WHAT'S THE REAL CAUSE OF HEART ATTACKS?

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http://www.fourwinds10.net/siterun_data/health/intentional_death/news.php?q=1418833899

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in a previous article in this journal ("What Causes Heart Attacks," Fall 2007), I presented the case that the spectrum of heart disease, which 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 happening in the coronary arteries (the arteries that supply the heart).

As we all know, the conventional view holds that the central event of heart disease occurs in the arteries, with the buildup of blockage called plaque.

In this follow-up article I will go into more detail about the conventional theory and why it is largely misleading; then I will describe the precise and well documented events that do lead to MIs (myocardial infarctions or heart attacks).

This understanding is crucial since during the last fifty years, the pursuit of the coronary artery theory has cost this nation billions of dollars in unnecessary surgical costs, billions in medications that cause as much harm as allow for any positive benefits, and, most seriously, has led many to adopt a low-fat diet, which only worsens the problem.

Newer twists on this theory only serve to further obscure the real cause. In contrast, by understanding the real patho-physiological events behind the evolution of MIs, we will be led to a proper nourishing traditional style of eating, the use of the safe and inexpensive heart tonic called g-strophanthin.

Most importantly, we will be forced to look at how heart disease is a true manifestation of the stresses of modern civilized life on the core of the human being.

To overcome the epidemic of heart disease, we literally need a new medical paradigm, a new economic system, 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 heartattacknew.com. For all interested in this important subject it is advised to read the entire website and watch the video on the website. The video above shows how the collateral circulation nourishes the heart even with a severe blockage of a coronary artery.

For health professionals and researchers, your understanding of this subject is incomplete without reading and studying the two articles found in the print version of the website.

The first is by G. Baroldi, "The Etiopathologies of Coronary Heart Disease: A Heretical Theory Based on Morphology," and the second by K. Sroka, "On the Genesis of Myocardial Ischemia." Both articles are reprinted in full on the website.

Rebuttal of Conventional Theory

Until recently I believed, along with most physicians, that most heart attacks were caused by the progressive blockage caused by plaque buildup in the four major coronary arteries leading to the heart.

These plaques were thought to be composed of cholesterol that built up 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 (the blockages) with either an angioplasty or stent, or, if that was not possible, then bypass this area with coronary bypass grafting (CABG). Simple problem, simple solution.

The problems with this approach became apparent to me through a number of avenues. The first emerged in a story related by the head of cardiology during a northern California heart symposium at which I was a speaker. He told us that during his residency he was part of a trial conducted in rural Alabama on black men.

In this trial, they did angiograms (injecting dye into the coronary arteries to detect blockages) on all the men presenting with chest pains. For the ones who had a single artery blocked, they did no interventions, only noting which part of the heart would have a subsequent heart attack if one occurred.

Of course, they all predicted it would be in the part of the heart supplied by that particular coronary artery. Then they waited. Eventually, many did return and did have heart attacks, but to the researchers' surprise less than ten percent had a heart attack in the area of the heart supplied by the original blocked artery.

This means, of course, that had they performed the usual angioplasty, stent, or bypass on that artery, the patient would have received no benefit. The second occurrence that helped change my mind was the publication in 2003 of a large study conducted by the Mayo Clinic on the efficacy of bypass surgeries, stents, and angioplasty.[1]

The study concluded that bypass surgery does relieve symptoms (chest pain); that bypass surgery does not prevent further heart attacks; and that only high risk patients benefit from bypass surgery with regard to a better chance of survival. In other words, the gold standard for treating arterial blockages provides at best only minimal benefits.

If you watch the video on www.heartattacknew.com and go to the FAQ called "The Riddle's Solution," it becomes clear why this is so. Large stable blockages, that is, sites that are over 90 percent blocked, in almost all cases compensate for the blockage by developing collateral or additional new blood vessels.

In fact, the view that the four coronary arteries supply all the blood to the heart is completely wrong. Starting soon after birth, the normal heart develops an extensive network of small blood vessels called collateral vessels that eventually compensate for the interruption of flow in any one (or more) of the major vessels.

As Sroka correctly points out in the above video, coronary angiograms fail to show the collateral circulation; furthermore the procedure creates spasms in the coronary arteries through the injection of heavy dye under high pressure. Thus, coronary angiograms are 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 performed on minimally symptomatic patients who show a greater than 90 percent blockage in one or more coronary artery. These arteries are almost always fully collateralized; it is not the surgery that restores blood flow, because the body has already done its own bypass.

If tests found a major coronary artery 90 percent blocked, with only 10 percent flow "squeezing through the bottleneck," how could you possibly still be alive if you did not have collateral blood vessels? And are we really to believe that the decisive thing that will cause the eventual heart attack is when the stenosis goes from 93 percent to 98 percent?

This is an insignificant difference, and the premise that this small increase will cause a heart attack is completely nonsensical. Yet this is what most of the procedures are meant to accomplish, to unblock the stenosis, which as the video on heartattacknew.com shows, does not actually improve blood flow.

It is no wonder that in study after study, these procedures fail to provide any significant benefit to the patients. For these reasons, conventional cardiology is abandoning the stable plaque model in favor of a different model for the etiology of heart attacks one that, as it turns out, is equally invalid.

Meet the Unstable Plaque

We can now all agree that the entire focus of cardiology—upon the stable, progressing calcified plaque: the thing we bypassed and stented for years, the thing we do CT scans of arteries for, the thing they told us is created from cholesterol buildup in arteries, the thing "alternative cardiology" like the Ornish program focused on eliminating—all this is not so important after all.

Don't worry, though, say the "experts," we know it must be the arteries, so let's introduce another concept—drum roll—that of unstable or friable plaque. This insidious scoundrel can attack at any time in any person, even when there is no large blockage. That's because these soft, "foamy" plaques can, under certain situations (we don't know which situations), rapidly evolve and abruptly close off the involved artery, creating an oxygen deficit downstream, with subsequent angina and then ischemia.

These soft plaques are thought to be the result of a combination of inflammatory "buildup" and LDL-cholesterol, the exact two components that are targeted by statin drugs. Therefore, since unstable plaque can come loose at any time, everyone should be on statin drugs to prevent this unfortunate occurrence. Some spokesmen have even suggested putting therapeutic doses of statins in the municipal water supplies.

Defendants of this theory point to angiogram studies that show the changes in these unstable plaques, claiming them as proof that unstable plaque is the true cause of the majority of MIs. As I will show, this acute thrombosis *does* happen in patients having heart attacks, but it is a *consequence*, not the cause of the MI. What can pathology reports—as opposed to angiography studies—tell us about the role of unstable plaque in heart attacks?

After all, pathology reports are the only accurate way of determining what actually happened during a heart attack, as opposed to angiograms, which are misleading and difficult to read. The first major autopsy study of patients dying of heart attack was carried out in Heidelberg in the 1970s.[2] The study found that sufficient thrombosis to cause the heart attack was found in only twenty percent of cases.

The largest such study found sufficient thrombosis in only 41 percent of cases.[3] The author, Baroldi, also found that the larger the area of the heart attack, the more often the pathology report found stenosis; in addition, the longer the time between heart attack and the death of the patient, the higher the percentage of stenosis. Some researchers have used these two facts to "cherry-pick" the numbers and make the stenosis rate seem high by studying only those with large MIs and those who live the longest after the heart attack event.

Another observation that puts into doubt the relevance of the coronary artery theory of heart attack is the fact that the proposed etiological mechanism of how thrombosed arteries cause ischemia is through cutting off the blood supply and thereby the oxygen supply to the tissues. To the enormous surprise of

many investigators, the reality is that when careful measurements are done assessing the oxygen level of the myocardial cells, there is no oxygen deficit ever shown in an evolving heart attack I.[4] The oxygen levels (measured as pO2) do not change at all throughout the entire event. I will come back to this fact 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 oxygen levels in the myocardial cells when in fact the oxygen levels don't change, then what exactly does happen? The conclusion is that while thrombosis associated with MI is a real phenomenon, it does not occur in more than 50 percent of cases—which leads to the question: why do the other 50 percent, those without an occlusion in the coronary arteries, even have an heart attack?

Second, it is clear from all pathology studies that thromboses of significant degrees evolve *after* the heart attack occurs, again leading to the question: what causes the heart attack in the first place? The fact that thrombosis does occur after a heart attack also explains why emergency procedures—remember, the only patients who benefit from bypass and stents are critical, acute patients—can be helpful immediately post-heart attack I 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 heart attack are fraught with inconsistencies. If this is so, what then does cause heart attacks?

The Etiology of Myocardial Ischemia

Any theory as to what causes myocardial ischemia must account for some consistent observations over the past fifty years. The most consistent risk factors for a person having heart disease are male sex, diabetes, cigarette use and psychological or emotional stress. Interestingly, in none of these is there a direct link to pathology of the coronary arteries—diabetes and cigarette use cause disease in the capillaries, not, as far as we know, in the large arteries. Also, we have learned over the past decades that the four main medicines of modern cardiology—beta-blockers, nitrates, aspirin, and statin drugs—all provide some benefits for heart patients (albeit all with serious drawbacks as well) and this observation must be accounted for in any comprehensive theory of myocardial ischemia.

Heart Rate Variability

The real revolution in the prevention and treatment of heart disease will come with increased understanding of the role played by the autonomic nervous system in the genesis of ischemia and its measurement through the tool of heart rate variability (HRV). We have two distinct nervous systems: the first, the central nervous system (CNS), controls conscious functions such as muscle and nerve function; the second nervous system, the autonomic (or unconscious) nervous system (ANS), controls the function of our internal organs.

The autonomic nervous system is divided into two branches, which in a healthy person are always in a balanced yet ready state. The sympathetic or "fight-or-flight" system is centered in our adrenal medulla; it uses the chemical adrenaline as its chemical transmission device and tells our bodies there is danger afoot; time to activate and run. It does so by activating a series of biochemical responses, the centerpiece of which are the glycolytic pathways, which accelerate the breakdown of glucose to be used as quick energy as we make our escape from the bear chasing us.

In contrast, the parasympathetic branch, centered in the adrenal cortex, uses the neurotransmitters acetylcholine (ACh), nitric oxide (NO), and cyclic guanosine monophosphate (cGMP) as its chemical mediators; this is the "rest-and-digest" arm of the autonomic nervous system. The particular nerve of the parasympathetic chain that supplies the heart with nervous activity is called the vagus nerve; it slows and relaxes the heart, whereas the sympathetic branches accelerate and constrict the heart. I believe it can be shown that an imbalance in these two branches is responsible for the vast majority of heart disease.

Using the techniques of heart rate variability (HRV) monitoring, which gives a real time accurate depiction of autonomic nervous system status, researchers have shown in multiple studies[5] that patients with ischemic heart disease have on average a reduction of parasympathetic activity of over one-third. Typically, the worse the ischemia, the lower the parasympathetic activity.[6] Furthermore about 80 percent of ischemic events are preceded by a significant, often drastic, reduction in parasympathetic activity.[7]

By contrast, those with normal parasympathetic activity, who experience an abrupt increase in sympathetic activity (such as physical activity or 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.[8] Presumably we are meant to experience times of excess sympathetic activity; this is normal life, with its challenges and disappointments. These shocks only become dangerous to our health in the face of an ongoing, persistent decrease in our parasympathetic, or life-restoring, activity. The decrease in parasympathetic activity is mediated by the three chemical transmitters of the parasympathetic nervous system: acetylcholine, NO, and cGMP. It is fascinating to note that women have stronger vagal activity than men, probably accounting for the sex difference in the incidence of MI.[9]

Hypertension causes a decrease in vagal activity,[10] smoking causes a decrease in vagal activity,[11] diabetes causes a decrease in vagal activity,[12] and physical and emotional stress cause a decrease in parasympathetic activity.[13] Thus, all the significant risk factors suppress the regenerative nervous system activity in our heart. On the other hand, the main drugs used in cardiology upregulate the parasympathetic nervous system.

Nitrates stimulate NO production while aspirin and statin drugs also stimulate the production of ACh along with NO—that is, until they cause a rebound decrease in these substances which then makes the parasympathetic activity even worse. Beta-blockers work by blocking the activity of the sympathetic nervous system, the increase of which is a central factor in the etiology of MI. The bottom line: the risk factors for heart disease and the interventions used all affect the balance in our ANS; whatever effects they may have on plaque and stenosis is of minor relevance.

How Heart Attacks Occur

So what is the sequence of events that leads to a heart attack? First comes a decrease in the tonic, healing activity of the parasympathetic nervous system—in the vast majority of cases the pathology for heart attack will not proceed unless this condition is met. Think of the person who is always pushing himself, who never takes time out, who has no hobbies, who constantly stimulates the adrenal cortex with caffeine or sugar, who does not nourish himself with real food and good fats, and who does not incorporate a regular pattern of eating and sleeping into his daily life.

Then comes an increase in the sympathetic nervous system activity, usually a physical or emotional stressor. This increase in sympathetic activity cannot be balanced because of chronic parasympathetic suppression. The result is an uncontrolled increase of adrenaline, which directs the myocardial cells to break down glucose using aerobic glycolysis. Remember that in a heart attack, there is no change in blood flow as measured by the p02 in the cells. This step shunts the metabolism of the heart away from its preferred and most efficient fuel sources, which are ketones and fatty acids.

This explains why heart patients often feel tired before their events. This also explains why a diet liberal in fat and low in sugar 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 one hundred percent of heart attacks, with no coronary artery mechanism required.[14, 15] As a result of the increase in lactic acid in the myocardial cells, a localized acidosis occurs. This acidosis prevents calcium from entering the cells,[16] making the cells less able to contract.

This inability to contract causes localized edema (swelling), dysfunction of the walls of the heart (hypokinesis, which is the hallmark of ischemic disease as seen on stress echoes and nuclear thallium stress tests), and eventually necrosis of the tissue—in other words, a heart attack. The localized tissue edema also alters the hemo-dynamics of the arteries embedded in that section of the heart, resulting in shear pressure, which causes the unstable plaques to rupture, further block the artery, and worsen the hemodynamics in that area of the heart.

Please note that this explanation alone explains why plaques rupture, what their role in the heart attack process is, and why they should indeed be addressed. Notice also that this explanation accounts for all the observable phenomena associated with heart disease and is substantiated by years of research. It could not be clearer as to the true origin of this epidemic of heart disease.

Nourishing the Parasympathetic Nervous System

If heart disease is fundamentally caused by a deficiency in the parasympathetic nervous system, then the solution is obviously to nurture and protect that system, which is the same as saying we should nurture and protect ourselves. Nourishing our parasympathetic nervous system is basically the same as dismantling a way of life for which humans are ill-suited. This means avoiding the excesses of industrial civilization. The known things that nourish our parasympathetic nervous system are contact with nature, loving relations, trust, economic security (a hallmark of indigenous peoples the world over) and sex—this is a whole new world of therapy for ailing hearts.

The medicine that supports all aspects of the parasympathetic nervous system is an extract from the strophanthus plant called ouabain or g-strophanthin. G-strophanthin is an endogenous (made within us) hormone manufactured in our adrenal cortex from cholesterol and therefore inhibited by statin drugs.

G-strophanthin does two things that are crucial in this process—two actions provided by no other known medicine. First, it stimulates the production and liberation of ACh, the main neurotransmitter of the parasympathetic nervous system; secondly, and crucially, it converts lactic acid—the main metabolic culprit in this process—into pyruvate, one of the main and preferred fuels of the myocardial cells. In other words, it converts the central poison in this process into a nutrient.

This may be what is meant in Chinese medicine when they say that the kidneys (that is, the adrenal glands, where ouabain is made) nourish the heart. In my many years of using ouabain, I have not had a single patient have an MI while taking it. It is truly a gift to the heart. Of course, I put all my patients on a WAPF-style heart-healthy diet, loaded with healthy fats and fat-soluble nutrients, and low in the processed carbs and sugars that are the hallmark of industrial, civilized life. There are homeopathic versions of strophanthus available, which could be used. Another option that is effective but not ideal is an extract of the plant. The drawback is that the amount of ouabain is unknown.

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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. He is the principal author of The Fourfold Path to Healing *and is co-author of* The Nourishing Traditions Book of Baby and Child Care. *Dr. Cowan lectures throughout the United States and Canada. Dr. Cowan is completing a book on the human heart that will be published by Chelsea Green Publishing in 2015.*

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