New Combination of Nutrients Addresses Three Critical Factors

Chris D. Meletis, ND (with permission from cpmedical.net, access pin: 587556)

In past articles in Vitamin Research News (“Anti-Hypertension Protocol Using Shark Cartilage and Cordyceps Sinesis”) we have discussed how too much parathyroid hypertensive factor (PHF) will increase intracellular calcium and how lowering PHF will reduce calcium entry into vascular smooth muscle, reducing blood pressure. This still remains a very important component of blood pressure control, yet, this is only part of the story. Only 30 percent or more of hypertensive patients have increased PHF levels, leaving a large number of individuals whose hypertension originates due to causes other than high PHF. Nitric Oxide deficiency and the activity of angiotensin-converting enzyme (ACE) are two other equally important factors. Additionally, minimizing arterial calcification also can have impressive effects on blood pressure by improving dilation and blood flow through the arteries. Because physicians can never be certain which of these factors are contributing to an individual patient’s hypertension, they will often experiment with various approaches that address each of these issues until they find the solution.

In this article, I will go beyond the issue of PHF to explain three other causes behind hypertension. I will explain the role that nitric oxide can play in maintaining healthy blood pressure levels. Furthermore, I will discuss how the ACE enzyme is often implicated in high blood pressure and why any blood pressure regimen should also seek to improve arterial function. Finally, I will explain how a new combination of nutritional substances can address all three of these factors.

Nitric Oxide—Say Yes to NO

Optimal blood pressure measurements rely on balanced levels of the gaseous molecule nitric oxide (NO), which signals the blood vessels to relax. NO is produced in the body from arginine and oxygen by various nitric oxide synthase (NOS) enzymes and by reduction of inorganic nitrate. The inner lining of the blood vessels, known as the endothelium, also produces NO and uses it to signal the surrounding smooth muscle to relax. This causes the widening of the blood vessels, known as vasodilation, which increases blood flow and decreases blood pressure. The higher levels of NO activate another enzyme that leads to further relaxation of the vascular smooth muscle. NO can reduce platelet aggregation (the sticking together of blood platelets), thus stopping an excessive number of platelets from adhering to the vessel walls.

Endothelial dysfunction that occurs with lowered NO levels has been linked to hypertension as well as coronary artery disease and atherosclerosis.
Overactive ACE

Kidney cells produce the enzyme renin, which causes an enzymatic cascade that eventually results in hypertension. In the blood, renin splits a globular protein called angiotensinogen into a smaller peptide called angiotensin 1, which is then converted into angiotensin 2 by another enzyme called the angiotensin-converting enzyme (ACE). This process causes blood pressure to rise in high-renin patients. In fact, many anti-hypertensive drugs (called ACE inhibitors) block this enzyme, helping decrease the conversion of angiotensin 1 into 2 that results in hypertension.

Angiotensin 2 is known to have a number of adverse effects. It constricts blood vessels (vasoconstriction) partially by inhibiting bradykinin, a substance responsible for relaxing blood vessels, thereby decreasing blood pressure. However, when bradykinin levels drop due to the release of ACE into the blood, the blood vessels will constrict. Angiotensin 2 also constricts the minute arteries that carry blood away from the kidney, causing increased pressure in the capillary networks of the kidney, and can cause ventricular remodeling of the heart, which may lead to ventricular hypertrophy and congestive heart failure. Furthermore, angiotensin 2 stimulates the adrenal cortex to release aldosterone, a hormone that acts on kidney tubules to retain sodium and chloride ions and excrete potassium. The increased sodium leads to retention of water, which in turn leads to increased blood volume and therefore a further increase in blood pressure. Angiotensin 2 also stimulates the posterior pituitary to release vasopressin (also known as anti-diuretic hormone, ADH), which acts on the kidneys to increase water retention.

Overactive ACE, through its role in the production of angiotensin 2, is implicated not only in hypertension but also congestive heart failure (CHF), left ventricular dysfunction, fibrosis, sarcoidosis and nephropathy in diabetes mellitus. Consequently, ACE inhibitors are frequently used in patients experiencing any of these conditions.

Arterial Calcification

When calcium deposits accumulate on arterial walls, it reduces the elasticity of the blood vessels and this stiffening interferes with the ability of the artery to expand and contract during each heartbeat. Studies have shown that high blood pressure is linked to coronary artery calcification even in healthy adults. In one study of women ages 20 to 35 years, each 10-mmHg change in systolic blood pressure while experiencing a stress-causing activity (playing video games) was associated with a 24 percent increase in the odds of having coronary artery calcification at follow-up. Blood pressure changes that occurred while the subjects played the video game predicted the presence of coronary artery calcification 13 years later.
“To our knowledge, this is the first study that reports blood pressure reactivity to a stressor being related to calcification in the coronary arteries,” the researchers wrote.1

Clearly, protecting the elasticity of the arteries is of concern to anyone who wants to maintain optimal blood pressure levels.

Three-Part Approach

Maintaining healthy blood pressure levels often involves improving nitric oxide levels, inhibiting the enzyme ACE and strengthening arterial health. This approach can be accomplished with a single formula that contains extracts of grape seed and blueberry combined with vitamin K2.

Increasing Nitric Oxide Levels

Grape seed extract is emerging as a nutrient that has profound effects on nitric oxide production. This mechanism of action is thought to be the reason why it can maintain healthy blood pressure levels.

A number of studies have investigated the beneficial effects of grape seed extract. In a study presented at the FASEB 2007 conference, researchers examined the effect of a unique, patented form of grape seed extract on blood pressure in 22 subjects with pre-hypertension. In the United States, more than 69 million adults are prehypertensive and are consequently at a higher risk for heart disease. The current study, therefore, sought to take a look at grape seed’s effect on this condition, which is a threat to cardiovascular health.

Subjects with a systolic blood pressure between 120 and 139 mmHg and/or a diastolic pressure between 80 and 89 mmHg were randomized into a placebo group or an experimental group. The experimental group was given grape seed extract at a dose of 300 mg/day for 8 weeks. The blood pressure was recorded using an ambulatory blood pressure monitoring device at the start of the treatment period and at the end. Subjects taking the grape seed experienced a reduction in systolic blood pressure of 8.3 mmHg and 5.7 mmHg diastolic blood pressure.2

Another study of subjects with the metabolic syndrome was undertaken to determine whether grape seed extract could lower blood pressure in this group of subjects. Researchers randomized the 24 subjects into three groups. One group received 300 mg per day of the extract, another group received 150 mg/day and a third group received a placebo. A 12-hour ambulatory BP recording was made at the start of the study and after 4 weeks of treatment. Serum lipids, blood glucose, plasma insulin and oxidized LDL were measured at the start and end of the study. After 4 weeks subjects receiving both 150 mg and 300 mg of grape seed extract significantly lowered their blood pressure while those on the placebo showed no significant change. The fasting blood glucose, plasma insulin, serum lipids and plasma oxidized LDL concentrations were unchanged. The study results showed that patients on the 150 mg dose of grape seed extract experienced a 13-mmHg drop in systolic blood pressure and a 6-mmHg
drop in diastolic measurements. The group receiving the 300 mg per day experienced a 12-mmHG reduction in systolic blood pressure and an 8-mmHg reduction in diastolic blood pressure.3

In exploring the reasoning behind grape seed’s blood-pressure lowering effects, researchers have determined that the extract causes an endothelium-dependent relaxation of blood vessels and that the mechanism behind this response involves the induction of endothelial nitric oxide synthase.4

In animal experiments, grape seed’s effects on blood pressure were the result of both raising nitric oxide levels and the extract’s ability to act as a calcium channel blocker. This led one group of researchers to conclude that grape seed “has a vasodilation effect not only in an endothelium-dependent, nitric oxide involved manner, but in inhibition of calcium release and blockage of potential-dependent calcium channels.”5

ACE Inhibition

As mentioned above, the enzyme ACE plays a significant role in the elevation of blood pressure as well as other conditions. Research is now showing that a special extract of blueberries may support healthy blood pressure levels by inhibiting ACE. In a preliminary study, researchers treated cells with ACE, then exposed the cells to two different concentrations of the blueberry extract. ACE was inhibited by 98 percent in the cells treated with the highest concentration of the blueberry extract. This preliminary study confirms other studies conducted in rats that showed that blueberry-rich diets affected the vascular smooth muscle contractile machinery of the animals.6

Improving Arterial Elasticity

Vitamin K2 (menaquinone) is a known promoter of arterial elasticity, while vitamin K1 (phyloquinone) is not thought to provide these effects. In a recent study, scientists sought to compare the effects of both forms of vitamin K on arterial calcification. Researchers evaluated the intake of vitamin K1 and vitamin K2 of 564 post-menopausal women by using a food-frequency questionnaire. The study authors then determined that 62 percent (360) of the women had coronary calcification.

Vitamin K1 intake was not associated with reduction of coronary calcification. However, intake of vitamin K2 was associated with decreased coronary calcification.7

“This study shows that high dietary menaquinone intake, but probably not phylloquinone, is associated with reduced coronary calcification,” the researchers wrote. “Adequate menaquinone intakes could therefore be important to prevent cardiovascular disease.”

The mechanism behind vitamin K2’s anti-calcification effect is thought to involve the Matrix Gla-Protein (MGP). Scientists have called this substance “the calcification inhibitor in need of vitamin K.”
MGP’s pivotal importance for vascular health is demonstrated by the fact that there seems to be no effective alternative mechanism for calcification inhibition in arteries other than that performed by MGP. In order to prevent calcification, Matrix Gla-protein must have sufficient supplies of vitamin K2.

Conclusion

High blood pressure can be caused by a number of factors including 1) parathyroid hypertensive factor’s role in increasing intracellular calcium; 2) a deficiency of nitric oxide; 3) increased activity of the ACE enzyme and 4) arterial calcification. In individuals whose blood pressure issues are caused by an increase in intracellular calcium, substances such as cordyceps and shark cartilage (PRESSURE-IX™) may be of interest. On the other hand, for individuals with nitric oxide deficiency, increased ACE activity and arterial calcification, a supplement that contains grape seed and blueberry extract plus vitamin K2 (Circutrol BP™) may play a positive role in achieving optimal blood pressure levels.

References: