Do Higher Doses of Vitamin C Lower Elevated Blood Pressure?
Three studies say higher doses can for those with elevated blood pressure.
By Michael Mooney

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Am J Physiol Heart Circ Physiol 2002 Jun;282(6):H2414-21
Vitamin C prevents hyperoxia-mediated vasoconstriction and impairment of endothelium-dependent vasodilation.
Mak S, Egri Z, Tanna G, Colman R, Newton GE.
Cardiovascular Division, Mount Sinai Hospital, University of Toronto, Toronto, Ontario, M5G 1X5 Canada.

High arterial blood oxygen tension increases vascular resistance, possibly related to an interaction between reactive oxygen species and endothelium-derived vasoactive factors. Vitamin C is a potent antioxidant capable of reversing endothelial dysfunction due to increased oxidant stress.

We tested the hypotheses that hyperoxic vasoconstriction would be prevented by vitamin C, and that acetylcholine-mediated vasodilation would be blunted by hyperoxia and restored by vitamin C.

Venous occlusion strain gauge plethysmography was used to measure forearm blood flow (FBF) in 11 healthy subjects and 15 congestive heart failure (CHF) patients, a population characterized by endothelial dysfunction and oxidative stress. The effect of hyperoxia on FBF and derived forearm vascular resistance (FVR) at rest and in response to intra-arterial acetylcholine was recorded.

In both healthy subjects and CHF patients, hyperoxia-mediated increases in basal FVR were prevented by the coinfusion of vitamin C.

In healthy subjects, hyperoxia impaired the acetylcholine-mediated increase in FBF, an effect also prevented by vitamin C.

In contrast, hyperoxia had no effect on verapamil-mediated increases in FBF. In CHF patients, hyperoxia did not affect FBF responses to acetylcholine or verapamil.

The addition of vitamin C during hyperoxia augmented FBF responses to acetylcholine. These results suggest that hyperoxic vasoconstriction is mediated by oxidative stress. Moreover, hyperoxia impairs acetylcholine-mediated vasodilation in the setting of intact endothelial function.

These effects of hyperoxia are prevented by vitamin C, providing evidence that hyperoxia-derived free radicals impair the activity of endothelium-derived vasoactive factors.

Does Vitamin C Lower Blood Pressure?
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Vitamin C has been traditionally used as a home remedy for lowering blood pressure, and a study by researchers at Boston University School of Medicine and the Linus Pauling Institute at Oregon State University showed promising results and may prompt more research into using vitamin c to lower blood pressure.

The study included 45 hypertensive adults and was double-blind and placebo controlled.

After patients took 500 milligrams of vitamin C per day for one month there was a decrease in both their systolic and diastolic readings.

Patients on the placebo also saw a small drop in their diastolic blood pressure and **vitamin C seemed to have no effect on people who already had normal blood pressure.**

"One theory that could explain the results is that vitamin C works as an antioxidant in the human body," said Balz Frei, a co-author of the research as well as a professor and director of the Linus Pauling Institute, "In doing that, it would help protect the body's level of nitric oxide, which is important to blood vessel function."

SOURCES:
Oregon State University (1999, December 21). Vitamin C Can Reduce High Blood Pressure, Study Finds. Oregon State University. Retrieved September 26, 2008, from http://oregonstate.edu/dept/ncs/newsarch/1999/Dec99/level.htm

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Aug 99 - Vitamin C improves function of arteries
Circulation, 1998, Vol 97, Iss 4, pp 363-368.

Chronic Heart Failure (CHF) is associated with endothelial cell dysfunction. These cells line the cavities of the heart. There is evidence for increased free radical formation in CHF.

This raises the possibility that nitric oxide (NO) is inactivated by the free radicals, thereby impairing endothelial function.

Nitric oxide causes dilation of the blood vessels and is derived from L-arginine in endothelial cells, macrophages, neutrophils, platelets, etc. It is a gaseous mediator of cell-to-cell communication formed in bone, brain, endothelium granulocytes, pancreatic cells, and peripheral nerves.

The short-lived NO molecule is manufactured by tissues, and plays a role in various processes, primarily by interacting between endothelium and smooth muscle cells. It is involved in dilation of blood vessels and penile erection, and possibly affects immune reactions and memory.

Shortage or inactivation of NO may contribute to high blood pressure and formation of atherosclerotic plaque. An excess of NO, which is a free radical, is toxic to brain cells, and NO is also responsible for the often fatal, drop in blood pressure accompanying shock from abdominal or pelvic infection.

This study tested the hypothesis that (NO) is inactivated by free radicals, by determining the effect of vitamin C (25 mg/min) on impaired dilation in patients with CHF.

It consisted of 15 patients with CHF and 8 healthy volunteers.

An excitotoxic amino acid was used to inhibit endothelial synthesis of nitric oxide. The result was that vitamin C restored impaired endothelium function in patients with heart failure after both intra-arterial administration (13.2% vs. 8.2%) and oral therapy (11.9% vs 8.2%) for 4 weeks.

The portion of dilation mediated by nitric oxide (which was inhibited by the excitotoxic amino acid) was increased after acute as well as after chronic treatment (CHF baseline: 4.2%, acute 9.1%, chronic 7.3%; normal subjects: 8.9%).

Thus vitamin C improved arterial dilation in patients with CHF as the result of increased availability of nitric oxide.

This supports the concept that endothelial cell dysfunction in patients with CHF is, at least in part, due to the accelerated degradation of nitric oxide by free radicals.

Arzneimittelforschung. 2006;56(7):535-40.
Effects of ascorbic acid on ambulatory blood pressure in elderly patients with refractory hypertension.
Sato K, Dohi Y, Kojima M, Miyagawa K, Takase H, Katada E, Suzuki S.
Source
Division of Clinical Research of Hypertension, Department of Internal Medicine, Nagoya City Johoku Hospital, Nagoya, Japan. ksato@sb.starcat.ne.jp

Abstract

The increased production of reactive oxygen species plays a role in the etiology of hypertension, but the effects of antioxidants on blood pressure are controversial.

However, antioxidants possibly lower blood pressure in elderly patients with hypertension, because vascular aging is also closely related to oxidative stress. Effects of chronic treatment with ascorbic acid (CAS 50-81-7; 600 mg/day for 6 months) on blood pressure and levels of C-reactive protein, 8-isoprostane, and malondialdehyde-modified low-density lipoproteins were examined in elderly patients (n = 12, six males/six females, age 78.3 +/- 5.0 years, mean +/- SD [range, 67 to 84 years]; elderly group) and adult patients (n = 12, five males/seven females, age 54.6 +/- 6.7 years [range, 39 to 621; adult group) with refractory hypertension.

Chronic treatment with ascorbic acid markedly reduced systolic blood pressure and pulse pressure in ambulatory blood pressure monitoring in the elderly group (from 154.9 +/- 21.6 to 134.8 +/- 19.7 mmHg, $p < 0.001$; and from 79.1 +/- 22.1 to 63.4 +/- 18.7, $p < 0.05$; respectively), which was accompanied by an increase in the serum levels of ascorbic acid and decreases in the levels of C-reactive protein, 8-isoprostane, and malondialdehyde-modified low-density lipoproteins. In contrast, ascorbic acid did not affect blood pressure in the adult group.

These results suggest that ascorbic acid is useful for controlling blood pressure in elderly patients with refractory hypertension.