETICS

Sodium restriction alone is not always effective, or without risk, in the disease conditions for which it has been advocated. Clearly, simultaneous use of sodium restriction and potent diuretics increases not only effectiveness but also increases likelihood of untoward effects.

As discussed earlier, the body has homeostatic mechanisms that maintain sodium and other ionic concentrations at normal levels. Normally, sodium that is ingested in excess of body need is excreted in urine. Diminished urinary excretion of both sodium and water as a consequence of disease states results in fluid retention, and edema (increased quantity of interstitial fluid) ensues. If sodium intake is restricted, excess fluid may be excreted in urine because water cannot be retained in the absence of sodium.

Clinical management of patients who evidence sodium and fluid retention has been greatly facilitated in the past two decades by availability of the newer diuretic agents. Some of these drugs are very potent and have often been used alone, i.e., with little attention given to dietary management. In view of the complications attending use of the more potent diuretics and of evidence that sodium restriction is effective in certain circumstances, it seems desirable to combine the two treatment modalities.

TYPES OF DIURETICS

- *Thiazide diuretics* Modern diuretic therapy began with introduction of benzothiadiazide diuretics (thiazides), which depress reabsorption of sodium and potassium in the renal tubule. Their potent effect on sodium excretion stimulates aldosterone secretion, which in turn further

augments excretion of potassium in urine. Therefore, some depletion of total body potassium and development of hypokalemia occurs in a substantial proportion of patients placed on continuing thiazide treatment. Depletion of body potassium may occur when moderate depletion of sodium resulting from excessive restriction of sodium intake coincides with chronic use of thiazides. Although thiazides are not commonly used in treating infants with mild to moderate edema, care must be taken to avoid simultaneous excessive restriction of sodium and use of thiazides (or other diuretics), because sodium depletion can occur rather promptly in infants. Modest elevation of serum uric acid is common, and there are occasional instances of glucose intolerance accompanying use of thiazides.

- *"Loop" diuretics* Two drugs--furosemide and ethacrynic acid--are even more potent than thiazides. Their special value lies in treating patients with markedly reduced renal function and in managing other situations where intense sodium retention occurs. As in the case of thiazides, these diuretics can produce hypokalemia and hyperuricemia. Their direct effect on glucose tolerance is questionable, but the hypokalemia that accompanies their use may reduce insulin secretion.
- *Diuretics causing potassium retention* Two types of diuretics cause potassium retention. One, presently exemplified by spironolactone, acts to block competitively the action of aldosterone and, therefore, is inactive in the absence of aldosterone. The other, represented by triamterine, blocks an aldosterone-independent component of sodium/potassium exchange in the renal tubule. Both drugs find their major role in limiting potassium loss produced by thiazide or "loop" diuretics. The use of spironolactone and triamterine is contraindicated in management of patients with renal failure and hyperkalemia. In any case, the level of potassium in the diet must be closely monitored if these drugs are used. Life-threatening hyperkalemia may occur when a high potassium intake and potassium-sparing diuretics are combined.

CONGESTIVE HEART FAILURE

The central role of sodium retention in the pathophysiology of congestive heart failure has been known for many years and has attuned physicians to the importance of sodium restriction in treating this syndrome. Before the advent of modern diuretics, moderate-to-marked sodium restriction

was necessary in practically all patients with congestive heart failure. Modern diuretics have lessened, but not removed, the need for sodium restriction. A diet containing 1,600-2,300 mg (70-100 mEq) of sodium daily will often be tolerated in patients receiving both digitalis and thiazides. If patients continue to show signs of congestive heart failure and are presumed to be following a diet with mild-to-moderate sodium restriction, the physician and dietitian should investigate if actual intakes are greatly in excess of the prescribed amount before increasing the dose of diuretic or restricting sodium more stringently. In hospitalized adults with very severe heart failure, it may be necessary to restrict dietary sodium to levels as low as 200-300 mg (9-13 mEq) daily, even when more potent "loop" diuretics are being given.

Intakes of sodium in excess of 230 mg (10 mEq/day)--2-3 times the requirement--seem undesirable for infants with congenital heart disease who develop congestive failure. Therefore, considerable care should be taken in the selection of milk or formula and of solid foods to provide sufficient energy intake, and in the provision of essential nutrients and water to meet infants' needs without giving them excessive sodium. As noted above, young infants are particularly vulnerable to sodium and potassium depletion from injudicious use of diuretics in addition to excessive sodium restriction.

HYPERTENSION

- *Restriction of sodium as sole therapy* Several very careful studies of severely hypertensive patients indicate that sodium must be restricted to approximately 200 mg (8.7 mEq) daily before a significant antihypertensive effect can be demonstrated (Kempner, 1948). More recently, Parijs *et al.* (1973) has shown that a 1,600 mg (70 mEq) sodium diet produces 5 mm mercury pressure drop in mild-to-moderately hypertensive patients, and that there is a significant positive correlation between blood pressure drop brought about by thiazide diuretic treatment and sodium deprivation (Parijs *et al.*, 1973). The minimal decrease in blood pressure accompanying modest dietary sodium restriction argues against use of sodium restriction alone as the primary therapy for most patients with mild essential hypertension. However, it is conceivable that an occasional patient, habitually ingesting large amounts of sodium, might show an adequate drop in blood

pressure as a consequence of sodium restriction alone. Although data from the older clinical literature, from epidemiological studies, and from experiments on rats suggest that potassium supplementation would further increase the antihypertensive effect of sodium deprivation, there is little clinical evidence attesting to the value of such an approach. Much more work is urgently needed on the significance of sodium:potassium ratio in hypertension.

- *Sodium restriction and antihypertensive drug therapy* The central approach to treating hypertension is to use diuretic drugs, especially of the thiazide type, as a means of reducing body sodium. It has been suggested that the antihypertensive effect of diuretics may be blocked by ingestion of increased amounts of sodium (Fallis and Ford, 1961; Langford *et al.*, 1977). The prudent course is to advise adult patients to restrict sodium intake modestly and to aim for 1,600-2,300 mg (70-100 mEq) per day.

The initially brisk diuresis that accompanies the use of ethacrynic acid or furosemide declines rather rapidly. Whether this is due to a decrease in glomerular filtration or to increased secretion of aldosterone caused by the hypovolemia is uncertain. Plasma renin activity has been shown to increase following administration of these drugs (Freis, 1976). Several other antihypertensive drugs (clonidine, propranolol, methyldopa) reduce renin secretion. However, this direct effect on renin secretion will be counteracted by a decrease in sodium excretion and an increase in renin secretion. In occasional patients the sodium retaining action of the compensatory mechanisms vitiates the original negative sodium and water balance and brings about a return of hypertension. However, this response is unusual.

- *Potassium supplementation* As is true of thiazide diuretics, furosemide and ethacrynic acid increase potassium excretion. Depletion of body potassium, leading at times to appreciable hypokalemia, is a complication of diuretic therapy, although its relative importance has been somewhat overemphasized. Nevertheless, ingestion of a high-potassium diet is an important aspect of dietary therapy in diuretic-treated hypertensive individuals. There are no valid data to indicate just how high the potassium intake should be, but a diet providing 3,900 mg (100 mEq) daily is a reasonable goal in treating adults. Supplements of potassium (as potassium chloride) may be needed to achieve this goal, as dietary means alone may not be adequate.

SODIUM RESTRICTION IN PREVENTION OF HYPERTENSION

A number of isolated populations that have been described show little increase in average blood pressure with age and have an infrequent occurrence of hypertension (Freis, 1976). Adults in some of these societies may regularly ingest less than 225 mg (10 mEq) of sodium per day and 7,800-11,700 g (200-300 mEq) of potassium per day. As noted above, Parijs *et al.* (1973) found that modest antihypertensive effects result from restricting sodium intake to 1,600 mg (70 mEq) per day as sole therapy. Therefore, it seems quite possible that persons with a pronounced family history of hypertension, or with borderline blood pressure elevation, should establish intakes less than 1,600-2,300 mg (70-100 mEq) sodium per day. It can be postulated also that increased potassium intake would aid in preventing hypertension. However, it must be noted that there are no well-controlled clinical studies to support the efficacy of these maneuvers, rational as they may seem.

Under ordinary circumstances, there is no known physiological benefit associated with high intakes of sodium. For the 20 percent of children who may be inclined to develop hypertension in adult life, it seems reasonable to control sodium intake during infancy and childhood (Committee on Nutrition, American Academy of Pediatrics, 1974). There are data indicating that normotensive adults have lower urinary sodium excretions (and presumably lower intakes of sodium) than do mildly hypertensive adults (Morgan *et al.*, 1975). Although Langford and Watson (1975) found no association between levels of urinary sodium and blood pressure among young children, they noted that the sodium: potassium ratio in 6-day urine collections correlated positively with diastolic blood pressure concurrently observed over an 8-day period. Meneely and Battarbee (1976) suggest that there may be individuals who are prone to hypertension because of "sensitivity" to sodium in the diet, but they may not develop hypertension because potassium intake is high enough to exert a protective effect.

CHRONIC LIVER DISEASE

Sodium intake and retention are important factors in the occurrence and treatment of ascites in patients with cirrhosis of the liver. Patients with cirrhosis and ascites excrete very little sodium in urine. Increase in ascitic

fluid is directly proportional to sodium intake and inversely related to sodium excretion (Davidson, 1970).

The mechanisms of sodium retention in cirrhotic patients are incompletely defined (Conn, 1972). Abnormalities thus far implicated include enhanced distal (convoluted) tubular sodium reabsorption, resulting from increased plasma aldosterone activity (Chaimovitz *et al.*, 1972). In some individuals it appears that proximal tubular sodium reabsorption may also be enhanced by decreased perfusion of the renal cortex or by the action of certain humoral factors (Papper and Saxon, 1959).

In the past, extreme dietary restriction of 200 mg/day (8.7 mEq) of sodium for the adult has been the mainstay of conservative management; it is more effective in preventing than in ameliorating ascites. Today, judicious use of diuretics is recommended as a concomitant of restricted dietary sodium (Baldus, 1972). Inclusion of diuretics in the management of such patients has the advantage of promoting elimination of sodium and fluid already retained. Overuse of diuretics can result in severe electrolyte disorders, oliguria, and death. Obviously, they must be used cautiously. Shorter-acting diuretics are preferable to long-acting ones, and kaliuretic agents (thiazides and "loop" diuretics) should not be used alone, but rather combined with a nonkaliuretic, naturetic drug (Sherlock, 1975). In general, patients are first put under a regime of severe sodium restriction [200 mg/day (8.7 mEq)], with supplements of potassium chloride and possibly magnesium acetate to replace the deficits in these two cations that may occur in association with cirrhosis. If diuresis does not occur, a nonkaliuretic, naturetic agent may then be given. Inhibitors of aldosterone, such as spironolactone, have proven very useful for this purpose (Conn, 1972).

Dose of diuretic and restriction of sodium should be adjusted to the functional response of the patient. If diuresis has not commenced 8 to 10 days after initiating the nonkaliuretic agent and dietary sodium restriction, small doses of a short-acting kaliuretic diuretic, such as furosamide (Sherlock, 1975), are often given. Throughout this treatment, the electrolyte and magnesium status of the patient should be carefully monitored, along with body weight, urine volume, and, if possible, urinary excretion of sodium and potassium. In adults, a daily weight loss of just 2 pounds resulting from diuresis (which is equivalent to about 900 ml of ascites) is unlikely to compromise the patient's plasma volume (Sherlock, 1975).

Should severe salt restriction impair appetite and if adequate and uncomplicated diuresis occurs, a modest liberalization of dietary sodium, up to 500-1,000 mg/day (22-44 mEq), may be allowed, as long as diuresis continues. At all times, the nutritional adequacy of the diet must be monitored.

RENAL DISEASE

Fluid accumulation and edema commonly occur in patients with various forms of renal disease, notably acute glomerulonephritis, nephrotic syndrome, chronic pyelonephritis, and intercapillary glomerulosclerosis. During the period of oliguria or anuria, which are associated with acute glomerulonephritis, sodium retention is marked and contributes to edema. Restriction of dietary sodium during this phase of acute glomerulonephritis is rational therapy.

In nephrotic syndrome, which is characterized by massive albuminuria and hypoalbuminemia, urinary sodium excretion is usually greatly reduced. Sodium is retained in the body and edema ensues. Some restriction of dietary sodium tends to prevent edema, but has very little beneficial effect alone in treating edema or ascites once these conditions have developed.

Furosemide produces diuresis in both adults and children, and a decreased glomerular filtration rate, which is characteristic of acute glomerulonephritis (Loggie *et al.*, 1975). There is limited information concerning the efficacy of diuretics in treating patients with nephrotic syndrome. Evidence suggests that ethacrynic acid and furosemide may be beneficial in treating some children with nephrotic syndrome (Loggie *et al.*, 1975). Urinary sodium wastage may occur in some cases of chronic renal diseases. Excessive restriction of dietary sodium in individuals in this situation may lead to depletion of body sodium and to development of a "low-sodium syndrome" with decreased glomerular filtration and increasing signs of uremia.

In patients with severe renal failure and markedly decreased urinary excretion of sodium, ingestion of excessive sodium may result in edema, increased plasma volume, and congestive heart failure. In general, the sodium tolerance of patients with severe renal failure should be determined by measuring urinary sodium excretion in response to graded levels of intake. Such a test will assess the capacity of the patient to excrete sodium and will identify those with

impaired capacity to conserve sodium. The findings should be used by the physician and dietitian in planning the diet of the patient in terms of allowable sodium intake. The increased use of hemodialysis in managing severe renal failure in recent years has facilitated management of sodium homeostasis in these patients.

PREECLAMPSIA-ECLAMPSIA (TOXEMIA OF PREGNANCY)

This disorder of pregnancy is characterized by hypertension, albuminuria, and generalized edema, usually appearing in the third trimester. It may occur earlier, particularly in women with preexisting hypertension or renal disease and with hydatid mole. The syndrome is more likely to occur in young primiparous or older multiparous women.

There is much controversy concerning the pathogenesis of toxemias of pregnancy and, in particular, the role of nutrition in causation. The inverse correlation between incidence of toxemias and socioeconomic status is of particular interest. Preeclampsia-eclampsia is more common in the south than elsewhere in the United States, and incidence is greater among clinic populations than private-practice patients. Mortality rates range from approximately 4 per 100,000 live births in Washington, D.C., to 30 per 100,000 in Mississippi (Committee on Maternal Nutrition, 1970). These data suggest that a number of factors closely associated with low income or poverty have an impact on the course and outcome of pregnancy. Among these factors are availability, quality and use of health care, prevalence of chronic cardiovascular and renal disorders, and general prevalence of poor nutritional status reflecting long-term dietary inadequacies.

The clinical onset of preeclampsia may be insidious and initially unaccompanied by symptoms. Rapid development of generalized edema and hypertension, followed by proteinuria, is the usual sequence. In any pregnant woman, a rise of 20 mm Hg systolic or 15 mm Hg diastolic pressure should be regarded as abnormal, since arterial blood pressure generally decreases during pregnancy (Ferris and Burrow, 1975).

Preeclampsia-eclampsia seems to result from widespread arteriolar vasospasm, the cause of which is unknown. The only known cure is termination of pregnancy. Presymptomatic gravidas who are likely to develop this syndrome tend to exhibit an increased pressor response to angiotensin II (Lindheimer *et al.*, 1976). Vasospasm presumably interferes with transport of water from the extracellular

to the intravascular fluid compartment, thus causing generalized edema. When vasospasm modifies glomerular blood flow, hypoxia may occur, resulting in escape of albumin into the glomerular filtrate. The widespread vasospasm results in increased peripheral resistance and causes elevation of systemic blood pressure.

The current concept is that the sodium retention of preeclampsia is not the cause of edema. Rather, it is a result of fluid accumulation (Kaminetzky and Baker, 1977; Lindheimer and Katz, 1973). Renin concentration and aldosterone secretion are lower in preeclamptic gravidas than in normal gravidas, despite the reduction in blood volume that accompanies this condition. Apparently there is interference with the mechanism that normally contributes to the positive sodium balance and increased blood volume during pregnancy.

The use of a sodium-restricted diet and diuretics for the management of preeclampsia-eclampsia addresses secondary and possible compensatory features of the disease. Despite the mobilization of the excessive extracellular fluid and its contained sodium, augmentation of other symptoms, including the onset of convulsions, is not preventable by such management. Furthermore, if sodium restriction and administration of diuretics are prolonged, volume contraction and pancreatitis can result (Gray, 1968; Pike and Smiciklas, 1972; Pitkin *et al.*, 1972). In addition, chlorthiazides have been reported to decrease placental transfer (Gant *et al.*, 1971) and induce fetal thrombocytopenia.

Mild preeclampsia is often managed by sedatives and bed rest, which usually result in an apparent decrease in the severity of the arteriolar vasospasm, leading in turn to reduction in systemic blood pressure and to a diuresis that eliminates excess extracellular fluid. When symptoms are not ameliorated or, in fact, are intensified, intravenous or intramuscular magnesium sulfate is often used to decrease neuromuscular irritability. A decline in systemic blood pressure frequently follows.

If the blood pressure rises despite these measures, an antihypertensive agent is indicated to forestall cerebrovascular accidents, especially when preeclampsia is superimposed upon essential hypertension.

Sodium restriction and diuretics are employed for gravidas with essential hypertension or renal disease and preeclampsia. However, the therapeutic objectives should be clearly defined, the side effects known, and the duration of use appropriately limited.

PLANNING, EVALUATING, AND USING SODIUM-RESTRICTED DIETS

The dietitian or other nutrition counselor should bring to the patient who needs dietary sodium restriction the health professional's understanding of pathology, treatment, and knowledge of nutrition; the educator's appreciation of the principles of learning and behavior change; and the humanist's empathy for the patient's situation.

Desired goals of the dietitian-patient relationship include patient recognition of his responsibility in the management of his condition, understanding of why sodium restriction is indicated and how the diet can be modified, and the clinical consequences of failure to comply.

The dietitian must respect and accept patients' values and feelings and their right to live their lives as they choose (even if they opt for a less-than-healthy existence, from the health professional's point of view) (Meenan, 1976). The dietitian can assist patients in achieving the desired goals by assessing their nutritional status and evaluating the multiple factors that may contribute to their status; assessing their attitudes toward dietary modification; developing with patients a realistic plan for modifying their diets; and instructing patients, evaluating their comprehension, and developing follow-up plans based on their initial comprehension, the extent of dietary behavior change entailed, and the need for reinforcement and support.

NUTRITIONAL ASSESSMENT

Dietitians are trained to obtain a detailed diet history. Assessment of the patients' nutritional status begins with a review of all available objective clinical (physical examination, anthropometry, and laboratory) data and

requires effective communciation between the physician and the dietitian for interpretation. Assessment of available data will frequently uncover problems (or potential problems) in areas that are related to diet other than the sodium restriction.

The patient interview is a critical aspect of the assessment process. Not only does it provide the dietitian with information about patients, their attitudes, dietary habits, life-style, and environment, but it also sets the stage for the entire dietitian-patient relationship. The quality of this relationship may govern, to a great extent, whether patients will in due course assume responsibility for their own behavior in management of the disease or condition. The dietitian can best help patients by communicating a sincere interest and respect for their ideas, feelings, and values and by indicating a willingness to provide information and practical assistance (Benjamin, 1969). The dietitian customarily demonstrates interest in patients by addressing their concerns about their health, their diets, their ability to follow the diet, and their "support" system at home.

It is necessary to ascertain what patients know about their disease or condition and whether they believe that dietary modification will improve their health status. This is an area in which patients are often confused or are under one or more misconceptions. Therefore, eliciting this information provides the dietitian with an opportunity to explain, in whatever detail seems appropriate, the clinical significance of sodium restriction. Unless patients fully understand the reason for such a major change in life-style, it is unrealistic to think that they will comply with instructions.

To obtain information about the patient's food habits and eating patterns, the dietitian may use a variety of techniques (e.g., 24-hour recall, food frequency lists, 3-day record, food diary). From this information it will be possible to evaluate the nutritional adequacy of the current food intake and to assess the magnitude of change in sodium intake that will be necessary to achieve the level considered therapeutically desirable. Information about patients' social and economic situation and ethnic background will often reveal factors that the dietitian must take into account in developing an appropriate plan.

The dietitian will need to learn if patients have previously tried unsuccessfully to change their diet. If they have, they may approach the interview and instructional setting with a negative attitude. Under such

circumstances the encouragement and support of the dietitian are critical if any measure of success is to be attained.

DEVELOPMENT OF THE DIET PLAN

When all data relevant to the patient's nutritional status have been reviewed, the dietitian should discuss the overall evaluation with the patient. In those instances where someone else regularly prepares the meals, this person should also participate in the discussions. Major sources of sodium in the current diet should be identified. Recognizing that permanent behavior changes come slowly for most people and that many individuals faced with making multiple changes simultaneously are likely to fail at some or all, the dietitian should stress changes believed to be of highest priority.

Sound judgment is required. In some cases, the traditional approach (i.e., insisting on a marked reduction in sodium intake to achieve an immediate physiological response) may not deserve highest priority. If long-term dietary compliance is essential, the dietitian may find it more important to deal first with a patient's negative attitude toward change. This approach is especially important in working with patients who have been unsuccessful in past attempts to change their eating habits. If these patients can achieve a few modest successes during the first few weeks and are supported and reinforced in their efforts, they may be inspired to work toward more ambitious goals.

It may be necessary to help patients set realistic short-term goals. If their expectations are very high (e.g., to change overnight from a 5-g sodium intake to a 1-g intake), they should be made aware that many patients with those expectations have failed. A more gradual approach to reduction of sodium intake should be suggested. Encouraging patients to identify areas in which dietary change would be desirable and allowing them to explore various options are important steps. Accepting these decisions is critical. In any case, patients will eventually do as they wish and are responsible for their behavior. The dietitian who understands this and acts accordingly will help patients recognize their responsibility in dealing with their disease or condition.

If extensive dietary changes are necessary in order to achieve a given therapeutic goal, or if it appears that the patient will be unsuccessful in complying with the diet order, the dietitian should discuss the clinical

implications with the patient's physician. Eventually, it may be possible for such a patient to restrict sodium severely if the long-term nutritional care plan capitalizes on incremental behavior change. The initial diet plan should be based on the patient's current habits and intakes. This is not possible with a preprinted diet sheet. The plan that the patient takes home may consist of no more than a few mutually-agreed-upon, short-term objectives (e.g., to lower sodium intake from 5 g to about 3.5 g/day) and steps toward implementation to be accomplished before the next visit (e.g., to substitute low-sodium bread for regular bread or fruit juice for canned soup and try one new low-sodium recipe that week).

Immediate and extensive restriction of sodium is best accomplished in a supervised setting, such as a hospital, where meals are prepared and served under the direction of a dietitian.

INSTRUCTING THE PATIENT

Patients needing sodium restriction should be familiar with the terminology (sodium vs. salt); the clinical significance of sodium (for themselves); where sodium is found; how to interpret food labels; how to prepare palatable foods with little or no salt; and how to fit a sodium restriction regime into their life-style (e.g., taking into account restaurant meals, social occasions, school lunches, traveling, etc.).

EVALUATION OF PATIENT

Before the counseling relationship is terminated, the dietitian must be certain that the patient truly understands the agreed-upon modified diet and the rationale for it. If the patient terminates the relationship before long-range goals are achieved, the dietitian should so inform the physician. It may at times be necessary to refer some patients to other agencies as a means to continue the process.

Such behavioral objectives as the following are recommended to enhance the patients' ability to apply the information that they have been given without the further assistance of a dietitian:

- Given a tray of food models representing assorted high-, moderate-, and low-sodium level foods, the patient (or other person responsible*) will identify the food items that contain large amounts of sodium and, therefore, should be limited or avoided, depending on the level of dietary restriction.
- Given a tray of assorted food labels, representing items containing high-, moderate-, and low-sodium levels, as well as specially processed or "dietetic" foods, the patient (or other person responsible*) will differentiate correctly between those that can be included and those that should be avoided.
- Given actual menus from two local restaurants, the patient (or other person responsible*) will select items for a complete meal from each menu that are consistent with the agreed upon level of sodium restriction.
- The patient (or other person responsible*) will plan at least 1 day's meals that are consistent with the indicated level of sodium restriction and normal nutritional needs, taking into account food preferences, available money, time, interest, skills, equipment, etc.
- The patient (or other person responsible*) will state one or more relevant reasons why sodium restriction is desirable in the management of the particular disease/condition.
- The patient (or other person responsible*) can state meaningfully what clinical consequences may result if sodium intake is not restricted.

FOLLOW-UP PLANS

It is only realistic to recognize that few people can or will permanently change such life-long behaviors as food habits simply as the result of a single encounter with a dietitian or any health professional. For dietitians to effect change, they must develop follow-up interactions with the patient (Peck, 1976).

*It may be necessary to instruct and evaluate the understanding of another person if, for example, the patient is a child or a person incompetent to take responsibility for his behavior or for managing his or her disease/condition. In most such instances it may be appropriate to include family members during the diet instruction.

The frequency and extent of follow-up sessions will vary with each patient, depending upon such factors as: the patients' understanding of diet as evaluated by behavioral objective tests; the extent of sodium restriction necessary to achieve a physiological effect; and the ability of patients to adhere to their decisions to achieve short-term objecitves and work toward ultimate goals.

The dietitian should recognize that retention of information and motivation often decrease over time. If long-term dietary modification must be achieved, periodic sessions are advisable for most patients so that the dietitian can reevaluate their understanding, reemphasize the clincial significance of diet modification, set new short-term objectives, answer questions, and encourage the patient to express frankly any difficulties encountered in sustaining interest and accomplishing change in behavior.

ASSISTANCE IN THE COMMUNITY

Dietitians who are knowledgeable about resources available in the community can be very helpful to the patient. Most communities have a variety of nutrition, social service, and health programs. Food and nutrition information programs commonly available include home-delivered meals programs, senior citizens nutrition programs, food stamps, emergency food programs, special supplemental food programs, and cooperative extension services. Such programs may well offer very useful support to patients who do not have necessary food preparation skills, whose financial resources are limited, or who may benefit from support from others of a group. It is unfortunate that therapeutic nutrition counseling services are not more widely available to patients outside of hospitals, although most communities do have access to health professionals from agencies such as health departments, home health agencies, and visiting nurse associations. If agencies such as these are to provide follow-up education or reinforcement and support, they should be given the dietitian's complete nutritional care plan, including specific short- and long-term objectives, and a summary of the nutritional assessment.

LEVELS OF SODIUM-RESTRICTED DIETS

Dietary sodium restriction should be thought of in terms of degrees of restriction, i.e., mild, moderate, severe, and

extreme. Attempts to calculate a diet to within a few milligrams of a therapeutic dietary goal are pointless. Values shown in available food consumption tables are imprecise, and actual sodium content of some foods may prove different from that listed in food composition tables. From the range of analyses of single food items, a 25 percent or greater deviation is not uncommon. Furthermore, the clinical effects of a specific level of sodium restriction will vary among individuals. *The least amount of restriction necessary to achieve a desired clinical response should be the goal.* Further restriction of sodium requires more changes in life-style of patients. Following a sodium-restricted diet is a complex process. Individuals must have time, energy, and skill to shop carefully for food; they must have access to food products that are appropriate for the diet; they must have the financial resources to purchase these often more expensive food items; and they must have the skill to prepare meals using raw materials (because many "convenience" and restaurant foods must be limited or avoided altogether).

For many adults, *mild* sodium restriction (defined as approximately 2,000-3,000 mg) often can be achieved by avoiding addition of salt in cooking or at the table and by avoiding highly salted foods. *Moderate* sodium restriction (defined as approxiamtely 1,000-1,500 mg) usually requires that food be prepared without salt and that some special low-sodium foods such as bread and margarine or butter be included. This level of restriction may be difficult for some to achieve because of their living situation. *Severe* sodium restriction (defined as approximately 500-700 mg) requires, in addition to the above, strict limitation of foods that naturally contain sodium, as well as incorporating in the diet many special low-sodium food products. This level of sodium restriction is impractical--and indeed may be impossible--for most ambulatory patients. *Extreme* sodium restriction (defined as approximately 200-300 mg) requires such careful monitoring that, with few exceptions, it should not be attempted except in hospitals.

Factors that may make *moderate* and *severe* sodium restriction difficult for ambulatory patients include:

- frequent occasion to eat in restaurants;
- dependence upon "convenience" foods because of a physical handicap, a lack of food preparation skills or interests, a lack of cooking appliances or facilities;

- previous heavy sodium intake because of acquired taste preference; cultural or religious food preference (e.g., Oriental foods containing soy sauce or monosodium glutamate; Kosher meats; foods cured, preserved, or processed in salt or brine, etc.);
- low income; and
- unavailability of specially processed products without salt or sodium.

OTHER CONSIDERATIONS

- *High-Potassium Diets*

The potassium content of the diet can be increased by very liberal intake of fruits, certain vegetables, or dried beans and peas. If the patient is able to consume 8-10 servings per day of high-potassium fruits or vegetables, dietary potassium may be adequate to avoid the potassium depletion caused by diuretics.

In some regions of the United States, fruits are relatively expensive during much of the year, especially in the quantity and variety that are needed by these patients. In addition, many persons do not ordinarily consume fruit regularly. If it seems unlikely that the patient will obtain adequate potassium from the diet, the patient's physician should be informed, so that alternative measures can be considered.

- *Support by the Physician and Other Health Professionals*

Physicians play a key role in assisting the patient to comply with a sodium-restricted diet. They should advise patients as to why a modified diet is necessary in the management of their condition and what effect the diet can be expected to have on their clinical symptoms. Reinforcement and encouragement of patients' efforts to change their behavior at each medical visit are important as a way of supporting this major change in life-style. All members of the health care team can assist the patient by providing such support.

- *Nutritional Adequacy*

The dietitian must be the key advocate for sound nutrition practices for protection of health as well as for

treatment of disease. Attention must be given to the overall nutritional adequacy of the diet in the course of trying to achieve desired restriction in sodium intake.

- *Food Preparation*

It is possible to serve palatable and attractive meals and still restrict sodium. Many spices, herbs, and other flavor aids can be used to enhance the palatability of low-sodium foods. Indeed, these diets might stimulate a wider appreciation of herb cookery. Flavoring aids that do not contribute significant amounts of sodium to the diet and that therefore may appropriately be used as desired are included in Table 1. If regular cookbooks are used, the patient must be sufficiently alerted not only to omit the obvious forms of salt in the recipe, but to recognize other

TABLE 1 Flavor Enhancers That Are Useful in Sodium-Restricted Diets

Allspice	Orange peel
Almond extract	Oregano
Anise	Paprika
Basil	Parsley (fresh)
Bay leaf	Pepper, black, red, or white
Caraway	
Cardamon	Peppermint extract
Chives	Pimento
Cinnamon	Poppyseed
Cocoa (not Dutch Process)	Poultry seasoning
Curry	Rosemary
Fennel	Saccharin
Garlic	Saffron
Ginger	Sage
Horseradish (not prepared)	Savory
Leeks	Sesame
Lemon juice or extract	Sucaryl calcium
Mace	Sugar, brown (in small amounts)
Maple extract	
Marjoram	Sugar, white
Mint	Tarragon
Mustard, dry	Thyme
Nutmeg	Tumeric
Onion juice	Vanilla extract
Orange extract	Wine

major sodium contributors and make appropriate substitutions.

- *Sources of Sodium and Potassium*

The committee believes that it is inappropriate to imply that a great deal of precision is necessary, or in fact achievable, in dietary therapy insofar as sodium or potassium are concerned. In part, this concern reflects the fact that diuretics have become important in managing patients with disorders that are characterized by sodium and water retention. In addition, it is now recognized that communication and cooperation among physician, dietitian, and patient are much more important than simply giving the patient a diet sheet purporting to show a precise level of sodium. Sodium content of foods is highly variable; therefore, it is misleading to imply a level of precision in sodium intake that is not attainable except on a metabolic ward.

Naturally occurring sodium content of water shows considerable variation, depending on geographic location. A survey of 2,100 water supplies, serving approximately half the U.S. population, showed a range of from 0.4 to 1,900 mg/l, 75 percent of which were below 100 mg (4.3 mEq) per liter (White *et al.*, 1967). For individuals requiring stringent restriction of sodium in the diet, water may be a significant source of sodium, depending upon the conditioning of the water supply and the quantity of water consumed. Zeolite water softeners may increase the sodium content by a factor of 10 to 40, depending upon the levels of calcium and magnesium in the water supply. In general, water containing more than 20, and certainly more than 40, milligrams of sodium per liter (as consumed) may be unacceptable for patients requiring severe or extreme restriction of sodium.

Physicians and dietitians must also be alerted to the sodium content of prescribed or over-the-counter medications. Among these are "anti-acid" or "alkalizing" preparations, saline cathartics, and salicylates. Similarly, toothpastes and powders may contain considerable sodium in various compounds that, if swallowed, represent additional intake of sodium.

SUMMARY

Sodium-restricted diets for the management of disorders of sodium and fluid balance and hypertension have become less popular with the availability of potent diuretics. However, diuretic therapy *per se* has led to complications, and it now appears that a judicious combination of dietary sodium restriction and an appropriate diuretic agent is the most rational approach to the management and treatment of diseases that are characterized by the retention of sodium.

Human beings have a very narrow range of tolerance for fluctuations in concentrations of electrolytes in plasma or in plasma osmolality. The kidney is the key organ in maintaining these ranges within tolerable limits despite wide fluctuations in external conditions and dietary intake.

The kidney exerts its control through hormonal action, effects of arterial blood pressure, and intrarenal distribution of blood flow. The hypothalamus assists in regulating fluid and electrolyte intakes by influencing thirst and the desire for salt. Acting singly, or in combination, the control system normally maintains the internal fluid environment at a level that is consistent with efficient cellular function.

In the United States, consumption of sodium in the diet well exceeds metabolic needs. It is not clear whether individual consumption is due to an innate neural organization, to learned eating behavior, or to both. The human organism can adapt to diets containing very low levels of sodium without apparent harm.

The newer diuretic agents have produced complications including sodium-potassium imbalance, glucose intolerance, hyperkalemia, and hyperuricemia. The likelihood of such complications can be reduced or avoided by combining

appropriate diuretics in proper dosage with judicious control of dietary intakes of sodium and potassium.

Sodium-restricted diets must be evolved from an empathic relationship between the patient and the physician or dietitian. To be effective and acceptable, the diet prescribed must be consistent with the life-style, needs, and constraints of the patient and must be developed and presented in such a way that the patient will recognize the need for the diet and the consequences of noncompliance. The goal should be the least amount of dietary restriction that is necessary, alone or in combination with diuretics, to achieve a desired clinical response.

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