Case Study & Review

An Epidemiological Approach to Subluxation-Based Chiropractic Care and CVD Risk Factors: A Case Study and Review of the Literature

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Abstract

Objective: We present the findings in which a 54 year old male experienced lipid panel normalization as following subluxation-based chiropractic care.

Clinical Features: 54 year old male first presented into the office with a chief complaint of dyslipidemia. He had a past history of myocardial infarction and angioplasty. One month prior to care the patient had a lipid panel drawn indicating that his total cholesterol levels were 124, LDL levels were 63, HDL levels were 38, and triglyceride levels were 116. Other complaints included anxiety, constipation, fatigue, irritability, mood swings, neck pain, and stiff neck. He also reported occupational and personal stress as well as depression.

Intervention and Outcome: Paraspinal surface electromyography, range of motion, and thermography readings were taken on the initial visit, on the twelfth visit one month later, and fifteen days after his second blood draw (4 ½ months into care). In conjunction with the above findings, vertebral subluxations were confirmed at the levels of C1, C5, pelvis, and sacrum. Care plan included thirty one patient visits over a five month period before blood draw confirmed that his cholesterol levels decreased. No reported lifestyle changes occurred beside chiropractic care. In response to the positive blood work results, the patients’ cardiologist reduced his medications.

Conclusion: We offer a brief historical account of the role cholesterol has played in CVD and provide the most recent global data. Our review reveals that the “cholesterol is harmful” hypothesis is not ubiquitously supported by the literature and there appears to be a growing paradigm shift that subscribes to the theory that stress-related inflammatory and hormonal responses are key components to atherosclerotic plaque build-up and subsequent CVD. A review of literature indicates that subluxation-based chiropractic care may be effective in managing atherosclerosis and other risk factors associated with CVD due to its potential effect on inflammatory markers; namely, C-reactive protein (CRP), immune system response(s), psychological and physiological stress. The results of this case study warrant further research to explore these relationships.

Key Words: Chiropractic, subluxation, cholesterol, hypercholesteremia, dyslipidemia, statins, stress, inflammation, cardiovascular disease, atherosclerosis

Introduction

The general consensus is that cholesterol is the main instigator in arterial occlusion much like septic sludge clogging up a drain pipe. A thorough review of the literature paints a clear picture as to how this “cholesterol is harmful” paradigm evolved to what it is today, in which billions are spent on cholesterol-
lowing drugs and related cardiovascular surgeries across the planet. (For an interesting historical perspective of the "cholesterol is harmful" hypothesis see the appendix).

To identify the common factors that contribute to CVD, the United States Federal Government sponsored a research initiative entitled The Framingham Heart Study, under the direction of the National Heart Institute (now known as the National Heart, Lung, and Blood Institute).\(^1\) Beginning in 1948, 5,209 men and women were recruited in this mega-study that would span decades; leading to a second generation study of 5,124 of the original participants’ adult children and spouses in 1971, and a more recent third generation study enrolling the grandchildren of the original cohort in 2002.\(^1\) Over the decades, this trial has been the gold standard and is the most heavily cited work to date on CVD.

Numerous data have been compiled regarding the lifestyle and dietary habits of people who, before participating in the study, did not have CVD. As we will see below, the Framingham data revealed that most heart attack victims had cholesterol levels no different than those who did not suffer from myocardial infarction (MI).\(^2\) The correlation between cholesterol and MI were seen in extreme cases such as people with very high cholesterol levels experiencing a lower incidence of MI and those with very low readings experiencing a higher incidence of MI.\(^2\) Nevertheless, the "cholesterol is harmful" hypothesis has prevailed partly due to an incorrect understanding of arterial anatomy and physiology.

An underlying concept in the oversimplified notion that cholesterol clogs arteries like sewage stops a drain pipe is that arteries function and have the same characteristics as pipes. In fact, quite the opposite is true. “The vascular tree,” according to Ozner, “is an active, living organ that expands and contracts in response to different stimuli, not a network of rigid metal conduits. Its walls are permeable – and cholesterol does not just build up inside an open space like so much drainpipe sludge.”\(^2\) “Initially,” according to Kronenberg et al., “It was thought that the [arterial] lumen was progressively narrowed by the accumulation of macrophages, the proliferation of smooth muscle cells, and the deposition of cholesterol.”\(^2\) Yet, as science continued its unending search to debunk the number one killer in the world, it became evident that, the truly dangerous lesion (the culprit lesion) may not cause marked luminal narrowing. As atherosclerosis progresses, there is compensatory expansion of the lumen that maintains lumen size rather constant. As the lesion develops within the intima, the complication of rupture of the overlying intima or endothelial erosion leads to exposure of the lesion content to platelets and initiates thrombosis. It is the acute thrombosis, not arterial lumen stenosis that is responsible for infarction in most cases. Rupture or erosion occurs where the fibrous cap is thin and where an acute inflammatory process is occurring.\(^3\)

We will look at inflammation’s role in acute thrombosis and subsequent myocardial infarction in more detail later. Yet, before we do, we need to take closer look at the molecule cholesterol.

**Cholesterol**

Cholesterol is a steroid alcohol, a waxy lipid-soluble substance found only in animal tissues. Slightly less than half of the cholesterol in the body derives from biosynthesis de novo. Biosynthesis in the liver accounts for approximately 10%, and in the intestines approximately 15%, of the amount produced each day. Cholesterol synthesis occurs in the cytoplasm and microsomes (ER) from the two-carbon acetate group of acetyl-CoA. The acetyl-CoA utilized for cholesterol biosynthesis is derived from an oxidation reaction (e.g., fatty acids or pyruvate) in the mitochondria and is transported to the cytoplasm by the same process as that described for fatty acid synthesis.

Almost all the fats in the diet are absorbed from the intestines into the intestinal lymph. During digestion most triglycerides are split into monoglycerides and fatty acids, being further resynthesized into new molecules of triglycerides that enter the lymph as minute droplets referred to as chylomicrons. In addition to the triglycerides, chylomicrons are comprised of phospholipids (9%), cholesterol (3%) absorbed from the GI tract, and apoprotein B (1%).\(^4\) In its post-absorptive state, after all chylomicrons have been removed from the blood, more than 95% of all plasma lipids exist in the form of lipoproteins; particles much smaller than chylomicrons, yet containing a similar constituency of particles. The significance of lipoproteins is their function as transportation molecules for non-polar cholesterol through blood plasma. With lipoproteins, cholesterol would not be able to travel through the body to fulfill its vital functions, thereby making life impossible.

Cholesterol is so absolutely vital for human life that if we do not consume enough of it in our diet (about 6 to 8 eggs yolks worth per day), the liver will make up the difference. The liver, therefore, is a type of cholesterol “thermostat” constantly regulating plasma levels via down-regulation.\(^5\) The significance of cholesterol is seen by its necessary contribution to the following:

- Brain synapses – made almost entirely of cholesterol.
- Bile – cholesterol being a key component.
- Cell membranes – in which cholesterol is a primary component.
- Sex hormones – cholesterol being the precursor to every one; namely, progesterone, estrogen and testosterone.
- Vitamin D – synthesized from cholesterol by sunlight acting on the skin.

According to Guyton, factors that affect plasma cholesterol concentrations are as follows:

- An increase in the amount of cholesterol ingested each day increases the plasma concentration slightly.
A highly saturated fat diet increases blood cholesterol concentration 15 to 25%.

Ingestion of highly unsaturated fatty acids generally depresses blood cholesterol levels.

Lack of insulin or thyroid hormone increases blood cholesterol concentration.4

It is documented that if dietary intake of fatty acids exceeds the immediate fuel needs of the liver, the excess is converted to triglycerols, which form very low density lipoproteins (VLDL) when combined with a variety of apolipoproteins, esterified cholesterol, and phospholipids.6 Subsequently secreted from the liver, the VLDL particles are depleted of triglycerol and eventually form low density lipoproteins (LDL). The LDL’s, in turn, travel to extra-hepatic tissues and the cholesterol molecules inside are used for further processing as described above. Due to their oxidized, free-radical nature, excess LDL presented in the blood are readily attacked by macrophages (scavenger cells) near arterial endothelial cells.

Because macrophages are not subject to down-regulation, the continued presence of oxidatively modified LDL causes them to get absorbed into the cell membrane, resulting in injury (lesion formation). This is known as the “response-to-retention” hypothesis.3 Becoming engorged with lipid, macrophages form foam cells, which accumulate in the subendothelial space and develop the first signs of atherosclerotic plaque (i.e. fatty streak). This is believed to induce an inflammatory response, triggering atherosclerosis.6

Other factors that injure endothelial cells are thought to be involved in fatty streak formation; namely, arterial hypertension, cigarette smoking, chronic elevated blood glucose levels, high circulations of the vasoconstricting angiotensin II.6 The resulting insult and subsequent adhesion molecule release can cause monocytic cells to accumulate between endothelial cells, resembling the classical inflammatory response to injury. “In fact,” as we have seen above and according to Lieberman et al., in Mark’s Essentials of Medical Biochemistry, “Some researchers have suggested that atherosclerosis is in fact an inflammatory disorder and could therefore be prevented or attenuated through the use of anti-inflammatory agents.” More on this later.

Microscopic separations are formed by foam cell accumulation and subsequent endothelial deformation. These exposed areas lead to platelet adhesion and aggregation; thus releasing cytokines that perpetuate this process, leading to local thrombus (clot) formation. Platelets are attracted to the clot which will either completely block the artery locally at that very same spot or will break off and block a smaller vessel further down the vasculature pathway (e.g. coronary artery leading to myocardial infarction or an artery in the brain leading to a stroke).

Not so simple in its formation, high density lipoproteins (HDL) are created by a number of mechanisms. Not much is known of HDL’s, but it is believed that they possess a very unique quality. In a process known as “reverse cholesterol transport,” they are purported to remove cholesterol crystals from cholesterol-saturated cells and return them to the liver for further processing into bile acids and other particles. Thus, the title commonly given to HDL’s, “Good Cholesterol.” See Graph #1 for the recommended cholesterol levels according to the CDC.

Since 1985, The National Cholesterol Education Program (NCEP), managed by the National Heart, Lung and Blood Institute, has worked to reduce increased cardiovascular disease rates due to hypercholesterolemia in the United States of America. The assigned goal of the NCEP committee is to meet on a recurring basis and review ongoing scientific research about atherosclerotic cardiovascular disease. Furthermore, their mission is to make simplified, consensus, committee recommendations to be promoted by the NIH, the American Heart Association, and other groups to both physicians and the public about how to reduce the incidence of disability and death resulting from atherosclerotic cardiovascular disease.

According to the NCEP Expert Panel there are three categories that modify LDL guidelines (see Graph #2).6

The NCEP concludes, “Any person with elevated LDL cholesterol or other form of hyperlipidemia should undergo clinical or laboratory assessment to rule out secondary dyslipidemia before initiation of lipid-lowering therapy…Once secondary causes have been excluded or, if appropriate, treated, the goals of LDL-lowering therapy in primary prevention are established according to a person’s risk category.”6 Causes of secondary dyslipidemia include:

- Chronic renal failure
- Drugs that harmfully affect LDL and HDL levels (e.g. anabolic steroids, corticosteroids, and progestins)
- Diabetes
- Obstructive liver disease 8

What’s intriguing about these numbers is that they have been anything but static since dyslipidemia has been on the forefront of thought in the health care community, nor do they promise to be so in the future. In 1979, for example, desired total cholesterol levels were less than 300.5 Now they’re less than 200. In 2002, desired HDL levels changed from 35 to 40.8 Now they’re 60+. In light of the rise in CVD’s globally, we suspect there to be a change in the near future further lowering LDL’s, triglycerides, total cholesterol, and possibly raising HDL’s again.

Note that the underlying factor in the inflammatory process as explained above is excess LDL (known as “Bad Cholesterol”) accumulation in the arterial lumen. Other than the genetic defect familial hypercholesterolemia – a genetic disorder in which LDL receptors are dramatically decreased, thus preventing LDL’s from being removed from circulation – the primary source of excessive plasma LDL can be traced to the diet and to lack of exercise. Furthermore, it is important to point out that having and measuring actual cholesterol levels in the blood is impossible.5

Granted, LDL contains cholesterol (approximately 3%), yet as far as we know there are no tests determining how many cholesterol molecules are in each of the LDL’s that are being measured in lipid panels around the world. We must remember that LDL and cholesterol are not synonyms. Thus,
the reference to “LDL Cholesterol” or “HDL Cholesterol” is very serious a scientific misnomer. They are two completely different molecules. Please keep this in mind as you read the remainder of this paper. Thus, the reference to “cholesterol” levels in the blood and related inferences are taken directly from the literature and used for continuity. We do not endorse its usage and propose a paradigm shift to refer to LDL’s as simply LDL’s and leave cholesterol out of the picture unless, to create a scientifically accurate arena, all the other components of LDL’s (i.e. phospholipids, etc.) are included in its description.

**A Global Look at Cardiovascular Disease**

Cardiovascular diseases are a group of disorders affecting the heart and blood vessels. Heart attacks (myocardial infarction) and strokes are usually acute events mainly caused by a blockage that prevents blood from flowing to the heart or brain, respectively. It is believed that the most common reason for this is fatty deposit accumulation on the inner walls of the blood vessels that supply the heart or brain. Bleeding from a blood vessel in the brain or from blood clots can also cause strokes.

According to the WHO, behavioral risk factors are responsible for about 80% of coronary heart disease and cerebrovascular disease. The effects of unhealthy diet and physical inactivity, for example, may manifest in individuals as raised blood glucose, raised blood lipids, raised blood pressure, and the onset of obesity. These are called intermediate risk factors or metabolic risk factors.

Shockingly, there are often no symptoms preceding CVD. The first warning oftentimes is a heart attack or stroke, giving the victim little time to react and make the necessary changes to amend their lifestyle. According to the WHO, “Individuals can reduce their risk of CVDs by engaging in regular physical activity, avoiding tobacco use and second-hand tobacco smoke, choosing a diet rich in fruit and vegetables and avoiding foods that are high in fat, sugar and salt, and maintaining a healthy body weight.”

The underlying premise above is that cholesterol, the suspected cause of “fatty deposit accumulation,” is harmful and great lengths need to be taken to avoid, counteract, and dismantle it. Interestingly, approximately 1/6 of Americans suffer from supposed “high total cholesterol.” Astoundingly, *this number has dropped by half* since the recorded statistics from 1960-1962. Interestingly, CVD’s are still on the rise, and have been for decades. According to the CDC:

- In 2006, 631,636 people died of heart disease. Heart disease caused 26% of deaths—more than one in every four—in the United States.
- Heart disease is the leading cause of death for both men and women. Half of the deaths due to heart disease in 2006 were women.
- Coronary heart disease is the most common type of heart disease. In 2005, 445,687 people died from coronary heart disease.
- Every year about 785,000 Americans have a first heart attack. Another 470,000 who have already had one or more heart attacks have another attack.
- In 2010, heart disease will cost the United States $316.4 billion. This total includes the cost of health care services, medications, and lost productivity.

CVD’s are not discriminatory of ethnicity. They prove to be the number one cause of death across the board as seen below. Of the risk factors below, 37% of adults reported two or more. See Graph #3. Sadly, this is not isolated to the U.S. According to the WHO:

- CVDs are the number one cause of death globally: more people die annually from CVDs than from any other cause.
- An estimated 17.3 million people died from CVDs in 2008, representing 30% of all global deaths. Of these deaths, an estimated 7.3 million were due to coronary heart disease and 6.2 million were due to stroke.
- Low- and middle-income countries are disproportionately affected: over 80% of CVD deaths take place in low- and middle-income countries and occur almost equally in men and women.
- By 2030, almost 23.6 million people will die from CVDs, mainly from heart disease and stroke. These are projected to remain the single leading causes of death.

**Cholesterol Not the Cause of CVD?**

We must not ignore that in spite of the fact that reported cholesterol levels have dropped by over 50% in the U.S. since first being reported in the early 60’s, CVD has been on the rise the past 5 decades. It appears quite clear from the literature that “cholesterol” levels have nothing at all to do with atherosclerotic plaque formation, and subsequent CVD. Research even suggests that LDL, HDL, and triglyceride levels are completely unrelated to mortality. Studies have proven, for example, that *lowered* cholesterol levels in certain age groups are significant risk factors, not elevated levels. For example, JAMA published an article, “Cholesterol and Mortality. 30 years of Follow-Up From the Framingham Study,” in which Anderson et al. conclude:

> “After age 50 years [of age] there is no increased overall mortality with either high or low serum cholesterol levels. There is a direct association between falling cholesterol levels over the first 14 years and mortality over the following 18 years. Under age 50 years these data suggest that having a very low cholesterol level improves longevity.”

Schatz and Schestov have also shown a similar aged-related risk factor for people older than 50 years old and low cholesterol levels. In the well known *Seven Countries Study*, CVD risk factors were measured between two Japanese cohorts of men aged 40-59; one from a farming village, the other from a fishing village. They found the following: “Multivariate models from data of the pooled cohorts showed that age was a significant predictor of coronary heart disease, stroke, cancer, all other causes and all-cause mortality. Cigarette smoking predicted
coronary heart disease, cancer and all-cause mortality. Systolic blood pressure predicted coronary heart disease, stroke and all-cause mortality. Serum cholesterol, body mass index and heart rate predicted none of these five causes of death [our emphasis].”14

In a 15 country statistical review comparing heart-disease rates in men aged 35-74 and average cholesterol levels, Kendrick found these interesting results:

- Australian Aboriginals, who have the lowest reported cholesterol levels, have the highest death rates.
- The Swiss, who have the highest reported cholesterol levels, have one of the lowest death rates.
- The French, who have above-average cholesterol levels, have the lowest death rates.
- The Russians, who have below-average cholesterol levels, have above average death rates.

His conclusion: “There is a complete and utter dissociation between cholesterol levels and heart disease.”15

Numerous documented reports suggest that dietary carbohydrate intake, not fat and cholesterol, affect serum lipid levels. It is suggested that eating fat lowers LDL levels, whereas eating carbs raises VLDL levels and subsequent LDL levels. Recalling from above that the liver is the cholesterol “thermostat,” this should not be a surprise, unless of course, hepatic failure or pathology is adding contributing factors.

Seshadri et al., for example, from their study, “A randomized study comparing the effects of a low-carbohydrate diet and a conventional diet on lipoprotein sub-fractions and C-reactive protein levels in patients with severe obesity,” state that, “Subjects on a low-carbohydrate diet experienced a greater decrease in large very low-density lipoprotein (VLDL) levels.” They conclude that, “In this 6-month study...we found an overall favorable effect of a low-carbohydrate diet on lipoprotein subfractions, and on inflammation in high-risk subjects. Both diets had similar effects on LDL and HDL subfractions.”15

Researcher and physiologist Ansel Keys documented similar results over 25 years ago, reporting that there is minimal, if any, connection between diet and serum cholesterol levels.16-17 He concludes, “Clearly, alteration, within practical, acceptable limits for free-living people, of the amount of cholesterol in the diet can have, by itself, only limited effects on the concentration of cholesterol in the serum [emphasis ours].”17

We have also found similar findings in regards to stroke. For instance, in a study published in Lancet, Qizilbash et al. investigated the associations of blood cholesterol and diastolic blood pressure with subsequent stroke rates by reviewing 45 prospective observational cohorts involving 450,000 individuals with 5-30 years of follow-up (mean 16 years, total 7.3 million person-years of observation), during which 13,397 participants were recorded as having had a stroke. They’re conclusion: “After standardization for age, there was no association between blood cholesterol and stroke except, perhaps, in those under 45 years of age when screened.”18

Treatment (Medical Community)

In the medical community, the set of behavior changes discussed above are generally the first line of treatment in an attempt to normalize serum cholesterol levels. If diet and exercise do not correct the problem, cholesterol-altering medications are usually recommended.19 Cholesterol-lowering drugs include: bile acid sequestrants; statins; niacin; fibrac acid derivatives; cholesterol absorption inhibitors; and aspirin. Each will be discussed briefly below, outlining the mechanisms pertaining to their function and documented side effects.

One of the original cholesterol lowering drugs, resins (bile acid sequestrants), bind bile acids in the GI tract, thus reducing bile acid in the liver. Since bile is largely made from cholesterol, the liver is forced to recruit plasma cholesterol via LDL transport molecules to form bile acids; thereby greatly reducing the body’s supply of cholesterol. Side effects include: constipation; gas; upset stomach. Examples include: Questran and Questran Light; Colestid; WelChol.2 19

Typically, the first medicine prescribed to treat hypercholesterolemia are Statins. These inhibit HMG-CoA reductase, a crucial enzyme needed for cholesterol production, thus lowering LDL and triglyceride production in the liver. Statins are also known for raising HDL’s, though, to a lesser degree. There is strong inference that they have a role in decreasing inflammation leading to atheromatous plaques. The side effects can be serious: muscle and liver toxicity; kidney failure; peripheral neuropathy; reduction of Coenzyme Q10.

Primarily in the mitochondria, CoQ10 is instrumental in the electron transport chain during the production of ATP. Since 95% of the body’s energy is generated this way, one can only suspect how staggering the effects can be on energy production and the slew of physiologic processes that are affected. Due to their anti-inflammatory role, multiple research studies purport that statins reduce the risk of CVD’s. Examples include: Atorvastatin (Lipitor); Fluvastatin (Lescol); Lovastatin (Mevaco); Pravastatin (Pravachol); Simvastatin (Zocor); Rosuvastatin (Crestor).2,19,20

B-complex vitamin, nicotinic acid (niacin), has been used for decades to lower LDL’s and triglycerides and raise HDL’s. It’s also known for increasing cholesterol size, preventing their migration into arterial walls and subsequent oxidization; though, recent research suggests that niacin is not associated with lowered risk of CVD. Side effects include: flushing; itching; tingling; headache. Examples include: Nicolar and Niaspan.2 19

Fibrates decrease VLDL’s released from the liver, thus lowering serum LDL’s and triglycerides and raising HDL’s. Side effects include: diarrhea; nausea; myopathy; liver inflammation; gallstone production (if taken for several years). Examples include: Atromid; Tricor; Lopid.2 19,21

Cholesterol absorption inhibitors (ezetimibe) block cholesterol absorption in the GI tract. They are often prescribed with statins in combination therapy. Recent research have not found any association with lowering risk of heart disease. Side
Aspirin prevents blood clots by blocking platelets and reduces inflammation. Thus, decreasing the risk of MI and stroke, diseases often attributed to dyslipidemia. Ubiquitous in its usage, most cardiovascular disease patients take aspirin daily. Side effects are minimal, the most common involving the GI system: duodenal ulcers; abdominal pain; nausea; gastritis. Counter indications include allergy and bleeding ulcers.\textsuperscript{2, 19, 21}

The IMS Institute For Healthcare Informatics reported in their most recent report, “The Use of Medicines in the United States: Review of 2010,”\textsuperscript{11} that spending on lipid regulators increased by $160 million in 2010.\textsuperscript{22} More than 255 million prescriptions were issued for these drugs in 2010, making them the most commonly prescribed medication in the U.S. Some other highlights from the IMS report include:

- Lipid regulators were the third largest therapy class by spending in 2010 at $18.7Bn, growing by only 0.9\% with much of the class now available generically.
- 2010 growth of $160Mn slowed from $490Mn in 2009.
- Dispensed prescriptions exceeded 255Mn in 2010, up from 210Mn in 2006, with 54\% of prescriptions filled with a generic and the remainder filled as brands, primarily Lipitor\textsuperscript{R} and Crestor\textsuperscript{R}.
- Crestor\textsuperscript{R} (rosuvastatin) led spending increases in 2010 with $717Mn in new growth.
- Lipitor\textsuperscript{R} (atorvastatin) continues to lead spending in the class, but declined by 4.1\% in 2010. It was second to generic simvastatin in prescription volume in the class, and is expected to face generic competition in late 2011.\textsuperscript{23}

It is important to note that patients with severe hypercholesteremia are oftentimes treated via “combination therapy,” in which drug combinations are employed at lower dosages than needed if used singly.\textsuperscript{3} For example, it is documented that statins combined with ezetimibe, niacin, or a bile acid sequestrant can reduce plasma cholesterol levels by 50\%. Vytorin, a preparation of simvastatin combined with ezetimibe, is purported to lower LDL levels by 60\%.\textsuperscript{3} Though, documented as reducing CVD risk factor(s), when a cocktail of pharmaceuticals are mixed together like this, the side effects become very complicated and oftentimes miserable for the patient to endure. As is the case in the patient from this study, it is no wonder that people are taking great strides to get off these medications.

Though the data appears conclusive and the presentation very attractive, some things just do not add up. We’ve asked the question before. How can it be possible that billions of dollars are spent annually across the globe on drugs to regulate serum cholesterol levels, yet CVD’s are on the rise? As just seen above, the literature indicates that these medicines are very effective in their role in monitoring HDL’s, LDL’s, and triglycerides. So what are we missing here?

Hadler puts it this way.

“There is no question that blood cholesterol is a risk factor, but it’s not much of a risk factor. If you have no extraordinary family history, yet have extraordinarily high LDL cholesterol and low HDL cholesterol, it will cost you a year or two of life expectancy. Nearly all who are labeled ‘high cholesterol’ are far from the extreme and have minimal risk. These people are contending with a reduction in life expectancy of months. Is this meaningful or even measurable?”\textsuperscript{23}

This begs the question as to whether or not unnecessary interventions are being employed. It’s becoming more than evident that there are. For instance, if pharmaceutical management is unsuccessful, then a more invasive approach usually ensues. In the U.S. alone, more than 1.5 million angioplasties and coronary bypass surgeries are performed each year, making heart surgery one of the most commonly performed surgical procedures for both men and women.\textsuperscript{2}

According to Ozner, as we have seen above, these interventions are based on the faulted logic that clogged arteries are seen as analogous to clogged drain pipes which need a simple, easy “snake” to clean out the sludge (angioplasty). Yet, if the “snaking” does not work, it is believed that adding a new pipe (bypass surgery) should keep blood flow regulated. Ozner states, “Although heart surgery can be lifesaving, the truth is that surgery benefits only a small fraction of the millions of patients who undergo these operations…In fact, except for a minority of patients, bypass surgery and angioplasty have never been shown to prolong life or prevent heart attacks.”\textsuperscript{2} He contends that 70 – 90\% of heart surgeries are unnecessary.\textsuperscript{2}

Treatment (Chiropractic Community)

In the chiropractic community treatment has historically been deferred to MD’s and following the above lifestyle guidelines above. A number of papers have been published advising the chiropractic community on attempting to manage dyslipidemia via supplements and other “natural” mechanisms like nutrition, exercise, etc.\textsuperscript{24, 30} A thorough review of Index to Chiropractic Literature, Mantis, PubMed, and Scopus search engines, revealed that only two subluxation-based articles have ever been published on chiropractic’s efficacy in treating dyslipidemia.\textsuperscript{31, 32} We have found that Loomis’ article “Nutritional Alternatives to Prescription Drugs,” epitomizes the chiropractic paradigm in its approach to treat dyslipidemia as evident in the peer-reviewed literature. Below are some bullet points:

- Prescription drugs are used to treat disease. They WORK by blocking a human enzyme system or by filling specific receptor sites, thus directing the body’s biochemistry
- However, nutritional supplements (whether protein, lipids, carbohydrates, vitamins, or minerals) do not, by themselves, perform WORK. They must be put to work by specific enzyme action. Nevertheless, the concept of considering nutritional alternatives to prescription drugs is well worth the effort.
- Nutritional supplements find their best use for maintaining health—not treating disease.
• Very useful physical finding that chiropractors can use to determine when a patient requires food enzyme supplementation—namely, loss of normal thoracic kyphosis in cases of indigestion, metabolic syndrome, and restless legs.
• Visceral dysfunction produces contraction(s) in the muscles that share spinal innervation with the stressed organ(s). This occurs not only in the periphery but at the spine as well. Thus, we have the occurrence of spinal subluxation contaminant with visceral dysfunction.
• Since all prescribed drugs interfere with normal body functions, by either blocking receptor sites or interfering with a human enzyme system, they all cause side effects that can be recognized very early by changes in muscle contraction and subluxation patterns.
• All symptoms are caused by the inability of an organ system to fulfill its role in maintaining homeostasis, either because it is nutrient deficient or there is excessive waste accumulation.

Your patients should be informed that prescription drugs are used for the treatment of disease and they do not restore normal function, nor can they maintain health.27

Case Report

Patient History

54 year old male patient first presented after hearing that chiropractic had positive effects on lowering plasma cholesterol levels. His chief complaint was dyslipidemia. Other complaints included anxiety, constipation, fatigue, irritability, mood swings, neck pain, and stiff neck. Occupational and personal stress levels were reported 8 out of 10 (this is particularly significant, as we will see later in our study of the relationship between stress and CVD).

He also indicated suffering from depression due to the death of his father, mother, brother, and uncle which all occurred within a year of each other. Health history included: angioplasty/stent; double hernia surgery; exploratory lung surgery; hydrocele correction; three rotary cuff surgeries. His main purpose in receiving care was to reduce the medications he was taking. Being diagnosed with high cholesterol 12 years prior after suffering a myocardial infarction (MI), the patient was taking several medications (see Graph #4).

One month prior to care patient had a lipid panel drawn indicating that his total cholesterol levels were 124, LDL levels were 63, HDL levels were 38, and triglyceride levels were 116. He has been and still is, at the time of publication of this article, an avid hockey player, playing on average twice weekly. Other exercise activities included walking 3 miles/day on treadmill. Diet typical of most Americans, eating refined foods, enjoying pizza, subs, and pasta.

Examination

Analysis involved leg checks as well as palpation. To evaluate for vertebral subluxation(s) physical and motion palpation examination were utilized, in addition to thermal and surface electromyography (sEMG), and full spine x-rays. On complete cervical, thoracic, and lateral film series the following findings were seen: C5 disc degeneration; anterior positioning of the cervical spine and anterior head carriage; decreased cervical curvature; curve reversal at C4 –5; hypertrophy of joint facets.

How a patient feels is such a poor indicator of health that it is necessary to have means of monitoring the 85+% of the nervous system that cannot be felt as it does not relay conscious sensory information. Even though the use of thermography in chiropractic has become increasingly common, the proper understanding and dissemination of this information to the public is unfortunately rare. With an in-depth understanding of the data provided by the scans, the chiropractor is given a window to autonomic and motor nerve function, sympathetic/parasympathetic balance, as well as range of motion changes taking place.

The Insight Millennium Subluxation Station® was used to provide objective results to monitor progress in performance of nervous system function. The accuracy of this technology is supported in the literature. McCoy et al., in the largest study of thermal scanning reliability conducted, documented excellent intra-examiner and inter-examiner reproducibility in regards to the thermal functions of this infrared scanner.33,34

In this study, “two practicing chiropractors conducted the measures on 100 subjects and found intra class correlation coefficients (ICCs) for agreement and consistency ranging from 0.959 to 0.976. Concordance correlation coefficients (CCCs) ranged from 0.783 to 0.859 with tight confidence intervals indicating robust estimates of these quantities.”34

Below is a brief description of scans we utilize from the Insight Millennium Subluxation Station® and how we interpret the data from these scans.

The rolling thermal scan offers insight to autonomic function. Every organ, tissue, gland and cell in the body receives and reports information to and from the brain. The theory is that when any area of the body does not receive proper, uninterrupted signals from the brain optimal health is not possible. Likewise, when the brain is not receiving clear and complete information from areas of the body it is operating and coordinating function with less than necessary data to express homeostasis. Areas of increased thermal imbalance indicate that this area of the nervous system has responded to stress present in the environment.

The sEMG provides insight into the motor function and balance of the paraspinal musculature. Areas of inadequate muscle tone can indicate a long standing issue in which the body is no longer actively working to resolve. These areas are also more prone to injury as supportive structures are not engaged in a manner necessary for proper responsive support. Likewise, the areas of excessive muscle engagement are likely limiting full ranges of motion, creating further stress on surrounding tissues and increasing fibrotic activity. We have found that posture corrective exercises and stretches are extremely important to continue progress of altering fibrotic activity. The ideal pattern, balance, and total energy scores for this scan in an optimally functioning and healthy individual are all 100.
The Pulse Wave Profile (PWP) uses the technology of Heart Rate Variability to monitor autonomic activity of the nervous system as well as the balance between parasympathetic and sympathetic systems. This is done by monitoring pulse rate, galvanic skin response (conductivity), and skin temperature. The coordination of changes in these factors indicates the body’s parasympathetic and sympathetic balance as well as the amount of energy available to deal with and react to stress in the environment.

A sympathetic shift will indicate that the body is operating with a baseline of sustained stress where stress hormones like cortisol, normally reserved for survival responses (fight/flight), are present a majority of the time. This sustained sympathetic tone has been linked in current literature to many of today’s chronic illnesses; namely, and of particular interest to this study, chronic heart failure. Shifts indicating a swing to the parasympathetic can indicate adrenal fatigue and are common when dealing with someone that has been stuck in a sympathetic shift for an extended period of time that is now taking steps to create balance.

Range of motion changes offer insight to posture changes and can indicate the cessation of fibrotic degenerative changes and decreased scar tissue presence. It is well documented that the loss of motion in just one segment of the spine decreases cerebellar input directly leading to increased thalamus, hypothalamus, and amygdala stimulation and increasing the stress response.

Intervention

The first adjustment was tonal using Torque Release Technique (TRT). We have found the dural stress that patients new to chiropractic care are under leads to increased symptomatology - most frequently leading to subluxations at C1, C5, and Sacrum. In other words, we have found it beneficial to “prime” the spine before performing a more osseous adjustment. In subsequent visits, we determine which protocol would be best to perform by whether or not there is a subluxation pattern; namely, whether segment(s) are found to be in the same subluxated position for 3 or more consecutive visits.

The patient in this study seemed to bounce back and forth with right/left C1 and right/left sacrum and pelvis. The fact that each visit was dynamic kept us from using tonal methods more often, so we utilized Diversified side posture, Diversified thoracic, and supine Diversified cervical adjustment techniques to correct the segments above. Post checks, adjustment by adjustment, were performed by looking for motion restoration and occasionally leg length balance.

The recommended care plan for the first year of care was 3 times per week for the first 5 weeks, 2 times per week for the following 5 weeks, and 1 time per week for the last 42 weeks. Overall, the patient was not compliant to this care schedule. There were 20 weeks of care from 1st adjustment to the 3rd thermal scan assessment and 18 weeks of care from 1st adjustment to the blood draw that indicated a normalization of lipid levels. At the time of publishing this paper, the patient is currently under care visiting the office generally once a week.

The data from the three thermography scans indicated that the patient improved in several areas. The thermal scan showed an overall improvement, indicating that the autonomic nervous system was dealing with environmental stressors differently. The sEMG pattern was less uniform indicating that the motor nerve system was undergoing an adaptive/growth response. ROM improved in all areas except cervical flexion/extension. The PWP indicated that the body was no longer in a sympathetic shift, which is likely due to decreased stress level(s) as a result of continued chiropractic care. See Scans #1, #2, #3 for thorough details and Graph #7 for a summary of the three scans taken on the patient.

Four months into regular care, patient had another lipid panel drawn indicating improvement in every area. His total cholesterol levels dropped to 104, LDL levels dropped to 43, HDL levels raised to 44, and triglyceride levels dropped to 83. In a letter mailed to the patient, his cardiologist, pleased with the results, stated, “Your liver functions and CPK were within normal limits. Your numbers looked great. Keep up the good work.” She advised him to continue taking Tricor 145mg daily, Welcheol three tablets daily, Vytorin until supply ran out, and to change to Simvastatin 80mg (a more conservative treatment plan) daily.

It is vital to note that, according to the patient, no other lifestyle changes were made during these 4 ½ months. One month after receiving care he wrote in a Wellness Progress Analysis, “I feel so much better and relaxed [our emphasis],” indicating as stated above that the patient was no longer in a sympathetic shift and, thus, experiencing a decrease in psychological and physiological stress levels.

Discussion

Inflammatory Components of Atherosclerosis Related to Chiropractic

It is vital to mention that, according to the National Heart Lung and Blood Institute, a division of the U.S. Department of Health & Human Services, “The cause of atherosclerosis is not known.” Current prevailing thought is that inflammation is responsible via oxidative stress, as explained above in the “response-to-retention” hypothesis. The big question here is what exactly causes the inflammation? As seen above, lipid and cholesterol have become the scape-goat, yet there is an entire body of research that contends contrary.

Below is an outline of some key components of inflammation that are not publicized very often, but we feel are quite worthy of mention due to their intricate connection to subluxation-based chiropractic care; namely, C-reactive protein (CRP) and immune system response. Understanding the science behind these two mechanisms may serve us well in unlocking the role chiropractic has played in normalizing our patient’s lipid panel.

It is of interest to note here that T-cells (predominantly CD4+ lymphocytes) are always present in atherosclerotic lesions. Oxidized LDL is presented by macrophages to type 1 helper T-cells (Th1), which ultimately produce the cytokine interferon-γ; thus, activating macrophages, initiating inflammatory and cytotoxic molecules, and promoting
Atherosclerosis. Furthermore, the inflammatory cytokines induce interleukin-6 production, which enters the general circulation and stimulates C-reactive protein (CRP) from the liver. Oftentimes, the serum CRP levels are used as clinical markers for atherosclerotic inflammation and subsequent coronary heart disease.3

The notion that atherosclerosis is associated with inflammation is well supported by the literature. Since a Russian article was first published in 1966 on the topic, over 10,000 articles have been published as of January 2012 according to a PubMed search of “inflammation and atherosclerosis.”4 As stated by Libby et al., Department of Medicine, Brigham and Women's Hospital, Harvard Medical School,

Recent advances in basic science have established a fundamental role for inflammation in mediating all stages of this disease from initiation through progression and, ultimately, the thrombotic complications of atherosclerosis. These new findings provide important links between risk factors and the mechanisms of atherogenesis…Elevation in markers of inflammation predicts outcomes of patients with acute coronary syndromes, independently of myocardial damage. In addition, low-grade chronic inflammation, as indicated by levels of the inflammatory marker C-reactive protein [CRP], prospectively defines risk of atherosclerotic complications, thus adding to prognostic information provided by traditional risk factors. Moreover, certain treatments that reduce coronary risk also limit inflammation. In the case of lipid lowering with statins, this anti-inflammatory effect does not [our emphasis] appear to correlate with reduction in low-density lipoprotein levels. These new insights into inflammation in atherosclerosis not only increase our understanding of this disease, but also have practical clinical applications in risk stratification and targeting of therapy for this scourge of growing worldwide importance.5

Abrahms confirms this association with CRP. “We must remember that CRP is but one of many inflammatory markers; nevertheless, the database supporting CRP measurements is far greater than for any of the other markers, and the widely available hs-CRP assay makes it extremely attractive to pursue the conundrum of inflammation and atherosclerosis with vigor.”6

Van-Assche et al. in some of the most recent research available, describes the immunological component to atherosclerosis,

The extensive cross-talk between the immune system and vasculature leading to the infiltration of immune cells into the vascular wall is a major step in atherogenesis…by activating a number of redox-sensitive transcriptional factors such as nuclear factor kappa B (NFkappa B) or activating protein 1 (AP1), that regulate the expression of multiple pro/anti inflammatory genes involved in atherogenesis. Delivery of genes encoding antioxidant defense enzymes (e.g. superoxide dismutase, catalase, glutathione peroxidase or heme oxygenase- 1) or endothelial nitric oxide synthase (eNOS), suppress atherogenesis in animal models. Similarly, delivery of genes encoding regulators of redox sensitive transcriptional factors (e.g. NF-kappa B, AP-1, Nrf2 etc) or reactive oxygen species scavengers have been successfully used in experimental studies.44

Main Contributor to CVD: Stress

Our intention in this section is to assert that stress is the main cause of CVD. We will offer some key biochemical and physiological explanations to stress that have a direct correlation to the body’s response to subluxation-based chiropractic care. We will also attempt to offer a clear definition of stress that will help health care professionals determine true risk factors for CVD.

In Mandarin, Cantonese, and Japanese, the words “death” and “four” are pronounced almost identically. This number evokes so much anxiety that the number 4 is oftentimes not used to identify floor and room numbers in some hospitals and phone numbers. The mainland Chinese air force even goes so far as to avoid “4” and uses other numbers to designate military aircraft. In a study published by the BMJ entitled, “The Hound of the Baskervilles Effect: Natural Experiment on the Influence of Psychological Stress on Timing of Death,” the objective of Phillips et al. was to determine whether their hypothesis that cardiac mortality is increased on days considered unlucky was supported by the data or not. What they found was staggering.

• All Chinese and Japanese (n=209,908) and white (n=47,328,762) Americans whose computerized death certificates were recorded between the beginning of January 1973 and the end of December 1998.

• Cardiac mortality in Chinese and Japanese people peaked on the fourth of the month. The peak was particularly large for deaths from chronic heart disease (ratio of observed to expected deaths = 1.13, 95% confidence interval 1.06 to 1.21) and still larger for deaths from chronic heart disease in California (1.27, 1.15 to 1.39). Within this group, inpatients showed a particularly large peak on the fourth day (1.45, 1.19 to 1.81). The peak was not followed by a compensatory drop in number of deaths. White controls, matched on age, sex, marital status, hospital status, location, and cause of death, showed no similar peak in cardiac mortality.

• [They concluded that their] findings of excess cardiac mortality on “unlucky” days are consistent with the hypothesis that cardiac mortality increases on psychologically stressful occasions. The results are inconsistent with nine other possible explanations for the findings—for example, the fourth day peak does not seem to occur because of changes in the patient’s diet, alcohol intake, exercise, or drug regimens.45

The 52 country INTERHEART study established a standardized case-control study in which 15,152 cases and 1,820 controls were enrolled. The relationship of smoking, history of hypertension or diabetes, waist/hip ratio, dietary patterns, physical activity, consumption of alcohol, blood apolipoproteins (Apo), and psychosocial factors to myocardial infarction are reported here. They concluded that, “Abnormal lipids, smoking, hypertension, diabetes, abdominal obesity,
psychosocial factors, consumption of fruits, vegetables, and alcohol, and regular physical activity account for most of the risk of myocardial infarction worldwide in both sexes and at all ages in all regions.\textsuperscript{1}\textsuperscript{36} The section that measured psychological stress indicated that people’s psychological well being collectively was responsible for 1/3 of the population studies. “Persistent severe stress makes it two and a half times more likely that an individual will have a heart attack compared to someone who is not stress [our emphasis].” \textsuperscript{4} They purport that stress and depression combined increase the risk threefold!

Without doubt, we can confidently say that stress is a killer. It affects every aspect of our anatomy and physiology to the point that billions are spent annually on stress-reducing methods. Pilates, yoga, psychotherapy, drugs, chiropractic, and a slew of other stress-reducing mechanisms are employed daily. Not only does various types of stress activate the sympathetic nervous system, it causes large quantities of corticotropins to be released by the anterior pituitary gland, precipitating the extra release of glucocorticoids (namely, cortisol) by the adrenal cortex.

Thus, activating hormone-sensitive triglyceride lipase, an enzyme that catalyze the hydrolysis of fats into glycerol and fatty acids,\textsuperscript{4} When corticosteroids and glucocorticoids are secreted in excessive amounts for extended periods of time, ketosis develops due to the frequent mobilization of fats\textsuperscript{4} It is important to note that cortisol also triggers glucose release from liver and, in most cases, acts as a direct antagonist to insulin. Thus, excess cortisol causes elevated blood-sugar/insulin levels and insulin-resistance, which can lead to diabetes.\textsuperscript{42} Moreover, high cortisol levels also raises VLDL, LDL, fibrinogen, PAI-1 (clotting factor), Von Willebrand (clotting factor), Lp(a) (clotting factor) levels and have an effect in lowering HDL levels.\textsuperscript{5}

Thus far, we have attempted to depict “stress” as a main culprit of CVD’s, yet defining and measuring it is a more daunting task. First of all, by “stress” we are not including physical stressors that contribute to advantageous health mechanisms (i.e. massage, exercise, competitive sports, thrill-seeking endeavors like rock climbing, etc.). Nor are we referring to psychological stressors like anticipation or excitement over successful life events such as winning a game or exceeding sales goals. See Graph #5 and #6 for a list of some common stressors that elicit pathogenesis as adapted by Liska et al.’s\textsuperscript{41} and Kendrick’s\textsuperscript{5} work concerning stress production by the body.

Of course, not intended to be a conclusive list, it is clear that most everyone across the globe has had in the past or is currently suffering from any number of these. Thus, suggesting that there is good reason to see the commonality we all share in the 21\textsuperscript{st} century and why CVD’s remain the #1 cause of death in every culture. The effects of this stress is staggering, affecting every aspect of our lives. One interesting function that is primarily affected and very often overlooked is the ability of our bodies to not only properly digest food, but to maintain metabolic homeostasis while under stress. We find that eating under stress has serious implications to our health.

Kendrick offers an explanation to this mechanism in which theautonomic nervous system responds to stress via the Hypothalamic-Pituitary-Adrenal axis (HPA-axis). This three-part hormonal system consists of the hypothalamus, pituitary gland, and adrenal gland in which stress hormones (adrenaline, cortisol, growth hormone, and glucagon) are controlled by the hypothalamus and pituitary gland working in unison, and consequently released by the adrenals. Knowing that the parasympathetic nervous system (representing an anabolic state) and sympathetic nervous system (representing a catabolic state) are generally antagonist to each other, he suggests that eating under stress is extremely unhealthy and is a major contributor to CVD. He reasons that,

If you were stressed, and then tried to eat, your metabolism would be thrown into confusion. You would be commanding the neurohormonal system to activate catabolism and anabolism simultaneously. This would mean high levels of adrenaline and cortisol [sympathetic response to stress], battling against high levels of insulin [parasympathetic response to eating]. Adipose tissue would be under instructions to both absorb and pump out fats into the bloodstream [a sympathetic response representing a catabolic state, gear the body burn up its energy stores, in preparation of a fight-flight response]. At the same time, the liver would be trying to store, and release, glucose [a parasympathetic response representing an anabolic state, rest-digest, to store energy reserves for a later time]. With food inside them, your guts would be automatically switched to “absorption.” But the sympathetic system would be fighting to direct blood away from the guts to the muscles. Wherever you looked, a fight for metabolic supremacy would be going on.\textsuperscript{5}

When the stress system breaks down, what Kendrick calls the “dysfunctional HPA-axis,” a tumor in the pituitary gland develops; which proceeds to release extreme stress hormones. If, for instance, a tumor develops eliciting excess ACTH (corticotropin) release, cortisol levels soar. And as we have seen just above, surging cortisol levels are directly associated with CVD’s.

\textbf{Chiropractic’s Efficacy in Managing CVD - Subluxation}

A cursory look at the above-mentioned term, “vertebral subluxation,” will guide our discussion on how chiropractic can successfully manage CVD. First of all, by “subluxation,” we are referring to mechanical compression and irritation to spinal joints and nerves creating neurological insult. Subluxation scrambles the neurological feedback loop by causing altered rhythms of neurological flow. There have been various attempts to explain this phenomenon. Several models pertaining to this case study will be discussed briefly.

The Subluxation Degeneration Model describes a progressive process associated with abnormal spinal mechanics.\textsuperscript{38} First explored in 1838, this model contends that in the cervical spine, for example, degeneration begins with the intervertebral discs, progressive changes in the vertebrae, and contiguous soft tissues. In the lumbar spine, pathomechanics and torsional stress have been suggestive causes of degeneration.\textsuperscript{48}

The neurological consequences of spinal degeneration include, but not limited to: cord compression; nerve root compression; local irritation; vertebral artery compromise; autonomic dysfunction.\textsuperscript{48}
In the Nerve Root Compression Model, we see that nerve roots are much more vulnerable to mechanical effects than even peripheral nerves. Their location within the intervertebral foramen, their lack of epineurium and perineurium protection, and their venous congestion especially make them vulnerable to insult. Moreover, circulation to the dorsal root ganglion has also been shown to be vulnerable. 

The Dyafferentation Model describes those situations in which biomechanical dysfunction result in alteration in normal nociception and/or mechanoreception. This is clearly evident as the intervertebral motion segment is especially endowed by nociceptive and mechanoreceptive structures. As Kent describes, “To use the contemporary jargon of the computer industry, ‘garbage in – garbage out.” Subsequently, abnormal afferent input to the CNS will lead to a plethora of efferent pathologies.

The Neurodystrophic Model, according to Kent, “suggests that neural dysfunction is stressful to body tissues and that ‘lowered tissue resistance’ can modulate specific and nonspecific immune responses.” Spinal lesions are suggested as being associated to increased sympathetic stimulation and paraspinal muscle tone. According to the literature, subluxation-based chiropractic care is directly related to correcting the main causes of atherosclerosis and CVD as discussed above; namely, inflammation, and elevated stress leading to increased serum cortisol levels.

In recent study published by Roy et al., chiropractic care, for example, has been shown to modify mediators of inflammation (IL-6 and CRP). Teodorczyk-Injeyah et al. confirmed these findings, claiming that spinal manipulative therapy reduces inflammatory cytokines. The literature also reports several instances in which various chiropractic techniques have aided in successful management of emotional and mental stress.

Our literature review also revealed 3 cases in which chiropractic care was directly involved in lowering plasma and salivary cortisol levels. Moreover, it has been preliminarily suggested that chiropractic adjustments directly affect autonomic responses, where cervical adjustments alter parasympathetic activity and thoracic adjustments altering sympathetic activity. This should be of no surprise as the literature is filled with examples of chiropractic’s efficacy in positively affecting a slew of physical and psychological stressors affecting CVD:

- Blood pressure/anxiety
- Enhanced immune response(s)
- Heart Rate Variability
- Dysrhythmic abnormalities
- Duodenal Ulcers
- Muscle tension
- Overall increased bodily function (objectively and subjectively observed)
- Neurocognitive function
- Dysmenorrhea
- Pulmonary function
- Decreased labor times for primigravidae and multiparous pregnancies
- Various mechanisms have been suggested and further research is greatly needed to substantiate these findings as they are comparably few in light of the tome of medical articles in the literature. Nevertheless, we see an indelible and undeniable link between chiropractic’s proven efficacy in treating inflammation and stress and decreasing our patient’s risk of CVD as was evidenced, according to his cardiologist, as normalized lipid levels. The models of vertebral subluxation discussed above help us suggest some proposed mechanisms.

In light of the Neurodystrophic Model, it is clear to see the relationship between vertebral subluxation correction and proper management of stress conditions, altered immune functions (i.e. inflammation, etc), and altered susceptibility to a variety of diseases (i.e. CVD, etc). This should be no surprise, as it is generally accepted that the nervous system has a direct relationship on the immune system. This has been widely known since 1993, in which a New York Times article citing Hosoi et al. popularized this fact. Quoting the article, “Scientists have found the first evidence of an anatomical connection between the nervous system and the immune system. Nerve cell endings in the skin and the white blood cells of the immune system are in intimate contact, and chemicals secreted by the nerves can shut down immune system cells nearby.” The implications of this relationship are remarkable for chiropractors. If, for instance, it is demonstrated that correcting vertebral subluxation(s) can affect the nervous system in such a way that nerve cells are stimulated to shut down inflammatory immune cells, then we will see a direct link substantiating chiropractors’ efficacy on managing a plethora of diseases; particularly, in our case, atherosclerosis and other CVD’s. Of course, further research is needed to make this claim, yet the legitimate need to research this topic is clearly evident.

In regards to stress hormones, it is important to note that cortisol is directly elevated in response to one of the key physical stressors discussed above; one in which chiropractic is intimately involved with; namely, spinal cord injury. According to an extensive literature review by Kendrick,

- Spinal-cord injuries leads to severe HPA-axis dysfunction and raised cortisol levels.
- Patients with spinal-cord injury have low HDL levels (and other lipid abnormalities, e.g. raised VLDL levels).
- Patients with spinal-cord injury have sharply raised blood-clotting factors, including fibrinogen, Lp(a), and plasminogen activator inhibitor-1 (PAI-1).
- Spinal-cord injury leads to insulin resistance, up to and including frank diabetes.
- Spinal-cord injury patients develop visceral obesity [a well-known risk factor for CVD].
- Spinal-cord injury patients are at a greatly increased risk of dying of heart disease.

As seen in our description of vertebral subluxation, it is at the very core of chiropractic philosophy to address and correct spinal-cord injuries. Thus, it can be inferred that by addressing the subluxation directly, chiropractors are possibly correcting pathological cortisol levels and subsequent CVD. As a
disclaimer, please note that by referring to “spinal-cord injury” we are inferring to any injury/insult to the cord that does not require immediate, emergency response intervention due to direct trauma such as severing, etc. In such cases, typical medical intervention is preferred and of course recommended.

In light of the Dysafferentation Model of vertebral subluxation, if vertebral subluxation causes dysafferentation in the hypothalamus, pituitary gland, and adrenal cortex, then it is plausible that related hormones (i.e. cortisol) could be pathogenically produced and therefore released. Furthermore, if, according to this theory, correcting a subluxation subsequently corrects an abnormal afferent response, we clearly see a direct connection in regulating abnormal hormone levels; thereby decreasing risk for CVD. Of course, further research is needed to substantiate this notion.

Limitations

There are two main limitations our case study.

When considering the fact that there are only 2 previously published studies concerning the relationship between subluxation-based chiropractic and normalizing pathogenic lipid levels, the primary limitation to this study is lack of published research substantiating the claim(s) made in this paper. We do not believe that the patient’s case in this study is an anomaly. We believe that there are hundreds, if not thousands of similar cases either collecting dust on chiropractors shelves or have never been documented around the globe.

There is no magic being done in our office or in the two offices in which similar situations occurred. The difference is that we are focused on research and we were focused on documentation and asking the right questions in order to substantiate chiropractic’s efficacy in treating various pathologies. So in reference to asking the right questions, how do we move forward? And what type of wellness perspective should we adapt to keep chiropractic on forefront of scientific research? The answers to these questions will be discussed in the conclusion below.

The second limitation to our case study is that no data on our patient’s cortisol levels or inflammatory factors exist. We would like to compare these levels to subjective emotional stress ratings and serum lipid levels to see if our speculations are valid. Our guess is that these levels would have decrease appropriately in response to care, but we will never know for sure.

Conclusion

The literature search included in this case study has led to several conclusions. CVD has been the number one cause of death globally for decades in spite of billions of dollars spent on cholesterol-lowering pharmaceuticals and surgical procedures that attempt to “unclog” arteries that are suspected of being filled with cholesterol. In fact, CVD’s are on the rise, in spite of recent data showing that overall serum cholesterol levels have decreased since the above interventions have been employed so aggressively. Moreover, the “cholesterol is harmful” hypothesis has been perpetuated by over a century of focused research, tens of thousands of peer-reviewed journal publications, and is thought to be the predominant factor contributing to CVD. There is a remarkable amount of evidence that supports a different theory. In it, serum cholesterol levels do not directly affect CVD and should not be a focus in managing heart disease. Inflammation, stress, and subsequent hormone and immune responses are purported as being responsible for atherosclerotic plaque, thrombi, and subsequent clotting that cause various CVD’s. Furthermore, the literature suggests that it is highly probable that subluxation-based chiropractic care has a direct contribution in helping manage inflammation, stress, and elevated cortisol levels.

The patient in this study suffered from medium to high (8 out of 10) personal and professional stress for years, depression, and anxiety; factors all contributing to increased sympathetic activity, increased cortisol levels and increased inflammation. As care progressed, patient’s subjective stress levels decreased. With no other lifestyle changes occurring, in just a few months his lipid panel improved so much that his medications were decreased. We suspect his lipid levels were normalized as a consequence of decreased stress and subsequent normalizing in cortisol and inflammatory factors.

Finally, the literature woefully lacks substantiating evidence that subluxation-based chiropractic care is effective in managing atherosclerosis and other factors leading to CVD. Chiropractors need to put their findings in print. The public has a right to know, through case-studies and random control trials, etc., that subluxation-based chiropractic care can possibly help people (without the use of drugs or surgery) who are apparently at risk of heart disease and a slew of other health concerns.

Our concluding proposal is, in order to substantiate chiropractic’s efficacy in positively affecting CVD’s and other significant global health risks, that we revisit our typical doctor-patient interactions within a broader scope of “wellness.” According to Jamison, American chiropractors practice “wellness” care under the mantel of “maintenance care.” She described the purpose of maintenance care “[optimizing] health, prevent conditions from developing, provide palliative care, and minimize recurrences or exacerbations.” Adapting the recommendations from the Institute of Alternative Futures. Jamison suggests that chiropractors take on the role of “healthy life doctors.” As healthy life doctors, chiropractors would specialize in the prevention of disease; a major tenant of vitalistic philosophy that all chiropractors should embrace.

The IAF prediction offers a possible futuristic scenario in which chronic diseases are being forecasted years in advance and lifestyle approaches to disease prevention are more widely accepted by patients; one in which a “healthy life” is regarded as powerful medicine. They note that chiropractors, who historically have argued that they have always incorporated lifestyle changes in their care plans, could greatly benefit in making a paradigm shift and become “healthy life doctors.”

Healthy life doctors would specialize in providing targeted health management plans in order to prevent the onset of
disease. In this model, there would also be increasing evidence that chiropractic adjustments not only help neuromuscular problems, but viscerosomatic conditions – something that the profession has always proposed and one in which the writers of this paper are particularly in agreement with. If adopted, according to Jamison, it would be “necessary that chiropractors routinely at least screen for, and preferably attempt to intercept, the risk factors considered responsible for the majority of diseases in this country [referring to Australia, but equally applicable globally].”

Like in most industrialized countries, there is strong evidence that in Australia, people are exposed to potentially modifiable risk factors (ranked in terms of disability-adjusted life years), “many of which are not specifically addressed in chiropractic clinics.” These risks are the standard ones that we have been discussing all along: illicit drug use, elevated serum cholesterol levels, tobacco use, high blood pressure, obesity, etc. Jamison conducted three surveys in which academic/political chiropractic leaders, practicing chiropractors, and chiropractic patients were surveyed. The results expressed strong agreement between all three groups that the majority welcomed a scope of wellness care in which practicing chiropractors would ask their patients about the listed factors above.

In fact, when surveying 347 patients, there was resounding agreement that chiropractors had every right in the world to question their patients on exercise, occupation, and weight (98%, 98%, 93% respectively). There was strong agreement on whether chiropractors should question about tobacco, alcohol use, and fruit/vegetable intake (70%, 60%, 71% respectively). Other factors questioned included taking blood pressure in which 54% said yes and 22% said no, and inquiring about cholesterol in which 47% said yes and 31% said no.

We would very much like to see a follow up study particularly in regards to blood pressure. Since Jamison’s paper was printed, Bakris et al. published a widely circulated and publicized paper showing chiropractic’s effectiveness in treating blood pressure. They concluded that one upper cervical specific adjustment has the same physiological effect in lowering blood pressure as two blood pressure pills.

Upper cervical organizations around the world have been reaping the reward(s) from these findings as this study has been spread across newspapers and Internet sites across the globe. It is becoming clear that the public is looking for more holistic ways to treat diseases, hoping to avoid drugs and surgery. We speculate that if chiropractic researchers can produce similar results in regards to lipid panels and other CVD risk factors, the public will start to storm chiropractic offices for non-surgical, non-prescription management of CVD.

We hope to have made the point clear enough that chiropractors need to know what risk factors our patients are experiencing in regards to CVD, diabetes, and other major health risks in order to substantiate chiropractic’s potential efficacy in vitalistically managing these diseases. We recognize that a vast majority of chiropractors see their primary focus in determining and correcting vertebral subluxations.

What we are suggesting is not to “treat” CVD’s or diabetes, but to simply give credit to chiropractic where it is deserved. How many thousands of people have been unknowingly saved from heart attacks, strokes, diabetes, and (you fill in the blank) all because of regular subluxation-based chiropractic care we will never know. Yet, just imagine not only the personal, but global benefits if we can make the connection(s) through various research studies. The patient in this case study heard from someone that chiropractic can normalize his lipid panel.

Definitely a bold claim, yet it still brought him into the office and he received care to his utter delight. In an interview, he told us, “There are definitely wonderful results that I’ve gotten since coming into [the clinic].” We can think of no better referral than a satisfied customer like this. What a motivator to going to work when you have people like this in your office(s) who believe in your profession because they are seeing results. Results, mind you, that they are seeing because they know what is going on inside of them and have undeniable proof in the form of lipid panels, blood glucose levels, blood pressure readings, etc.

We recommend that every 6 to 12 months patients be asked to report their personal findings to the above risk factors. What this looks like in each office, of course, will differ according to personality and policy. Whether or not they receive a short form to fill out or have someone ask them the questions directly does not matter. If they do not know what their stats are, then they should be referred to a local clinic to get them checked. This information should be collected and reviewed periodically for significant patterns or changes. The data retrieved will be of extreme importance when, for instance, lipid panels start to regulate and the only known lifestyle change in a patient’s life is regular chiropractic care, as was the case in this study. The rest is simply connecting the dots and sharing with the world how chiropractic was able to help another person outside of neck and back pain.

References
7. Centers for Disease Control and Prevention. Heart Disease [Internet]. Atlanta; c2010 [last updated 2010; cited 2012 January].


36. Lymeropoulos A, Rengo G, Koch WJ. GRK2


In the thermal scan it is evident that there is significant autonomic disturbance in the C2 area as well as the lumbo/sacral area, and moderate disturbance in the lower cervical spine. These are all areas of dural attachment and were a contributing factor for choosing a tonal method for the first adjustment. This initial sEMG illustrated that the motor nervous system was not demonstrating the same amount of disturbance as the autonomic scans. As stated above, this is common of disturbances of a long stand nature where the body is no longer in growth/repair phase despite the fact that issues remain as demonstrated in the autonomic scan. The ROM indicates significant restrictions in cervical rotation and lateral bend and the flexion/extension balance is congruent with the forward head posture found on radiographic exam. The PWP scan reveals a severe decrease in autonomic activity (energy reserves) and a moderate sympathetic shift indicating an increased likelihood of stress response initiation with a less than normal threshold environmental stress.
Scan #2. (2nd Scan – 1 Month Into Care)

The thermal scan illustrates that the lumbo/sacral and lower cervical autonomic activity has decreased, while the C2 swing remained. The sEMG scan indicates the body has become more active in mechanical spinal adaptation and the upper thoracic tone increase indicates potential for cervical curve and forward head posture improvement. Range of motion illustrates improvement in cervical lateral bend and rotation. The PWP shows a sizable increase in autonomic activity, where the parasympathetic and sympathetic balance is greatly improved. It is important to note that normalizing the balance of the parasympathetic and sympathetic tone can cause the body’s change in physiology and perception of its environment leading to the normalization of lipid levels, as seen in this case study.
The thermal scan shows an improvement throughout, an indication that the autonomic nervous system is now dealing with environmental stressors differently. The sEMG has a pattern that is less uniform indicating that the motor nerve system is continuing to go through an adaptive phase. All initial indicators/scans indicated that the patient was in a significant state of defense initially and now that his nervous perception of the environment, if more acute, is now reentering a growth phase. During phases of growth modifications in tone and support must take place and, during these times, it is not uncommon to see the pattern and symmetry ebb and flow for 8-12 months before becoming more symmetrical. ROM continues to improve in all areas except cervical flexion/extension. The PWP indicates that the body is no longer in a sympathetic shift and has moved out of the fight/flight response despite the fact that the overall autonomic energy is lower again. This is likely due to decreased stress level(s) as a result of continued chiropractic care.

### Graph #1

**Cholesterol level guidelines**

<table>
<thead>
<tr>
<th>Desirable Cholesterol Levels</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>Less than 200 mg/dL</td>
</tr>
<tr>
<td>LDL (&quot;bad&quot; cholesterol)</td>
<td>Less than 100 mg/dL*</td>
</tr>
<tr>
<td>HDL (&quot;good&quot; cholesterol)</td>
<td>60 mg/dL or higher</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Less than 150 mg/dL</td>
</tr>
</tbody>
</table>

Note: LDL = total cholesterol – HDL – VLDL – (Triglycerides/5).
Graph #2

CVD risk category & LDL guidelines

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>LDL Goal (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD and CHD risk equivalents</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Multiple (2+) risk factors</td>
<td>&lt;130</td>
</tr>
<tr>
<td>Zero to one risk factor</td>
<td>&lt;160</td>
</tr>
</tbody>
</table>

Graph #3

CVD risk factors

<table>
<thead>
<tr>
<th>Race of Ethnic Group</th>
<th>% of Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>African Americans</td>
<td>25.8</td>
</tr>
<tr>
<td>American Indians or Alaska Natives</td>
<td>19.8</td>
</tr>
<tr>
<td>Asians or Pacific Islanders</td>
<td>24.6</td>
</tr>
<tr>
<td>Hispanics</td>
<td>22.7</td>
</tr>
<tr>
<td>Whites</td>
<td>27.5</td>
</tr>
<tr>
<td>All</td>
<td>27.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inactivity</td>
<td>39.5</td>
</tr>
<tr>
<td>Obesity</td>
<td>33.9</td>
</tr>
<tr>
<td>High Blood Pressure</td>
<td>30.5</td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>20.8</td>
</tr>
<tr>
<td>High Cholesterol</td>
<td>15.6</td>
</tr>
<tr>
<td>Diabetes</td>
<td>10.1</td>
</tr>
</tbody>
</table>

Graph #4

List of medications patient was taken at the time of presenting into office.

- Toprol XL – 50 Mg – 1 per day
- Tricor – 145 Mg – 1 per day
- Vytorin – 10/80 Mg – 1 per day
- Lexapro – 10 Mg – 1 per day
- Wellbutrin – 150 Mg – 1 per day
- Ecotrin – 81 Mg – 1 per day
- Omega 3 – Fish Oil – 2 per day
- Metformin – 500 Mg – 2 per day
- Januvia – 100 Mg – 1 per day
### Graph #5

Physical stressors that elicit pathogenesis

<table>
<thead>
<tr>
<th>Overeating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Eating a diet not consistent with one’s biochemical individuality</td>
</tr>
<tr>
<td>Metabolic imbalances including</td>
</tr>
<tr>
<td>Digesting, absorptive, and microbiological imbalances</td>
</tr>
<tr>
<td>Detoxification and biotransformation imbalances</td>
</tr>
<tr>
<td>Oxidation-reduction imbalances and mitochondropathies</td>
</tr>
<tr>
<td>Excessive, intense, forced exercise or work in adverse conditions</td>
</tr>
<tr>
<td>Extreme environmental change/rapid alteration in temperature</td>
</tr>
<tr>
<td>Drug use</td>
</tr>
<tr>
<td>Major trauma/surgery</td>
</tr>
<tr>
<td>Spinal cord injury</td>
</tr>
<tr>
<td>Steroid use</td>
</tr>
<tr>
<td>Disease of the hormonal system, e.g. Cushing’s disease, phaeochromocytoma, diabetes, acromegaly</td>
</tr>
</tbody>
</table>

### Graph #6

Psychological stressors that elicit pathogenesis

<table>
<thead>
<tr>
<th>Bullying boss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suffering racism</td>
</tr>
<tr>
<td>Being “dislocated” from the surrounding population/culture</td>
</tr>
<tr>
<td>Money worries, long-term debt</td>
</tr>
<tr>
<td>Low status in social hierarchy</td>
</tr>
<tr>
<td>Poor social network</td>
</tr>
<tr>
<td>Non-supportive unloving spouse or abusive spouse</td>
</tr>
<tr>
<td>Significant disappoint Football team losing</td>
</tr>
<tr>
<td>Getting caught in an earthquake (though this is a physical stressor, too)</td>
</tr>
<tr>
<td>Getting up on Monday morning</td>
</tr>
<tr>
<td>Forced emigration/social dislocation</td>
</tr>
<tr>
<td>The number four for many Chinese and Japanese men and women</td>
</tr>
<tr>
<td>The number thirteen for many Americans</td>
</tr>
</tbody>
</table>
Graph #7 – Summary of Scans

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic activity index:</td>
<td>55.08</td>
<td>69.72</td>
<td>52.66</td>
</tr>
<tr>
<td>Autonomic balance index:</td>
<td>76.44</td>
<td>93.99</td>
<td>79</td>
</tr>
<tr>
<td>NSFI:</td>
<td>82.24</td>
<td>84</td>
<td>79</td>
</tr>
<tr>
<td>ALG:</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROM:</td>
<td>84.49</td>
<td>91</td>
<td>89</td>
</tr>
<tr>
<td>EMG:</td>
<td>95.05</td>
<td>77</td>
<td>71</td>
</tr>
<tr>
<td>Thermal:</td>
<td>79.09</td>
<td>86</td>
<td>85</td>
</tr>
<tr>
<td>PWP:</td>
<td>62.56</td>
<td>78</td>
<td>66</td>
</tr>
<tr>
<td>BP:</td>
<td>111/77</td>
<td>116/82</td>
<td>113/79</td>
</tr>
<tr>
<td>Wt:</td>
<td>172 lbs</td>
<td>170</td>
<td>170</td>
</tr>
<tr>
<td>W HR:</td>
<td>0.92</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Range of Motion (Normal in Parentheses)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical Flexion (50)</td>
<td>59</td>
<td>58</td>
<td>58</td>
</tr>
<tr>
<td>Cervical Extension (60)</td>
<td>53</td>
<td>45</td>
<td>37</td>
</tr>
<tr>
<td>Cervical Left Lateral (45)</td>
<td>31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervical Right Lateral (45)</td>
<td>40</td>
<td>48</td>
<td>45</td>
</tr>
<tr>
<td>Cervical Left Rotation (80)</td>
<td>50</td>
<td>67</td>
<td>75</td>
</tr>
<tr>
<td>Cervical Right Rotation (80)</td>
<td>57</td>
<td>56</td>
<td>67</td>
</tr>
<tr>
<td>Lumbosacral Left Lateral (25)</td>
<td>21</td>
<td>25</td>
<td>23</td>
</tr>
<tr>
<td>Lumbosacral Right Lateral (25)</td>
<td>24</td>
<td>36</td>
<td>23</td>
</tr>
<tr>
<td>Lumbosacral Flexion (60)</td>
<td>51</td>
<td>51</td>
<td>48</td>
</tr>
<tr>
<td>Lumbosacral Extension (26)</td>
<td>34</td>
<td>37</td>
<td>27</td>
</tr>
</tbody>
</table>

**Thermal Scan**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Red Zones</td>
<td>C2, L4, S1</td>
<td>C2</td>
<td></td>
</tr>
<tr>
<td>Blue Zones</td>
<td>L5</td>
<td>T2, T12</td>
<td></td>
</tr>
<tr>
<td>Green Zones</td>
<td>C6, T1</td>
<td>L1, L2</td>
<td>T1</td>
</tr>
</tbody>
</table>

**Static EMG**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pattern:</td>
<td>98.32</td>
<td>69.25</td>
<td>64.31</td>
</tr>
<tr>
<td>Symmetry:</td>
<td>90.14</td>
<td>87.77</td>
<td>80.45</td>
</tr>
<tr>
<td>Total Energy:</td>
<td>101.74</td>
<td>105.73</td>
<td>108.72</td>
</tr>
</tbody>
</table>
Appendix

The historical account below of the “cholesterol is harmful” hypothesis is taken from Dale Peterson’s article Copy of Cholesterol Limbo: How Low Can You Go? Dr. Peterson is a graduate of the University of Minnesota College of Medicine. He completed his residency in Family Medicine at the University of Oklahoma. He is a past president of the Oklahoma Academy of Family Physicians. He had a full-time family practice in Edmond, Oklahoma, for over 20 years and was a Chief of Staff of the Edmond Hospital. He was active in teaching for many years as a Clinical Professor of Family Medicine through the Oklahoma University Health Sciences Center.

The “cholesterol is harmful” hypothesis is but one of several theories that have been advanced over the past 200 years to explain the phenomenon of atherosclerosis (hardening of the arteries). The story of how our understanding of atherosclerosis and coronary heart disease develop is interesting. Current research suggests that a theory first advanced in 1815 may be correct, but it is receiving as little attention today as it did 200 years ago.

The first recorded mention of atherosclerosis is in the writings of Huang Ti, the Yellow Emperor of China in approximately 2650 BC. Huang Ti recorded a “hardened pulse” and suggested that it was associated with a high salt intake.

Hippocrates, considered the Father of Medicine, suggested in about 400 BC that illness was the result of an imbalance of four bodily humours. These he called yellow bile, black bile, blood, and phlegm. It could be argued that the “cholesterol is harmful” hypothesis is an argument that heart attacks arise from an excess of yellow bile, as cholesterol means “solid bile”.

No significant progress was made over the next 2000 years. Around 1500 A.D. Leonardo da Vinci described what we now refer to as atherosclerosis. He stated that blood vessels in the elderly restrict the transit of blood due to thickening of their walls, which he referred to as “tunics”.

The first to specifically advance the “cholesterol is harmful” hypothesis was an English physician, William Heberden, who did so in 1772. He reported that the blood serum of an obese patient who experienced a sudden death was thick like cream.

Coronary artery hardening was first described by another English physician, Caleb Hiller who, in 1799, found a gritty substance in coronary arteries while doing an autopsy. His first impression was that some plaster had fallen from the ceiling, but upon closer investigation he discovered that the plaster-like substance was within the arteries themselves.

In 1815 a London surgeon, Joseph Hodgson, advanced a novel theory of atherosclerosis. Hodgson suggested that inflammation was the underlying cause of the disease rather than a natural part of the aging process. In that same year, however, cholesterol was discovered by a French researcher and Hodgson’s theory was largely ignored.

It was in 1841 that Carl Von Rokitansk, one of the first pathologists, proposed that the deposits he observed in the inner layer of arteries were derived from substances circulating in the blood. The primary component of arterial plaque was shown to be cholesterol just two years later. The “cholesterol is harmful” hypothesis was advanced in 1949 by J. W. Gofman, an American physician who was researching fats in the bloodstream. He and his team suggested that LDL cholesterol was the cause of atherosclerotic plaque. The hypothesis gained additional support when autopsies of young soldiers killed in the Korean War revealed that 77.3 % had cholesterol deposits in their coronary arteries.

Spurred by the observation that the death rate from heart attacks dropped in areas where the food supply was low during World War II, a University of Minnesota researcher, Dr. Ansel Keys, conducted studies on dietary fat and heart disease beginning in the 1950s. As a result of his studies Dr. Keys became an advocate of what is now known as the Mediterranean Diet, a diet high in vegetable oils and low in saturated fat.

Dr. Keys’ findings were eagerly endorsed by “cholesterol is harmful” advocates, but he himself did not state that cholesterol was the direct cause of heart disease or atherosclerosis. He pointed out that just because cholesterol is present in arterial plaque does not mean that cholesterol is the cause of arterial plaque. The “cholesterol is harmful” train had left the station, however, and nearly all dieticians, physicians, and medical researchers ran to jump on the bandwagon. The movement steadily picked up momentum during the 1960s and 1970s. By the time an alternative theory was advanced in the late 1980s hardly anyone was willing to listen.

Despite its popularity, the “cholesterol is harmful” theory remains unproven. After tens of thousands of studies and billions of dollars invested in research, the best answer advocates can give to the question, “Is lowering cholesterol beneficial?” remains, “We think so.” Conclusive proof does not exist. While lowering cholesterol has been shown to decrease the number of deaths from heart attacks in some age groups, evidence that lowering cholesterol increases longevity and prolongs vitality is lacking.