

Nutritional Treatments for Hypertension

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Introduction

Over the last 15 years, medicine has gone through a revolutionary change. The medical dictum was that nutrition and lifestyle made no contribution to chronic disease. Medicine has done a complete turn-around and has started a war against bad lifestyle habits like smoking, fat consumption, sedentary behavior, etc. Modern medicine has accepted its responsibility to direct the lifestyles of people toward health. Possibly no movement other than Orthomolecular medicine was shouting like a voice in the wilderness before the rest of the profession identified the important role of nutrition. Although Orthomolecular physicians and scientists first touted the important role of nutrition in mental health, mental health is actually now known to be the foundation for all health. Indirectly, and often directly, Orthomolecular physicians and scientists have heralded the way for the complete nutrition revolution in the United States. This nutritional revolution is most evident in the transformation of the American doctor's treatment of hypertension. More than any other illness, it is now accepted by mainstream medicine that nutritional and dietary factors and therapies should be utilized by physicians in treating hypertension. It is fitting, therefore, to bring this review to the readership which helped begin the revolution that is now transforming all aspects of American health.

Epidemiology

Hypertension is clinically defined as systolic blood pressure greater than 140 mmHg and/or diastolic blood pressure greater than 90 mmHg. It is a leading problem in the United States, where nearly 20 percent of Americans are affected by this disease (Kaplan, 1984; McCarron, 1984). More than ten million Americans are being treated for this disease at a cost of over 2.5 billion dollars, the largest medical expenditure for a single disease in the United States (Chobanian, 1986).

High blood pressure afflicts over 60 million Americans and contributes to one million deaths per year in the United States, adding 18 billion dollars per year to United States health expenditures (Lavash, 1987). Genetic, psychological, and environmental factors play a role in hypertension. In 1975, over half (54 percent) of all United States deaths were from cardiovascular disease. Hypertension is the most significant and preventable contributing factor (Froehlich, 1986). Hypertension is associated with an increased risk of heart failure, kidney failure, and stroke. It is virtually an epidemic in the black population (Check, 1980; Harburg et al., 1982; Schachter et al., 1984; Tyroller and James, 1978). About 1/3 of blacks between 18 and 49 have hypertension and 2/3 over 50 have hypertension. However, the ratio of black to white hypertensives is decreasing, probably due to the better treatment of high blood pressure in blacks than in whites. American blacks are twice as likely to suffer from kidney failure, and have the world's second highest incidence of stroke, behind the Japanese (Williams, 1986; Check, 1986).

A study in Rochester, Minnesota showed that controlling hypertension led to a decrease in the incidence of stroke (Ganarrax and Whishant, 1957). In addition to increasing incidence of stroke, high blood pressure appears as a contributing factor in some cancer deaths as well (*Journal of National Cancer Institute* 77:1-63, 1986). Vigorous treatment of elevated blood pressure reduces strokes (Daughtery, et al., 1986). Another complication of hypertension in the portal system is that it impairs nutrient absorption from the diet (Sarteh, et al., 1986).

Hypertension is also an increasing problem among children, possibly due to dietary factors, such as high fat and refined carbohydrate consumption (Doheny, 1986). Hypertension has been correlated to a diet high in calories, sodium, sodium/potassium ratio, alcohol, low in protein, calcium, magnesium, micronutrients, and vitamins. Hypertension increases with age and occurs more frequently in men.

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A study done in Tel Aviv, Israel showed that 67 percent of the elderly population had some degree of hypertension (Golan, et al., 1986). Women who get pregnant at a later age (35 or over) have increased complications, especially essential hypertension (Weisley, 1983). Paradoxically, one study supports the conjecture that elevated systolic and/or diastolic blood pressure in the aged composes a risk reduction factor (Burch, 1983).

Five percent of all hypertension has been classified as "secondary", that is, associated with some other disease (usually renal or adrenocortical tumor) (Chobanian, 1982). Ninety-five percent of hypertension is classified as "essential" hypertension, primarily related to stress, nutrition and other lifestyle-like factors (Chobanian, 1982; Davidman and Opsahl, 1984). Most hypertension cases are probably due to arteriosclerosis. Patients with sustained hypertension show increased peripheral resistance. This is possibly due to a decrease in the number of arterioles and increased viscosity. The physician should treat only after making an acceptable benefit/risk ratio, and then involve the patient in his/her treatment (Bass, 1987).

Atherosclerosis formation is a very complex problem and may be related to an intracellular deficiency in essential fatty acids. One study suggests that there are four categories of hypertensive patients, each with a different pathophysiology and pharmacological profile: The young patient often has an increased cardiac output, the middle age patient's total peripheral resistance is elevated, and the elderly patient's total peripheral resistance is even further increased while their intravascular volume is contracted. Obese and black patients have elevated total blood volume and cardiac output (Messerli, 1987).

Treatment

Our paper is devoted to the nutritional treatment of essential hypertension. This is a very successful program and should be the first approach to hypertension. A whole array of symptoms and effects manifest themselves with the usage of standard hypertensive pharmacological therapy (Curb, et al., 1986). Any regimen for hypertension may have detrimental effects on the cerebral functioning of the aged (Middleton, 1984). Approximately 30-50 percent of elderly patients experience side

effects from antihypertensive therapy (Gifford, et al., 1986). Rapid treatment of hypertension in the elderly can cause quick drops in blood pressure and possibly lead to stroke (Jansen, et al., 1986). Antihypertensive drugs have been postulated to be related to the genesis of acute, as well as chronic pancreatitis (Weaver, 1987). Drug therapy for mild hypertension (systolic 140-150, diastolic 90-100) will only help a small percentage of patients, and the side effects may far outweigh the potential gain (Chobanian, 1986). Antihypertensive therapy should be matched to the underlying biochemical problems. This would give more efficacious therapy than the standard stepped care approach (Frohlick, 1987).

The financial cost of an antihypertensive regimen should be considered for long-term patient compliance (Sahler, 1987). Labarthe (1986) further supports the use of nonpharmacological therapy in mild hypertension while condemning drug use. The great many side effects of antihypertensive medications for treating mild hypertension has caused many cases of noncompliance and ineffective long-term therapy (Croog, et al., 1986). It is becoming apparent that drug regimens for the treatment of hypertension have become increasingly unsatisfactory to modern physicians. Even mild hypertension poses risks in the long run and should be treated (Schoenlenger, 1986). This is where our nutritional and lifestyle program has a tremendous input.

The Dangers of Drugs

Diuretics

There are approximately five categories of drug treatments: diuretics, beta-blockers, alpha blockers, angiotensin-2 inhibitors, and calcium channel blockers. The most commonly used treatment is diuretics and continues to have a large variety of side effects (Ames, et al., 1984; Reyes, et al., 1984; Morgan, et al., 1984; Field and Lawrence, 1986; Weinberger, 1985; Kaplan, 1986). Patients who receive diuretics as their sole therapy have an increased risk of mortality due to myocardial infarction or sudden death (Morgan, et al., 1984). Diuretics deplete magnesium and potassium (Reyes and Leary, 1984). Further support for early plasma and urinary changes in potassium from diuretic-treated patients comes from the study of

Papademetriou and colleagues. Potassium sparing diuretics can cause serious hyperkalemia when administered, while hyponatremia can be a result of thiazide diuretic therapy (Phan, 1986). Thiazide diuretic therapy in the elderly leads to almost 50 percent of the patients displaying hypokalemia or hypomagnesemia (Petri, et al., 1986). Further support for intracellular magnesium loss during diuretic therapy comes from Dyckner and Wester, 1983. They demonstrate that 42 percent of patients with arterial hypertension had subnormal levels of skeletal muscle magnesium. Potassium deficiency can usually be corrected, but the loss of magnesium is rarely addressed. Red blood cell sodium increased, the membrane sodium potassium ATPase activity decreased, and potassium decreased when patients were studied who were receiving the diuretic hydrochlorothiazide. Even at low doses, diuretics, like hydrochlorothiazide, will have adverse effects on serum lipid levels but will not then produce significant hypokalemia (Mokenney, et al., 1986). We have found decreased RBC magnesium to be a common occurrence among patients using diuretics as well as beta-blockers. Moreover, as Ames and Peacock (1984) have pointed out, serum cholesterol as well as other serum lipids are increased during treatment with diuretics. This includes triglycerides and LDL levels.

Diuretic drugs prescribed for hypertension cause glucose intolerance and raise glycohemoglobin concentrations as well as increase blood cholesterol and triglycerides. One study showed that with up to one year of treatment with diuretics, plasma cholesterol increased accordingly (Williams, 1986). The side effects of diuretics are still disputed by some physicians (Freis, 1986). Hollifield pointed out the problems of thiazide diuretics relative to potassium and magnesium metabolism, and ventricular ectopy.

Beta-Blockers

The second most commonly used therapy are beta-blockers. Beta-blockers have similar side effects as diuretics. Weinberger has pointed out undesirable serum lipid fractions in patients treated with beta-blockers. At least 25 percent of all patients using beta-blockers will develop a need for antidepressants (Avon, 1986). Moreover, they have negative inotropic effects which can cause an increased risk of

heart failure (Chobanian, 1982). Miettinen suggested that patients on beta-blockers had an increased risk of coronary heart attacks, as did patients on anticholesterol drugs or diuretics. Yet, Floras tried to argue against the side effects of beta-blockers.

Beta-blockers, like most antihypertensive drugs, can cause sexual dysfunction (Croog, et al., 1986). Twenty-eight percent of patients on the beta-blocker timolol maleate experienced adverse reactions which most commonly consisted of fatigue, dizziness, and nausea. Lipid-soluble beta-blockers that cross the blood brain barrier have been known to produce neurotoxic side effects as well as cold in the bodily extremities (Thodani, 1983). Some evidence indicates that beta-blockers are more effective in Caucasian than Negro hypertensives (Veiga and Taylor, 1986). Long-term use of beta-blockers, more than two to three years, is probably contraindicated for most patients.

Alpha Blockers

Alpha-blockers such as Catapres have a significant amount of side effects, notably hypotension, constipation, sedation, dry mouth, and dizziness. I have not found them to be particularly helpful in long-term treatment of hypertension.

Methyldopa and Angiotensin

Methyldopa, for instance, seems to lower work performance and general well-being, as compared to other antihypertensive agents (Croog, et al., 1986). In the same study, methyldopa was compared to Propranolol and Captopril and rated worse in causing the following conditions: fatigue, sexual disorder, headache, neck pressure, insomnia, and nightmares. Up to 50 percent of patients on one of these three drugs experienced fatigue or lethargy; up to 30 percent had some form of sexual disorder; and over 10 percent had sleep disorder, nightmares, headaches, anxiety, irritability, palpitation, dry mouth, dizziness, nausea, and muscle cramps (Croog, et al., 1986). Captopril, an angiotensin inhibitor, is one of the safer drugs for hypertension, wherein it does not affect a patient's glucose tolerance (Shinodin, 1987). Nevertheless, angiotensin-II inhibitors seem to affect trace elements significantly. Selenium and zinc are decreased and copper increased, which may be a prob-

lem in the psychologically sensitive (Braverman and Pfeiffer, 1982). Calcium channel blockers are seen to be more efficient and give fewer side effects as compared to the traditional hypertension therapy of diuretics and/or beta-blockers (Tarazi and Tarazi, 1986).

Vasodilators

Vasodilators like nitrates are frequently accompanied by headaches. Nitrates have been used for the treatment of angina pectoris and congestive heart failure, but have not been systematically studied for efficacy for clinical usage in hypertension (Simon, et al., 1986). Drugs like Hydralazine also produce depression in 10 to 15 percent of the patients taking it. Dopamine-metabolite inhibitors (i.e., Methyldopa or Aldomet) are frequently linked with depression and other negative side effects. Hence, we have found virtually all drug regimens have side effects significant enough to warrant searching for other modalities.

It has become evident from articles in *The New Scientist* that treating hypertension with drugs is not cost effective given the current efficacy of drug regimens (Lesser, 1985; Kaplan, 1985). As Dr. Grimm and McAlister (1983) suggest, the treatment of mild hypertension may not be beneficial. Mild hypertensive patients have a diastolic blood pressure between 90 and 104. Over the age of 80, there seems to be little benefit from treating hypertension (Amery, et al., 1986).

It appears that once drug regimens are opted for, drug therapies will spiral. However, the Framingham study indicates that a certain small percentage of formerly treated hypertensives maintain normal blood pressure when treatment is stopped. After abrupt withdrawal from antihypertensives, blood pressure usually rebounds (Greenberg, 1986). The need for drugs continues to increase. This is why a suitable nutritional program is necessary. Ironically, it has come from places like the *Annals of Internal Medicine* and the *AMA News* to suggest that dietary changes and not drugs are the best option (Kaplan, 1985; *AMA News*, 1986). The focus of treatment in hypertension should move towards elimination of pharmacological side effects and reduction of risk factors for coronary heart disease (Weinberger, 1987). A recent article in *JAMA* (1987) states that "Nutritional therapy may substitute for drugs in a sizeable portion of

hypertensives, and if drugs are still needed it can lessen some unwanted biochemical effects of drug treatment." A study in Finland where people restructured their diet found that the mortality from coronary heart disease decreased up to 49 percent in some segments of the population. In hypertensive therapy, more than any other aspect of medicine, the role of dietary factors has entered into orthodox medical thinking.

Lifestyle, Obesity, and Dietary Considerations

Numerous lifestyle factors have been identified in hypertension by McCarron and colleagues. A study in New York City, where school children maintained ideal weight, decreased total and saturated fat, cholesterol, and sodium while increasing consumption of complex carbohydrates and fibers, showed improved blood pressure, plasma cholesterol, body mass index, and overall cardiovascular fitness. Even men with a genetic history of familial hypercholesteremia can greatly reduce cardiovascular risks by eating a low fat diet, doing regular aerobic exercise, strict avoidance of cigarettes, and monitoring blood pressure and blood cholesterol (Williams, et al., 1986).

The sympathetic nervous system, which is activated by stress, isometrics, etc., plays an important role in creating hypertension (Tuck, 1986). It has become increasingly clear that lifestyle changes can reduce excess catecholamine levels, which are potentially harmful chemicals when inappropriately distributed in the body and increase under stress (Eliasson, et al., 1984; Msus, 1984). Nicotine from cigarette smoking causes small sretioles to constrict, blocks the useful effects of antihypertensive medicines, and is associated with malignant hypertension. A reduction of blood pressure was found with exercise when hypertensive rats were given the opportunity to do so (Fregly, 1984).

A cold environment might correlate with higher blood pressure levels. Differences between winter and summer blood pressure may be predictive of future hypertension (Tanaka, 1989).

There is some evidence that the roots of hypertension are found in early childhood and preventive attention should begin as early as adult blood pressures are achieved.

Obesity is the number one lifestyle factor related to hypertension and probably overall health and longevity. Therefore, weight loss is an essential part of a high blood pressure regimen (Garrison, et al., 1987). We do not, however, recommend appetite suppressants. One of these, phenylpropanolamine, can induce significant hypertension. Obesity is a major cardiovascular risk factor having a very complex socioeconomic, cross-cultural interrelationship with various other risk factors. One study established that long-term changes in blood pressure correlate with decreases in body weight (Dornfeld, et al., 1985). Hypertension has been shown to be directly proportional to obesity and glucose intolerance.

In a study with Urban Bantus of Zaire, body weight and age were the major predictors of systolic and diastolic blood pressure (M'Buyamba-Kabangu, et al., 1986).

There is a wide variability of blood pressures among black people in Africa suggesting that factors other than race play a role. A simple genetic explanation for the blood pressure differences between blacks and whites is inadequate and socioeconomic issues must be considered (Anderson, 1989).

Diet and exercise such as walking, swimming, and biking have beneficial effects on blood lipid levels. We always encourage our patients to exercise, if capable, usually after an EKG, 24-hour blood pressure monitor, and echocardiogram have been done, and, in some cases, after a stress Thallium test. Exercise has been shown not to depress appetite but rather help to control it and is almost essential in a weight loss plan for hypertension. Simple exercise such as walking or swimming can add years to one's life. In a study where energy expenditure per week approached 3 500 calories, morbidity (illness) also decreased significantly.

Seventh Day Adventist lactovegetarians were compared with omnivorous Mormons (theoretically matching groups for effects of religiosity and abstention from alcohol, tobacco, and caffeine). The lactovegetarians had lower blood pressure, even after adjusting for the effects of weight. "Long-term adherence to a vegetarian diet is associated with less of a rise of blood pressure with age and a decreased prevalence of hypertension. Specific mechanisms and nutrients involved have not been clarified." (Beillin, Armstrong,

Marqetts, Rouse, Vandongen, 1986).

Psychological, emotional, and environmental factors also play a large role in cardiovascular disease, and this knowledge can be used to complement treatment regimens. Psychosocial and behavioral modification techniques are safe and somewhat effective in hypertension therapy. Feedback monitoring of blood pressure at intervals of several weeks was shown to be as effective as relaxation and biofeedback. Cranial electrotherapy stimulation (CES) a stress and anxiety reduction technique also probably lower, blood pressure.

An Australian study showed that after adjustment for different variables, the level of education was inversely related to blood pressure levels. Learning and education correlates with better lifestyle and lower blood pressure.

Serum cholesterol correlates very closely to blood pressure levels and helps to identify the segment of the population in need of treatment. Elevated serum cholesterol (above 240 mg/dl) is the single, most important risk factor in coronary heart disease. In a study with more than 360,000 men, cardiovascular mortality rises steadily with increasing serum cholesterol levels (718 mg/dl). Aggressive dietary modifications are very useful to lower blood cholesterol levels which are linked to atherosclerotic vascular disease and coronary artery disease. Elevated serum and arterial cholesterol is a major entity in hypertension and cholesterol and can be reduced by dietary fibers such as bran and pectin. The positive role of fiber in reducing cholesterol is further supported by Fletcher and Rogers. Dietary fibers contained in foods such as carrots and other vegetables lower body cholesterol levels by binding bile salts. Dietary fiber has an important moderating effect on serum cholesterol. Insoluble dietary fibers such as guar gum and pectin have been shown to be hypocholesterolemic and hypertriglyceridemic.

High quality fresh and whole food sources of oils and animal products are important. Fatty acids (including polyunsaturated fats) and cholesterol are susceptible to degradation by oxidation and free radical reactions. Studies on animals show the resultant "oxy-cholesterols" have atherogenic properties. Powders of egg and moldy cheeses (found in many fast foods) are especially susceptible.

Serum cholesterol and changes in serum

cholesterol were correlated to consumption of fats. However, serum cholesterol levels are not significantly related to dietary cholesterol in conjunction with a diet rich in polyunsaturated fats. A very high cholesterol intake by rural South African blacks caused no meaningful blood lipid fluctuations. The cholesterol synthesis-inhibiting drug lovastatin may have the side effect of promoting cataracts.

Egg intake coupled with a diet low in other saturated fats, high in polyunsaturated fats does not significantly raise blood cholesterol. Polyunsaturated fats can be used to lower total serum cholesterol and to raise HDL level, and thus can help to prevent atherosclerosis. Therefore, up to 7 eggs per week are permitted for most hypertensive patients (unless they have an extremely elevated or refractory high cholesterol level). In one study with renal patients, egg consumption did not significantly increase cholesterol or triglyceride levels. No significant correlation between egg consumption and serum cholesterol level was suggested by Pfeiffer.

Animal studies suggest that sucrose (found in cane sugar and some fruits and vegetables) has the effect of raising blood pressure. At high levels of carbohydrate consumption (50 to 80 percent) increased blood pressure is also noted (McDonald, 1987). Kannel pointed out that the dietary factors in hypertension may relate to the excess calories of saturated fat intake as well as high cholesterol and salt intake.

Fruits, vegetables, whole grains, and low fat dairy items protect against hypertension. An epidemiological study showed that one Chinese group with a history of hypertension had a high intake of added salt to their milk and tea, and consumed little starchy food, fresh fruits, and vegetables. Consumption of proteins, animal fats, disaccharides, animal products, refined foods, and high daily energy content of food were directly related to congestive heart disease (CHD) morbidity, arteriosclerosis, myocardial infarction, and arteriosclerosis mortality, whereas consumption of vegetable fats, starch, cellulose, hemicellulose, pectin, vegetables and fruit shared an inverse correlation.

Hypertensive patients may have impaired glucose tolerance, especially when treated with diuretics. Glucose tolerance tests in hypertensive patients are frequently abnormal.

A high carbohydrate (sucrose) diet has been shown to induce sodium retention. Sucrose or glucose can mediate a sodium retention effect, and thus, through this retention of sodium, raise systolic blood pressure. A diet high in sucrose will raise blood pressure in animals significantly, possibly due to a relative decrease in potassium intake. Glucose intolerance, obesity, and blood pressure are tightly interrelated, so a derangement in one will cause problems in the others.

Significant decreases in the consumption of calcium, potassium, vitamin A, and vitamin C have been identified as nutritional factors that distinguish hypertensive from normotensive subjects. Calcium intake was the most consistent factor in hypertensive individuals. Previous reports showed a significant negative correlation between water hardness and mortality rates. A study comparing the twin Kansas cities in the United States showed the opposite to hold true; hard-watered Kansas City, Kansas had more cardiovascular problems including a ten-fold higher serum cadmium level. Coffee has been shown to increase coronary heart disease risk by almost 250 percent. Smoking and hypertension are the two main risk factors for ischemic heart disease. Youngsters who smoke even less than one pack of cigarettes per day increase blood cholesterol and triglycerides.

Harlan and others have suggested that alcohol plays a role in hypertension. Moderate use of alcohol may lower blood pressure, but excessive use may elevate it. At moderate levels of one drink per day, alcohol has been shown in some cases to be protective against coronary artery disease. Alcohol in large doses may lead to rhythmic disturbances in the electrophysiology of the heart (Greenspan and Schaal, 1983). Alcohol use may lead to depression and increase carbohydrate consumption which will lead to hypertension.

In light of these findings, we recommend the following dietary guideline to most of our hypertensive patients: low sodium, low saturated fat, and low refined carbohydrate intake, with high vegetable intake from the starch group, high salad and protein intake (particularly fish). Fresh cheeses are emphasized above aged cheeses. Simple sugar, alcohol, caffeine, nicotine, and refined carbohydrates should be reduced drastically or eliminated.

Saturated Fat and Fish Oil

Numerous researchers have suggested that saturated fats can raise blood pressure, while Singer and colleagues (1985), Knapp et al. (1986), and Nestel (1985) have suggested the potential blood pressure-lowering effect of fish oil. Polyunsaturated fats can be used to lower total serum cholesterol and to raise HDL level, and thus can help to prevent atherosclerosis. Dietary fat modifications, such as an increase in polyunsaturated to saturated fat ratio and an overall decrease in percentage of fat in the diet, lower blood pressure and have favorable effects on serum lipid levels. Greenland and Icelandic eskimos, whose diet is rich in saturated fats, have a much lower incidence of coronary heart disease than controls because of high fish consumption. An inverse relationship was found with fish consumption and twenty-year mortality from coronary heart disease. Those who consumed 30 grams or more of fish per day had a 50 percent lower cardiac mortality rate than those who did not. Fish oils (Omega 3 fatty acids) reduce high levels of plasma lipids, lipoproteins, and apolipoproteins in patients with hypertriglyceridemia. They also have effects on serum lipid levels in healthy humans (Gehily, et al., 1983, *Experimental Nutrition*, 1986). Eicosapentaenoic acid (EPA or fish oil) lowers abnormal blood lipid levels and decreases blood viscosity. Fish oil, like niacin, raises HDL and reduces risk from heart disease (Messim, et al., 1983). Atherosclerosis formation is a very complex problem and may be related to an intracellular deficiency in essential fatty acids. Halberg (1983) suggests that dietary lipid controls may be even more important than salt restriction in the control of hypertension.

Polyunsaturates and Hypertension

Mogenson and Box (1982) and Puska and colleagues (1985) have suggested that both linoleic acid and dihomogammalinolenic acid (found in evening primrose oil) can be extremely useful in the treatment of hypertension. Fish oil, rich especially in Omega 3 fatty acids, has been shown to lower blood pressure. Increasing consumption of monounsaturated fat is beneficial in lowering high blood pressure (Williams, et al., 1987). Dietary fat modification is an essential part of the treat-

ment of hypertension. Saturated fats have been definitively linked to high serum cholesterol. Dietary supplementation with linoleic acid, gamma linoleic acid, or other polyunsaturated fatty acids is of use in controlling hypertension. These agents lower blood pressure and have both a diuretic effect (particularly linoleic acid and gammalinolenic acid) and a prostaglandin-E2 inhibitory effect. A diet with fish, which is high in EPA, for example mackerel, has been shown to lower high blood pressure, serum, triglycerides, cholesterol, LDL, and raise HDL. A diet high in linoleic acid lessened a rise in blood pressure in Nephrectomized rats (Izumi, et al., 1986). Calcium supplementation may lower elevated blood pressure by increasing naturesis (sodium excretion) (Gilland, et al., 1987). Linolenic acid, a polyunsaturate, is helpful in the treatment and prevention of hypertension probably due to its conversion to prostaglandins and/or other vascular regulators (Benz and Hirsch, 1986). Linoleic and linolenic acids are both prostaglandin precursors and are useful in hypertension therapy (Adam, 1985).

Cis-linoleic acid is converted to gamma linoleic acid and eventually to prostaglandin E which is a vasodilator and inhibitor of platelet aggregation (Fletcher and Rogers, 1985). Smith and Dunn (1985) of Case Western Reserve University in Cleveland did at least eight different studies where safflower oil, linoleic acid, cod liver oil, and eicosapentaenoic acid all lowered blood pressure significantly. Fish oils, especially the Omega-3 fatty acids, have been shown to decrease risk of coronary heart disease. A diet high in fish or fish oil supplementation is recommended in patients with increased risk of coronary heart disease (Neutze and Starlins, 1986). In doses of up to 16.5 grams, fish oil has been shown to significantly lower blood pressure and cardiovascular risk factors (Norris, et al., 1980).

Omega 3 fatty acids prevent elevated triglycerides induced by carbohydrates by blocking VLDL and triglyceride metabolism (Harris, et al., 1984). Angina patients showed a lower ration of EPA to AA (arachidonic acid) (Kords, et al., 1986). The authors of the study consider this a new cardiovascular risk factor. Six grams of fish oil per day lowered VLDL and raised HDL while greatly decreasing plasma triglycerides and cholesterol. A

diet high in fish as compared to one high in cold cuts or meat lowered serum cholesterol, blood pressure, and raised HDL (*Atherosclerosis*, 1986). Fatty acids, especially linoleic, oleic, and arachidonic acids, have been shown to reduce angiotensin receptor affinity (Good, Friend, and Ball, 1986). An olive oil-rich diet has been shown to decrease non-HDL cholesterol while leaving triglyceride levels constant (Mesink and Katan, 1987). Eicosapentaenoic acid in the form of cod liver oil or mackerel is an excellent polyunsaturate and lowers cardiovascular risk factors (Singer, 1986).

Hence, all our patients were treated with eicosapentaenoic acid (fish oils - Omega 3), linoleic acid (safflower oil) or gammalinolenic acid (primrose oil) or all three (Smith and Dunn, 1985). Dietary fatty acid intake is of particular importance in relation to blood pressure when weight reduction is occurring (Katz and Knittle, 1985), as is the case with our patients.

Calcium

Numerous studies suggest that calcium may have an important role in hypertension (Bloomfield, et al., 1986; Belizan, et al., 1983; McCarron, 1984; Schleiffer, et al., 1984; *The Lancet*, 1982). An oral calcium load has been shown to decrease systolic and diastolic blood pressure, elevate PGE₂, decrease PTH, decrease norepinephrine, and decrease 1,25 dihydroxy-Vitamin D (Yoshikatsu, et al., 1986). Hypertensive patients showed significant deficiencies in dietary calcium, potassium, vitamin A, and vitamin C with calcium being the most consistent dietary risk factor for hypertension (McCarron, et al., 1984). Preliminary reports show that oral calcium supplements (1 to 2 grams per day) lower blood pressure in some patients, particularly in young adults, possibly more so in women (Belizan, 1983). However, manipulation of dietary calcium may not be very useful in older women (Schramm, et al., 1986). Oral calcium carbonate administration also seems to have an effect on mild hypertensives (Bloomfield, 1986). Calcium citrate is probably the best therapy.

In one study, calcium supplementation reduced blood pressure in young adults (Belizan, et al., 1983). Calcium supplementation of up to 1000 grams has been shown to lower blood

pressure in mild to moderate hypertension (McCarron and Morris, 1985). Furthermore, surveys have shown a positive relationship between blood pressure and serum calcium levels. Acute elevation of circulating calcium levels during elevation of blood pressure, chronic hypercalcemia or hyperthyroidism, and vitamin D intoxication are all associated with increased chronic hypertension (Sowers, et al., 1985). Calcium supplementation may lower elevated blood pressure by increasing natriuresis (sodium excretion) (Gilland, et al., 1987). Calcium can partially alleviate high blood pressure in the spontaneously hypertensive rat due to its renal productions of dopamine, a probable natriuretic factor (Felsiceita, et al., 1986). Three clinically paradoxical findings in the relationship of calcium and hypertension are as follows: calcium mediates vascular smooth muscle; calcium channel blockers lower blood pressure; and increased calcium intake can also relieve hypertension (McCarron, et al., 1987). A recent hypothesis says that there is a circulating plasma factor which increases intracellular platelet coagulation in hypertension. This factor may on cells and thus increase peripheral vascular resistance (Lindrer, et al., 1987).

In contrast, several studies have shown that calcium can be a factor in elevating hypertension. Therefore, we use calcium sparingly except in the case of a woman suspected of having osteoporosis or in cases of/in normal plasma, ionized calcium or red blood cell calcium (Schleiffer, et al., 1984; Bloomfield, et al., 1986; Cappuccio, et al., 1985; *Nutrition Reviews*, 1984; Belizan, et al., 1983; Kesteloot and Beboers, 1982; Sica, et al., 1984; Staessen, et al., 1983; Weinsier and Norris, 1985; McCarron, et al., 1985; Johnson, et al., 1985; Stern, et al., 1984).

Furthermore, Schedl (1984) pointed out the need for vitamin D in blood pressure control. Sowers et al. (1985) also correlates vitamin D and calcium intake with blood pressure among women. When we use calcium supplements, we use them with vitamin D.

Magnesium

Magnesium, in contrast to calcium, is well-known to lower blood pressure, and has been used in the treatment of hypertension in pregnancy for a number of decades (Lee, et al., 1984; Dyckner and Wester, 1983). Magne-

sium, calcium, phosphorous, potassium, fiber, vegetable proteins, starch, vitamin C, and vitamin D showed an inverse relationship with blood pressure with magnesium's correlation being the strongest (Joffres, et al., 1987).

Magnesium is a vasodilator, according to Wallach and Verch (1986), and can at high levels cause low blood pressure (Fassler, et al., 1985). The use of various nutritional substances as pharmacological agents for hypertension has produced many success stories. Nevertheless, magnesium therapy has been instituted for hypertension to combat a deficiency state often inflicted by diuretic usage (Braverman, 1987). In a study with Finish ewes, hypomagnesium was correlated with hypertension (Weaver, 1986). Magnesium deficiency may relate to high blood pressure by increasing microcirculatory changes or microcirculatory arteriosclerosis (Altura, et al., 1984). Intracellular free magnesium levels are inversely linked to blood pressure independent of calcium metabolism (Resnick, et al., 1986). Direct lowering of blood pressure with magnesium in patients with high blood pressure has been demonstrated by Dyckner and Wester (1983). Magnesium works like a calcium channel blocking drug (i.e., Verapamil, Diltiazem) (Iseri and French, 1984; Sjogren and Edvinson, 1985; Platonoff, et al., 1985; Flodin, 1985).

Altura and colleagues suggested that magnesium supplements have a valuable effect on diabetic and hypertensive rats (Altura and Altura, 1984). Magnesium's use has been documented in cardiac situations, such as digitalis toxic arrhythmias due to magnesium depletion and myocardial infarctions due to decreases in potassium (Rasmussen, et al., 1986; Cohen and Kitzes, 1983; Delhumea, et al., 1985; Cassadonte, et al., 1985). Magnesium may be an important prophylaxis in hypertensive patients prone to arrhythmia. Untreated hypertensives showed lower levels of intracellular free magnesium which strongly correlates to systolic and diastolic blood pressure (Resnick, et al., 1984). Sempos and colleagues (1983) found that hypertensive patients using diuretics had a magnesium level of 1.79 mg to 100 ml compared to normotensive patients with 1.92 mg to 100 ml, a significant difference. Magnesium is also low in blood mononuclear cells in intensive care

cardiac patients (Ryzen, 1986). Serum magnesium deficiency was noted as well in a medical ICU unit in Los Angeles County (Ryzen, 1985). Furthermore, we have shown in seven patients a significant decrease in red blood cell magnesium (see Table 1, page 239) as compared to the mean of normotensive individuals. Type A personalities have been shown to lose red blood cell magnesium under stress and thus show a correlation to their behavioral tendency to eventually develop hypertension, ischemic heart disease, and coronary vasospasms (Henrotte and Plovin, 1985). Further support for magnesium's use in hypertension treatment is documented by Wester and Dyckner (1985) who claim that magnesium acts by vasodilation or by a sodium potassium ATPase metabolism. Magnesium metabolism was abnormal in the spontaneously hypertensive rat (Berthelot, et al., 1985). Hypomagnesia in acute myocardial infarction patients was probably due to magnesium's migration from a cellular to intracellular space and not from renal losses (Rasmussen, et al., 1986). Magnesium deficiency has been shown to be sometimes related to dietary habits (Sheehan, et al., 1984). Hence, many of our patients receive magnesium. In addition, many of our hypertensive patients tend to have constipation which is relieved by magnesium.

Sulfur Amino Acids

A study by Ogawa and colleagues (1985) suggested that decreases in plasma taurine and methionine were significant in patients with essential hypertension. Decreases in plasma serine and threonine were also significant although not therapeutically relevant. Taurine may lower blood pressure. Furthermore, all sulfur amino acids - methionine, cysteine, and taurine - lower heavy metals which are often factors in hypertension.

In our study we found a trend toward decreases in plasma cystine, probably due to B₆ deficiency. Further studies by Paasonen, et al. (1980) suggested there may be an elevation in platelet taurine. Hence, most of our patients with hypertension receive supplemental sulfur amino acid treatment. Three grams of taurine daily could elevate blood taurine levels 2 to 3 times normal (Braverman and Lamola, 1986). We considered this an appropriate level to reach for hypertensives. Paradoxically, plasma

taurine was elevated in our patients (see Table 2, page 239).

Sodium and Potassium

The role of dietary sodium in hypertension is long-standing and well-known (Liegman, 1985). It has been suggested that the average person consumes 10 to 12 grams of sodium, which should be reduced to 2300 mg per day (Tufts University Diet and Nutrition Letter, 1985). This can be counterbalanced by increasing potassium intake (Emsley, 1984), which may lower blood pressure. A higher ratio of potassium to sodium has been shown to lower moderately high blood pressure. Potassium therapy is useful in lowering blood pressure induced by diuretic-induced hypercalcemia (Carnegie, et al., 1983). An inverse relationship between serum potassium and blood pressure was shown by Loft, et al., 1985. High potassium intake greatly reduced brain hemorrhages, infarctions, and death rate in spontaneous hypertensive rats (Tobian, 1986). A high potassium intake may help to alleviate high blood pressure, the leading risk factor for smokers (Khaw, Barnet-Carment, 1987).

Biochemical abnormalities, including hypokalemia and alkalosis due to amnesia, have been correlated with vascular headaches for hypertensive patients (Colen, 1986). Linolenic acid is the precursor for prostaglandins and omega 3 fatty acids and has a profound effect on blood pressure (*The American Journal of Clinical Nutrition*, 1986).

The hazards of high sodium intake are beyond hypertension and include gastric cancer. Dietary sodium affects urinary calcium and potassium excretion in men with regular blood pressure and differing calcium intakes (Castermiller, 1985). Anderson (1984) suggested that stress and salt are cyclical, meaning increased salt intake produces stress and craving salt is a sign of stress. Dietary sodium and copper have long-term effects on elevating blood pressure in the Long-Evans group of rats (Wu, et al., 1984). Decreased sodium intake can decrease stress (Castenmiller, et al., 1985; Richards, et al., 1984; Vollmer, et al., 1984; Karppanen, et al., 1984; Kaplan, et al., 1985; Montesetal., 1985; Treasure, et al., 1983; Voors, et al., 1983).

Some essential hypertensives have a low sodium to potassium and/or a high lithium to

sodium counterpart. One study shows that hypertension in spontaneous hypertensive rats is caused by a circulating hypertensive agent produced by the kidneys and adrenals whose secretion can be suppressed by volume or salt depletion (Spieker, et al., 1986). Hence, all of our patients are asked to restrict sodium as completely as possible and use salt substitute. We suggest to all our patients that they use high potassium salt substitutes.

Trace Elements and Hypertension

Numerous studies have suggested that elevations in serum copper can raise blood pressure. Excess dietary copper can increase systolic blood pressure in rats, according to Wu and colleagues (1984) and Liu and Medeiros (1986). Elevations in serum copper and cadmium have been found in smokers, which may be the reason why they have elevated blood pressure, according to Davidoff and colleagues (1978) and Kromhout, et al. (1985). Serum copper was inversely related to HDL level (Kromhout, et al., 1985). Contraceptive pill users have elevations in serum copper and elevations in arterial pressure (Staessen, et al., 1984). Patients who suffered from myocardial infarctions had decreased levels of zinc and iron but increased nickel levels (Khan, et al., 1984). Hypertensive subjects that use diuretics have significantly higher serum copper levels. Increased serum copper has a role in primary or pulmonary hypertension (Ahmed and Sackner, 1985). Zinc lowers serum copper and may actually lower blood pressure (Ahmed, Sackner, 1985). Higher dietary zinc intake has been associated with lower blood pressure (Pfeiffer, 1975; Medeiros and Brown, 1983). Zinc is depleted by diuretics (Olness, 1985).

Increased red cell content of zinc in essential hypertension has been found by Frithz and Tonquist (1979) and Henrotte, et al. (1985). Zinc is a well-known antagonist of heavy metals such as cadmium and lead (Pfeiffer, 1977), which even in chronic dosages has been found to elevate blood pressure. Hence, all our hypertensive patients receive zinc to lower copper, lead, cadmium, and manganese. Studies suggesting that sub-acute elevations in cadmium and lead have a role in the elevation in blood pressure have been done by Staessen, et al., 1984; Hulon, et al., 1985; Perry, et al., 1979; and the *AMA News*, 1985.

Blood lead levels, which are elevated in chronic alcoholism, have been correlated with increases in blood pressure (Dally, et al., 1986). The correlation of blood lead to blood pressure is stronger for systolic than diastolic blood pressure (Kromhout, et al., 1985). An overabundance of lead can lead to a form of hypertension with renal impairment (Batuman, et al., 1983). The lead content of the ventricles and aorta of myocardial infarction victims was consistently greater than for normal patients though not significant. Further evidence for a relationship between blood lead levels and blood pressure is presented by Prickle and colleagues (1985). Serum zinc levels were significantly lower for older hypertensive women and older men with high systolic readings (Medeiros and Pllum, 1984; Harlan, et al., 1985). Elevations of lead and cadmium with decreases in zinc are a factor in many inner city patients with hypertension. Plasma zinc levels were significantly lower in patients having coronary heart disease risk factors (Kushliedaite, et al., 1984). Furthermore, it has been shown by Pfeiffer (1977) that vitamin C in combination with zinc may be an even more effective way of reducing subacute levels of lead and cadmium. Hence, manganese levels are directly correlated to LDL and inversely correlated to HDL (Kushliedaite, et al., 1984). We have had every patient follow a treatment plan which included zinc therapy. It is a consistent clinical observation to see rises in blood pressure with as little as 20 mg of manganese per day. A group of hypertensive females has been shown to have decreased intake of phosphorous, potassium, and magnesium (Karanja, et al., 1987).

Vitamin B₆ and Hypertension

It has been established by Dakshinamurti, et al. (1986) that Pyridoxine deficiency has a role in hypertension. Vitamin B₆ inhibits platelet aggregations through its metabolite pyridoxal 5' phosphate (Fletcher and Rogers, 1985). Pyridoxine deficiencies which can cause hypothalamus 5-Ht and GABA deficiencies (neurotransmitter involved in blood pressure regulation) as well as general increases in sympathetic stimulation can cause blood pressure to become elevated (Paulos, et al., 1986). Besides being a co-factor for transamination, vitamin B₆ seems to relieve

edema and swelling and thus has mild diuretic properties. It is known that Pyridoxine (vitamin B₆) has diuretic properties; therefore, all of our patients receive Pyridoxine.

Niacin

Niacin, possibly because of its flush or vasodilating producing properties, can lower blood pressure as a vasodilator and can raise HDL fraction which is frequently reduced in hypertensive patients. Niacin administration is a very effective agent against an increased level of LDL in patients with type II hyperlipoproteinemia. It also significantly raises HDL levels (Hoeg, et al., 1984). Niacin has also been shown to reduce the average numbers of lesions per subject and block new atheroma formation. Niacin, when used alone or in conjunction with the drug colestipol, can effectively lower cholesterol and triglyceride levels to the normal physiological range (*Journal of Lipid Research*, 1981). Niacin is used as an adjunct therapy in our treatment.

Selenium

Serum selenium of patients with acute myocardial infarction was determined to be low before this condition occurred and not as a result (Oster, et al., 1986). Further evidence for serum selenium levels and cardiovascular death correlation comes from the work of Virtamo and colleagues (1985). Chromium concentrations in aortas of patients dying from atherosclerotic disease are significantly lower as compared to a control group. Low plasma chromium was found in patients with coronary artery and heart diseases (Somonoff et al., 1984). Both selenium and chromium may have a role in the nutritional control of hypertension, at least in the protection from myocardial infarction during a difficult dietary period.

Tryptophane

Tryptophane may have a role in hypertension too. It has been established by Feltkamp and colleagues (1984) and Wolf and Duhn (1984) that tryptophan in dosages of 3.5 g/day can lower blood pressure.

Other Nutrients

Vitamin C stabilizes vascular walls and helps metabolism of cholesterol into bile acids. When elderly patients receive 3 grams of

inositol, their total blood lipid and cholesterol levels decreased (Leingard and Moore, 1949).

Garlic has been shown to be of great benefit in hypertension therapy, raising HDL and lowering both the total cholesterol and LDL. Garlic oil decreases platelet aggregation, serum cholesterol, and mean blood pressure, while it raises HDL and red blood cell arachidonic acid. Thus garlic has been shown to be an antiatherosclerotic, antithrombotic, and an anti-hypertensive agent (Banie, et al., 1987). Vitamin E lowers cholesterol and effects prostaglandin synthesis (Fletcher and Rogers, 1985), yet vitamin E, by clinical observation, raises blood pressure (Sharma et al, 1976; Keyes, 1980; Bordia et al, 1977).

Melatonin, according to Birau and colleagues (1981) and Kawashima and colleagues (1984) may have a role in regulating hypertension. Manipulation of dietary calcium may not be very useful in older women (Schramm, et al., 1986). CoQ10 has been deficient in approximately 40 percent of hypertensives and has a possibly beneficial effect on hypertensive therapy (Cardiovascular Research Ltd., 1985).

One study has shown that nutritional and hormonal treatments can enhance the sodium potassium ATPase activity level and in turn helps to prevent or treat essential hypertension (McCarty, 1984). One study has shown estrogen given to postmenopausal women reduces heart attack risk (Healthline, 1986). Alcohol abuse, lead poisoning, birth control pills (estrogen), licorice (glycyrrhizicals), diseases of the kidney, adrenal or pituitary glands, pregnancy, and pre-eclampsia are some common causes of hypertension (Laragh, 1987).

Although there are some things written on progesterone by Rylance (1985), melatonin by Birau and colleagues (1981), and atrial peptides by Cantin and Genest (1986), as hypertensive agents their use is unclear. Patki, et al., 1990 once again demonstrated the benefits of potassium 60 mg. a day in lowering arterial blood pressure. Total body potassium content may be more useful in producing and utilizing potassium in hypertension treatment. Abu Hamdon et al., 1987, suggested that side effects of drugs like Captopril and angiotensin 2 blockers might be helped by the addition of zinc. Saito et al., 1988, suggested the benefits of magnesium therapy. Boulos et al., 1988,

suggests warnings about calcium supplements being contaminated. Numerous studies suggest the benefits of 24-hour blood pressure monitoring (Weber et al., 1988). This is an extremely important breakthrough in the management of hypertension.

Chronic lithium administration, because of its reduction in stress and increased secretion of sodium, may also benefit blood pressure according to Ide et al., 1988. Kestaltloot et al., 1988, suggests that the relationship of sodium, potassium, calcium, and magnesium is critical to normal blood pressure. Cootman et al., 1990, suggests the benefits of dietary treatment in hypertension. J. David Spence et al., 1990, suggests the effect of any stress reduction technique on blood pressure. Numerous studies suggest the benefit of doing body composition analysis in patients with high blood pressure to monitor weight loss and fluid retention such as Troisi et al., 1990.

Body composition testing can help predict and follow overall recovery from obesity and the natural approaches toward blood pressure. Iacona et al., 1990 again show the detrimental effects of high fat diets. Levinson et al., 1990 have again demonstrated the beneficial antihypertensive effects of fish oil. Seelig et al., 1990, suggests the benefits of magnesium therapy in numerous groups. Sauter and Rudin, 1990 suggest once again that if one has to use hypertension drugs, calcium antagonists may be the best because they can reduce brain damage from stroke and damage to the heart and other organs. Jule et al., 1990, has again suggested the benefits of nonpharmacological nutritional therapies in the treatment of hypertension. Digiesi et al., 1990, suggested the benefits of 100 mg or more of CoQ 10. Mills et al., 1989, have suggested that the borage oil supplement may be even better than the primrose oil supplement in lowering blood pressure. Hypertension and nutrition research marches onward. Everyone with hypertension and/or a family history needs a dietary and nutritional regimen.

Biochemical Individuality/Genetic Differences

It is very important for both the physician and hypertension patient to realize that every human being is genetically, and thus, biochemically distinct. Dietary or drug regimens have different effects on different patients.

The influence of diet on blood lipid levels is not predictable for each individual due to different genetic traits. Sodium restriction is generally recommended in an anti-hypertensive diet, and in most cases, this reduces blood pressure through volume effects. Sodium restriction is beneficial for the majority of hypertensives (Huddy, 1986). A recent epidemiologic study showed sodium restriction to be of no value in a small subgroup of the population at large (Cardiology Observer, 1987). Furthermore, in a small group of patients, sodium restriction actually increases the activity of the angiotensin system, and thus raises blood pressure. According to Dr. Weinberger of Indiana University, dietary sodium restrictions shows a heterogeneity of responses due to genetic differences in the renin-angiotensin-aldosterone system (Weinberger, 1986). Not all people respond similarly to the same levels of electrolyte intake or patterns of multiple electrolyte intake, hence biochemical individuality (Harlan and Hadan, 1986).

Dietary cholesterol, as found in eggs for example, usually does not significantly raise serum cholesterol in most patients if the patient is on a proper dietary regimen. Nevertheless, not all patients can consume large quantities of eggs without an increase in serum cholesterol. The dietary recommendations made in this paper work exceptionally well in a vast majority of the hypertensive population, but some trial and error might be needed to tailor the program to a patient's specific biochemical needs. Clinical judgement of which nutrient and diet to use can be refined by measuring, plasma fatty acids, plasma amino acid, red blood cell trace elements, hair analysis and vitamin levels. Following Sed rates, cholesterol epolepsotermis, and fibrinogen levels is also useful.

New Data on Hypertension

Who will have a heart attack? Does hypertension increase risk? We know that individuals who have a high risk for heart attack have some of the following biochemical features: elevated fibrinogen, elevated renin levels, elevated cholesterol, low HDL, low apolipoprotein A.

Therapies for Hypertension, Updated

Patki et al., 1990, once again demonstrated the benefits of potassium, 60 millimols a day, in lowering arterial blood pressure. Total body potassium content may be more useful in predicting and utilizing potassium in hypertension treatment. Polert et al., 1990, has again shown the fact that blood sugar problems and insulin resistance are characteristic of hypertension. Geygeretal., 1989, has again shown the role of cadmium in contributing to high blood pressure. Cappuccio et al., 1989 has again shown the benefits of calcium therapy in hypertension. Rinner et al., 1989 has again shown the benefits of sodium, potassium, calcium, magnesium modifications in blood pressure. Lauten et al. have also shown the benefits of dietary potassium in blood pressure. Marraccini et al., 1989 have shown the dangers on overall health of hypertension. Hoffman et al., 1988 have again shown the possible problems of increased sodium. Morris et al., 1989 have again shown how low level lead can raise blood pressure. Lind et al., 1987 have again shown the benefits of vitamin D which can help the absorption of calcium. Yuricomi et al., 1988 have again shown the role that sulphur and amino acids can regulate blood pressure through changing brain chemistry. Radak and Deck, et al., 1989 have again shown the roll of fish oil in blood pressure. Bak et al., have again shown the fact that caffeine can have an impact on blood pressure. Stamler et al have again shown that overall approach to hypertension must first be nutritional, dietary, and hygienic. Luft et al., 1989 have again shown that dietary interventions are critical to monitoring blood pressure.

Depet et al., 1990 suggested the possible benefits of calcium supplementation to hypertension. Salvaggio et al 1990 has suggested the role of caffeine in raising blood pressure. Steiner et al., 1989 suggested the benefit of fish oil in hypertensive patients. Ashry et al, 1989 suggested the benefit of linoleic acid and safflower oil in lowering blood pressure. Baksi et al 1989 suggested a role for low calcium in elevated blood pressure. Tractman et al., 1989 again suggested the role of taurine in lowering blood pressure. Oberman et al., 1990 again suggested of nonpharmacological and nutritional treatments in lowering blood pressure. Triber et al., 1989 has again shown the relationship

between hostility and raising blood pressure. Rouse et al., 1984 has again shown the role of vegetarian diet when all other approaches fail in lowering blood pressure. Kaplan, 1990 has again shown the role and benefit of nonpharmacological therapy. Multiple studies have shown the benefit of nutritional and nonpharmacological therapy and it is time to make use of hypertensive nutrients.

Our antihypertensive nutrient called the heart formula is the best nutritional supplement to date. Dye et al., 1990 have shown that sucrose and glucose ingestion can raise blood pressure. Golub et al., 1990 have suggested the possible benefits of bio flavonoids in lowering blood pressure. Hsieh et al., 1990 suggested again the benefits of magnesium therapy in hypertension.

Jule et al., 1990, has again suggested the benefits of nonpharmacological nutritional therapies in the treatment of hypertension. Digiesi et al., 1990 suggested the benefits of 100 mg or more of CoQ10. Mills et al., 1989 have suggested that the borage oil supplement may be even better than the primrose oil supplement in lowering blood pressure. Hui et al., 1989 again suggested the benefits of fish oil therapy.

Therapies for Hypertension

Chronic lithium administration, because of its reduction in stress and increased secretion of sodium lithium therapy, may, on occasion, also benefit blood pressure according to Ide et al, 1988. Kestaltloot et al., 1988 again suggests that the relationship of sodium, potassium, calcium, and magnesium is critical to normal blood pressure.

Iacona et al., 1990, have again shown the detrimental effects of high fat diets. Levinson et al., 1990 have again demonstrated the beneficial antihypertensive effects of fish oil. Seelig et al., 1990 suggest the benefits of magnesium therapy. Boulos et al., 1988 suggests warnings about calcium supplements being contaminated by lead.

Side Effects of Drugs

AbuHamdonetal., 1987 suggested that the side effects of drugs like Capropril and angiotensin 2 blockers might be helped by the addition of zinc.

New Tests

Numerous studies suggest the benefits of 24-hour blood pressure monitoring (Weber et al., 1988). This is an extremely important breakthrough in the management of hypertension because of the diagnostic accuracy of thirty blood pressure readings.

Numerous studies suggest the benefit of doing body composition analysis in patients, such as Troisi et al., 1990 suggest the benefits of body composition testing in predicting overall recovery from obesity and beneficial approaches toward blood pressure. Drayer, 1985 and Pickering et al., 1988 have suggested at least 20 percent of hypertension patients are misdiagnosed because ambulatory blood pressure monitoring is not used. O'Brien et al., 1990 has again shown the benefits of 24-hour blood pressure monitoring in determining blood pressure. Belini et al., 1990 has again shown the benefits of the influence of lowered body composition on blood pressure.

Sauter and Rudin, 1990 suggest once again that if one has to use hypertension drugs, that calcium antagonists may be the best because they can reduce brain damage from stroke and damage to the heart and other organs.

Case Histories

1. Removal of Multiple Drugs

G.F. is a 51 -year-old male on multiple medications, weighing 265 pounds with a 25-year history of smoking 2 packs of cigarettes per day. He stopped smoking 3 years ago. He had BP of 150/100 and 140/100 with a pulse of 74. He was taking Aldomet, Klotrix, Hydrochlorothiazide for 10 years and Nitropatch nightly. He was put on a weight reducing, low carbohydrate diet and started on a multivitamin, 6 per day; B₆, 500 mg; magnesium Orotate, 3 grams; garlic, 1440 mg; taurine, 3 grams; primrose oil (dihomogammolinolenic acid), 3 grams; Max-EPA (eicosapentanoic acid), 6 grams; magnesium oxide, 1.5 grams per day; and Klotrix, 4 per day or 40 mcg. Blockadrin was reduced to 2 and Aldomet reduced to one.

After one month, his BP was 144/104 (increase in BP can occur in early reversal of drugs), weight 248, and on 1/28 his BP was 120/88 and weight 249. Aldomet was stopped and Blockadrin was maintained. On 2/11 his BP was 140/90, pulse 78, and weight 235. Blockodrin was reduced to one pill, but he

still used Nitropatch. On 3/11 BP was 140/94 and weight 226, and Blockadrin was stopped. Taurine was reduced to 2 grams and garlic to 960 mg. He was no longer on any medication except Nitropatch for BP. Klotrix was reduced to 3 tablets, and fish oil was switched to Mega-EPA, a more potent brand of EPA. On 4/10 his BP was 150/90, pulse 78, and weight 216. On 5/23 his BP was 130/70, pulse 80, and weight 214 pounds, and Nitropatch was stopped. Medication was reduced to 4 multivitamins, 4 garlic, 60 mg; taurine, 3 grams; primrose oil, 2 grams; fish oil, 6 grams, and his antihypertensive formula was stopped. From 3/11 on he was taking 2 zinc pills per day, magnesium oxide 1000 mg (substituted for magnesium orotate), and niacin, 1 gram per day. Safflower oil, 2 tbsp. per day was also prescribed from 3/11 on, and vitamin C, 2 grams per day from 4/10 on. Chromium 200 mcg/1 tab/day was taken from 5/22 on.

Hence, this patient, through the use of mega-nutrient therapy, was completely removed from drugs. His BP remains stable at 130/70. On 12/19 his cholesterol was 290 and triglycerides were 280. On 3/27 his triglycerides were 122 and cholesterol 223. He occasionally used vodka, coffee, and tea. His sex drive was increased gradually throughout the treatment, and exercise (walking) gradually increased.

2. Removal of B-Blockers

A 42-year-old male, 5'10", weighing 179.5, was on Corgard for 2 years, drinking 2 cups of coffee a day, with a high sex drive and craving for salt. His BP was 150/90 on 5/16, and he was started on multivitamins, 500 mg vitamin B₆, 200 mcg folic acid, 250 mcg vitamin B₁₂, 3 grams magnesium Orotate, 3 grams taurine, 1500 mg garlic, 3 grams primrose oil, and 6 antihypertensive heart formula. On 5/30 his BP was very good at 128/82. He was off Corgard, with a pulse of 86 and weight 173.

3. Removal of B-Blockers

A 51-year-old female with a 10 year history of hypertension was presented to us for treatment. She weighed 150 pounds at 5'3", and was taking Lopressor 50 mg morning and evening. She did not smoke or use alcohol or tea. On 5/23 her BP was 194/120, with a pulse of 116 and weight of 150. She began taking multivitamins, 2 vitamin B₆, 500 mg, 60 mg

folic acid (for atrophic vaginitis), 3 grams magnesium Orotate, 2 grams magnesium oxide, 3 grams taurine, 1440 mg of garlic, 6 grams Mega-EPA, 6 pills antihypertensive or heart formula. She returned on 6/12 with BP 160/100, having gone 3 weeks without a migraine for the first time in years.

Her regimen was adjusted to 50 mg magnesium Orotate and 3 grams magnesium oxide, 2 grams taurine, 600 mg calcium carbonate, 3 grams primrose oil, 100 mg niacin, 200 mcg chromium and 200 mcg selenium and 50 mg Lopressor with instructions to go off 50 mg of Lopressor if there was improvement in 2 weeks. She returned drug free and her BP was 130/80. The rapid recovery of this patient was due to following a stricter diet of fish 2 times daily, meat 2-3 times per week, 3 tbsp. safflower oil, and frequent use of ginger, garlic and onions.

4. Removal of Diuretics

A 57-year-old male, 5'6" came to us for treatment in December with a BP of 160/100. He was taking Corgard, had a moderate sex drive, did not use caffeine, did not exercise, and had a 30-year history of hypertension. He started on 2 GTF morning and evening, 1 gram vitamin C morning and evening, Ziman (zinc 10 mg, manganese 2 mg) 10 drops, selenium 200 mcg morning, molybdenum, Max-EPA 6 grams day, taurine 500 mg day, magnesium Orotate 2 grams day, and Corgard was reduced to 30 mg day.

On 1/8 niacin was added (timed-release evening), and his weight had fallen to 154, with BP 120/75. On 2/5, Corgard was reduced to half a pill every other day, and he was advised to stop it in 2 weeks. Safflower oil 1 tbsp. morning and evening was added, zinc 50 mg morning and evening, dolomite (routine dose) one morning and evening, and all medications remained stable. On 3/17 he was feeling lightheaded and came in with a BP of 85/70 and a pulse of 90. Medication remained the same (he should have stopped Corgard 2/19), but safflower oil was stopped. He returned on 4/1 with BP 132/62, pulse 62, and weight 149. Initially his triglycerides were 256, cholesterol 190, and HDL fraction was 26 (high coronary risk). On 3/17 triglycerides were normal at 153, with cholesterol of 176 and an HDL fraction of 41 with all drugs removed.

5. Removal of Diuretics

A 62-year-old female, 5'3", with a 15-year history of hypertension came to us for treatment. She had been treated for 20 years with Hygroten 50 mg/day and Zylprim (because of gout induced by Hygroten). Twenty-eight years ago she went through menopause and as a result had a diminished sex drive. Her BP was 160/100 and her weight was 204. Hygroten was stopped, and she was put on a dyazide (instead of a diuretic) every other day. She was then permitted no fried or salted foods and was asked to follow a low carbohydrate diet (Appendix). She was started on a multivitamin one per day in July, chromium, 200 mcg morning and night, vitamin C, 2 grams morning and night, vitamin B₆, 500 mg in the morning; taurine, 1 gram per day; primrose oil, 2 grams morning and evening; zinc, 50 mg morning and evening; and safflower oil, 1 tsp. morning and evening. The diuretic was finally stopped on 12/9, and her medication was changed to 1 multivitamin morning and night; vitamin B-complex-50,1 per night; GTF, 1 morning and night; niacin, 500 mg morning and night, vitamin B₆, 500 mg morning; vitamin C, 1/2 tsp. morning and evening; thyroid, 1 grain morning (per lab results); selenium, 200 mcg morning; kelp, 2 morning and evening; Max-EPA, 1 gram morning and evening; taurine, 500 mg morning and evening; zinc, 50 mg evening. On 4/30 her BP was 130/70, and she was without the use of diuretics and her vitamins were reduced gradually without elevation of blood pressure.

6. 15-year History of Hypertension

A 53-year-old male, 5'11 1/2", with a 15-year history of hypertension, taking one Maxzide a day, 300 mg Logressor a day, 20 mg Minipress a day, and one Zylprim, 300 mg a day for the treatment of gout. He had a moderate sex drive, drank two cups of coffee a day, and had as many as three to seven drinks per week. His BP was initially 130/85, pulse 66, and weight 209 pounds. His triglycerides were 327, HDL 46 percent, and had a high cholesterol of 255. He was started on one multivitamin a day, GTF chromium 2 mcg morning and evening, niacin 400 mg time release/am and pm, vitamin B₆ 500 mg/AM, vitamin C, calcium and magnesium powder 1/2 tsp. am and pm, magnesium orotate 500 mg/2 am and pm, taurine 500 mg/2 am and pm, methionine 500 mg/am and pm, mega-

EPA 2 grams am and pm, and zinc gluconate am and pm.

Maxzide was changed to every other day, and he was instructed to stop it if lightheadedness developed. He was put on a carbohydrate-deprivation diet, a protein and vegetable diet with no bread or fruit. On 5/6 he returned with BP of 118/78, pulse 60, and weight 196 pounds. During that period, Lopressor had been reduced to 100 mg am and pm by phone conversation, Minipress was 5 mg am and pm, and Maxzide was stopped based on BP's had done at home on his machine. On 5/6, vitamin C was reduced due to diarrhea, but the other supplements were virtually the same, but generally increased GTF chromium up to 200 mcg/2 tabs am and pm, multivitamin am and pm, niacin increased to 3 times a day, vitamin B₆ 500 mg/am, selenium 200 mcg, methionine 500 mg/am and pm, taurine 1 gram am and pm, magnesium Orotate (1 tab reduced because of diarrhea), Mega Epa 3 gram/am and pm, primrose oil am and pm, and zinc 15 mg am/30 mg pm.

On 5/27 patient returned with BP of 120/80, weight 188, triglycerides now normal at 97, cholesterol reduced to 238, and HDL increased 51 percent. Patient had now gradually reduced Minipress to 5 mg/day and Lopressor 100 mg/every other day, magnesium oxide was stopped due to diarrhea, zinc gluconate 15 mg am/30 mg pm, lithium 1/pm, mega-EPA 3 gram/am and pm, taurine 1 gram am and pm, tryptophan 500 mg/am and pm, methionine 100 mg/am and pm, niacin reduced back to 2/day, GTF 2/day, and multivitamin doubled/am and pm.

On 6/25 the patient returned with weight of 184, BP 110/80, having been on Lopressor 100 mg every other day and pulse 75. He was not put on Lopressor (50mg/day). On 7/15 the patient was drug free and BP of 120/80 consistently over multiple readings.

7. Heart Formula for Blood Pressure Lowering

A 53-year-old male with a 15-year history of poorly controlled hypertension came to us, being treated with diuretics, Minipress, Lopressor, and Zylprim. He was taken off all drugs with the simple application of weight loss and the mega-nutrient therapy, 10 pills a day of antihypertensive or heart formula, in the pharmacological treatment of hyperten-

sion.

8. Getting Off Diuretics

A 65-year-old female, 5'6", with a long history of hypertension, who had been treated with diuretics, with BP 170/110, pulse 102, weight 170, triglycerides 70, cholesterol 234, HDL 65, was presented to us and was placed on a low carbohydrate diet and supplement regimen. This included: GTF 1 am and pm, niacin timed-release 2 pm, 1 gram pm, vitamin B₆ 500 mg am, vitamin E 400 mg am and pm, vitamin A 25000 IU am, selenium 200 mcg am, Max-EPA 3 grams am, 2 grams noon, 3 grams pm, tyrosine 2 grams am, methionine 1 gram am, dolomite 2 at bedtime, and Ziman fortified 1 am and pm.

The patient returned on 1/21 with BP 152/90, with a weight of 164, and was put on Max-EPA and primrose oil 1 gram am and pm, vitamin C 2 1/2 grams am and pm, safflower oil 1 tsp. am and pm. She then returned on 4/15 with BP 110/85 without medication, pulse 78 and weight of 162. Her BP is well controlled on this regimen and her caffeine consumption has been stopped.

9. Formerly Treated with Dyazide and Lopressor

A 45-year-old female, 5'5", treated with Dyazide and Lopressor, had a BP of 130/80, weight 105, triglycerides 115, and cholesterol 178. She started with multivitamins 1/day, GTF chromium 200 mcg 1 am, niacin 400 mg timed release am and pm, vitamin B₆ 500 mg am, methionine 500 mg am and pm, tryptophane 1 gram before sleep, taurine 500 mg am, primrose oil 1 1/2 grams am and pm, Max-EPA 1 gram am and pm, bone meal (a calcium supplement) 1 am and pm, and zinc 15 mg am and pm. Dyazide was reduced to two a day and one the next, and Lopressor was stopped. On 8/1 she was taking Lopressor every other day as well as Dyazide, with essentially the same regimen of vitamins. By 11/12 her BP was 110/80, pulse 80, and weight 109. She was off all BP medication.

In sum, this patient highlights the growing effect of nutrients over time, with very little change in her diet except for the reduction of fried foods, caffeine, and white flour.

10. Taken Off Diuretics

A 59-year-old man, 5'8", with a weight of 227, on Hydrodiuril for 10 years, was presented to us with BP 120/80, pulse 60, triglycerides 298, and cholesterol 173. He was started on GTF chromium 200 mcg 1/am and pm, vitamin C 500 mg am and pm, vitamin B₆ 500 mg am, beta carotene 50 mg/day, selenium 200 mcg am, Max-EPA 2 am and pm, cysteine 100 mg am and pm, dolomite 2 am and pm, and Ziman fortified 1 am and pm. He was put on a high vegetable, low fruit, low carbohydrate diet, and Maxzide was changed to 1 tab every other day. He was put on safflower oil 2 tsp. am and pm and taken off of caffeine beverages. Due to lightheadedness the patient had to stop Maxzide shortly thereafter.

On 12/18 his BP was 120/90 without a diuretic, and his weight was up to 238. Vitamins were kept the same except for Max-EPA which was increased to 4 grams am and pm, and taurine was started at 500 mg am and pm. On 3/17 his triglycerides were normal at 153, with cholesterol of 176, and the HDL fraction was improved. By 5/14 his BP was 120/80, and his pulse was 60 without medication. His weight is 227, and he still continues to do well without medication, diuretic free, with reducing the vitamins by half the dose.

11. Ten-Year History of Hypertension

A 56-year-old female, 5' 10", and a 10-year history of hypertension. She drank one to two cups of coffee a day, and was presented to us with a BP of 160/90, pulse 80, weight 185, triglycerides 154, and cholesterol 233, while taking one dyazide daily. She was started on one tab multivitamin a day, GTF one am and pm, niacin 400 mg time-release, vitamin B₆ 500 mg am, taurine 500 mg am and pm, Mega-EPA 1 gram am and pm, magnesium oxide 1 am and pm, and primrose oil 1 gram am and pm.

She was presented on 5/20 with a BP of 140/84, after having stopped diuretics, with a weight of 181 pounds. She had not even begun the magnesium or niacin. Later, she began these treatments and her BP was 120/80 and is well controlled.

12. On Diuretics for Twenty Years

An 80-year-old female, 4'11", on diuretics for 20 years, drinking 4 cups of coffee daily, had a BP of 200/98, pulse 88, and weight

114 3/4. She was started on multivitamins, 500 mg vitamin B₆, 2 grams calcium panthoate, 1 zinc, 1 manganese, 3 grams magnesium Orotate, 1500 mg calcium Orotate, 3 grams taurine, 3 tabs of tyrosine and dhyphenylalanine. On 1/23 her BP was 180/90, pulse 78, and she was removed from diuretics.

Medication was changed to 4 multivitamins/day, 1 gram calcium, 15 mg zinc 2/day, manganese was stopped, 2 grams primrose oil was added, 3 grams vitamin C, and 2000 mg fish oil (EPA). On 2/20 her BP was 174/80, pulse 76, and weight was stable at 113/4. Taurine was reduced to 2 gram, primrose oil increased to 3/4 grams, vitamin C increased to 5 grams, and fish oil to 3 grams. On 3/21 her BP was 150/90, pulse 76 and weight was 116. One tbsp. safflower oil was added with 1 gram magnesium. On 4/18 her BP was 150/80, pulse 76, and weight 114.

Conclusion

In summary, it appears that mega-nutrient therapy can replace much drug treatment, although there may be some difficulty in the age group of 75 and older due to a more advanced stage of the disease and individuals who present on two or more drugs. Catching the disease early and treating with the Orthomolecular approach is the best answer. Treating hypertension can be an art and require thyroid function tests 24-hour free Cortisol renal scan, IVP, 24 hour urine steroids, and plasma renin for the patients that do not respond to either drug or nutrient regimens. At this point we have had an extremely high success rate using mega-nutrients, which have emphasized large dosages of magnesium (particularly in oxide form), large dosages of eicosapentanoic acid (up to 7 grams), large dosages of primrose oil 2-3 grams, safflower oil, vitamin B₆ 500 mg, taurine up to 3 grams, methionine up to 1 gram, niacin up to 2 grams, zinc up to 60 mg, and garlic up to 1500 mg.

Although the number of nutrients replacing drugs can be an enormous amount, we have not seen significant side-effects other than diarrhea from vitamin C and/or magnesium. We have seen virtually no side effects from these nutrients, and patients claim to feel better, do better, feel good about being drug free, and have far less side-effects than any

other regimen so far reported. We usually suggest that all hypertension patients have an initial complete chemical screen, triglycerides, cholesterol, thyroid, as well as BRBC magnesium, trace elements (selenium, chromium, zinc, lead, cadmium) plus amino acids and IgE allergy screen. Orthomolecular treatment of hypertension through diet and nutrients has arrived as a documented and successful approach.

Data

Baseline Therapy for Typical Hypertensive

200 mg vitamin B₆
 200 mg selenium
 1 gram primrose oil am & pm
 GTF 200 mcg am
 2 grams fish oil am & pm 500 mg
 Mag oxide am & pm 15 mg am & pm zinc
 500 mg am & pm taurine 500 mg am & pm
 cysteine garlic am & pm niacin 400 mg am & pm
 tryptophan 1 gram at bedtime
 vitamin C 500 mg am & pm Co-Q10 60-90 mg daily 10-25 meg potassium

PATH (Products for Achieving Total Health

Heart Formula (1-15 pills daily with other vitamins and medications as directed by physician)

Each tablet contains:

Garlic Powder (odorless)	300 mg
Taurine	300 mg
Magnesium (Oxide)	75 mg
Potassium (Chloride)	10 mg
Selenium (Sodium Selenite)	30 mcg
Zinc (Chelate)	6 mg
Chromium (Chloride)	40 mcg
Niacinamide	75 mg
Vitamin C	60 mg
Molybdenum (Chelate)	60 mcg
Vitamin B ₆	75 mg
Beta Carotene	2000 IU

This supplement has been the most useful for treating hypertension.

Diet

- 2 tbsp. safflower oil
- fish daily; poultry cooked without skin often
- no food additives, sugar, refined foods, caffeine, or alcohol
- red meat once a week
- high vegetables (non-starchy type)
- high salad
- 1 slice whole wheat bread (now frequently not allowed until any needed weight loss is accomplished)
- 1/2 fruit

Table 1
RBC Magnesium in Hypertension

	Patient Groups	
	Control	Hypertension Beta Blockers
RBC g	n = 50	n = 7
(RBC mg p < 0.00)	4.4 ± 0.22 meg/L	3.79 ± 1.57

Table 2
Plasma Sulfur Amino Acids in Hypertension

	Control	Hypertension
Cystine	n = 16	n = 7
	2.3 ± 1.5	2.97 ± 1.57
Taurine	n = 16	n = 7
	5 ± 1.3	6.86 ± 2.34
Methionine	n = 16	n = 7
(Taurine p < 0.03)	3 ± 0.9	2.71 ± 0.76

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