

What Causes Heart Attacks

by Dr. Thomas Cowan

In this article, I lay out the case that the spectrum of heart disease that includes angina, unstable angina, and myocardial infarction (heart attack) is better understood from the perspective of events happening in the myocardium (heart) as opposed to events in the coronary arteries (the arteries that supply the heart). As we all know, the conventional view of the cause of heart disease is that the central events occur in the arteries. In this article, I will go into more detail about why the conventional theory is largely misleading, and then I will describe the precise and well-documented events that do lead to heart attacks.

This new understanding is crucial because, as the past 50 or so years have shown, pursuing the coronary-artery theory has cost this nation billions of dollars in surgical costs – most of which are unnecessary – and billions in medications that cause as much harm as any benefits, and has led many people to adopt a low-fat diet and which only worsens the problem. In contrast, by understanding the real pathophysiological events behind the evolution of MIs, we will be led to a proper “Nourishing Traditions”-style diet, the use of the safe and inexpensive medicine g-strophanthin, and most important, we will be forced to look at how heart disease is a true manifestation of the cost of modern life to human health. To overcome the epidemic of heart disease, we need a new medical paradigm, a new economic system, and a new ecological consciousness; in short, a new way of life. The coronary theory misses all of this, just as it

misinterprets the actual pathological events.

In writing this article, I am indebted to the work of Dr. Knut Sroka and his website www.heartattacknew.com. For all who are interested in this important subject, it is advised to read the entire website and watch the video on the website. For health professionals and researchers, your understanding of this subject is incomplete without reading and studying two items found in the print version of the website: *The Etiopathogenesis of Coronary Heart Disease: A Heretical Theory Based on Morphology*, by G. Baroldi, and “On the Genesis of Myocardial Ischemia,” by Sroka.

Rebuttal of the Conventional Theory

Until recently, it was thought that most MIs were caused by progressive blockage created by plaque buildup in the major arteries leading to the heart (there are four major coronary arteries). The plaque was thought to be cholesterol buildup in the arterial lumen (inside of the vessel), which eventually cut off blood supply to a certain area of the heart, resulting in oxygen deficiency in that area, causing first pain (angina), then progressing to ischemia (heart attack). The simple solution was to unblock the stenosis (blockage) with either an angioplasty or stent, or, if that was not possible, to bypass the area with coronary bypass grafting (CABG). Simple problem, simple solution.

However, problems became apparent through a number of avenues. First, a story related by a cardiologist at a Northern California

heart symposium, at which I was a speaker, points to the first problem. He told us that during his residency he was part of a trial conducted in rural Alabama with black men. In this trial, angiograms (injections of dye into the coronary arteries to detect blockages) were done on all the men presenting with chest pains. For the ones who had a single artery blocked, no treatment was prescribed, and the researchers predicted in their notes which part of the heart would have a subsequent heart attack if one occurred. Of course, they all predicted that it would be in the part of the heart supplied by the blocked coronary artery. Then they waited.

Eventually, many of the men did return and did have MIs, but to the researchers’ surprise, less than 10% had a heart attack in the area of the heart supplied by the original blocked artery.

Second, a large study conducted in 2003 by the Mayo Clinic on the efficacy of bypasses, stents, and angioplasty concluded that:

- a. bypass surgery relieves symptoms (chest pain);
- b. bypass surgery does not prevent further heart attacks;
- c. only high-risk patients, those whose lives are in acute danger, benefit from bypass surgery with regard to a better chance of survival.

In other words, the gold standard for treating arterial blockages has, at best, only minimal benefits. If you watch the video on the heartattacknew.com website and go to the FAQ called “The Riddle’s



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➤ Solution,” it becomes clear why this is so. Large, stable blockages – those over 90% – are in almost 100% of the cases completely compensated by collateral blood vessels. In fact, the view that the heart gets its blood only from the four major vessels is itself false. Starting soon after birth, the normal heart develops an extensive network of small blood vessels called collateral vessels, which eventually compensate for the interruption of flow in any one (or more) of the major vessels.

As Sroka correctly points out in the video, coronary angiogram – by failing to show the collateral circulation, and by creating spasms in the coronary arteries through the injection of heavy dye under high pressure – is notoriously inaccurate at assessing the amount of stenosis in the vessels as well as the true blood flow in the heart. To this day, most of the bypasses, stents, and angioplasties are done on minimally symptomatic patients who show a greater than 90% blockage in one or more coronary arteries. These arteries are almost always fully collateralized; the surgery does not restore blood flow because the body had already done its own bypass. Ask yourself: If it were true that an artery more than 90% blocked had no collateral circulation, how would that person still be alive? And does it really make sense that the eventual MI is caused when the stenosis goes from 93% to 98%? This is an insignificant difference and is completely nonsensical. Yet this is what most of the procedures are meant to accomplish, to unblock the stenosis, which, as the video shows, actually has no blood-flow repercussions. It is no wonder that in study after study these procedures fail to provide any significant benefit to patients.

For these reasons, the stable-plaque model is being abandoned by conventional cardiology, in favor of a different model for the etiology of

MIs, which, as it turns out, is almost equally invalid.

Meet the Unstable Plaque

So, we all agree that the entire focus of cardiology – the stable, progressing, calcified plaque, the thing that we bypassed and stented for years, the thing that we do CT scans of your arteries for, the thing that we told you is from cholesterol buildup in your arteries, the thing that “alternative cardiology,” such as the Ornish program, focused on – actually is not so important in the etiology of heart attacks. Don’t worry, though. We know that the focus of the problem *must* be the arteries, or so conventional thinking goes. Therefore, enter the unstable, or friable, plaque. This insidious fellow doesn’t actually create a large blockage; rather, it’s a soft, “foamy” plaque that under certain situations (we don’t know which situations) rapidly evolves and abruptly closes off the involved artery, creating a downstream oxygen deficit, followed by angina, then ischemia. These soft plaques are thought to be a combination of inflammatory “buildup” and LDL, the exact two things targeted by statin drugs. Therefore, the thinking goes, because this type of plaque can build up in anyone’s arteries, at any time, everyone should be on statin drugs to prevent heart attacks. (Some people even advocate putting therapeutic doses of statins in municipal water supplies.) Angiogram studies have been presented to show the evolution of these unstable plaques as proof that they are the true cause of the majority of MIs.

As I will show in the next section, acute thrombosis does happen in patients having MIs, but it is a *consequence*, not the cause, of the MI. But how often does it actually happen? Let’s look at pathology studies, which are the only accurate way to determine what actually happened, as opposed to angiograms, which are misleading and create many artifacts. The first major autopsy/pathology study of people who died

of MIs was done in Heidelberg in the 1970s.¹ The conclusion was that thrombosis sufficient to cause the MI was found in only 20% of cases. Baroldi, in the largest such study ever done, found sufficient thrombosis in 41% of cases.² He also found that the larger the area of the MI, the more often a stenosis was present, and the longer the time between MI and death, the higher the percentage of stenosis. These two facts allow some researchers to “cherry-pick” the numbers and make the stenosis rate high, as they choose to study only those with large MIs and who live the longest after the MI event. Later in this article, I will explain how the stenosis comes about as a consequence of the MI.

Another observation that casts doubt on the relevance of the coronary artery theory of MI has to do with the proposed mechanism of how thrombosed arteries cause ischemia, which is by cutting off the blood supply and thereby the oxygen to the tissues. The reality is that when careful measurements are done assessing the pO₂ (oxygen level) of the myocardial cells during an MI, to the huge surprise of many, there is no oxygen deficit ever shown in an evolving MI.³ The pO₂ levels do not change at all throughout the entire event. I will come back to this later when I describe what does change in every evolving MI ever studied. Again, the question must be asked, if this theory is predicated on the lowering of the pO₂ in the myocardial cells, and in fact the pO₂ doesn’t change, then exactly what did happen?

The conclusion is that thrombosis associated with MI is a real phenomenon, but in no pathological study has it been found in more than 50% of deaths, which raises the question, why did the other 50% even have an MI? Second, it’s clear from all pathology studies that thrombosis of significant degrees evolve *after* the MI occurs, again leading to the question of what caused the MI in the first place. The fact that thrombosis does occur also

explains why emergency procedures (remember, the only patients who benefit from bypass and stents are the most critical, acute patients) can be helpful immediately post-MI to restore flow in those patients who do not have adequate collateral circulation to that part of their heart. So again, all the existing theories as to the relevance of the coronary arteries in the evolution of the MI are fraught with inconsistencies.

If this is so, what then does cause MIs?

The Etiology of Myocardial Ischemia

Any accurate theory of the cause of myocardial ischemia must account for the risk factors most associated with heart disease. These are: being male, having diabetes, smoking cigarettes, and experiencing chronic psychological/emotional stress. Interestingly, none of these risk factors directly link to pathology of the coronary arteries. Diabetes and cigarette use cause disease in the capillaries, not the large vessels, and stress has no direct effect on coronary arteries that we know of.

In addition, during the past five decades or so, the four main medicines of modern cardiology – B-blockers, nitrates, aspirin, and statin drugs – all have some benefits for heart patients (albeit all with serious drawbacks as well), which must be addressed in any comprehensive theory of myocardial ischemia, which I will attempt to do here.

The real revolution to come in the prevention and treatment of heart disease has been inaugurated through our understanding of the role of the autonomic nervous system in the genesis of ischemia and its measurement through the tool of heart-rate variability. First, some brief background. We have two distinct nervous systems. The first, the central nervous system, controls conscious functions such as muscle and nerve function. The second nervous system is called the autonomic (or unconscious) nervous system (ANS), which controls the function of our internal organs. The autonomic

nervous system is divided into two branches, which in health are always in a balanced but ready state. The sympathetic, or fight-or-flight, system is centered in our adrenal medulla and uses the chemical adrenaline to tell our bodies that danger is afoot, it's time to run. It does so by activating a series of biochemical responses, the center of which are the glycolytic pathways that accelerate the breakdown of glucose to be used as quick energy so that we can make our escape.

In contrast, the parasympathetic branch, centered in the adrenal cortex, is the rest-and-digest arm of the autonomic nervous system. The particular nerve of the parasympathetic chain that innervates the heart is called the vagus nerve. It slows and relaxes the heart whereas the sympathetic branch accelerates and constricts the heart. It is the imbalance of these two branches that is responsible for the vast majority of heart disease.

Using heart-rate variability monitoring, which gives a real-time, accurate depiction of the status of these two branches of the ANS, it has been shown in four studies that patients with ischemic heart disease have, on average, a reduction of parasympathetic activity of more than a third. Typically, the worse the ischemia, the lower the parasympathetic activity (5/134). Furthermore, about 80% of ischemic events have been shown to be preceded by significant, often drastic, reductions in parasympathetic activity related to physical activity, emotional upset, or other causes.⁶ This finding contrasts with others that show that people who have normal parasympathetic activity, then have an abrupt increase in sympathetic activity (physical activity, or often an emotional shock), never suffer from ischemia. In other words, without a preceding decrease in parasympathetic activity, activation of the sympathetic nervous system does not lead to MI.⁷ Presumably, we are meant to have times of excess sympathetic activity; that is normal

life. What's dangerous to our health is the ongoing, persistent decrease in our parasympathetic, or life-restoring, activity. This decrease in parasympathetic activity is mediated by the three chemical transmitters of the parasympathetic nervous system: acetylcholine, NO, and cGMP. This is where it becomes fascinating, for it has been shown that women have stronger vagal activity than men, probably accounting for the sex difference in the incidence of MI.⁸ Hypertension causes a decrease in vagal activity, smoking causes a decrease in vagal activity, diabetes causes a decrease in vagal activity, and physical and emotional stress also causes a decrease in parasympathetic activity.⁹⁻¹² So all the significant risk factors have been shown to downregulate the activity of the regenerative nervous system in the heart.

On the other hand, the main drugs used in cardiology – nitrates – stimulate NO (nitrous oxide) production, which upregulates the parasympathetic nervous system. Aspirin and statin drugs also stimulate the production of ACH and NO, two of the principal mediators of the parasympathetic nervous system (until they cause a rebound decrease in these substances, which then makes the parasympathetic activity even worse). Finally, B blockers are called B-blockers because they block the activity of the sympathetic nervous system. To summarize, the risk factors and interventions that have actually been borne out through time all help balance the ANS. Whatever their effect on plaque and stenosis development is of minor relevance.

So, what is the sequence of events that leads to an MI?

First, and in the vast majority of cases the pathology will not proceed unless this condition is met, there is a decreased tonic activity of the parasympathetic nervous system. Then comes an increase in the sympathetic



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➤ nervous system activity, usually a physical or emotional stressor, which raises adrenaline production, which directs the myocardial cells to break down glucose using aerobic glycolysis (remember, no change in blood flow as measured by the pO₂ in the cells has occurred). This development redirects the metabolism of the heart away from its preferred and most efficient fuel source, which is ketones and fatty acids. This explains why heart patients often feel tired before their events. This also explains why a high-fat, low-glucose diet is crucial for heart health. As a result of the sympathetic increase and resulting glycolysis, a dramatic increase in lactic acid production occurs in the myocardial cells. This happens in virtually 100% of MIs, with no coronary artery mechanism required.^{13,14} As a result of the increase in lactic acid in the myocardial cells, a localized acidosis occurs. This acidosis causes the calcium to be unable to enter the cells, making the cells less able to contract.¹⁵ This inability to contract causes localized edema, dysfunction of the walls of the heart (called hypokinesis, the hallmark of ischemic disease as seen on stress echoes and nuclear thallium stress tests), and eventually necrosis of the tissue, which we call an MI. The localized tissue edema also alters

the hemodynamics of the arteries embedded in that section of the heart, causing the sheer pressure that ruptures the unstable plaques, which further blocks the artery and worsens the hemodynamics in that area of the heart. Only this explanation tells us why plaques rupture, what their role in the MI process is, and when and how they should be addressed; that is, only in the most critical, acute situations. Only this explanation accounts for all the observable phenomena associated with heart disease. The true origin of heart disease could not be more clear.

The final question is, why is this understanding of heart disease relevant, besides being of academic interest? The first and obvious answer is that if you don't know the cause, you can't find the solution. The solution is to protect our parasympathetic activity, use medicines that support it, and nourish the heart with what it needs. Nourishing our parasympathetic nervous system is basically the same as dismantling a way of life for which humans are ill suited. This way of life, in my view, is otherwise known as industrial civilization. The known things that nourish the parasympathetic nervous system are contact with nature, loving relations, trust, economic security (a hallmark of indigenous peoples the world over), and sex – in a sense, a whole new world.

The medicine that supports all aspects of the parasympathetic

nervous system is a medicine from the strophanthus plant called ouabain, or g-strophanthin. G-strophanthin is an endogenous hormone made in the adrenal cortex from cholesterol, whose production is inhibited by statin drugs, that does two things that are crucial for heart health and are done by no other medicine. First, it stimulates the production and liberation of ACH, the main neurotransmitter of the parasympathetic nervous system. Second, and crucially, it converts lactic acid – the main metabolic poison in this process – into pyruvate, one of the main and preferred fuels of the myocardial cells. In other words, it converts a “poison” into a nutrient.

Perhaps this “magic” is why Chinese medicine practitioners say that the kidneys (i.e., adrenals, where ouabain is made) nourish the heart. In my years of using ouabain in my practice, I have not had a single patient who had an MI while taking it. It is truly the gift to the heart.

Finally, this understanding of heart disease leads us to the correct diet, one that is loaded with healthful fats and fat-soluble nutrients, and is low in the processed carbohydrates and sugars that are the hallmark of industrial, civilized life.

Notes

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Dr. Tom Cowan discovered the work of the two men who would have the most influence on his career while teaching gardening as a Peace Corps volunteer in Swaziland, South Africa. He read *Nutrition and Physical Degeneration*, by Weston Price, and a fellow volunteer explained the arcane principles of Rudolf Steiner's biodynamic agriculture. These events inspired him to pursue a medical degree. Tom graduated from Michigan State University College of Human Medicine in 1984. After his residency in family practice at Johnson City Hospital in Johnson City, New York, he set up an anthroposophical medical practice in Peterborough, New Hampshire. Dr. Cowan relocated to San Francisco in 2003.

Dr. Cowan has served as vice president of the Physicians Association for Anthroposophical Medicine and is a founding board member of the Weston A. Price Foundation. During his career he has studied and written about many subjects in medicine. These include nutrition, homeopathy, anthroposophical medicine, and herbal medicine. He is the principal author of the book *The Fourfold Path to Healing*, which was published in 2004 by New Trends Publishing, and is the coauthor of *The Nourishing Traditions Book of Baby and*

Child Care, published in 2013. He writes the “Ask the Doctor” column in *Wise Traditions in Food, Farming and the Healing Arts*, the foundation's quarterly magazine, and has lectured throughout the US and Canada. He has three grown children and currently practices medicine in San Francisco, where he resides with his wife, Lynda Smith Cowan. Dr. Cowan sees patients at his office in San Francisco, does long-distance consults by telephone, and is accepting new patients. He also gives lectures and presentations.