



Protecting the Heart by "Thinning" the Blood

You've probably heard that so-called blood thinners are sometimes used to treat heart and hypertensive patients. This term conjures images of blood, thick as molasses, that requires diluting in some way so that it can course more freely through your blood vessels. In reality, this picture of blood thinners as a kind of physiological paint thinner is erroneous. What they really do is simply prevent platelets from becoming sticky and forming clots. A more accurate term for such medicine is anticoagulant.

Physicians and their patients have actually been benefiting from anticoagulant therapy since antiquity, though explanations for how this worked were often fanciful at best. For example, an ill patient would sometimes have his or her back covered with a species of bloodsucking leech (*Hirudo medicinalis*) that would quickly become engorged with blood. The good doctor would then pry off and discard the little feasters, confident they had sucked all the poisons from the patient's blood. The fact that the health of many of these patients sometimes improved seemed at the time to validate this toxin-removal theory. Indeed, so common was this practice that the word **leech** actually derives from the middle English term **leche**, meaning "physician."

Today, researchers have a better understanding of some of the mechanisms of "leech therapy." It turns out that leeches have an extremely potent natural anticoagulant called **hirudin** in their saliva. From the leeches' point of view, this is essential to their style of feeding because it prevents blood from clotting and thus keeps an uninterrupted stream of it flowing into their digestive tract. Natural hirudin, as well as synthetic versions of it, are now being studied in the fight against heart attacks. Squeamish patients take note: A hungry leech is no longer directly required to deliver the drug!

There is another potent anticoagulant that has been used since antiquity, though not necessarily because of its anti-coagulant properties: the drug in the painkilling, anti-inflammatory compound aspirin. About ten years ago, physician volunteers were recruited to see if taking a single aspirin tablet containing 230 milligrams daily could affect the risk of heart disease and death by heart attacks. Researchers already knew that one of aspirin's many properties included the inhibition of platelet clumping. But was this inhibition powerful enough to affect cardiovascular health? The results of the physicians' study, which were published in 1997 in ***The New England Journal of Medicine***, concluded that a daily aspirin does indeed have a significant impact on heart health, lowering the risk of heart disease and heart attacks. (1)



Other researchers have also shown that aspirin can slash the risk of a second heart attack in patients who have already suffered a first heart attack. And because unchecked platelet clumping has also been implicated as one cause for chronic high blood pressure, aspirin and other anticoagulants may help in the treatment of hypertension as well.

Unfortunately, many of these anticoagulant drugs, aspirin included, can have pernicious side effects for many patients, side effects that can range from serious stomach bleeding to kidney damage. Indeed, further analysis of the same landmark physician study itself found that those doctors in a control group who received a placebo instead of aspirin had the same overall incidence of death as those who received the aspirin. How could this be, if the aspirin takers were enjoying such a comparative reduction of heart disease and heart attacks? (2)

Well, it turns out that the physicians on aspirin increased their odds of another, often fatal condition: hemorrhagic stroke, that is, unchecked bleeding into the brain. This kind of stroke is a prime example of where you need some protective blood clotting, but the anticoagulants have tuned off the capacity to do so.

A more recent report from the Boston University School of Medicine cautioned that aspirin can irritate the stomach lining, causing severe upper gastrointestinal bleeding and, in rare instances, death (3)

The bottom line: For some patients, "blood thinning" with daily aspirin can be a lifesaver, but for others it can result in a kind of disease substitution whereby reducing the odds of one bad outcome simply ups the odds of another.

If you are a blood donor, take heart! Regularly giving blood was found to be heart-protective, presumably because it reduces the volume of hemoglobin in the blood, a risk factor for heart attacks and stroke, according to the Framingham studies. (4)

ADNO (Arginine Derived Nitric Oxide): The Body's Safe and Natural Blood Thinner

The good news is that researchers have found another "blood thinning" approach that is equally effective in controlling platelet aggregation, but without the side effects of the conventional anticoagulants from aspirin to leech saliva. This discovery came after Drs. M.W.Radomski, R.M.J. Palmer, and Salvador Moncada learned that platelets themselves contain their own form of the enzyme nitric oxide synthase, which lets them create NO from arginine. (5)

Why, they wondered, would platelets need to do this? Further research by these pioneers eventually suggested that the ability to form NO was a sort of "fail-safe" mechanism that limits the capacity of the platelets to do inadvertent damage to the blood vessels they're designed to save. It's as if nature has equipped us with our own emergency clot-busters.



Alas, in people whose cardiovascular system is already severely damaged in multiple ways, the NO produced by platelets may sometimes be too little, too late.

But supplemental arginine can help a hypertensive patient's remaining undamaged endothelial cells produce additional NO to keep the arteries open and prevent platelets from clumping and sticking to vessel walls. In fact, in 1994, researchers at the Hanover Medical School in Germany reported that intravenous arginine resulted in a 33 percent decrease in platelet aggregation – a very impressive result. Moreover, the researchers concluded that the arginine inhibits platelet aggregation specifically "by enhancing nitric oxide formation" (6)

The effect of oral arginine on platelet "stickiness" is also surprisingly long-lived. In a report published in the *Journal of the American College of Cardiology*, cardiologists at Stanford University Medical School gave their patients with high serum cholesterol levels a daily dose of 8.4 grams of oral arginine. After two weeks, these patients' platelets had become significantly less sticky. What's more, the beneficial effect remained undiminished for two more weeks after the supplements stopped. Indeed it took a full eighteen weeks before the platelets returned to their pre-arginine levels of stickiness. (7) By the way, the patients experienced no significant side effects throughout the duration of the experiment.

If you are currently on a regimen of anticoagulant therapy prescribed by your doctor, you might want to consider discussing with him or her the possibility of adding three to six grams of daily arginine supplements to your treatment. If you haven't been prescribed blood thinners but have wondered lately if maybe taking a daily aspirin could be a prudent insurance policy for your future heart health, the same three to six grams of supplementary arginine could be a better, safer choice.

Not only will arginine help prevent your platelets from clotting, it will also provide your hemoglobin with more of the nitric oxide "hitchhiker" that is at the ready to hop off and dilate the blood vessels supplying tissues with a high demand for oxygen and nutrients. The Arginine Solution may just be the closest thing there is to an engine additive to rev up the performance of your human machine.

The delivery of oxygen from the lungs to the body tissues, and the subsequent removal of carbon dioxide waste back to the lungs for exhalation is a complex event largely mediated by hemoglobin, an iron-containing protein found in red blood cells. Recent groundbreaking research has found that hemoglobin also binds to a third gas, nitric oxide. This is ferried along with the oxygen and carbon dioxide and can be released when needed to dilate arterial vessels and thus allow more blood flow to tissues requiring extra oxygen.



Nitric oxide also figures predominantly as a kind of fail-safe mechanism for blood platelets, the body's smallest cells. When a vessel sustains an injury, platelets become "sticky" and aggregate into clots that serve as miniature dams to stanch further blood loss. This protective mechanism can sometimes go awry in patients whose arteries have undergone narrowing and occlusion due to atherosclerotic plaque. Large blood clots can be deadly, especially when they occur in vital areas, such as the coronary arteries that supply the heart muscles or the arteries that feed the brain. Even a small clot can be dangerous, especially if it travels as an embolus and winds up clogging a small or narrowed artery. To prevent such an untoward outcome, heart patients are often prescribed anti-coagulants to inhibit platelet stickiness.

Unfortunately, many of the conventional "blood thinners" including aspirin, can trigger other side effects, from stomach ulcers to hemorrhagic stroke (or bleeding into the brain)

Researchers have recently discovered that platelets contain their own form of an enzyme that allows them to manufacture nitric oxide when needed. This NO, in turn, can help open arteries and reduce the stickiness of platelets. Supplemental arginine holds great promise as a safe and natural anti-coagulant, one that can help keep platelets in check when their "healing" function becomes overzealous enough to imperil cardiovascular health

From the book "**THE ARGININE SOLUTION**"
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