Anticoagulant Natural Alternative

Natural Anticoagulant Regimen

A natural anticoagulant routine. How could this benefit you? How can you implement this? Why is this a time-proven alternative to conventional approaches?

Hopefully, you have read part1, part2, and part 3 of this 4 part series. Primers on coagulation and thrombosis. I have carefully explained the process of coagulation resulting in heart attacks and strokes. We have talked about platelet aggregation. Which then leads to fibrin aggregation. Which leads to the final clot. All clots cause obstruction. Obstruction causes heart attacks and strokes. How can we prevent this?

I have alluded to the most powerful natural anticoagulants. These include the following:

- Nattokinase — 100 mg twice daily
- Ginkgo biloba — 120mg daily
- High-dose fish oils — 1 tablespoon (10 grams) daily
- Vitamin E — 800-1200 units daily
- Adequate hydration — many glasses of pure water daily
Nattokinase

Let’s start with Nattokinase. Nattokinase is a derivative of the natto bean. This is a Japanese food source. Nattokinase is a serine protease produced by 

* Bacillus subtilis 
during fermentation. Nattokinase is very bitter to the taste! Unpalatable. Therefore, the standardized extract is preferable and convenient.

Nattokinase is a fibrinolytic agent. You can refer back to all previous fibrin pathways. The goal is to prevent fibrinogen converting to fibrin.

Nattokinase has been studied extensively by the Japanese. Nattokinase biochemical activity is labeled in FU. That is fibrinolytic units. These activity units have a mg equivalent. A typical dose is **100 mg (2000 FU) twice daily. It is essential that you take this twice daily.** Once daily is insufficient. This has been time proven through empirical observation.

I have treated thousands of cases with Nattokinase preventing recurrent thrombophlebitis, strokes and heart attacks. It is essential that you source NSK-SD.

There are alternatives to Nattokinase. Lumbrokinase is derived from the earthworm. It is commonly sold as Bolouke in Canada. By the manufacturer’s claims, it is even more potent. Higher biological activity. Serrapetase has similar but not identical properties. I use Serrapetase to treat arterial plaques.

Here is a great reference paper on the fibrinolytic and anticoagulant properties of [Nattokinase](#).
Ginkgo Biloba

Ginkgo biloba has many actions. This is a time-honored and venerated Chinese herb. Ginkgo biloba has multiple constituents. These are flavonol and flavone glycosides, lactone derivatives (ginkgolides), bilobalide, and much more. We use a standardized 26% ginkgo extract. The Germans have studied this in detail. Reference the [German E Commission](#). Here is a more readable guide.

The dose I recommend is **120 mg daily**. Or 60 mg split twice daily. German studies have advocated as high as 240 mg. Through empirical observation, I find this dose to be way too aggressive. Ginkgo is so potent that 240 mg will probably cause adverse bleeding.

Ginkgo Biloba exerts its action primarily as an anti-platelet anticoagulant drug. It inhibits platelet aggregating factor. Ginkgo has many other uses. It is an antioxidant. Ginkgo may have vasodilatory effects. It is used for cognitive enhancement. I find it much more effective as a cardiac protective drug.

Be careful with your dosing ginkgo biloba. I advocate the use of 4Sight. This is a potent combination used to prevent eyesight deterioration. It includes 60 mg of ginkgo biloba. It is one example of adjusting the total dose of ginkgo biloba. And that is, again, 120 mg.

Because of its action, ginkgo biloba should not be combined with aspirin or NSAIDs. It will have synergistic effect.

High-dose fish oils
High-dose fish oils have a “rheological” effect. That is the prevent aggregation of red blood cells. It is the aggregation of platelets and/or red blood cells is the initiating process. High-dose fish oils act like Teflon to prevent this aggregating effect. The dose that I recommend is aggressive. 1 tablespoon daily. You will rarely achieve these doses with oral capsules. I highly recommend fish oils in liquid form.

Fish oils contain EPA (eicosapentanoic acid) and DHA (docsoahexanoic acid). I recommend 4000-5000 mg of EPA and 3000-4000 mg of DHA. That would total approximately 10 grams of fish oil. 1 tablespoon of high potency fish oil will yield this high dose of 5000 mg EPA and 4000 mg of DHA. I stress again, you will not achieve these doses with fish oil capsules.

Fish oils, in addition to their anticoagulant effect, may have multiple benefits:

- enhance brain function – cognitive enhancement
- prevent cardiovascular events
- antidepressant
- improve skin turgor
- lower blood pressure
- treat gastric reflux

Vitamin E
Vitamin E also has rheological properties. It prevents red blood cell aggregation. Vitamin E exists as a family of tocopherols.

I highly recommend mixed tocopherols. This is a isomeric mix of natural tocopherols including the alpha, beta, gamma and delta forms of vitamin E. There is a difference between “natural” vitamin E and “synthetic” natural vitamin E. Synthetic vitamin E is a tocopheryl not tocopherol.

I routinely recommend a **400-800 units of mixed tocopherols** (vitamin E) daily. In many instances I will recommend 800-1200 units of vitamin E. Studies have shown that Gamma tocopherol is much more potent than alpha tocopherol. Most studies only investigate the alpha form of Vitamin E.

**Water - Hydration**

And last but not least, hydration. Keep well hydrated. Hydration will also prevent red blood cell and platelet aggregation. It will improve skin turgor. I highly recommend water in glass bottles, not plastic bottles. You should avoid all plasticizers. PVCs, phthalates and BPA. The softer the plastic bottle the higher the plasticizer content. Never tap drink water. Most likely, all your municipal water sources are contaminated or polluted. Chlorine, chloramine, flouride, and heavy metals. Flint Michigan is just the tip of the iceberg.
Potency and efficacy

So this routine when fully implemented will prevent platelet and red blood cell aggregation. It prevents the conversion of fibrinogen to fibrin. This treats all pathways of coagulation. It is a much more comprehensive approach to anticoagulant therapy than conventional drugs. Less expensive. With fewer complications. Is it effective?

Virtually every surgeon now has been taught to ask, “what other vitamins and herbs are you taking?” Every surgeon knows that each and all of these agents clearly have anticoagulant effects. Surgeons see the effects of drugs. Internists only infer the effects of drugs. Surgeons know that prior to surgery anticoagulants can significantly increase complications. However, just following surgery the opposite effect is noted. The body may react with a vigorous coagulation response.

So empirically and rationally you can see this routine has the ability to effectively prevent heart attacks, strokes and recurrent thrombophlebitis.

Medical Supervision is Essential

A major caveat! I never advocate abruptly stopping conventional anticoagulant routines. You need medical supervision to start and monitor your progress. This anticoagulant routine requires special knowledge of potency, dosing and the value of these natural sources. I have been treating thousands of cases over the past 20 years. This is time-tested. It is effective. But this regimen is not “validated” through conventional guidelines or task force committees. It is not the “standard of care.” For this reason, your internist, cardiologist or family practitioner will have little understanding of the rationale or efficacy of this routine.

This is not a DIY — do it yourself advocacy.

So where do we go from here? Let me give you some suggestions. Call us or
write for further information.

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AntiPlatelet AntiCoagulant Drugs

Coagulation and Anticoagulant Therapy

Read [part I](#) and [part II](#) of this series for background. So you will have a basic understanding of coagulation pathways. Yes, it is complex.

Remember, coagulation or clotting starts with platelet aggregation. That initiates the complex coagulation cascade. This causes the mature thrombus or blood clot. Aggregated platelets with a thick fibrin mesh causes the thrombus. The thrombus causes heart attacks, strokes or thrombophlebitis. Here is the final complete picture:

![Diagram of coagulation and anticoagulant pathways](#)
So let’s discuss anticoagulant therapy medications.

**Antiplatelet Agents - Aspirin and Plavix are first line of defense**

Figure 2 shows how we prevent platelet aggregation and activation at various stages. Look at figure 1. *Aspirin* has been the mainstay of antiplatelet therapy. There has been a succession of drugs over the last three decades. Newer antiplatelet drugs supersede older ones. Are these true advances or simply marketing campaigns?

![Diagram of antiplatelet drugs](fig2.png)

Aspirin has been well studied in the literature. A full 325 mg dose will prevent platelet aggregation. Over time, aspirin has significant adverse reactions. These are direct and indirect reactions. It can cause tinnitus (ringing or buzzing noises in the ear). It can cause major gastrointestinal bleeding. This is a significant problem.

Therefore, baby aspirin (81 mg) is a reputedly safer dose. This lower dose is effective. We now know this is subject to individual variations. Fast or slow metabolizers. There are ways of testing for aspirin efficacy. Aspirin Resistance
(11-Dehydrothromboxane B2) is one available test. Realistically, we rarely perform this test. So that empirically, an 81 mg (baby aspirin) dose is less toxic. Interestingly, we rarely treat babies and toddlers with baby aspirin any longer.

**Aspirin is a COX inhibitor.** COX is an acronym for cyclo-oxygenase. There are COX-1 and COX-2 enzymes.

Fish oils are potent COX inhibitors. I will talk about Fish Oils in the final chapter - part 4.

**Plavix** (Clopidogrel) is now the second most commonly prescribed drug in the US. A 75 mg dose is common. **Plavix is an ADP antagonist.** A totally different site of action from aspirin. Plavix combined with aspirin has never been a rational choice. It is over treating, potentially causing more complications. Various studies have proven this. **Lancet** argument against using Plavix and aspirin together:

> Adding aspirin to clopidogrel in high-risk patients with recent ischaemic stroke or transient ischaemic attack is associated with a non-significant difference in reducing major vascular events. However, the risk of life-threatening or major bleeding is increased by the addition of aspirin.

Dr Mercola also writes:

> Sadly, Plavix also has serious side effects you may not be aware of. Six years ago, I warned my readers that when combined with aspirin, the drug nearly **doubled the death rate** from heart disease among patients who had not had a previous heart attack but were at risk, compared to those taking aspirin alone.

**Anti Fibrin and Standard Anticoagulant Drugs - Coumadin**

During acute episodes in hospital, physicians use heparin. This is intravenous dosing. Once discharged, physicians prescribe **Coumadin (Warfarin)** for long-term therapy. Yes, Warfarin is rat poison.
Typically, Coumadin anticoagulant therapy is continued for a variable period of time depending on the condition. **Coumadin blocks vitamin K-dependent conversion steps.** So you are advised not to ingest any extra vitamin K. Or foods that are high in vitamin K such as leafy green vegetables.

Prothrombin time (PT) was used in the past to monitor the efficacy and dosing of Coumadin which is quite variable. Today we use the INR (international normalized ratio). Weekly monitoring is necessary initially. Then less frequently as the INR activity stabilizes. But I stress, there is considerable variability in dosing from 2 mg up to 12 mg.

**A typical Coumadin dose is 5-6 mg daily.**

Coumadin has serious adverse consequences because it blocks vitamin K. **Matrix GLA** is a vitamin K-dependent protein that regulates calcium flow. It prevents “reverse calcium flow.” That is, calcium flowing out of the bones into the arteries (and heart valves). Coumadin can accelerate osteoporosis and arterial calcification. That is why high doses of vitamin K in healthy adults is so vital. So the risk-benefit ratio seems questionable. Here is one journal citation

*Fourteen years ago in this journal, Price and colleagues reported that 2 weeks of warfarin treatment in young rats “caused massive focal calcification of the artery media” (1). It had been known for years that Warfarin could induce mineral deposition in the arteries of rodents (2), and the phenomenon was so robust that Warfarin was often used as an experimental model for vascular calcification, but the mechanism was unknown.*

**Newer Anticoagulant – Xa (10a) blockers**

So time marches on. Newer drugs, with which are not vitamin K antagonists, are now the “latest advance.” These are Xa (10a) antagonists. They eliminate the variability of Coumadin dosing and the necessity for frequent testing. And we suspect marketing decisions are in play.

What are these newer drugs?

- **Dabigatran Etxilate - Pradaxa 150 mg twice daily**
- **Rivaroxaban - Xarelto 20 mg daily**
**Apixaban - Eliquis 5 mg twice daily**  
**Edoxaban - Savaysa 60 mg daily**

These newer drugs avoid frequent monitoring. What are the downsides? There are no currently available “antidotes” to unintentional overdose. Vitamin K is a simple antidote to Coumadin. And, long term, there are consequences yet to be fully determined. In one study from *BMC Musculo-skeletal Disorders*, there was a direct toxic effect on osteoblasts. These are the cells that build new bone:

*In conclusion, rivaroxaban and enoxaparin treatment led to a reduction in alkaline phosphatase activity and a reduction in BMP-2, osteocalcin and Runx2 mRNA expression, indicating that treatment with both drugs leads to a general negative effect on osteoblast activity.*

The Medical Letter lists these as the major toxic effects. Common to all anticoagulants:

*The most common adverse effect of edoxaban in clinical trials was bleeding. Edoxaban has no established antidote to reverse its anticoagulant effect, which persists for about 24 hours after the last dose, and it is not dialyzable. Epidural or spinal hematomas resulting in permanent paralysis could occur in patients taking the drug who require neuraxial anesthesia or spinal puncture.*

We will eventually discover other long term consequences. This class is certainly far more convenient. And way more expensive.

So let us turn our attention in the final Part 4 to a comprehensive and time-tested natural anticoagulant routine.

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**Coagulation Heart Attack and**
Stroke - Part 2

It is necessary to understand the interaction of fibrin coagulation pathways and platelet aggregation. Once we understand these interactions we can then discuss current treatment modalities. My goal is to show you a more creative and natural approach to anti-coagulation.

Initial Platelet Aggregation

In part one I emphasized the importance of platelet aggregation as the initial step. There is an initial injury to an arterial or venous wall. As a response, activated platelets cause a clumping reaction to repair the injury. Somewhat like Hans Brinker’s “finger in the dike.”

This injury could be a laceration. It could be a rupture of a atheromatous plaque. It could simply be a tear in an arterial wall. Or simply from low-flow stasis. Atrial fibrillation is an example.
You can see from fig 2 above that the platelets initiate an array of reactions. I will elaborate even more detail in the next post. This is ever-increasing overview.

**Aspirin** is the most common drug to prevent platelet aggregation. We will look at an array of drugs that have been used over the years. But there is a more creative approach.

Once the initial platelet plug has formed the long-term fibrin coagulation pathway is initiated.

### Fibrinogen pathways

#### Prothrombin Time

The classic fibrin clotting pathway is divided into the intrinsic and extrinsic systems. The intrinsic system starts with Factor XII. There is a cascading series of reactions to the final common pathway. This is prothrombin activating **thrombin**. Thrombin then catalyzes the reaction of fibrinogen to **fibrin**. Finally, fibrin is polymerized to an insoluble mesh. The extrinsic arm interacts with the intrinsic arm.

As discussed previously, the production of fibrin can be blocked at various stages. The conversion of fibrinogen to fibrin can be reversed through a process called
fibrinolysis. An important concept.

So now we have a complete picture. The following diagram summarizes the evolution of this thrombus from platelet aggregation to fibrin clot.

**Decision-making - Blocking Which Pathway?**

Now we have rapidly covered the two aspects of thrombosis (clot) production. This is where the subject becomes indeterminate. Some “guidelines” emphasize the prevention of platelet aggregation. While other guidelines emphasize the long-term use of anti-fibrin drugs. What is the best strategy? Depends on the guidelines. These evolving guidelines are determined by age, associated risk factors, and preceding condition.

These guidelines were originally developed to make decision-making simple. And uniform. And coherent. The community standard of care. But guidelines eventually become bloated and incoherent. Much like IRS tax code. I anticipate this decision-making process will soon be totally computerized. Remove the human discretionary input. I do not agree. Look here to see just how complex this [decision tree for anticoagulant therapies](#) can be.

The entire purpose of this series of posts is to present a novel combination of anti-fibrin anti-platelet modalities.

Now you have an overview of the basic flow. In part 3 we will examine current and past drug interventions.

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**Coagulation Stroke Heart Attack**
Part 1

Heart Attack and Stroke Coagulation Basics

Let’s talk about heart attack and stroke causes and the complexity of the underlying coagulation (clotting) pathways. What a heady subject. You need a PhD in medical hematology, just to fully understand the complexities of coagulation. So let’s work through this step by step. This will be a multi part series. I will help you achieve a clearer understanding. None of us wants to suffer a heart attack or stroke!

What causes these catastrophic vascular events? What are the risks? What medications are commonly used? Why are they not necessarily the best or even the healthiest choice? Can we assess risk benefit ratios? And what are the natural herbal-based alternatives? Ones that can be just as effective with fewer side effects and less costly. This is what you will not hear from your personal internist, cardiologist or even family physician.

Blood Clot is a Thrombus or Embolus

A blood clot usually starts with an injury to a vascular wall. This could be one of your arteries or veins. An intricate series of reparative or reactive events is set in motion. Your body tries to rapidly repair injury to the vascular wall. Atrial fibrillation is an alternative source of thrombus formation. In this instance, stasis
and not injury, initiates the reaction.

Fig 1 above shows you a representation of the well formed blood clot. It has the potential for blocking blood flow (vascular occlusion) which deprives tissues distal to the block of vital cell oxygenation. The medical term is myocardial infarction or cerebral infarction. Cells die from lack of oxygen.

*The initial early phase begins with activation and aggregation of platelets.*

![fig 2](image)

Fig 2 shows you the beginning of the activated platelet aggregation sequence.
The final result of thrombus formation is illustrated in fig 3. An artistic rendering of the platelet-rich polymerized fibrin lattice.

Now, I want you to look at this animated video of this rapidly evolving sequence of events. It will more visually summarize this first part. This two-minute video is well worth watching. Then we will move on to the individual steps of each coagulation pathway. Once we understand the sequence of events can begin to understand the rationale for various treatment modalities.

Thrombin Fibrinogen Fibrin Pathways -
Here is the classic series of reactions in the fibrin producing pathway. What a complex picture! Let’s see if we make some sense of this rich set of reactions. How does this affect stroke and heart attack (myocardial infarction) risk?

First look at the pink box. That is the fibrin cascade. The common pathway is the activation of thrombin. This cascades to fibrinogen. Fibrinogen is converted to fibrin. Mono-filament fibrin is elaborated. It will become a fibrin polymer. This is not yet a mature thrombus (clot). We will talk about the platelet contribution in Part 2. We routinely measure fibrinogen in all our patients — every time.

What is important in this diagram is the internal and homeostatic balance. Bleeding vs clotting (thrombosis). There are various counter mechanisms to thin the blood. Your body has its own internal set of reactions. Now look at the green box. The most common is plasmin. Another is a less recognized but highly important PAI-1 (plasminogen activator inhibitor). The PAI-1 is susceptible to 3 critical genetic variations. If it is mutated, you will see decreased fibrinolysis (clot breakdown). This can accelerate thrombus or clotting formation. This diagnosis is frequently missed by most hematologists. It can be source of severe pulmonary embolus or deep vein thrombophlebitis.

We will explain fibrin and platelet pathways in greater detail in Part 2.