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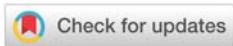
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Review Article

The Cheney Syndrome: To a man with a hammer a lot of things look like nails that need pounding. The Importance of Diet and Lifestyle Modification for treatment of Heart disease

significantly reduced 30 day and 6 month mortality compared with patients who do not. Unfortunately, many patients still do not receive the necessary dietary and lifestyle counseling required to accomplish this goal.

Instead of emphasizing the prevention aspect of coronary artery disease, most individuals have elected to depend upon physician intervention and treatment of acute coronary events, rather than make the necessary dietary and lifestyle changes required to reduce their risk of heart disease. Several approaches to, and publications regarding, the treatment of acute coronary syndromes (ACS) through interventional therapy have existed since the 1960s. This includes 1,488 references on thrombolytic therapy, 11,370 references on angioplasty, 2,865 references on stents including both radioactive and non-radioactive varieties, 1,144 references on atherectomy, 79 references on rotator therapy, 209 published studies on transmyocardial laser revascularization (TMR) therapy, 174 gene therapy articles, and 11,020 published articles on coronary artery bypass surgery, including both venous and arterial grafts. Clearly if the use of interventional procedures alone provided the answer, the battle on heart disease would have already been won. Acute interventional procedures should be viewed as a means to prolong life during the immediate period of crises and provide “short term”, rather than “long term” solutions to the problem.

Most patients and many doctors believe that once interventional procedures have been performed, everything will be fine and the problem has been solved. Many patients believe they no longer have coronary artery disease once they have undergone interventional treatment and cardiac rehabilitation, which while extremely important as part of a dietary and lifestyle change, does not unfortunately mean they no longer have coronary artery disease. While there are no clinical trials suggesting medications should be discontinued following interventional procedures, some physicians reinforce

Coronary artery disease is the leading cause of death in the United States and has been so for several decades affecting an estimated 12 million Americans. This year approximately 1.1 million Americans are expected to have an acute cardiac event with almost 2/3rds of these (650,000) being recurrent cardiac episodes in people already being treated for heart disease; accounting for 1 out of every 3 deaths [1]. This high rate of recurrence clearly illustrates that the problem doesn't go away following initial medical treatment, interventional procedures and/ or by simply telling people to change their diet and lifestyle; particularly given all the confusion about what is a health diet and lifestyle.

Despite the staggering statistics and the knowledge that elevated cholesterol, homocysteine, fibrinogen, lipoprotein (a) and other factors related to dietary and lifestyle practices are responsible for heart disease, little if any significant improvement in preventive behaviors have occurred in either the United States [2], or European [3], communities. While the dietary and lifestyle practices leading to the development and progression of atherosclerosis are formed during our adolescence [4], years, people who are willing to make the necessary dietary and lifestyle practices later in life can reduce their risk of heart disease. Those people who lower their LDLc levels by as little as 5% during 6 weeks of dietary and lifestyle changes, can further lower their risk of heart disease with these dietary and lifestyle changes [5], alone. Further research now shows [6], that following a heart attack, patients started on a regimen to lower their serum cholesterol level have a

this idea by taking patients off their medications following angioplasty, bypass surgery, et cetera, and fail to discuss and reinforce the necessary changes in diet and lifestyle needed to halt the disease and potentially even reverse it.

Despite recommendations by the American Heart Association, some patients are advised to focus on “getting their strength back” with less attention paid on lower saturated fat intake. This dichotomy in messages about which foods are healthy and which are not is even more confusing to patients and their families, and does nothing to make the needed changes in diet and lifestyle required to reduce the risk of future events. It is after all, these very foods which lead to the buildup of inflammatory cholesterol plaques. These are the very foods which led to the need for surgery or treatment in the first place. It is important to note, that there has not been a single study, showing an interventional procedure which has reversed or controlled heart disease, yet we have continued to approach heart disease as if interventional procedures and/or surgery are the cure to the problem, instead of the temporary fix that they provide. The benefits of interventional procedures lie in their ability to buy the time necessary to make the changes required to treat the underlying heart disease, assuming the patient lives long enough to make these changes.

For many people, their first real treatment for heart disease isn't diet or lifestyle change, but the use of thrombolytic agents following their presentation to the emergency room with chest pain resulting from a blood clot blocking blood flow through a diseased coronary artery. These blood clots can occur in arteries with or without significant narrowings and are frequently associated with an inflammatory cholesterol laden plaque rupture. These thrombolytic agents include agents such as tissue plasminogen activator (t-PA), streptokinase, urokinase, and a variety of other agents used to dissolve the blood clot, while other agents are used to reduce the risk of further blood clot formation. These drugs are not without risk and the treatment of an acute myocardial infarction (AMI) and the selection of thrombolytic agents has continued to change [7], over the years along with the consideration of whether thrombolytics should be used, or if angioplasty (PTCA) or stent placement, or a combination of methods, should be used in the acute setting [8].

Angioplasty itself is associated with the dislodgement of plaques which go downstream and result in infarctions distal to the site of angioplasty. The use of PTCA and stents are not without risk and include re-stenosis [8], heart attacks, strokes and other issues. Many studies now discuss the outcome of initial treatment on the basis of initial reperfusion of occluded arteries (TIMI flow) versus longer terms outcomes, such as actual patient survival. Again the point must be made that treatment of the initial occlusion is a “short term” response to a “long term” problem which doesn't get better as the result of a mechanical intervention alone. The underlying problem lies within the artery and “long term” treatment must focus on the causes of the problem and their treatment.

In the absence of an ACS, studies have also shown that there is little or no substantial difference in the survival of people

treated either medically or surgically [9-11]. The relief of angina does not guarantee longer survival. It is after all simple treating the symptom and not the problem. The exceptions, noted in the Coronary Artery Surgery Studies (CASS), are those patients whose left main or triple vessel [12], disease with left ventricular dysfunction have placed them at an even greater risk of sudden cardiac death. In this instance surgery will keep them alive for now, only to be revisited by the same problem unless they change their diet and lifestyle.

While interventional procedures like angioplasty and atherectomy can be safely performed [13], in many instances, there is an increased incidence of major adverse cardiac events [14], in older individuals, people with prior myocardial infarctions and those with reduced left ventricular function. These recurrent episodes of angina, and/or myocardial infarction, are the result of inflammatory reactions [15,16], which atherectomy and other interventions cannot change, even if they are used for the treatment of recurrent angina following stent [17], or vessel re-stenosis. Given the re-stenosis rates of stents and angioplasties [18-20], the initial advantage is more one of cost savings [21], with cardiac events occurring in 26.2% of those undergoing stent placement in contrast to 12.2% in those undergoing bypass surgery. Efforts to use laser therapy or rotablator for in-stent re-stenosis have also not been particularly effective [22]. While stents may be less expensive than bypass surgery, they have a greater need for repeat revascularization and repeat risk of heart attacks and stroke. The benefits of medical regimen and bypass surgery appear to be equivalent for most individuals, but in the final analysis there is progression of native coronary artery and bypass vessel (both venous and arterial) disease unless risk factor modification is implemented, which includes dietary and lifestyle changes beginning with immediate intervention of such within the hospital setting itself. The use of vitamin supplements, especially in the face of cardiomyopathies have not yet been clearly established.

Given the problems with prior interventional procedures to correct coronary lumen disease (CLD), a term much more descriptive of the disease extending from the coronary artery into the lumen space, newer efforts to change myocardial blood flow have included transmyocardial laser revascularization (TMR), gene therapy and even the use of radioactive stents. Despite initial optimism, these procedures have not shown the promise once hoped for. Laser therapy has only demonstrated improvement in coronary blood flow for one-third [23], of those studied 3 months after TMR is performed, while other studies have shown deterioration of blood flow [24], in the regions of laser treatment, with any beneficial effects occurring in the nonlasered [25], regions of the heart. While TMR may help some individuals in the “short term” [26], like other interventional procedures it does not solve “long term” problems, and it is not being enthusiastically recommended [26].

The addition of gene therapy (vascular endothelial growth factor, aka. VEGF) which was once thought to enhance angiogenesis through the promotion of collateral blood flow, resulting in the reduction of angina by reducing regional blood

flow differences responsible for angina [27,28], has since been shown to potentially promote further atherosclerotic [29], development, resulting in progression of atherosclerotic lesions. Finally, the use of radioactive stents [30,31] have failed to produce any better results than earlier interventional procedures with the additional development of “candy-wraper” lesions at the edges of the stents.

While interventional procedures do and must play an important role in the initial treatment of acute coronary syndromes, prevention remains the key [32] to reducing coronary heart disease (CHD) morbidity and mortality. Poor dietary and lifestyle practices have resulted in greater numbers of Americans and people throughout the world becoming overweight [33], with an increasing incidence of heart disease. Our current unsuccessful prevention of, and intervention driven treatment of heart disease is responsible for greater numbers of individuals either dying from heart attacks or living with congestive heart failure [34,35] following AMIs. In 2001, 700,000 Americans will die in heart failure [36], a result of failure to correct the underlying cause of heart disease and culminating myocardial infarctions. However, people need not die from or even necessarily experience a heart attack, if they make the appropriate changes in their diet, lifestyle and medical management.

As already discussed, risk factor modification if done right, can significantly reduce mortality in people who have experienced, and survived, a heart attack. Changes in dietary consumption of saturated fat has been shown to be associated with a reduction in serum lipid levels [37]. For some, even a moderate fat diet [38] may be detrimental if they are used to diets with lower fat content. There are now several studies [39-44] which have shown either improvement in cardiac risk factors or regression of heart disease when dietary and lifestyle changes are instituted. Still other studies have shown actual recovery of viable myocardium [45-48] when aggressive medical and dietary programs are undertaken to reverse atherosclerosis by reducing cholesterol, triglycerides, homocysteine, fibrinogen, lipoprotein (a) levels and the other risk factors responsible for heart attacks and strokes. These dietary and lifestyle practices not only reduce the risk of heart attacks; but, strokes and heart failure as well. Additionally, other types of dietary patterns [49-51] have been shown to be associated with the progression of heart disease and to impair coronary blood flow, including diets which are high in saturated fat and excess calories. These diets can result in the rapid progression [52] of heart disease.

The use of today's interventional procedures can truly be lifesaving for many individuals, at least in the acute setting; but, they provide only a temporary measure which can allow us the necessary time needed to correct the underlying problem, which must be corrected through the treatment of the cause(s) and not the symptom(s) of coronary artery disease. Sometimes, the use of these procedures prevents the correction of the underlying problem by leading physicians and patients both down the path of complacency. It is easy to forget the underlying problem once the symptoms have been temporarily controlled even though the cause of those symptoms continues to worsen and even easier to resume the old dietary and

lifestyle practices which led to the problem in the first place. The following is a case example of how the use of interventional procedures help for a while, but the long term treatment of the problem requires a different approach. An approach which can successfully reduce the risk of heart disease and the risk of death.

Case Presentation

Dr. SK is a 63 year old gentleman referred for evaluation from Washington, D.C. where he lives, after recurrent angina resulted in chest pain with any activity immediately after eating or with walking less than a quarter of a block. The presentation of his angina described as a “squeezing” (Levine sign) retrosternally discomfort began in October of 1979 when as a student he sought medical attention at student health. He was initially told he was too young and in too good a shape to be having a heart problem and presented with his first myocardial infarction four-hours later in the local hospital emergency room.

He was treated interventionally and by January of 1980 he was scheduled for his first coronary artery bypass surgery (CABG), which included a saphenous vein (SV) graft to his right coronary (RCA) and left circumflex (LCx) artery. Following this surgery he remained essentially angina free for the next 8 years, with angina returning in 1988. The angina persisted and by 1990 he began the first in a series of 8 angioplasties (PTCAs) he would undergo between 1990 and 1994. In 1995 a stent was placed which closed three weeks later. Following this a left internal mammary artery (LIMA) graft was placed to the left anterior descending (LAD) artery and the patient was finally enrolled in a lifestyle modification program.

Twelve weeks later the angina ceased. By November of 2000, the angina had returned, happening if he tried walking one-quarter block, or with any exertion following a meal. On the 15th of December 2000 he underwent a thallium-201 imaging study which revealed ischemia in all three major epicardial artery territories. An angiogram performed on the 8th of January 2001 confirmed these findings and revealed a normal left main (LM) with 100% occlusion of the LAD at its origin, 100% occlusion of OM2 (LCx) which was now receiving collateral blood flow from a dominant RCA which had “several” lesions with greater than 70%DS narrowing of the coronary lumen. The SV grafts to both the RCA and LCx were 100% occluded. Only the LIMA graft remained open; but, to a “diffusely” diseased LAD.

Of additional concern was a history of diabetes mellitus, hyperlipidemia, hypertension, prior myocardial infarctions and left carotid disease. Given these findings the patient reports that he was advised by his physicians including 2 cardiologists and 1 internist, to do one of three things: (1) find a program that could help him reverse his heart disease, (2) try for a heart transplant, and/or (3) update the status of his “will”. The interventional procedures including two bypass surgeries, a stent and 8 angioplasties had failed along with an unsuccessful effort at lifestyle modification. The patient was subsequently referred to us in late January of 2001 following a cardiology presentation one of his physicians had heard in Dallas, TX.

SK was initially seen at the Institute on the 23rd of January 2001 taking Zestril 2.5 mg b.i.d., Imdur 30 mg qD, Atenolol 5.0 mg b.i.d., Norvasc 5 mg daily, Lipitor 10 mg qHS, Claritin 10 mg daily, Hytrin 2 mg qD, aspirin 325 mg daily, Insulin 12U NPH & 5U reg qAM and 10U NPH & 4U reg qPM. He is also taking alpha lipoic acid 200 mcg TID, L-carnitine 1 gm qD, Chromium Polynicotinate 200 mcg TID, CoQ-10 100 mg qD, Vit C. 1.5 gm qD, Fish Oil 1 mg TID, Folate 1600 mcg qD, Mg Glycinate 400 mg qD, Vit E 800 IU qD and MVI qD.

His initial fasting blood work showed an elevated glucose of 152 (65-115), a homocysteine level of 12.2 (<9.0), C-reactive protein <1.0 (0-1), triglycerides 75 (<150), total cholesterol 110 (<150), HDLc 30 (>34), LDLc 65 (<100), fibrinogen 327 (200-400) and lipoprotein (a) [Lp(a)] of 14.0 (<30). His initial electrocardiogram revealed sinus bradycardia with left anterior fascicular block (LAFB), right bundle branch block (rBBB), bifascicular block and right ventricular hypertrophy (RVH). His echocardiogram revealed no regional wall motion abnormalities at rest. His cardiac chambers all measured "normal" in size and resting function with a left ventricular ejection fraction (LVEF) of 74.14% and a Cardiac Output (CO) of 5.58 L/Min. Doppler interrogation revealed "mild" pulmonary regurgitation. Evaluation of peripheral vascular disease revealed "mild" intimal thickening of both carotid arteries including a 19% narrowing of the right internal carotid artery (RICA) and an 18% narrowing of the left internal carotid artery (LICA) in addition to ankle brachial indexes (ABIs) of 1.0 bilaterally.

He underwent myocardial perfusion imaging (MPI) to determine the extent of prior myocardial injury, LVEF, regional wall motion abnormalities and coronary regional blood flow [27,28,53]. Prior to our understanding of sestamibi redistribution and quantification of true ASCAD, we had conducted MPI with semi-quantification. His initial resting image demonstrated prior myocardial injury in the inferoposterior, posterolateral, distal anterior and apical regions as shown and described in panel 3 of Figure one. The LVEF was 84.5% and there were no regional wall motion abnormalities at rest. He was then pharmacologically "stressed" with dipyridamole. Four minutes into "stress" he reported "chest and throat" discomfort which radiated into his left mandibular region. The patient reported these symptoms as identical to his "typical" angina. This was associated with QRS widening on the electrocardiogram. The study demonstrated decreased coronary blood flow in all three vascular territories as described in table 1. Following pharmacologic stress, regional wall motion abnormalities were noted in the posterolateral wall with a reduction in LVEF (71.0%) of 13.5% compared with the resting study.

Dr. SK was placed on a dietary program [33], eliminating essentially all fats from the diet. The fish oils were discontinued as well, to eliminate any reduction in blood flow [50] and endothelial function. He was additionally started on L-arginine which was used both as a free radical scavenger mechanism and to enhance endothelial function. The L-arginine was started at 500 mg TID and was slowly increased over the next several weeks to 3 grams TID. His ACEI was changed to quinapril beginning at 10 mg daily and increased over the next several

weeks to a maximal dose of 20 mg BID. With the additional dietary changes, his fasting blood glucose levels decreased, requiring a reduction in insulin treatment to 8U NPH & 2U reg qAM with 6U NPH & no reg qPM. He was also started on Vit C. 1.5 gm qD, Feosol 45 mg TID for treatment of an iron deficiency anemia. Over the course of the next 2-3 weeks he noted increased exercise tolerance and had started to use his treadmill again.

On April 2nd, approximately 10 weeks after his initial dietary, lifestyle and medication changes had been made, Dr. SK returned for re-evaluation. He was now able to walk 20 minutes on the treadmill before noting anginal symptoms and longer if he "warmed-up" first. His medications at this time included Accupril 20 mg BID, Imdur 15 mg daily, Atenolol 5.0 mg qD, Norvasc 5 mg daily, Lipitor 10 mg qHS, Claritin 10 mg daily, Hytrin 2 mg qD, aspirin 325 mg daily, insulin 8U NPH & 2U reg qAM and 6U NPH & zero (currently) U reg qPM. He was also taking alpha lipoic acid 200 mcg TID, L-carnitine 1 gm qD, Chromium Polynicotinate 200 mcg TID, CoQ-10 100 mg qD, Vit C. 1.5 gm qD, Feosol 45 mg TID, Folate 4.8 mg qD, Mg Glycinate 400 mg qD, Vit E 800 IU qD and MVI qD.

Re-evaluation of his blood work demonstrated a glucose of 145 (65-115), a hematology profile reflecting a Hb/Hct of 12.2/34.9% respectively with normal indices, a homocysteine level of 8.7 (<9.0), C-reactive protein <1.0 (0-1), triglycerides 79 (<150), total cholesterol 93 (<150), HDLc 29 (>34), LDLc 48 (<100), fibrinogen 399 (200-400) and lipoprotein (a) [Lp(a)] of 7.0 (<30).

He underwent repeat evaluations including an electrocardiogram which was unchanged from his original study 10 weeks earlier. His echocardiogram revealed no regional wall motion abnormalities. The chambers remained "normal" in size and function with a left ventricular ejection fraction (LVEF) of 86.38% and a Cardiac Output (CO) of 6.28 L/Min. Doppler interrogation no longer demonstrated pulmonary regurgitation. Evaluation of peripheral vascular disease again demonstrated "mild" intimal thickening with a 19% narrowing of the RICA and an 13% narrowing of the LICA with no change in the ABIs. His resting MPI study demonstrated improvement compared with the study completed 10 weeks (Table 1) earlier.

Recovery of viable myocardium was confirmed by comparing wall motion following stress. On the initial study of Jan 24th 2001 there was akinesis of the posterolateral wall. On the second study of April 3rd 2001, the posterolateral wall moved "normally" and the ejection fraction increased following "stress" compared with the drop in ejection fraction seen on the initial study.

Table 1: Changes in the extent and severity of perfusion deficit in each of the arterial beds before treatment (Jan 2001) and ten weeks later (April 2001).

Region (Jan 2001)	Extent (%)	Severity (%)	Region (April 2001)	Extent (%)	Severity (%)
LAD	22	35	LAD	19	29
RCA	26	49	RCA	12	47
LCx	80	61	LCx	61	59
LAD/LCx	89	68	LAD/LCx	85	56

Finally, the “stress” perfusion results of the second study showed improvement in all three vascular territories (Table 1) consistent with improvement in coronary blood flow and not a coronary steal phenomena or collateral blood flow. During this second MPI study, Dr. SK reported anginal symptoms which he rated at 4 out of 10 compared with 10 out of 10 reported during his initial study. After 10 weeks of dietary and lifestyle changes, Dr. SK had noticed a reduction in his angina, a reduction in the disease in his left carotid artery, an improvement in blood flow throughout all of his heart and a recovery of viable myocardium which now functioned to improve his cardiac output, exercise capacity and quality of life.

It is important to remember that whether a patient undergoes interventional procedures with either a cardiologist or cardiovascular surgeon, the final outcome [32-56] is determined by what is done to prevent recurrence and reverse coronary artery disease. Clearly the role of the patient and their primary physician becomes the ultimate determinant of their fate. During the last 4 weeks (14 weeks of treatment), Dr. SK continued to make improvement and reported that he was able to walk on his treadmill for 40 minutes at 5 METS. This is equivalent to shoveling dirt or swimming the breast stroke.

His occasional episodes of angina resolved when he reduced his activity to 3.5 METS, in contrast to the angina he originally experienced after walking a mere quarter block. Like many patients Dr. SK found himself confronted with recurrent angina following multiple interventional procedures. These state of the art interventional procedures performed in the hands of skilled clinicians provide “short term” answers to a “long term” problem. The reclosure rate of arteries opened mechanically is 1 in 3 during the first 6 months, which may sound good to the 2/3rds who do well initially, until one realizes that even these vessels will continue to have progression of coronary artery disease unless something is done to prevent it.

The answer has always been to look at what causes the problem and to correct it. Treating the symptom and not the cause has never worked “long term” in medicine and never will. To many physicians we have been taught to address the “short term” problem with an immediate fix, but we have forgotten to deal with correcting the “long term” problem which is admittedly more difficult to address. The short term solution is much as Mark Twain suggested, “To a man with a hammer a lot of things look like nails that need pounding.” As cardiologists and cardiovascular surgeons we all have our own hammers which we are inclined to use. The limitations of these hammers in the treatment of heart disease is that they treat the symptom and not the underlying cause of heart disease.

This approach at best simply delays the problem for another day. A day which may come tomorrow or six months from now, but a day that will come. The disease is after all, an inflammatory reaction occurring all too often in response to poor dietary and lifestyle practices, and only changes in these dietary and lifestyle practices can change the course of the disease. Dr. SK is a classic example of just such a lesson. The physicians taking care of him had provided the best interventional procedures possible and while they provided

temporary solutions, they did not provide long term answers. This case is not unlikely many other Americans, some of whom we have heard of, and others we have not. Their course like his requires a change in lifestyle, diet and medications designed to address the inflammatory character of the underlying coronary artery disease and promoted its regression.

As Dr. SK’s case points out, this approach may not only improve the coronary blood flow, but also allow the recovery of viable myocardium reducing the ever imposing threat of heart failure or even death. The obvious ramifications to Dr. SK was improvement in exercise tolerance, reduction in angina, an improvement in his diabetes and the opportunity to grow older with his wife. The ramifications to other patients are the same.

Conclusion

The process of successful intervention in individuals with coronary artery disease (CAD) is determined by when intervention begins; primary or secondary. Clearly, once an Major Adverse Cardiac Event (MACE) has occurred, physicians have little choice but to mechanically intervene. That does not however mean the problem of CAD has been resolved; time has merely been bought to make the necessary changes needed to reduce or potentially reverse the underlying “Inflammatory Process” of CAD. [57,58].

Independent of the next steps taken, there remains considerable debate over the role of dietary and lifestyle changes and the effect of vitamin supplementation, which appears to be dependent upon the type of dietary and lifestyle changes recommended [59]. What is needed now is a clinical study to “measure” the actual changes in ischemic CAD looking at these various dietary and lifestyle changes to determine what works and what doesn’t [60].

References

1. Benjamin EJ, Virani SS, Callaway CW, Chamberlain AM, Chang AR, et al. (2018) Heart Disease and Stroke Statistics-2018: A Report From the American Heart Association. *Circulation* 137: e67-e492. [Link: https://bit.ly/31Jxs2W](https://bit.ly/31Jxs2W)
2. News From the Centers for Disease Control and Prevention (2001) Mortality from coronary heart disease and acute myocardial infarction—United States, 1998. *JAMA* 285: 1287-1288. [Link: https://bit.ly/2L8f72x](https://bit.ly/2L8f72x)
3. EUROASPIRE I and II Group; European Action on Secondary Prevention by Intervention to Reduce Events (2001) Clinical reality of coronary prevention guidelines: a comparison of EUROASPIRE I and II in nine countries. EUROASPIRE I and II Group. *European Action on Secondary Prevention by Intervention to Reduce Events. Lancet* 357: 995-1001. [Link: https://bit.ly/2WP0mLm](https://bit.ly/2WP0mLm)
4. American Heart Association (2001) Bad habits, Non-cholesterol risk factors in youth linked to fatty plaques. *Circulation*. [Link: https://bit.ly/2Fp7nwC](https://bit.ly/2Fp7nwC)
5. Henkin Y (2007) Six weeks of dietary therapy for cholesterol reduction predicts long-term success. 48th annual Israel Heart Society, Jerusalem.
6. Aronow HD, Topol EJ, Roe MT, Houghtaling PL, Wolski KE, et al. (2001) Effect of lipid-lowering therapy on early mortality after acute coronary syndromes: an observational study. *Lancet* 357: 1063-1068. [Link: https://bit.ly/2XoHp6w](https://bit.ly/2XoHp6w)
7. Aronow WS (2001) Thrombolysis and antithrombotic therapy for coronary artery disease. *Clin Geriatr Med* 17: 173-188. [Link: https://bit.ly/2WSLLhS](https://bit.ly/2WSLLhS)

8. Le May MR, Labinaz M, Davies RF, Marquis JF, Laramée LA, et al. (2001) Stenting versus thrombolysis in acute myocardial infarction trial (STAT). *J Am Coll Cardiol* 37: 985-991. [Link: https://bit.ly/2WSYhD6](https://bit.ly/2WSYhD6)
9. Davis KB, Fisher L, Pettinger M (1985) The effect of clinical characteristics on the comparison of medical and surgical therapy in the Coronary Artery Surgery Study (CASS) and the Veterans Administration Cooperative trial. *Circulation* 72: V117-122. [Link: https://bit.ly/2Kwp6Gu](https://bit.ly/2Kwp6Gu)
10. Espinola-Klein C, Rupprecht HJ, Erbel R, Nafe B, Brennecke R, et al. (2000) Ten-year outcome after coronary angioplasty in patients with single-vessel coronary artery disease and comparison with the results of the Coronary Artery Surgery Study (CASS). *Am J Cardiol* 85: 321-326. [Link: https://bit.ly/2Y1CkOq](https://bit.ly/2Y1CkOq)
11. Caracciolo EA, Davis KB, Sopko G (1995) Comparison of surgical and medical group survival in patients with left main equivalent coronary artery disease. Long-term CASS experience. *Circulation* 91: 2335-2344. [Link: https://bit.ly/2Y1DhpY](https://bit.ly/2Y1DhpY)
12. Bonow RO, Epstein SE (1985) Indications for coronary artery bypass surgery in patients with chronic angina pectoris: implications of the multicenter randomized trials. *Circulation* 72: V23-30. [Link: https://bit.ly/2N0YHTd](https://bit.ly/2N0YHTd)
13. Kosuga K, Tamai H, Ueda K (2001) Initial and long-term results of directional coronary atherectomy in unprotected left main coronary artery. *Am J Cardiol* 87: 838-843. [Link: https://bit.ly/2x52jJh](https://bit.ly/2x52jJh)
14. Soriano J, Lopez-Sendon J, Moreno R (2001) In-Hospital major complications associated with rotational atherectomy: Experience with 800 patients in a single center. *Rev Esp Cardiol* 54: 460-468. [Link: https://bit.ly/2IVqyz4](https://bit.ly/2IVqyz4)
15. Meuwissen M, Piek JJ, van der Wal AC (2001) Recurrent unstable angina after directional coronary atherectomy is related to the extent of initial coronary plaque inflammation. *J Am Coll Cardiol* 37: 1271-1276. [Link: https://bit.ly/2Y1DVno](https://bit.ly/2Y1DVno)
16. Depre C, Tibichini F, Wijns W (2000) Morphological analysis of atherosclerotic plaque retrieved by coronary atherectomy. *Semin Interv Cardiol* 5: 175-184. [Link: https://bit.ly/2XpqNeP](https://bit.ly/2XpqNeP)
17. Palacios IF, Sanchez PL, Mahdi NA (2000) The place of directional coronary atherectomy for the treatment of in-stent restenosis. *Semin Interv Cardiol* 5: 209-216. [Link: https://bit.ly/2L4k6Z8](https://bit.ly/2L4k6Z8)
18. Rath PC, Tripathy MP, Panigrahi NK (1998) Elective coronary artery stenting—immediate and follow up results. *J Assoc Physicians India* 46: 263-267. [Link: https://bit.ly/2L5x88H](https://bit.ly/2L5x88H)
19. Galassi AR, Foti R, Azzarelli S (2001) Long-term angiographic follow-up after successful repeat balloon angioplasty for in-stent restenosis. *Clin Cardiol* 24: 334-340. [Link: https://bit.ly/2WSzizy](https://bit.ly/2WSzizy)
20. Whitlow PL, Bass TA, Kipperman RM, Sharaf BL, Ho KK, et al. (2001) Results of the study to determine rotablator and transluminal angioplasty strategy (STRATAS). *Am J Cardiol* 87: 699-705. [Link: https://bit.ly/2RoNOco](https://bit.ly/2RoNOco)
21. Serruys PW, Unger F, Sousa JE, Jatene A, Bonnier HJ, et al. (2001) Comparison of coronary-artery bypass surgery and stenting for the treatment of multivessel disease. *N Engl J Med* 344: 1117-1124. [Link: https://bit.ly/2It0yf9](https://bit.ly/2It0yf9)
22. Reith S, Radke PW, Volk O, vom Dahl J, Klues HG (2000) The place of rotablator for treatment of in-stent restenosis. *Semin Interv Cardiol* 5: 199-208. [Link: https://bit.ly/2Fpa4ye](https://bit.ly/2Fpa4ye)
23. Kostkiewicz M, Rudzinski P, Tracz W, Dziatkowiak A (2000) Changes in myocardial perfusion after transmyocardial laser revascularization in patients with end-stage angina pectoris. *Cardiology* 94: 173-178. [Link: https://bit.ly/2x4KTMH](https://bit.ly/2x4KTMH)
24. Burns SM, Brown S, White CA, Tait S, Sharples L, et al. (2001) Quantitative analysis of myocardial perfusion changes with transmyocardial laser revascularization. *Am J Cardiol* 87: 861-867. [Link: https://bit.ly/2XmDffr](https://bit.ly/2XmDffr)
25. Kraatz EG, Misfeld M, Jungbluth B, Sievers HH (2001) Survival after transmyocardial laser revascularization in relation to nonlasered perfused myocardial zones. *Ann Thorac Surg* 71: 532-536. [Link: https://bit.ly/2XoD8zP](https://bit.ly/2XoD8zP)
26. Hayat N, Shafie M, Gumaa MK, Khan N (2001) Transmyocardial laser revascularization: is the enthusiasm justified? *Clin Cardiol* 24: 321-324. [Link: https://bit.ly/2lsmBmd](https://bit.ly/2lsmBmd)
27. Fleming RM, Boyd L, Forster M (2000) Angina is Caused by Regional Blood Flow Differences - Proof of a Physiologic (Not Anatomic) Narrowing. Joint Session of the European Society of Cardiology and the American College of Cardiology, Annual American College of Cardiology Scientific Sessions, Anaheim, California, USA. [Link: https://bit.ly/2XsvOTX](https://bit.ly/2XsvOTX)
28. Fleming RM (2000) Regional Blood Flow Differences Induced by High Dose Dipyridamole Explain Etiology of Angina. 3rd International College of Coronary Artery Disease from Prevention to Intervention. Lyon, France.
29. Dake M (2001) Recombinant VEGF may have atherosclerotic side effects. *Nature Med* 7: 403-4, 425-429.
30. Virmani R, Farb A, Carter AJ, Jones RM (1999) Pathology of radiation-induced coronary artery disease in human and pig. *Cardiovasc Radiat Med* 1: 98-101. [Link: https://bit.ly/2FkDJZc](https://bit.ly/2FkDJZc)
31. Wardeh AJ, Knook AH, Kay IP (2001) Clinical and angiographic follow-up after implantation of a 6-12 mCi radioactive stent in patients with coronary artery disease. *Eur Heart J* 22: 669-675. [Link: https://bit.ly/31LPqBK](https://bit.ly/31LPqBK)
32. (2001) Mortality from coronary heart disease and acute myocardial infarction—United States, 1998. *MMWR* 50: 90-93. [Link: https://bit.ly/2IP7VMU](https://bit.ly/2IP7VMU)
33. Fleming RM (2001) Obesity and related health problems are the result of too many calories and too much saturated fat regardless of the misconceptions promoted by many popular weight loss diets. USDA Research program on health and nutrition effects of popular weight-loss diets. Washington, DC.
34. He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, et al. (2001) Risk factors for Congestive Heart Failure in US men and women: NHANES I Epidemiologic follow-up study. *Arch Intern Med* 161: 996-1002. [Link: https://bit.ly/31LAG5N](https://bit.ly/31LAG5N)
35. Jayakar DV (2001) Surgical treatment of chronic heart failure. What to tell patients about heart-saving options. *Postgrad Med* 109: 67-70. [Link: https://bit.ly/2KXgok2](https://bit.ly/2KXgok2)
36. (2001) Doctors to implant artificial heart. Associated Press.
37. Sacks FM, Ornish D, Rosner B, McLanahan S, Castelli WP, et al. (1985) Plasma lipoprotein levels in vegetarians. The effect of ingestion of fats from dairy products. *JAMA* 254: 1337-1341. [Link: https://bit.ly/2lrdQoA](https://bit.ly/2lrdQoA)
38. Suh I, Oh KW, Lee KH, Psaty BM, Nam CM, et al. (2001) Moderate dietary fat consumption as a risk factor for ischemic heart disease in a population with a low fat intake: a case-control study in Korean men. *Am J Clin Nutr* 73: 722-727. [Link: https://bit.ly/2WV9tyY](https://bit.ly/2WV9tyY)
39. Ornish D, Brown SE, Scherwitz LW, Billings JH, Armstrong WT, et al. (1990) Can lifestyle changes reverse coronary heart disease? The lifestyle heart trial. *Lancet* 336: 129-133. [Link: https://bit.ly/2KrTAtf](https://bit.ly/2KrTAtf)
40. Barnard RJ (1991) Effects of life-style modification on serum lipids. *Arch Intern Med* 151: 1389-1394. [Link: https://bit.ly/2xbDmrv](https://bit.ly/2xbDmrv)
41. Barnard RJ, Inkeles SB (1999) Effects on an intensive diet and exercise program on lipids in postmenopausal women. *Womens Health Issues* 9: 155-161. [Link: https://bit.ly/2RtsXVp](https://bit.ly/2RtsXVp)
42. Fleming RM, Ketchum K, Fleming DM, Gaede R (1996) Assessing the independent effect of dietary counseling and hypolipidemic medications on serum lipids. *Angiology* 47: 831-840. [Link: https://bit.ly/2RoTcFl](https://bit.ly/2RoTcFl)
43. Fleming RM, Ketchum K, Fleming DM, Gaede R (1995) Treating hyperlipidemia in the elderly. *Angiology* 46: 1075-1083. [Link: https://bit.ly/2Kt9cN2](https://bit.ly/2Kt9cN2)

44. Czernin J, Barnard RJ, Sun KT (1995) Effect of short-term cardiovascular conditioning and low-fat diet on myocardial blood flow and flow reserve. *Circulation* 92: 197-204. [Link: https://bit.ly/2IXYWZQ](https://bit.ly/2IXYWZQ)
45. Fleming RM (2000) The clinical importance of risk factor modification: looking at both myocardial viability (MV) and myocardial perfusion imaging (MPI). *Int J Angiol* 9: 65-69. [Link: https://bit.ly/2WXzHkj](https://bit.ly/2WXzHkj)
46. Fleming RM (2000) The Fleming Unified Theory of Vascular Disease: a link between atherosclerosis, inflammation, and bacterially aggravated atherosclerosis (BAA). *Angiology* 51: 87-89. [Link: https://bit.ly/2FgiojJ](https://bit.ly/2FgiojJ)
47. Fleming RM (1999) Chapter 64. The Pathogenesis of Vascular Disease. *Textbook of Angiology*. John C. Chang Editor, Springer-Verlag New York, NY 787-798.
48. Fleming RM (2000) Reversing heart disease in the new millennium—the Fleming Unified Theory. *Angiology* 51: 617-629. [Link: https://bit.ly/2IsHUEo](https://bit.ly/2IsHUEo)
49. Fonseca V, Dicker-Brown A, Ranganathan S (2000) Effects of a high-fat-sucrose diet on enzymes in homocysteine metabolism in the rat. *Metabolism* 49: 736-741. [Link: https://bit.ly/2RvHT5y](https://bit.ly/2RvHT5y)
50. Vogel RA, Corretti MC, Plotnick GD (1997) Effect of a single high-fat meal on endothelial function in healthy subjects. *Am J Cardiol* 79: 350-354. [Link: https://bit.ly/2iyaSFZ](https://bit.ly/2iyaSFZ)
51. Fleming RM (2000) The effect of high-protein diets on coronary blood flow. *Angiology* 51: 817-826. [Link: https://bit.ly/2L4VwqY](https://bit.ly/2L4VwqY)
52. Fleming RM (2000) The natural progression of atherosclerosis in an untreated patient with hyperlipidemia: assessment via cardiac PET. *Int J Angiol* 9: 70-73. [Link: https://bit.ly/2XknOo9](https://bit.ly/2XknOo9)
53. Fleming RM (2000) Ch. 31 Nuclear cardiology: Its role in the detection and management of coronary artery disease. *Textbook of Angiology* 397-406. [Link: https://bit.ly/2IVSW45](https://bit.ly/2IVSW45)
54. Vlietstra RE (1991) Advances in coronary interventional techniques. *Int J Cardiol* 32: 175-181. [Link: https://bit.ly/2FmlP7I](https://bit.ly/2FmlP7I)
55. Manchanda SC, Narang R, Reddy KS (2000) Retardation of coronary atherosclerosis with yoga lifestyle intervention. *J Assoc Physicians India* 48: 687-694. [Link: https://bit.ly/2KtAUJM](https://bit.ly/2KtAUJM)
56. Pocock SJ, Henderson RA, Seed P, Treasure T, Hampton JR (1996) Quality of life, employment status, and anginal symptoms after coronary angioplasty or bypass surgery. 3-year follow-up in the Randomized Intervention Treatment of Angina (RITA) Trial. *Circulation* 94: 135-142. [Link: https://bit.ly/2Zy0PTV](https://bit.ly/2Zy0PTV)
57. Inflammation and Heart Disease (2011) Fleming Unified Theory of Vascular Disease. 1-655833842. Started 9-1-2011. *Effective TX* 7: 451-244.
58. Fleming RM (1999) Chapter 64. The Pathogenesis of Vascular Disease. *Textbook of Angiology*. John C. Chang Editor, Springer-Verlag New York, NY 787-798.
59. Fleming RM, Fleming MR, Harrington GM, Ayoob KT, Chaudhuri TK (2019) The Weight, Inflammation, Diet and Heart (WIDtH) Study Looking at Potential Primary Prevention of Coronary Artery Disease. *Acta Scientific Pharmaceutical Sciences* 3: 33-44.
60. Fleming RM, Fleming MR, Chaudhuri TK, Mckusick A (2019) A Call to Quantify the Impact of KETO Diets. *ACTA Scientific Pharm Sci* 3.

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