Blood Clotting Disease

Blood Clotting Disease can be from too Much or too little Clot Formation

Blood Clot
Normal

Red blood cell
Broken blood vessel wall

Platelet

Too much
build-up of red blood cells
build-up of platelets

Activated platelet
Clot
Fibrin

Too little
Stomach
Peptic ulcers may lead to bleeding, perforation, or other emergencies

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Evidence Based Natural Energetic Medicine Education
Reduced platelet production (thrombocytopenia) by the bone marrow caused by ionizing radiation damage, disease, or toxic exposure of the bone marrow to drugs is another cause of deficient clotting. A third cause is dietary deficiency of vitamin K. This vitamin, normally provided in the food or by the intestinal bacteria, does not take part in clotting directly but is required for the synthesis of prothrombin in the liver. Newborn infants in whom the digestive tract is still devoid of bacteria are deficient in vitamin K and are therefore more susceptible to bleeding if injured.
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Questions to Patient

1. Have you or a blood relative ever needed medical attention for a bleeding problem or been told you have a bleeding disorder or problem:
   - During/after surgery
   - With dental procedures, extractions?
   - With trauma?
   - During childbirth or for heavy menses?
   - Ever had bruises with lumps?
2. Do you have or have you ever had:
   - Liver or kidney disease, a blood or bone marrow disorder; a high or low platelet count?
3. Do you take aspirin, NSAIDs (provide common names), clopidogrel (Plavix™), warfarin, heparin?

History From Patient

- Personal history of VWD
- Abnormal laboratory test
- Positive family history of bleeding disorder or bleeding
- Patient is concerned about bleeding; patient who has unexplained anemia or history of previous DDAVP use

No evaluation; usual care
No further evaluation; usual care
Evaluates further: initial laboratory tests and possible referral (figure 4, p 25)

Estrogen Inhibits Clogging of the Arteries

Vitamin E

Vitamin E is found in corn, nuts, olives, green, leafy vegetables, vegetable oils and wheat germ

Tocopherol
Deep Vein Thrombosis (DVT)

June 8, 2011

Nearly 2 million people in the US are affected by blood clots every year so this disease also called as Deep Vein Thrombosis (in medical terms) is deeply embedded in the public’s consciousness now, following several newspaper reports.

It mainly affects people who are more than 40 years of age. DVT is better understood as blood clot in the veins. Blood flow is a continuous and a natural process of our body.

Pure blood flows through the arteries and the impure blood which is to be cleaned and re-oxygenated flows through the veins.
Basically, there are two types of veins in our body:

**Superficial veins**—lie just below the skin and can be seen on the surface.

**Deep Veins**—located deep within the leg muscles.

**Deep Vein Thrombosis (DVT) of the Leg**
What is Deep Vein Thrombosis?

The formation of a blood clot in the deep veins is called Deep Vein Thrombosis. In most of the cases clots are formed in the deep veins of legs or thighs, pelvis.

The formation of clots in the leg or thigh is itself not so serious, but it becomes a life threatening situation when the clots get broken and start travelling towards heart, lungs, brain. You need not panic yet; there are a number of natural and effective home remedies for Deep Vein Thrombosis.

This situation, when the clot breaks and starts travelling is known as pulmonary embolism. Clots in superficial veins don’t pose to be a threat as the perforator vein acts as a sieve and does not allows the clot to enter the deep venous system.
DEEP VEIN THROMBOSIS

2.5 Million Americans are affected by DVT annually.

get screened today and find out if you’re at risk

schedule your personal consultation today

The intrinsic and extrinsic pathways of blood coagulation

**EXTRINSIC SYSTEM**

- XII (on contact with vascular endothelium) → XIIa
- Tissue factor + VII + Ca²⁺ → Xa
- X + V + Ca²⁺ + phospholipid → Xa
- Xa + VIII + Ca²⁺ + phospholipid
- Prothrombin → Thrombin
- Fibrinogen → Fibrin

**INTRINSIC SYSTEM**

- XI → Xla
- IX + VIII + Ca²⁺ → IXa + phospholipid
- XII → XIIIa by crosslinking
The coagulation phase of hemostasis

**Coagulation Phase**

Does not start until 30 seconds or more after the vessel has been damaged; involves a complex sequence of steps leading to the conversion of circulating fibrinogen into the insoluble protein fibrin; as the fibrin network grows, blood cells and additional platelets are trapped in the fibrous tangle, forming a blood clot that seals off the damaged portion of the vessel; procoagulants (clotting factors) in the plasma, including Ca\(^{2+}\) and 11 different proteins identified by Roman numerals, play a key role in this phase; many clotting factors are proenzymes; the activation of one proenzyme commonly creates a chain reaction, or cascade.

**Common Pathway**

The common pathway begins when enzymes from either the extrinsic or intrinsic pathway activate Factor X, forming the enzyme prothrombinase. Prothrombinase converts the proenzyme prothrombin into the enzyme thrombin (THROM-bin). Thrombin then completes the clotting process by converting fibrinogen, a soluble plasma protein, to insoluble strands of fibrin.

**Extrinsic Pathway**

The extrinsic pathway begins with the release of tissue factor (Factor III) by damaged endothelial cells or peripheral tissues. The greater the damage, the more tissue factor is released and the faster clotting occurs. Tissue factor then combines with Ca\(^{2+}\) and another clotting factor to form an enzyme complex capable of activating Factor X, the first step in the common pathway.

**Intrinsic Pathway**

The intrinsic pathway begins with the activation of proenzymes exposed to collagen fibers at the injury site. This pathway proceeds with the assistance of PF-3, a platelet factor released by aggregating platelets. After a series of linked reactions, activated clotting factors combine to form an enzyme complex capable of activating Factor X.

**Clot Retraction**

Once the fibrin meshwork has formed, platelets and red blood cells stick to the fibrin strands. The platelets then contract, and the entire clot begins to undergo clot retraction, a process that continues over a period of 30–60 minutes and pulls the cut edges together.

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What NOT to EAT

SUPER CARDIO Diet Tips

STARTS With

What NOT To EAT

1. AVOID Synthetic Foods
2. AVOID Hi Glycemic Foods
3. AVOID Processed Foods
4. AVOID White Sugars
5. AVOID Foods Boiled in Oil
6. AVOID Nitrite/Nitrate meat
1. Eat Natural Foods with little preservatives
2. Eat more fruits, seed products, leafy greens, salads
3. Let Fruit be your Sweetener,
4. Drink ONLY 100% Fruit juice diluted with water
5. Boil foods in WATER, NOT OIL
6. Use fresh, cold processed UNHEATED olive oil, sunflower oil, safflower oil etc.
7. Less Cooking, Use stir fry well washed veggies
8. Foods made with Love and Nature is Blessed Nutrition, Foods made and eaten with Hate and Anger are poisons.
9. Celebrate each meal with friends, family or at least your joyous self. Celebrate
10. Listen to your inner self what to eat, and when to stop, do not eat with your eyes
Causes Of Deep Vein Thrombosis (DVT)

The clot formed in DVT can block the flow of blood and results in swelling and pain. Amazingly, DVT can also occur in fit and healthy people who have no previous cardiovascular problems.

People doing work outs regularly are somehow having a greater risk of DVT complications as they have low resting pulse, that can help prompt DVT during the long periods of inactivity. Nonetheless, there can be certain reasons too, most likely to develop DVT:

- After a pacemaker catheter has been passed through the vein in the groin
- Bed rest
- Cigarette smoking
- Family history of blood clots
- Fractures in the pelvis or legs
• **Giving birth** within the last 6 months
• **Heart failure**
• **Obesity**
• Recent surgery (especially hip, knee, or female pelvic surgery)
• Too many blood cells being made by the bone marrow (polycythemia vera), causing the blood to be thicker and slower than normal
• **Blood that is more likely to clot (hyper coagulability)**
• Cancer
• Taking estrogens or birth control pills
• **Pregnancy**

One more reason which is most likely, when one or more of the above listed risk factors are also present—**sitting for long periods when traveling** (long duration flights, rail, and road journeys).

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**Signs and Symptoms of Deep Vein Thrombosis (DVT)**

Some of the symptoms which are observed in the patient are summed up below—

• Excessive pain at the site of clot formation
• Swelling and tenderness may appear
• **Blue colored bulge**
• skin Ulcers
• **Difficulty in breathing**, discomfort in chest is experienced in case of Pulmonary Embolism (PE)
• Changes in skin color (redness) in one leg
• Increased warmth in one leg
• Sometimes Palpitations also occurs
• The vein often gets as a firm, thickened cord
• Inflammation may occur during the course of the vein but no infection is there

Tests for diagnosing DVT

• D-dimer blood test
• Doppler ultrasound exam of the legs
• Plethysmography (measurement of blood flow) of the legs
• X-rays to show veins in the affected area (venography)
• Activated protein C resistance (checks for the Factor V Leiden mutation)
• Anti thrombin III levels
• Anti phosphor lipid antibodies
• Genetic testing to look for mutations that make you more likely to develop blood clots, such as the pro thrombin G20210A mutation
• Lupus anticoagulant
• Protein C and protein S levels
• Screening for disseminated intravascular coagulation (DIC)

The above list of tests is not all inclusive.
Natural Home Remedies for Deep Vein Thrombosis (DVT)

There are no specific Deep Vein Thrombosis home remedies for the removal of blood clot, but DVT should not be overlooked merely as a blood clot as in worst cases of PE (Pulmonary Embolism) the situation becomes life threatening.

For the first 15 – 20 days you have to depend upon the treatment given by doctors.

- Usually the first drug given to the patient is Heparin.
- If heparin is given through a vein (IV), you must stay in the hospital.
- Newer forms of heparin can be given by injection once or twice a day. You may not need to stay in the hospital as long, or at all, if you are prescribed this newer form of heparin.
- Another drug called Warfarin (Coumadin) or any anti coagulant (as suggested by the physician) is also started along with heparin. These anticoagulants keep more clots from forming or old ones from getting bigger. Generally, these drugs do not dissolve clots.
- Most likely warfarin is administered for minimum 3 months but in some cases people have to take it for the rest of their lives.

Although no natural remedies are there which can cure DVT, but some can help in the process of blood thinning and prevention of the disease. Here our primary goal is to prevent the disease from re occurring.

Some of the home remedies for DVT prevention from re occurrence are:

- Regular intake of a glass of water with 1 tbsp lemon juice and chopped slices of ginger is the easiest solution for blood clot problem. Lemon and ginger is blood circulation improver.
- Have one garlic clove daily as it has favorable effects on cardiac factors.
- Keep your body hydrated with the intake of lots of water.
- Cold water fish should be preferred over other animal proteins.
- Most useful herb for improving the circulation of blood is ashvaganda. Hence, it works as one of the most effective home remedies for Deep Vein Thrombosis (DVT) natural treatment.
- Broccoli being rich in fiber content is considered good for cardiovascular patients.
- An aggravated stress hormone narrows down the blood vessels. **Celery** is an active compound and helps in **reducing the stress hormone** and should be included in diet.
- Proper vascular state is maintained with the proper amount of **Vitamin C** in the body.
- **Banana, apricot, spinach juice** help in keeping the blood pressure normal.
- **Skimmed milk** should be used, as non skimmed milk offers extra fat which deposits on the walls of blood vessels thereby thinning their diameter.
- **Capsicum and pepper** help in preventing the platelets to stick together, so you can also include them in your diet.
- Prepare a juice of leaves of spinach and one part each of pepper, garlic and clove. This is one of the best **herbal remedies** for Deep Vein Thrombosis (DVT).
- Use **mustard oil, canola oil** instead of high fat cooking oils.

**Dos and Don’ts for DVT**

- **Avoid food rich in high Vitamin K.**
- Avoid smoking.
- **Avoid the intake of alcohol**, as it dehydrates you.
- **Lose weight** if overweight.
- **Control your blood pressure.**
- Wear compression stockings if advised by your doctor.
- Avoid drinking too much coffee or tea.
- Daily walk for 15 – 20 min.
- Cut down your daily dose of margarine, processed foods.
- **Avoid long duration flights**, if cannot then try walking on aisle after every 2 hours or so.
- Don’t sit with an obstruction to the thigh muscle.
- Elevate your leg up to 6 inches while sleeping.
- **Don’t sit idle for long time**, move your legs.
- Exercise your **lower calf muscles**, only after consulting your doctor.
- Avoid doing **risky things** which may **cause bleeding**.

Natural therapies consisting of **home remedies** for Deep Vein Thrombosis (DVT) prevention are made with common herbs, vegetables, fruits to heal problems of blood clotting and maintain a proper blood circulation.
HEMOSTASIS & PHYSIOLOGY OF BLOOD CLOTTING

Because blood flows continuously in the vascular bed, it is prone to leave the body quickly whenever there is either an external or internal injury to the tissues. The vital importance of blood to tissue survival has produced a variety of preventive and defense mechanisms aimed at minimizing blood loss during injury.

IMPORTANCE OF VASOCONSTRICTION. Tissue injury often severs the connective tissue and a portion of vasculature, exposing collagen fibers in the blood vessel wall. The fragile blood platelets flowing by these rough surfaces adhere and rupture, releasing their serotonin, a potent vasoconstrictor agent that immediately stimulates contraction of smooth muscle cells in the wall of injured arterioles and even the smaller arteries. This constriction effectively reduces and/or blocks blood flow in these vessels.

PLATELET PLUG AND BLOOD CLOT FORMATION. The vasoconstriction is a highly effective but temporary hemostatic (blood stopping) measure. This initial defense mechanism is followed by a longer lasting response consisting of formation of a plug to fill the site of injury with a temporary protective tissue until tissue regeneration repairs the wall. Thus, platelet rupture releases another substance, ADP (adenosine diphosphate), at the injury site. ADP, like serotonin, is normally stored in the platelet vesicles. ADP causes the neighboring platelets to adhere to those already bound to the injured wall, causing a clumping of the platelets (platelet aggregation). The aggregate gradually grows, finally forming a temporary hemostatic plug to prevent blood leakage. This plug resembles a blood clot when blood is allowed to stand outside the body. Next, this platelet plug is reinforced by deposition of a meshwork of fibrin fibers. This fibrin net traps the RBCs and platelets, forming a fairly rigid and strong barrier against further blood loss. Initially loose, the fibrin net becomes gradually tight, at which point it is called a blood clot.

BIOCHEMISTRY OF CLOT FORMATION. Fibrin is a fibrous protein formed by the action of the protease enzyme thrombin on fibrinogen (profibrin), a circulating protein made by the liver. Thrombin is normally present in the blood as its inactive form, prothrombin. The activation of prothrombin, the key step in the clotting mechanism, requires the presence of calcium ions and a protein factor called factor X (ten). Activation of factor X can occur by either of two pathways: the intrinsic (blood) pathway involves the activation of factor XII, which originates from blood-related sources. The extrinsic (tissue) pathway involves the production from the injured tissue of another enzyme called thromboplastin (factor III). Thromboplastin can directly activate factor X, but factor XII must activate several other factors, which in turn activate factor X. The precipitated fibrin is initially loose; in the presence of another blood factor (factor XIII), it becomes tight, rigidifying the clot. In the absence of injury, circulating anticoagulant factors such as antithrombin or possibly heparin prevent thrombin activation and clot formation.

CLOT CONTRACTION AND DISSOLUTION. Once a clot forms, it begins to contract. Contraction is an active process involving utilization of ATP and contraction of actin filaments in the platelet pseudopods. Clot contraction causes extrusion of the plasma trapped within the clot and shortening of the pseudopods. Because the edges of the clot are attached to the edges of the injured tissue, clot contraction is believed to bring the injured edges closer together, improving hemostasis and facilitating wound closure and repair.

The final stage in the life of a blood clot is its dissolution, brought about by the action of the enzyme plasminogen, which digests the fibrin net, resulting in clot breakdown. Plasmin is formed from a precursor called plasminogen. ABNORMALITIES OF CLOT FORMATION. Several disease conditions or certain nutritional deficiencies interfere with proper clotting and pose serious hazards to the individual. Hemophilia (bleeding sickness) is a series of hereditary diseases characterized by deficient hemostasis and continued blood loss after injury. The causes of these hereditary diseases are the lack of one of the blood clotting factors. In type A hemophilia, which is most frequently (75%) observed, the individual is deficient in factor VIII. The disease mainly affects males. The most famous case is the family of Queen Victoria of England, in which many of the male children fell victim to hemophilia. To prevent hemophilia, the missing clotting protein must be provided externally. Large scale production of such proteins by the application of modern bioengineering methods promises to prevent hemophilic bleeding.

Reduced platelet production (thrombocytopenia) by the bone marrow caused by ionizing radiation damage, disease, or toxic exposure of the bone marrow to drugs is another cause of deficient clotting. A third cause is dietary deficiency of vitamin K. This vitamin, normally provided in the food or by the intestinal bacteria, does not take part in clotting directly but is required for the synthesis of prothrombin in the liver. Newborn infants in whom the digestive tract is still devoid of bacteria are deficient in vitamin K and are therefore more susceptible to bleeding if injured.
How to Thin Blood Naturally

If you are prone to blood clots, strokes or other blood thickening concerns, you will likely have to work on thinning your blood. There are many ways in which you can thin the blood safely and "do it yourself" (D.I.Y.), but with your doctor's supervision and advise. Either thinning or thickening the blood can be achieved via medication or by consuming certain foods. Options should be discussed with your doctor prior to incorporating any methods on your own. Ongoing thinning of the blood helps prevent blood clots that can cause deep vein thrombosis, pulmonary embolism, myocardial infarction and various sizes of brain ischemic strokes. Some small or micro attacks may go undetected, but still blood vessel and organ damage does add up as clogging increases and blood flow is less or is blocked in small blood vessels (capillaries).

Steps

Opt for foods that thin the blood (are anti-coagulant). To avoid promoting blood clotting and thickening, select foods that are low in vitamin K (which promotes coagulation) but high in vitamin E, instead. Some good choices are peanuts, hazelnuts, almonds, walnuts and
pistachios. You can also find vitamin E in rice, oats, chickpeas (to make a dip called humus, similar to refried beans) and lentils (flat, disc shaped legumes often used in soups).

- Sometimes natural thinners are not sufficient depending for instance on how many of up to nine risk factors/points you present; follow your cardiovascular, specialist doctor's orders. *Never give medicine prescribed for you to another person nor take any not prescribed to you.*

![Image of spices and vegetables]

**Select foods that are rich in salicylates.** This is an aspirin-like substance that is beneficial, if you are looking for blood thinning foods. Foods that are high in salicylates include: *avocados, cabbage, oranges, tangerines, grapes, strawberries, cranberries, raisins and cherries.* Try to use plenty of salicylates rich spices including: *ginger, real cinnamon, curry powders, cayenne pepper, turmeric, paprika, oregano, licorice and real peppermint.*
Choose blood thinning beverages in your diet. These include orange juice, cider, wine and pomegranate juice. Note that pomegranate juice has proven to be a great blood thinner. It stimulates the flow of blood to the heart. Additionally, pomegranate juice reduces plaque levels in the arteries, building upon the good cholesterol and decreasing the cholesterol levels that are considered bad.

Note that alcohol is also a blood thinner, but only in very limited amounts. Women are limited to one alcoholic beverage a day to thin the blood, whereas men are allowed two.
Consuming more than the one or two recommended drinks of alcohol a day can result in the opposite effect - thickening the blood, lowered brain activity and hardening of the liver.

Consider incorporating an exercise regimen into your daily routine. Exercise reduces vitamin K in the blood stream and increases your blood circulation, which helps thin the blood. Opt for cardio training, swimming or yoga as exercise options.

Take aspirin, if you want to thin your blood. Be aware, though, that aspirin introduces additional stomach bleeding risks. Aspirin absorbs vitamin K in your blood stream which is beneficial, if you have concerns with blood clots. Aspirin thins your blood by preventing blood cells from sticking together, thus decreasing the risk of clots. Caution: Always follow your doctor's orders for any form of prescription drugs, dietary restrictions or medicinal intervention.
Ask your doctor, if you should take a prescription blood thinner and discuss the side effects of any prescription medications, particularly those that **require daily or weekly testing**. There are Coumarin-based medications such as **Coumadin or Warfarin** that work to reduce the formation of vitamin K dependent clotting factors in the blood. Coumarin/Cumarin-based drugs (including the brand names of Coumadin/Warfarin) block or fight vitamin K (called "vitamin K antagonist"). Discuss your options with your doctor, and determine whether a prescription blood thinner is the best option for you, considering all the side effects.

- Depletion of vitamin K by Coumadin/Warfarin therapy increases risk of arteriosclerosis (hardening tissues) including arterial calcification and heart valve calcification, especially if much of the essential vitamin D is present.\(^1\)
- **Caution**: If you take a Coumarin based prescription blood thinner, never eat cranberries as it compromises the validity of the INR blood coagulation testing, that is drawn regularly when using Coumadin/Warfarin for thinning blood under a doctor’s supervision.

**Don’t eat large amounts of vegetables with vitamin K, because they badly affect blood thinners; also don’t increase and decrease amounts of foods rich in vitamin K, especially if using Coumadin/Warfarin**: "be consistent", as vitamin K thickens the blood.

- Avoid eating much of: Liver, broccoli, cauliflower, brussel sprouts, cabbage, kale, spinach and other green leafy vegetables, green beans, green tea, some cheeses and consuming vitamin K supplements.\(^2\)
Ask about newer anticoagulants (available since about the years 2009-10; they include: Eliquis, Pradaxa, and Xarelto) that do not require daily or weekly testing because they do not work by blocking vitamin K; realizing, however, they are much more costly than the older K blocking type.

**Bleeding may not stop:** Urgent! Call your doctor or get medical help right away, if you develop any of these signs or symptoms of significant amount of bleeding (possibly hidden, internal bleeding, hemorrhage; they may give you vitamin $\text{K}_1$ to "thicken" the blood). Here is a checklist of problem bleeding issues:[3]

- Unexpected bleeding and any that may last a long time, such as:
- Nosebleeds that happen repeatedly
- Unusual bleeding from gums
- Menstrual or vaginal bleeding heavier than normal
- Bleeding that is severe, i.e.: that you can not control
- Red, pink, or brown urine
- Bright red, red streaked or black stools (looks like tar, i.e.: digested blood)
- Coughing up blood or blood clots
- Vomiting blood or your vomit looks granular like “coffee grounds”
- Headaches, feeling dizzy, faint or weak
- Pain, swelling, or new drainage at wound or drip-IV insertion sites
Cardiovascular Disease Linked to High Homocysteine Levels in the Blood

Are B Vitamins the Answer? Research Says Yes!
HOME REMEDIES FOR BLOOD CLOTS

Consumption of vitamin C rich food helps to maintain good vascular health

Broccoli rich in fiber is good for patients suffering from vascular disorder

Bread, beans, cereals, brussels sprouts & asparagus improves flexibility of brittle arteries

Ginger, turmeric, bilberry reduces blood clotting

Capsicum & pepper prevents platelets from sticking together

Use canola oil & mustard oil for cooking rather than high fat oils

Banana, apricot, spinach, orange juice regulates blood pressure & keeps vascular system active

Skim milk contains calcium needed for platelet functioning

Gingko, onion & garlic reduces fibrin content, a protein that is important in forming blood clots

Physical activity like walking helps maintain constant blood flow
NATURAL EXCHANGE FOR WARFARIN VS. PRESCRIPTION: WHAT ARE THE RISKS?

There are some potentially useful natural remedies that have shown some promise in preliminary research regarding blood clot treatment and prevention. While these treatments may be helpful, it is important to fully understand the risks associated with natural alternatives. This article is going to cover the dangers of alternative, herbal remedies, the differences between natural and pharmaceutical medications, the types of natural effects that are needed to treat pulmonary embolisms and other clotting problems, and a few of the more common natural remedies that are used in patients with a DVT or pulmonary embolism. Please remember that this material is not medical advice. It, like everything on this site, is meant to be used to facilitate discussions between a patient and his or her medical team.

Many people come looking for natural cures with the idea that a natural cure is a chemical-free and safe cure. The popular view is that if a pharmacy sells the medication it is unsafe and if a natural food store sells it, the cure is safe. It is very important that we stop looking at the issue in this way. Natural remedies have potential benefits; however, they are not risk free. Natural remedies are chemicals that have side effects.

As a society we are being taught that chemicals are bad and plants are good. The problem with this view is that "herbal remedies" work by using chemicals that are present in a substance. Plants, minerals, enzymes, and vitamins sound safe because we do not use their chemical names. Plant chemicals, no matter how gentle they sound, can be dangerous. One example is Kava Kava. Kava Kava, Piper methysticum, is used to treat anxiety and sleeplessness. Xanax sounds far more dangerous than Kava Kava. After all, Xanax needs a prescription and Kava Kava is sold in a drugstore and can be grown in a garden. Kava Kava may sound safe; it is not. It is known for causing liver damage. Any substance, when used as a treatment, is a drug, contains chemicals, and has potential side effects. It does not matter whether the treatment came from a garden or from a pharmacist, the facts are the same. Everything that you put into your body is a potential risk.

Medications that are produced as a medication are required by the FDA to publish the possible risks. Herbs do not have to publish the possible risks. If you are interested in a natural remedy do not assume it is risk free. Find an authoritative source and research all drug interactions and possible side effects. Know what you are putting into your body. Some remedies are fairly harmless while others can cause fatal reactions. Label free does not mean risk free.
Please talk with your doctors and pharmacist before adding a natural remedy, no matter how mundane, to your treatment plan. This is especially important if you are using it for a life threatening condition, in place of another medication, or in addition to other medications. Also, if you are on warfarin you will need to schedule extra INR checks to see if the natural treatment affects your INR.

WHAT ARE THE DIFFERENCES BETWEEN NATURAL AND CONVENTIONAL TREATMENTS?
There are two major differences between a mass produced pharmaceutical and a natural remedy. The first difference is that a mass produced medication has been tested. The risks are known and have been published. It is much more difficult to find risk information for natural remedies. This does not mean that the natural formulation is safer than the man made formulation.

The second major difference between natural formulations and man made formulations is that FDA does not test natural remedies to ensure that one brand has the same amount of a chemical.

Blood thinners can be very dangerous in a prescription form. Keep in mind that natural remedy amounts vary from pill to pill and treatment to treatment. The same amount of warfarin within a certain measure of error. Natural remedies do not come wit

WHAT TYPES OF NATURAL TREATMENTS WILL HELP WITH BLOOD CLOTS AND PULMONARY EMBOLISMS?
In order to know which supplements and alternative treatments will help heal a pulmonary embolism, you must first know a little about blood clotting. Pulmonary embolism and deep vein thrombosis are both types of blood clots that form in the veins of an individual. Veins are the blood vessels that return blood to the heart. Veins have no muscles and rely on the body's movement to assist in moving blood back to the heart. When the body moves, the movement moves the blood. When the body is stationary, the blood pools and sits still.

Thrombin, a glue-like chemical in the blood, begins to set up and form clots when it sits still. The thrombin system, these drugs are known as anticoagulants. They prevent the blood from coagulating. Warfarin is an example.

Other "blood thinners" work on platelets. Platelets are the blood cells that help to form the clots. An example of this type of medication is aspirin.

Lastly, once blood has formed a clot, thrombolytics are responsible for breaking down the clots. Scientists have created drugs that are able to replicate this mechanism as well. Thrombolytics only work AFTER a clot has formed.

Any alternative or natural remedy that is used to prevent new clots from forming in the veins must work on thrombin's ability to glue the clots together. If it works on platelets or already formed clots, th
NATURAL TREATMENTS THAT ARE KNOWN TO WORK

There are a few alternative treatments that have a strong track record preventing pulmonary embolism, DVT, and other venous clots. These treatments are the ones that have had substantial research and are generally accepted by the medical community. They include treatments that work on thrombin and are good for venous clots. If you are looking for an established treatment, here are some options to discuss with your doctor:

- Graduated compression stockings
- Staying well hydrated, especially with a hydration beverage like Pedialyte or Gatorade
- Pneumatic leg cuffs during surgery
- Never sit still for longer than 2 hours without a movement break

These options are typically very safe, compatible with warfarin and other medications, are inexpensive, and are the best methods for clot prevention outside of anticoagulation therapy. Many people are surprised when they are told that these are some of the best clot prevention options out there.

NATURAL TREATMENTS WITHOUT SUBSTANTIATING RESEARCH

There are some other natural treatments that are often discussed in support groups and online. These treatments include fish oil, nattokinase, vitamin E, and pycnogenol. Here is a little background on each of these supplement options. Keep in mind that none of these supplements have substantial (if any) in vitro (in human) testing. Much of this research was done using test tube blood samples. Also keep in mind that all of these treatments raise the risk of major bleeding problems. Talk to your doctor before trying any of them. The clotting system is a very important part of your body!

FISH OIL
Fish oil really is just fish oil. It is very high in Omega-3 fatty acids. The blood thinning effects of fish oil happen in the arterial system. A tiny amount of research shows that it is possibly effective in preventing strokes due to its ability to decrease platelets. This effect is not seen in patients already on aspirin. The studies have been small and conflicting. More research needs to be done before this can be seen as a large help for stroke prevention. Strokes happen due to arterial clots. No research has been done on pulmonary embolism. Due to its arterial effects, it is unlikely to help in pulmonary embolism or DVT.

NATTOKINASE (NATTO)
Nattokinase is an enzyme that occurs in soybean fermentation. Nattokinase seems to have
thrombolytic properties. This means that nattokinase breaks down clots that have already happened. It will not prevent new clots from forming. There have been no well organized human trials using nattokinase. All of the testing on nattokinase has been in animals or in test tubes. One study using a blend of natto and pycnogenol showed that it may prevent clots when taken before a flight. This study had design flaws and has been thrown into question. The safety of natto has not been established. It may raise the risk of bleeding and should be avoided in people who are already taking an anticoagulant or anti-platelet medication. Natto contains high levels of vitamin K and will affect INR.

PYCOGENOL
Pycogenol is a product derived from pine trees. Currently there is insufficient evidence to rate its effectiveness on blood clots. It was used with natto in the one study mentioned above. This study showed promise but had poor methodology. No other research has been done on pycogenol. This means that while it shows promise, we do not know if it is effective or if it has major side effects.

VITAMIN E
Vitamin E has been shown to have blood thinning effects. In fact, the blood thinning effects make this a dangerous supplement. It increases the risk of bleeding in people who have a vitamin K deficiency, are on warfarin, are using aspirin, or are on other blood thinning medication. Multiple studies have shown that it raises the risk of death.

What Are the Contraindications for Thrombolytics?

Clots in the blood can block arteries, preventing oxygen from getting to tissues in the body and causing damage.

Patients with peptic ulcer disease and other conditions may experience complications from drugs that dissolve blood clots.

Thrombolytics are drugs that dissolve clots in the blood. These clots can block arteries, preventing oxygen from getting to tissues in the body and causing damage. The drugs are commonly used as emergency treatment in conditions such as heart attack and stroke. Thrombolytics cannot, however, be used in every circumstance. Contraindications for thrombolytics, or situations where the use of these drugs is not advisable, can be either absolute or relative. Absolute contraindications mean that the drugs should not be administered to the patient. The risk of the patient taking these drugs outweighs any positive effect that the drugs might have. Absolute contraindications for thrombolytics include if the patient has active internal bleeding or a suspected aortic dissection. As the patient in these situations is already bleeding heavily, or has the potential to bleed, medications that prevent blood from clotting could be life threatening.

Other situations where the drugs should be avoided include if the patient has undergone traumatic cardiopulmonary resuscitation or has had an intercranial condition, such as injury, tumor,
or aneurysm, in the previous six months. The extent of the damage in these conditions is often not known for a number of months, and thrombolytic drugs could result in internal bleeding. Thrombolytic drugs should also not be given to patients who have severe hypertension, are pregnant, or have undergone major surgery in the previous two weeks. Finally, under no circumstances should the drugs be given to patients who have had a previous allergic reaction.

Relative contraindications for thrombolytics include situations where giving the drug to the patient can have significant risk, but this risk can be outweighed by the potential benefit. Doctors decide these situations on an individual basis. Relative contraindications for thrombolytics include if the patient has a known bleeding disorder such as hemophilia or is currently using anticoagulant medications, such as warfarin, which reduce the ability of the blood to clot. Patients with peptic ulcer disease, renal disease, or diabetic retinopathy are also contraindicated for thrombolytics due to bleeding complications; however, these risks may be outweighed by the need for the drug. Other relative contraindications for thrombolytics include a recent trauma to the patient or a major surgery performed in the previous two months. Patients whose blood pressure was very high but is currently controlled may also be considered candidates for the drugs if they are otherwise in good health. Finally, patients who have suffered cerebrovascular accidents in the past can be given the drugs but must be closely supervised to make sure there are no ill effects.

**Elevated Factor VIII Levels and the Risk of Thrombosis**

Factor VIII (FVIII) is an essential blood-clotting protein, also known as anti-hemophilic factor (AHF). In humans, factor VIII is encoded by the *F8* gene. Defects in this gene result in hemophilia A, a recessive X-linked coagulation disorder.

**Genetic Determinants of Plasma Factor VIII Levels**

In healthy individuals, family studies have indicated a genetic influence on the level of factor VIII:C. Factor VIII levels varied less among twins than among unrelated individuals.

Classic acquired risk factors for venous thrombosis include trauma, immobilization, pregnancy, surgery, malignancy, and infection. These are all factors that may cause tissue damage, stasis of the blood, or changes in blood composition. Inherited risk factors for venous thrombosis

Sustained rises in factor VIII are seen during pregnancy, surgery, chronic inflammation, malignancy, liver disease, hyperthyroidism, intravascular hemolysis, and renal disease. In most conditions, there is a concordant increase of factor VIII and vWF:Ag levels.

In humans, the majority of genetic factors regulating vWF remain to be determined. Candidate genes include a variety of genes coding for proteins involved in the biosynthesis and clearance of vWF. In mice, 2 modifier loci of vWF have been identified, 1 of which concerns an *N*-acetylgalactosaminyltransferase gene. Other important determinants of vWF level are age, acute phase, stress, and endothelial dysfunction.
High Factor VIII Levels
The first reports on a possible association between factor VIII and coronary artery disease date from the early 1960s. In the same period, blood group non-O and high factor VIII-related antigen (vWF) were identified as candidate risk factors for atherothrombotic disease.

Atherosclerosis itself could have affected the clotting factor levels by chronic inflammatory responses and elevated factor VIII, or vWF levels may reflect the inflammation and progression of atherosclerosis.

People with high levels of factor VIII are at increased risk for deep vein thrombosis and pulmonary embolism. Copper is a required cofactor for factor VIII and copper deficiency is known to increase levels of factor VII.

In 1969, Jick et al reported that blood group non-O is associated with an increased risk of venous thrombosis. Today, we know that individuals with blood group non-O have higher levels of vWF and factor VIII than do those with blood group O.

Body mass index (positively correlated with factor VIII levels) and higher levels of glucose (diabetes mellitus), insulin, fibrinogen, and triglycerides are also associated with increased factor VIII levels. Factor VIII levels increase with age, with an average rise of 5 to 6 IU/dL per decade. Oral contraceptives seem to have no effect on factor VIII levels.

Several stimuli can cause a transient or sustained increase in factor VIII levels. Exercise transiently induces a rise of factor VIII that is probably a result of adrenalin and β2-adrenoreceptor stimulation. Also, 8-arginine vasopressin and its analogue 1-deamino-8-D-arginine vasopressin enhance plasma vWF and factor VIII levels indirectly or directly via signaling via the V1 receptor. Sustained rises in factor VIII are seen during pregnancy, surgery, chronic inflammation, malignancy, liver disease, hyperthyroidism, intravascular hemolysis, and renal disease. In most conditions, there is a concordant increase of factor VIII and vWF:Ag levels.

Inherited Causes of Blood Clots
Inherited causes of blood clots are related to a genetic (inherited) tendency for clot formation that generally occur at a young age (for example, occurring before 40 or 45 years), with or without an apparent cause, and with a tendency to recur.

Low levels of natural anticoagulants such as antithrombin, protein C, and protein S account for less than 15% of selected cases of juvenile and/or recurrent clots, and less than 10% of unselected cases. Resistance to the anticoagulant action of activated protein C (APC) has now been shown to be the most common cause of an inherited clotting disorder, accounting for 20% to 50% of cases. Mild hyperhomocysteinemia, which is inherited, has been found in 19% of cases of venous clotting in children.

An alteration in the prothrombin gene (P20210) that results in an increased expression of prothrombin has been linked to an increased risk of clotting. Age-related increases in coagulation proteins, specifically increased levels of factors VIII, IX and XI, have also been linked to an increased risk of clotting.

Table 1 provides a list of inherited causes of blood clots. Click on the individual condition to learn more about specific causes.
Table 1. Inherited Causes of Blood Clots

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*The Factor V Leiden mutation does not result in increased FV levels but a resistance to the anticoagulant action of activated protein C.

Increased Levels of Natural Procoagulants

Factor V Leiden (Activated Protein C Resistance)

What Is Factor V Leiden Mutation?
The factor V Leiden mutation or alteration (FVL) was identified in 1993 and has since been found to be a leading cause of blood clots among white populations. In fact, the FVL alteration is the most common genetic risk factor for blood clots. This mutation produces an altered coagulation factor V (FV) protein, commonly known as “Leiden” protein. In normal blood clotting, activated protein C, a natural anticoagulant, controls the clotting activity of FV. In people with the altered “Leiden” protein, the FV is resistant to regulation by activated protein C (APC). As a result, clotting is uncontrolled.

Population Frequency of Factor V Leiden (FVL)
The population frequency of the FVL gene alteration is high. Heterozygous FVL mutation (having two or more different versions of a gene) is found in 5% to 10% of white individuals and in up to 30% of patients with a clotting disorder. Thus FVL gene alteration is by far the most common inherited risk factor for a clotting disorder. FVL is very uncommon in African Americans, Hispanics and Asians.

Why Do Patients With Factor V Leiden Mutation Develop a Clotting Disorder?
Factor V is an important coagulation protein that is made in the liver. Normal or wild type FV has a dual role in coagulation. When it is activated, FV acts as a procoagulant (promotes clotting), whereas when it is inactivated by activated protein C (APC), it acts as an anticoagulant (prevents clotting).

As a procoagulant, activated FV (FVα) works with activated factor Xa (FXa) to generate thrombin. Thrombin generation is the key step in coagulation to form a fibrin clot, the end result of coagulation.
The anticoagulant function of FV is related to its role as a cofactor in the APC/protein S complex. This complex breaks down activated FVIII (FVIIa), which is a coagulation protein that plays an essential role in clot formation. Normally FV circulates in the blood in an inactive form. APC directly cuts FVa to create the anticoagulant form of FV. In patients with the FVL mutation, however, the FVa is altered and therefore cannot be cut by APC. As a result, FV cannot work as a cofactor for the APC/protein S complex in the breakdown of FVIIIa. This phenomenon is known as “APC resistance” and results in unchecked generation of FVIIIa. This, in turn, leads to uncontrolled thrombin generation and excessive clotting.

Risk of Clotting Disorder Associated With FVL Mutation

- **Risk of a Clotting Disorder in FVL homozygotes:** FVL homozygotes (people with two identical genes at the same position on two chromosomes) have only the Leiden protein and an 80-fold increased risk of a clotting disorder compared to the general unaffected population.
- **Risk of a Clotting Disorder in FVL heterozygotes:** FVL heterozygotes (people with two dissimilar gene forms) are believed to produce about 50% of Leiden protein and have a 5- to 7-fold increased risk of a clotting disorder compared to the general population. Remember that Factor V Leiden by itself does not cause blood clotting in individuals who are FVL heterozygotes; usually a triggering event is required.

Most blood clots in a heterozygous individual occur in association with another cause or “trigger.” In heterozygous women, the most common triggers are pregnancy, the use of birth control pills, and hormone replacement therapy after menopause. This is because the balance of coagulation is tipped towards clotting during pregnancy. Hormonal replacement and birth control pills mimic this condition. In one study from Europe, 60% of women who experienced clots during pregnancy were found to have the FVL mutation. The risk of blood clots is actually highest 6 to 8 weeks after the birth of a baby. In heterozygous men with FVL, blood clots may occur after surgery or an injury, especially injuries to the leg. **Not everyone with heterozygous FVL develops a blood clot.** For some people with heterozygous FVL, there is no history of abnormal blood clotting in their parents, even if one of their parents has this gene.

Other Mutations within Factor V gene

Besides the FVL mutation, several other alterations in the FV gene also contribute to APC resistance. People with the Factor V Cambridge mutation and the Factor V Hong Kong mutation have mild APC resistance. Recently a complex set of closely linked genetic markers of the FV gene (FV HR2) have been found to contribute to APC resistance, although to a lesser degree than FVL.

Acquired causes of APC resistance

Acquired causes of APC resistance include states associated with increased clotting, such as pregnancy, use of estrogen therapy, antiphospholipid antibody syndrome, and conditions with inflammation, such as sepsis syndrome or disseminated intravascular coagulation (DIC).

Prothrombin 20210 Mutation

A specific alteration of the prothrombin gene, which has been found to be present in 18% of people with a clotting disorder, increases the risk of blood clots almost threefold. This gene alteration or mutation, called the prothrombin 20210 mutation, is associated with higher levels of prothrombin and therefore increases an individual’s risk for blood clots. This mutation is also linked to other clotting events such as coronary artery disease (especially in young women and people with stroke), venous blood clots, clots in the mesenteric vein, and clots in the central retinal artery or portal vein.

Hyperhomocysteinemia

Hyperhomocysteinemia, or increased levels of the amino acid homocysteine, is estimated to affect 5% of the general population. Among persons with symptoms of coronary artery disease, the prevalence of hyperhomocysteinemia is estimated to be 13% to 47%. Mild to moderate hyperhomocysteinemia is an independent risk factor for stroke,
Heart attack, peripheral arterial disease, and narrowing of the extracranial carotid artery. High levels of homocysteine are associated with enzyme defects or shortages of folate or vitamin B6, particularly in the elderly. Mild or moderate hyperhomocysteinemia has been associated with venous blood clots in the young and recurrent venous clots. The condition also has been shown to have a high frequency (10%) in patients with first episodes of venous blood clots. Several inherited or acquired conditions may lead to an increase in homocysteine levels.

Inherited causes of hyperhomocysteinemia include low levels of an enzyme necessary for the conversion of homocysteine to cysteine, which increase the risk of a clotting event. Studies in families with low levels of the enzyme suggest that this disorder may be inherited.

Acquired causes of hyperhomocysteinemia include advanced age, tobacco use, coffee intake, low levels of folate in the diet, and low intake of vitamin B. Higher homocysteine levels are also associated with diabetes mellitus, cancers, low level of thyroid function, lupus, inflammatory bowel disease, and certain medications such as cholesterol-lowering agents, metformin, methotrexate, anticonvulsants, theophylline, and levodopa.

Elevated Levels of Clotting Factors

High levels of other procoagulants such as factors VIII, IX, XI, VII, fibrinogen, and Von Willebrand factor (VWF) are associated with an increased risk of clotting. Specifically, persistently high levels of FVIII have been shown to be associated with recurrence of a clotting disorder. Currently there are no specific recommendations to include analysis of these clotting factor levels in an evaluation for a clotting disorder.

Elevated Factor VIII

Coagulation factor VIII (FVIII) activity levels may vary widely due to various reasons, such as pregnancy, use of hormonal therapy, stress, exercise, or presence of an inflammatory state. It is often difficult to know whether a high FVIII level is caused by an acute event or leads to it. A high level of FVIII is a known independent risk factor for blood clotting. High levels of FVIII are an even stronger risk factor for recurrent blood clots. The likelihood of recurrence of a clotting event at 2 years was found to be 37% in people with a high FVIII level versus 5% among persons with a lower FVIII level.

Elevated Factor IX Levels

High levels of coagulation factor IX (FIX) may play a role in clotting disorders. The Leiden Thrombophilia Study found that levels of FIX in the 90th percentile and higher increased the risk of blood clots by 2- to 3-fold.

Elevated Factor XI Levels

Coagulation factor XI (FXI) has procoagulant and antifibrinolytic roles in blood clotting. FXI contributes to the formation of fibrin. It also protects the fibrin that has formed from being broken down. People with high FXI levels have an age- and sex-adjusted increased risk of a blood clot in a deep vein, such as a vein in the leg. This type of blood clot is called a deep vein thrombosis (DVT). The higher the FXI level, the greater the risk of a blood clot. Increased levels of FXI also have been associated with an increased risk of heart disease in women.

Elevated Factor VII Levels

Some studies have shown an increased risk of heart disease with high levels of coagulation factor VII (FVII). FVII levels, however, are not an independent risk factor after controlling for cholesterol, LDL-cholesterol, and triglycerides. Elevated FVIIa levels have been reported in people with blockage of a retinal vein.

Elevated Von Willebrand Factor Levels

Von Willebrand factor (VWF) is produced in cells that line the blood vessels (the endothelium). Damage to or swelling of the endothelial lining lead to increased VWF levels. FVIII circulates with VWF and often the levels of these two clotting factors are similarly affected by stress, inflammatory states, or endothelial injury. Continuously high levels of FVIII lead to an increased risk of blood clots; therefore it might be reasonable to assume that elevated levels of VWF would also be associated with and contribute to an increased risk of clots. Additionally, VWF plays an important role in platelet adhesion to areas of damaged endothelium. Elevated VWF levels may have more than one mechanism through which they contribute to clots.

Decreased Levels of Natural Anticoagulants

Antithrombin (AT)
Antithrombin is a naturally occurring anticoagulant that inactivates thrombin and clotting factors IXa, Xa, XIa, and XIIa. Heparin increases this inactivating effect of antithrombin. Changes in the antithrombin gene may cause deficiencies or abnormal activity of antithrombin. Antithrombin deficiency is an inherited condition.

Patients with a deficiency of AT are at risk for clotting in both arteries (arterial) and veins (venous). The frequency of people with symptoms of AT deficiency is estimated to be 1 in 2,000 to 1 in 5,000 people. AT deficiency without symptoms may occur as frequently as 1 in 600 people. In patients with a history of a clotting disorder, the incidence of AT deficiency ranges from 0.5% to 4.9%. People with AT deficiency who have defects in the heparin-binding site have a severe clotting tendency that presents early in life and often involves a clotting disorder in the arteries.

**Types of AT deficiency**

There are two types of AT deficiency: **Type I and II**. In people with Type I AT deficiency, a genetic alteration leads to **low levels of the AT protein**. People with Type II AT deficiency have an **abnormally functioning protein** because of a genetic alteration in the gene that codes for AT. The genetic alteration can affect how AT binds to heparin or how AT neutralizes the effect of thrombin in the absence of heparin.

**AT Levels**

The levels of AT reach normal adult range by around age 6 months and depend on the patient’s age and other associated conditions. Several conditions can reduce AT levels. These conditions include liver abnormalities, consumptive coagulopathy, complications during pregnancy or labor and delivery, kidney disease, cancer, malnutrition, gastrointestinal abnormalities, use of oral contraceptives, and other medications. Use of medicines like Coumadin® may lead to increases in AT levels. In some people with an altered AT gene, the levels of AT range between 40% and 70% of normal.

**Protein C**

Protein C, a vitamin K dependent protein, is made in the liver and contributes to the inactivation of FVIII. Protein C deficiency is an inherited condition. Protein C is slowly activated by thrombin to **activated protein C (APC)**. The activation is increased 20,000 fold when protein C forms a complex with thrombin that is bound to a receptor in the blood vessel lining. This receptor is called **thrombomodulin**. APC helps regulate the coagulation pathway by inactivating FVa and FVIIIa that are bound to membranes. Aside from its role in coagulation, APC also has anti-inflammatory and cell-protective functions.

The frequency of protein C deficiency ranges from 1.4% to 8.6%. In a study of healthy subjects, the frequency of the deficiency was found to be 1 in 200 to 1 in 300, while a study of almost 10,000 blood donors found a frequency of 1 in 500 to 1 in 700.

**Types of Protein C deficiency**

Protein C deficiency is divided into **Type I or Type II deficiency**. People with Type I deficiency have low levels of protein C and proportionally low levels of protein C activity. In people with Type II deficiency, the activity of protein C is low because the genetic alteration produces an abnormally functioning protein.

Homozygous protein C deficiency usually appears in newborn infants as a rare and potentially catastrophic skin condition called **purpura fulminans**. Patients with purpura fulminans have sudden massive areas of bleeding in the skin that can become severely infected. Purpura fulminans is associated with severe illness if not death unless promptly identified and treated. The symptoms are caused by the formation of blood clots in capillaries and small blood vessels, which lead to tissue death as a result of lack of blood flow in the affected skin. Laboratory testing of babies with this condition reveals a severe deficiency (protein C levels of <1% of normal). Some infants who do not have neonatal purpura fulminans but still have low levels of protein C (5% to 20%) often have a severe tendency to clot at an early age. These patients need lifelong anticoagulation to prevent recurrent blood clots.

**Warfarin-Induced Skin Necrosis (WISN)**

People with protein C deficiency can experience a potentially catastrophic complication of warfarin therapy, commonly known as **warfarin induced skin necrosis (WISN)**. When warfarin therapy is started, it can lead to a rapid drop in levels of protein C and coagulation factor FVII. The levels of other clotting factors remain relatively high. This upsets the normal balance between bleeding and clotting states, resulting in a temporary super-clotting state.
particularly in the small blood vessels of the extremities. This imbalance between procoagulants (promote clotting) and anticoagulants (prevent clotting) is further exaggerated in protein C deficiency. This effect may be more pronounced when large loading doses of warfarin are used. WISN typically occurs during the first few days of warfarin therapy. The skin damage of WISN is distributed on the extremities, torso, breasts, and penis. The symptoms begin as redness of the skin. If appropriate therapy is not started promptly the redness progresses to become purplish blotches on the skin (purpura). The skin tissue can eventually die as a result of blood clots that interrupt the blood flow in skin tissue. To avoid this catastrophic complication, people with protein C deficiency are treated simultaneously with other blood thinners such as heparins until the appropriate level of blood thinning is achieved through warfarin. Infusion of protein C concentrate or fresh frozen plasma may be used to increase protein C levels.

Protein C Levels
Protein C levels depend on the patient's age and other conditions, with adult levels being reached at late adolescence. Many medical conditions may reduce protein C levels. These conditions include liver disease, disseminated intravascular coagulation (DIC), clotting disorders, respiratory distress syndrome (RDS) in newborns, preeclampsia, acquired purpura fulminans, systemic lupus erythematosus, ulcerative colitis, oral contraceptives, and oral blood thinners.

Protein S
Protein S, a vitamin K-dependent protein, is made by the liver and acts as the principal cofactor to protein C. Protein S exists as two forms in the blood circulation: a free form and a bound form. Approximately 60% to 65% of total protein S in the circulation exists in the bound form and ~35% to 40% in the free form. Free protein S is the form involved in the activated protein C (APC) blood thinning activity.

There are no data on the frequency of protein S deficiency in the general population, but the frequency is believed to be roughly the same as for protein C deficiency (1.4% to 7.5%).

Types of Protein S deficiency

- **Type I deficiency**: The decrease in the activity of protein S is proportional to the decrease in the level of protein S.
- **Type II deficiency**: The levels of the free and bound forms of the protein are normal, but they do not function properly because of a gene alteration.
- **Type III deficiency**: There is a normal level of total protein S, but the level of free protein S is abnormally low.

Protein S deficiency is rare in the healthy population, with an estimated frequency of approximately 1 in 700. When considering a selected group of patients with recurrent blood clots or a family history of clotting, the frequency of protein S deficiency ranges from 3% to 6%. The frequency of homozygous deficiency has been estimated to be 1 in 160,000 to 1 in 360,000. Infants and babies within the first year of life who have homozygous protein S deficiency characteristically have purpura fulminans.

Protein S Levels
Adult levels of protein S levels are reached when a child is approximately 6 months to 1 year old. Compared to men, women tend to have on average a lower level of free protein S, especially when pregnant or taking oral contraceptives. Newborn infants also have lower free and total protein S levels. Many medical conditions may be associated with abnormal protein S levels including liver disease, DIC, a clotting disorder, herpes infections, systemic lupus erythematosus, ulcerative colitis, and use of oral contraceptives and oral blood thinners. Levels in heterozygotes are approximately 40% to 70% of the normal level.

Thrombomodulin
Thrombomodulin is a transmembrane protein found on the surface of cells lining the blood vessels (endothelium). It acts as a receptor for thrombin and plays an important role in coagulation and clot breakdown (fibrinolysis).
Thrombomodulin-bound thrombin starts the protein C anticoagulant pathway by activating protein C. Defects in thrombomodulin result in increased coagulation. Thrombomodulin also activates thrombin-activated fibrinolysis inhibitor (TAFI), which affects clot breakdown. Small thrombomodulin fragments circulate in soluble form in plasma of healthy individuals. These soluble fragments retain their functional activity and can be measured in plasma. Increased levels are seen in patients with venous and arterial clotting conditions, including clots in the brain and eyes, and DIC. The clinical relevance of soluble thrombomodulin levels in treating clotting disorders is not fully known.

**Heparin Cofactor II**

Heparin cofactor II is found in plasma and rapidly inhibits thrombin in the presence of dermatan sulfate or heparin. Heparin cofactor II deficiency is classified into:

- **Type I (quantitative):** There is a decrease in both cofactor level and its functioning.
- **Type II (qualitative):** There is a decrease in the functional activity of the protein with normal cofactor levels.

Only a few cases of heparin cofactor II deficiency have been described. Further research is needed to find out the clinical importance of heparin cofactor II deficiency.

**Tissue Factor Pathway Inhibitor (TFPI)**

Tissue factor pathway inhibitor (TFPI) inhibits a complex that starts the process of clotting. Most of TFPI (60% – 80%) is bound to the lining of the blood vessels (endothelium), with only 20% free in the blood. Recent evidence suggests that low levels of TFPI are a risk factor for clotting disorders. Interestingly, different forms of the TFPI gene have been found that result in higher levels of TFPI in the circulation. One report suggested that these higher levels “correct the balance” in patients with Factor V Leiden, and normalize their risk for a clotting event.

**Abnormalities of Fibrinolysis**

**Plasminogen Deficiency**

Plasminogen is synthesized in the liver and is present in most tissues. Plasminogen is converted to the enzyme plasmin by plasminogen activators such as tissue-plasminogen activator (tPA) and urokinase-plasminogen activator (uPA). The main action of plasmin is to break down fibrin. Defective clot breakdown (fibrinolysis) has been associated with clotting diseases.

**Types of Plasminogen Deficiency**

There are two types of plasminogen deficiency. In people with **Type I** deficiency, there is a proportionate decrease in both the level of plasminogen and its activity. In **Type II** deficiency (also called dysplasminogenemia), there is a decrease in the functional activity of the protein, although the plasminogen levels are normal. Plasminogen levels do not reach the healthy adult range until late adolescence. Higher levels of plasminogen are found in women in the last trimester of pregnancy; newborns have levels approximately one-half of levels in healthy adults.

The most common clinical symptom of plasminogen deficiency is ligneous (‘wood-like’) conjunctivitis (inflammation of conjunctiva in the eye). The conjunctiva becomes irritated because of a build-up of white, yellow-white, or red thick masses with a wood-like consistency that may replace normal tissue. The build-up (also called lesions) occurs mostly on the eyelids and may be triggered by injury or infection. The build-up often recurs after it has been removed. The wood-like lesions have been reported to occur in other mucous membranes, such as the mouth, nasopharynx, windpipe, and female genital tract. Some affected children may experience congenital occlusive hydrocephalus (increased fluid around the brain). Removal of the lesions does not cure the condition and may promote recurrence. The lesions are responsive to systemic plasminogen replacement or to local therapy in the eyes. The incidence of plasminogen deficiency is not well known and may be underestimated because ophthalmologists, dentists, obstetricians, gynecologists, and ENT physicians may see these patients and not refer them or not recognize the symptoms to be related to plasminogen deficiency.

Plasminogen deficiency that runs in families appears to be an uncommon but recognized cause of an inherited clotting disease. The clotting complications of this deficiency predominantly involve the veins and include
thrombophlebitis, PE, and stroke. It is interesting to note that in affected individuals, there have not been reports of clotting disease in association with pregnancy or oral contraceptive use, and even more intriguing is the report that plasminogen levels become normal in a deficient patient during pregnancy or with use of oral hormonal therapy. This finding indicates that in heterozygotes, the normal gene form may be able to increase plasminogen synthesis. Because homozygous patients infrequently develop clots, especially spontaneous events, the possibility of heterozygous plasminogen deficiency as a cause for a clotting event may be dismissed or overlooked.

**Decreased Levels of Tissue Plasminogen Activator (tPA)**

Tissue plasminogen activator (tPA) is synthesized by endothelial cells. When tPA is released, it converts plasminogen to plasmin. Theoretically, decreased release of tPA could lead to a super-clotting state (also called hypercoagulable) due to decreased clot breakdown (fibrinolysis).

**Increased Levels of Plasminogen activator inhibitor 1 (PAI-1)**

Plasminogen activator inhibitor 1 (PAI-1) functions as the primary inhibitor of plasminogen activator in plasma. Increased levels of PAI-1 could lead to excessive inhibition of tPA, leading to decreased activation of fibrinolysis and a clotting tendency. Increased PAI-1 levels have been shown in some cases to be an inheritable trait.

**Elevated levels of thrombin-activatable fibrinolysis inhibitor (TAFI)**

Increased levels of thrombin are needed for clot formation and to prevent clot breakdown. If the thrombin levels are too high, they activate thrombin-activatable fibrinolysis inhibitor (TAFI). TAFI helps to stop (inhibit) clot breakdown by preventing plasminogen from binding to the fibrin clot. Increased levels of TAFI may prevent the start of normal clot breakdown and therefore could theoretically increase the tendency for a clotting state. The Leiden Thrombophilia Study suggests that high levels of TAFI may be a mild risk factor for a super-clotting state.17 However, these results require confirmation.

**Other Inherited Causes Associated with Increased Risk of Blood Clots**

**Paroxysmal nocturnal hemoglobinuria**

Paroxysmal nocturnal hemoglobinuria (PNH) is a rare disorder of stem cells and is caused by a gene alteration on the X chromosome. PNH is known to be associated with an increased risk of clotting. PNH results in the breakdown of red blood cells (hemolysis), which causes the release of hemoglobin into the blood. Ultimately the hemoglobin is released in the urine. This release produces dark-colored urine most often in the morning (also called hemoglobinuria). This type of hemoglobinuria was called “nocturnal” because it was believed that the breakdown of red blood cells occurred during sleep. This observation was later disproved. Hemolysis has been shown to occur throughout the day, but the concentration of urine that occurs through sleep results in the dramatic color change. Once considered to be an acquired hemolytic anemia, PNH has been reclassified as an inherited condition related to a genetic alteration in stem cells. In people with PNH, surface proteins are missing not only in the membrane of red blood cells but also in all blood cells, including platelets and white cells.18 This disorder usually presents in adulthood and is less common in childhood. In adults PNH is most commonly seen as hemolytic anemia with nighttime episodes while in children PNH is most commonly associated with bone marrow failure. Blood clots may occur in 39% of adults and 31% of children with PNH. The clots usually occur in the veins, particularly in the veins of the liver (Budd-Chiari syndrome), but the portal veins, central nervous system, and peripheral venous system also may be involved. Increased circulating activated platelets have been implicated in clotting events due to PNH, but no consistent fibrinolytic or coagulation abnormality has been documented.

**Want to Learn More?**

- Signs and Symptoms of Blood Clots
- Diagnosis of Blood Clots
- Treatment of Blood Clots
- Acquired Causes of Blood Clots
- Special Considerations for People with Blood Clots
- What Can the IHTC Do for You?
Deep vein thrombosis is a condition of formation of blood clots in lower part of legs or thigh. The thrombus (blood clot) – from which the condition derives its name – poses serious health risks when a broken part of the blood clot reaches pulmonary circulation system or the heart. This may lead to pulmonary embolism – condition in which blood clot rests on pulmonary artery of lung. Herbal remedies help prevent deep vein thrombosis without major side effects. Here are some of the best herbal remedies for deep vein thrombosis.

AMAZING HERBAL REMEDIES FOR DEEP VEIN THROMBOSIS

Cayenne

Cayenne For Deep Vein Thrombosis
This particular herb is a well known **natural blood thinner**. It normalizes blood pressure and promotes blood circulation in the body. **The active agent in cayenne pepper is capsaicin that reduces cholesterol in blood and triglyceride levels.** It prevents platelet aggregation and hence helps the body in dissolving fibrin, which is crucial in the formation of blood clots. It stimulates CNS (central nervous system) and improves functioning of heart. Hence, it has been traditionally used as a remedy for heart disorders. Regular use of cayenne strengthens the heart. You can apply cayenne pepper directly on the affected part of legs for relief from pain.

**Rosemary**

![Rosemary](https://via.placeholder.com/150)

**Rosemary For Deep Vein Thrombosis**

This particular herb has amazing benefits for blood circulatory system and hence is a popular herbal remedy for sprains, bruises, low blood pressure, varicose veins, etc. **It contains flavonoid called diosmin that reduces fragility in capillaries and hence boosts blood flow.** It is also rich in vitamin B6 (pyridoxine) that metabolises protein like hemoglobin and aids it in carrying oxygen to tissues in body.

**Butcher’s Broom**

![Butcher’s Broom](https://via.placeholder.com/150)

**Butcher’s Broom For Deep Vein Thrombosis**
This particular herb has been in use for its action against circulatory disorders like spider veins, varicose veins, chronic venous insufficiency, etc. **It has strong anti-inflammatory properties that reduce swelling in legs caused by deep vein thrombosis.** It also promotes better blood circulation thereby preventing risks of deep vein thrombosis and further pulmonary embolism. The active agents in butcher’s broom are sterols, fatty acids and few rare flavonoids like sparteine and ruscogenin. These flavonoids improve condition of blood arteries and blood vessels.

**Gingko Biloba**

![Gingko Biloba For Deep Vein Thrombosis](image)

It improves blood flow through dilation of blood vessels. The active agents in **gingko biloba** are flavonoids that protect heart muscles, blood vessels, nerves, and retina. Hence, it treats various diseases and disorders related to poor blood circulation like heart disease, brain disorders like **Alzheimer’s disease**, cancer, balding, etc. Besides, Gingko biloba has strong antioxidant properties that prevent damage by free radicals.

**Hawthorn**

This particular herb has amazing antioxidants such as quercetin and OPCs (oligomeric procyandins) that free the body from the harmful effect of free radicals. The antioxidant properties of the herb also prove helpful in dilating blood vessels, protecting blood vessels, improving blood flow and thereby benefiting the heart. The herb avoids damage to blood vessels.
**Skullcap**

It contains many anti-inflammatory and antioxidant compounds such as flavonoids namely wogonin, baicalein and baicalin. **Due to the anti-inflammatory properties, the herb cures leg pain caused by deep vein thrombosis.** It improves blood circulation and inhibits platelet aggregation.

**Ginger**

Apart from being a cure for plenty of diseases, ginger plays a great role in curing deep vein thrombosis. **Its anti-platelet activity proves to be a boon for patients of deep vein thrombosis.** Besides, it is effective in breaking down fibrin that causes the disease. It boosts blood circulation in arteries as well as veins.
References

Vitamin K - key to help blood clotting

fat-soluble vitamin

- MAKES PROTEINS FOR BLOOD CLOTTING & HEALTHY BONES
- Leafy green vegetables are the best source of Vitamin K
- JUICED PROVIDES 80 MCG which is 100%DV

SOURCE: Physicians Reference Desk – Page 1547