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A Guarantee of Arterial Wellness: New Era of Cardiovascular Medicine

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Abstract

The door is open to change the United States platform of healthcare for Cardiovascular Disease (CVD) in terms of maintaining arterial wellness. Accomplishments in this arena of healthcare have been phenomenal, but the vast majority of them deal with end-stage arterial disease. The price tag has been substantial in terms of financial resources and quality of life. Current projections indicate CVD driven mainly by arterial disease will bankrupt our country. Those estimates are based on the current system of treating end-stage arterial disease. We can avoid fiscal insolvency and deteriorating quality of life by shifting to a platform that prevents arterial disease or, at minimum, treats it before it is evident. We live in an era of technology sufficient to identify subclinical arterial disease and knowledge necessary to halt atherosclerosis. For example, safe, inexpensive, painless, and reliable imaging techniques now exist that detect silent non-obstructing arterial plaque, or atheroma; a prerequisite for a majority of cardiovascular events. Additionally, numerous health issues are known to produce inflammation of the arteries, which is causal of atherosclerosis. Each can be managed effectively to extinguish the inflammation. When this is accomplished the disease will halt, which significantly mitigates risk for an acute obstructive cardiovascular event. CV healthcare can now enter a new era of maintaining arterial wellness. We must seize this opportunity for fiscal and humanitarian reasons.

Keywords: Arterial inflammation; Subclinical arterial disease; Prevention platform of healthcare; Cardiovascular wellness; Bankruptcy of healthcare

Cardiovascular (CV) healthcare was established on a platform that treats end-stage disease. At the time, existent technology and knowledge could only manage evident arterial disease. The financial and intellectual commitment in this area of healthcare have resulted in astonishing accomplishments such as coronary bypass grafting, angioplasty, stents, acute antithrombotic therapies, left ventricular assistant devices, transplants, and stem cell research. The consequence has been dramatic reduction in CV mortality. Unfortunately, the incidence of end-stage arterial disease has not been significantly impacted. In addition, the price tag for these phenomenal advances in therapy has been devastating. Based on the current system of healthcare, projections for the incidence and cost of CV disease over the next decade are dismal [1]. The current CV healthcare system is not financially sustainable [2]. Our system must enter a new era, anchored on a platform of maintaining arterial wellness. Fortunately, we now possess the technology and knowledge to do so.

Traditional methods of dealing with Cardiovascular Events (CVE) are reactionary and often inefficient. For example, predicting CVEs on the basis of risk factors frequently fails to identify individuals that actually experience a heart attack or stroke [3]. Identifying arterial disease is a superior predictor of CVEs compared to the presence of a conventional risk factor because the presence of arterial disease in any arterial bed elevates event potential regardless of risk factors [4-6]. According to an important CAFES-CAVE study, individuals who harbor such disease are high risk without treatment for a cardiovascular event [7]. The issue has been the difficulty of detecting arterial disease prior to an event. This is in part due to the fact that while the presence of an atheroma is an essential ingredient for a cardiovascular event, the majority are non-obstructing prior to an atherothrombotic event [8-10]. Waiting for a CVE to prove the presence of arterial disease is no longer necessary. There are safe inexpensive means to detect subclinical arterial plaques [11].

The current era of medicine possesses several non-invasive, innocuous, low-cost means to detect atheroma. X-rays that demonstrate calcification in arterial beds documents the presence of arterial disease. This type of incidental finding is associated with higher CVE risk [12,13]. Ankle Brachial Index (ABI) testing for peripheral artery disease is simple and inexpensive. An abnormal ABI portends significant increased CVE risk [14]. Coronary Calcification (CAC) via Computerized Tomography (CT) is widely available, painless, and involves little radiation. A positive score documents the presence of atherosclerosis [15]. CAC has been associated with increased risk of CVEs [16,17]. Carotid intimal media thickness (CIMT) via B-mode ultrasound is safe, valid, reliable and low-priced [18]. CIMT is associated with increased CVE risk [19]. This imaging technique can also identify carotid plaque, which is associated with increased CVE risk [20,21]. We have multiple reasonable modalities to utilize clinically to evaluate each patient for the presence of arterial disease.

The CAFES-CAVE study referenced earlier demonstrated that patients with atherosclerosis who do not receive treatment have increased risk for CVEs. When arterial disease is identified, these individuals should be treated. Therapy should be directed to the cause

of their arterial disease. Evidence shows that inflammation is causal of atherosclerosis and CVEs [22,23]. Numerous pathologies can contribute to arterial inflammation, which include lipids, smoking, hypertension, insulin resistance, vitamin D deficiency, obstructive sleep apnea, obesity, diet, physical inactivity, psychosocial issues, oral health issues, systemic inflammatory conditions such as rheumatoid arthritis, lupus, genetic influences, and systemic infectious disease [24-37]. Thankfully, therapies for all of these conditions are available, and evaluation of all potential "root" pathologies contributing to arterial inflammation can be performed in an economical manner with each patient.

A priori, halting arterial disease requires extinguishing arterial inflammation. There are objective inflammatory biomarkers that allow for clinical monitoring of arterial inflammation. The acute phase reactants fibrinogen and high sensitivity C Reactive Protein (hs-CRP) can indicate endothelial inflammation. These are as predictive for CVEs as high-density lipoprotein and total cholesterol [38]. Urinary microalbumin-creatinine ratio is an excellent biomarker of endothelial wellness and an independent predictor of CVEs [39,40]. Lipoprotein associated phospholipase A2 (Lp-PLA2) indicates inflammation in the arterial wall [41,42]. Oxidative stress can drive arterial inflammation, and the urinary biomarker F2-isoprostane is an excellent test for this [43]. Myeloperoxidase can result in inflammation of the endothelium and/or arterial wall [44]. The effectiveness of reducing arterial inflammation by managing the "root" inflammatory pathologies can be determined objectively by these serum and urinary biomarkers.

Emphasis upon preventative care has gained momentum over the past few years. The concept of clinically utilizing the aforementioned screening tests for subclinical atherosclerosis was promoted during a recent American Heart Association conference [45]. Subsequent to this publication, we now have appropriate use documents for CAC and CIMT [46-48]. In June 2009, the state of Texas passed the Texas Heart Attack Prevention Bill, which mandates health-benefit plans to provide coverage for CAC and CIMT testing [49]. Additionally, there is an increase in publications that discuss the merit of basing an individual's CVE risk on the detection of subclinical atherosclerosis with such tools as CAC [50]. Recent publications have also elucidated the importance of reducing arterial inflammation as measured by biomarkers discussed above [51-54]. Yet policy change has not kept pace. Despite these endorsements, national health care policy does not yet mandate this type of arterial disease care. This is not surprising, as it is well known that translation of excellent science into the clinical arena can take decades [55]. Green and Seifert demonstrated there is too much delay in the utilization of innovative information by health care systems and providers. As a consequence, patients' outcomes suffer. They suggested health policies should support expert physicians who integrate novel evidence-based knowledge into practice [56].

Fortunately, healthcare providers do not need to wait for a national directive. In the arena of arterial disease, which is so costly from a humanitarian and financial standpoint, healthcare providers may choose, as the authors have, to enter this new era of arterial disease care now. It is exciting to practice medicine in an era where we possess clinical tools and knowledge allowing us to migrate to a more superior platform of healthcare. The platform that focuses on managing end-stage arterial disease is too expensive and devastating to an individual's wellness. We now have the opportunity to shift to a platform designed to prevent disease, or at minimum treating it before it is evident. We do not need to wait for huge randomized double blind prospective outcome studies to prove such a platform will be superior. We have no choice. We have proven that the current platform leads to insolvency. It is possible with a personalized, comprehensive, and holistic approach to determine the causes of the arterial disease in each patient. There are effective therapies for all the inflammatory conditions and adequate biomarkers to judge the effectiveness of the treatment. Arterial inflammation can be extinguished. The new era of CV healthcare can guarantee arterial wellness. The authors have been guaranteeing their patients' arterial wellness for years with the method articulated in their book, Beat the Heart Attack Gene: The Revolutionary Plan to Prevent Heart Disease, Stroke, and Diabetes [57]. We invite you to join us in this satisfying endeavor.

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