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AS WE SEE IT

What the News Media Did Not Reveal About Bill Clinton's Heart Problem

When a celebrity develops a serious illness, the news media reports not only on the famous person, but also on the disease itself. The media conducts interviews with physicians and discusses what may have caused the celebrity's ailment.

We have long argued that medical ignorance is the leading cause of death in the US. We thus believe that the news media provides a partial public service by revealing intimate details about a celebrity's disorder, and information about what average people can do to reduce their risk of contracting the same disease.

In early September, former President Bill Clinton underwent quadruple coronary artery bypass surgery. This operation was performed after Clinton went to his doctor complaining of chest pains and shortness of breath. An angiogram revealed significant (90%) atherosclerotic occlusion in the major arteries feeding his heart. Immediate bypass surgery was prescribed to prevent the 58-year-old former president from suffering a major heart attack.



William Faloon



The news media did a good job of educating the public about coronary artery disease, how it is diagnosed, and what happens during bypass surgery. There was also a lot of reporting on what may have caused the apparently robust former president to develop such a severe case of coronary artery occlusion.

Bill Clinton's penchant for eating artery-clogging fast food was noted, along with his mild hypertension. If these news media reports motivate some Americans to alter their food choices and maintain optimal blood pressure levels, then Clinton's ordeal will have provided some benefit to the public.

Regrettably, the news media spent so much time focusing on Clinton's cholesterol level that people could have been misled into believing that keeping cholesterol low is all it takes prevent coronary artery occlusion. While cholesterol (especially the more dangerous low-density lipoprotein, or LDL) facilitates arterial blockage, it represents only part of the reason why heart attacks continue to strike Americans at epidemic levels.

Misconceptions About Atherosclerosis

Atherosclerosis is the underlying cause of most heart attacks and strokes, yet doctors remain confused as to how this artery-blocking process occurs. Most cardiologists overlook specific mechanisms that inflict arterial wall damage and the ensuing progression to occlusive atherosclerotic disease. The usual result is that only a few of the factors that accelerate arterial blockage (such as elevated LDL) are addressed in today's clinical setting.

Most doctors think of an atherosclerotic lesion as a "clog" consisting of fat, cholesterol, and platelets that have accumulated on an inner arterial wall. As a result, they tell their patients to eat less fat, take a statin drug (if cholesterol levels are high), and use a baby aspirin to prevent arterial platelet aggregation. The problem with these approaches is that while they may postpone a heart attack or stroke, they fail to correct the underlying pathologies that cause atherosclerotic lesions to form and progress.

If people are to live long lives free of the ravages of atherosclerosis, these lethal misconceptions must be cleared up. Otherwise, there will be an epidemic of aging people receiving coronary stents, undergoing bypass surgeries, and dropping dead from sudden heart attacks.

In reporting on Bill Clinton's coronary bypass surgery, the news media stated that over 300,000 of these "routine" procedures are performed every year. Considering the miserable adverse consequences these operations can inflict, coronary bypass surgery should be considered only as a last resort rather than as a "routine" procedure.

BILL CLINTON'S CHOLESTEROL, LDL, AND BLOOD PRESSURE

One of the unique aspects of being president of the United States is that details of your medical history are made public every

year. According to the last exam performed before he left the White House, Bill Clinton's total cholesterol was 233 mg/dL, which is not outrageously high. Since optimal cholesterol levels are now considered below 200 mg/dL,¹ the news media made a big deal over the fact that Clinton's cholesterol was too high.

A more significant problem that the news media only touched on was that the former president's LDL level was a dangerous 177 mg/dL. New guidelines call for LDL to be below 100 mg/dL, and some doctors want LDL to be below 70 mg/dL in those suffering from coronary artery disease.² The Life Extension Foundation was first to advocate that LDL levels should be below 100 mg/dL. Until recently, most mainstream doctors believed that an LDL of 130 mg/dL was all right.

Clinton's blood pressure of 136/84 used to be considered acceptable, but conventional medicine has finally realized that blood pressure over 119/79 exposes one to greater risks of coronary artery disease, stroke, kidney failure, and other diseases.³ One of the drawbacks of being president is that you get free health care provided by government doctors who are not always up to date on new disease risk factors.

Interestingly, Clinton was prescribed cholesterol-lowering medication before he left the White House, but decided on his own to stop taking the drug after losing some weight. Without first verifying that LDL and cholesterol levels are in safe ranges using a simple blood test, discontinuing any lipid-lowering therapy is a risky strategy. Regrettably, side effects from cholesterol-lowering drugs cause some patients to stop taking them without informing their doctors.⁴

Why Arteries Clog as We Age

The aging process damages blood vessels, even when conventional risk factors such as cholesterol and blood pressure are within normal ranges.

For the past 35 years, the standard treatment for coronary atherosclerosis has been to bypass the blocked arteries. Recuperation from this procedure can take months, and some patients are afflicted with lifetime impairments such as chronic inflammation, memory loss, and depression.⁵⁻¹⁵

A review of the scientific literature reveals that atherosclerosis is associated with high blood levels of homocysteine,¹⁶⁻²⁴ fibrinogen,²⁵⁻²⁸ C-reactive protein,²⁹⁻³⁶ glucose,^{37,38} cholesterol,³⁹⁻⁴³ insulin,⁴⁴⁻⁴⁷ iron,⁴⁸⁻⁵¹ LDL,³⁹⁻⁴³ and triglycerides,⁵²⁻⁵⁴ along with low levels of HDL⁵⁵⁻⁵⁷ and testosterone.^{45,58-64} Optimizing blood levels of these substances can dramatically reduce heart attack and stroke risk.

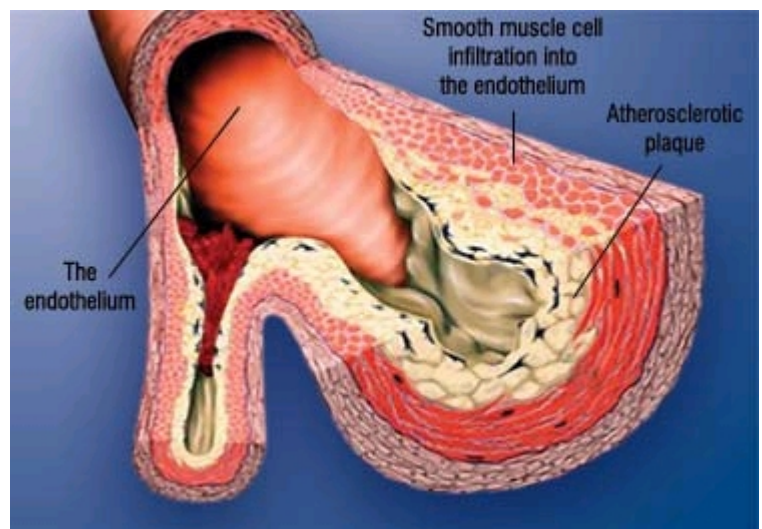
Despite thousands of studies validating that atherosclerosis is a multifactorial process, today's doctors often prescribe a statin drug as the sole therapy to prevent and treat coronary atherosclerosis. Mainstream cardiologists fail to appreciate that coronary atherosclerosis is a sign of systemic arterial dysfunction requiring aggressive therapy to correct it. Conventional medicine's failure became self-evident when the news media interviewed cardiologists about Bill Clinton's diseased arteries. The doctors focused on his elevated cholesterol as the cause of his problem.

Life Extension members, on the other hand, have grown impatient with doctors who fail to translate research findings into improved therapies. More than ever before, health-conscious people are taking responsibility for the health of their arteries by correcting as many of the known risk factors as possible.

Anatomy of the Artery

Arteries are the blood vessels that bear the full force of each heartbeat. Most people think of arteries as flexible tubes whose only function is to carry blood that flows continuously throughout the body. In fact, arteries are dynamic, functioning muscular structures that, when healthy, expand and contract to facilitate circulation and maintain optimal blood pressure.

The outer layer of the artery comprises mostly connective tissue and provides structural containment for the two layers beneath. The middle area comprises elastic smooth muscle that provides the contractile strength to make possible the artery's expansion and contraction with each heartbeat. The inner layer, known as the endothelium, comprises a thin area of endothelial cells whose integrity is crucial if atherosclerosis is to be prevented.



Cross-section of an artery demonstrating plaque.

Poor health habits and normal aging result in endothelial

dysfunction, a process in which the endothelium boundary is broken, arterial flexibility is diminished, abnormal platelet aggregation occurs, and atherosclerotic lesions form in response to arterial wall (endothelium) injuries.

Folic acid,⁶⁵⁻⁷¹ vitamin C,⁷²⁻⁷⁶ fish oil,⁷⁷⁻⁷⁹ and lipoic acid⁸⁰⁻⁸⁴ are just a few of the nutrients that help maintain healthy endothelial function. It is no coincidence that these same nutrients have been shown to reduce cardiovascular incidence in both animals and people.⁸⁵⁻⁹⁴ Agents that suppress chronic inflammation also help protect the endothelium.⁹⁵⁻¹³³

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How Atherosclerosis Develops

At least one of every two Americans over the age of 65 has atherosclerosis.¹³⁴ It is so common in older people that some experts used to think that it was part of the normal aging process.

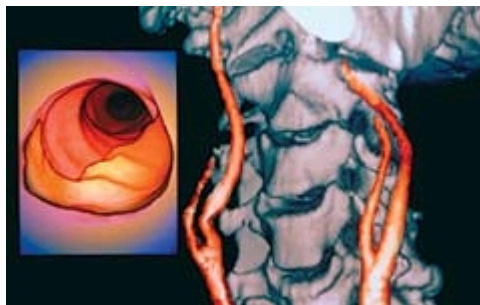
Atherosclerosis begins with changes in endothelial cell function that cause white blood cells moving through the blood to stick to the endothelium (inner arterial wall) instead of flowing by normally. The endothelium then becomes weakened. This allows blood cells and toxic substances circulating in the blood to pass through the endothelium and enter the artery's sub-endothelial compartment. Lipid or fat-cell-like substances such as LDL and triglycerides in the blood then accumulate in this area.

The lipids that accumulate in the broken endothelium become oxidized, causing the smooth muscle cells to try to "repair" the damaged endothelium. The result of this repair process is smooth muscle cell infiltration into the endothelium causing the formation of the initial atherosclerotic lesion. Depending on an individual's risk factors—such as poor diet, lack of exercise, smoking, high blood pressure, and the aging process itself—fat accumulation continues and the atherosclerotic process accelerates.

Immune cells called macro-phages then invade the damaged arterial area to digest the fat. But smooth muscle cells that have migrated to the area have already changed their nature to scavenge fat. These fat-laden white blood cells and smooth muscle cells are called "foam cells," and provoke a chronic inflammatory attack by various immune components.

Smooth muscle cells try to curtail the injury to the endothelium by producing collagen, which forms a cap over the injury site. Calcium then accumulates over the injury site to form a material resembling bone. This is why atherosclerosis used to be referred to as "hardening of the arteries."

This complex array of foam cells, calcification, and lipid accumulation is called an atherosclerotic plaque. The plaque grows, and if it becomes unstable, it is vulnerable to acute rupture that exposes the contents of the plaque to blood. Platelets can then rapidly accumulate around this ruptured plaque, resulting in an acute blockage (or blood clot) on the inner surface of the blood vessel wall. This clot can become very large and occlude the vessel. Even small plaques, if they rupture, can interfere with blood flow and cause an acute heart attack.



A 3-D spiral CT scan of carotid atherosclerosis. Inset: Plaque attached to the wall of left internal carotid artery. This angiogram was taken with a helicoidal scanner.

Alternatively, atherosclerotic plaques can grow to such a degree as to restrict blood flow severely, as was the case with former President Clinton. When blood flow within an artery is gravely compromised by a large plaque or blood clot, the cells of tissues that depend on blood flow from that artery become damaged or die. Coronary atherosclerosis cuts off the heart's blood supply by occluding the heart's arteries, thus stopping the oxygen supply to the heart and causing a heart attack. A stroke results when atherosclerotic processes cut off the oxygen supply to a portion of the brain.

As you can see, therefore, much more is involved in the development of atherosclerosis than just high cholesterol and LDL. We must emphasize, however, that maintaining optimal LDL and cholesterol levels is an important component of an atherosclerosis-prevention program.

Protecting Your Arterial Walls

High blood pressure,¹³⁵⁻¹⁴⁰ elevated LDL-cholesterol-triglycerides,¹⁴¹⁻¹⁴⁶ low HDL,¹⁴⁷ smoking¹⁴⁸⁻¹⁵¹, diabetes,¹⁵²⁻¹⁵⁷ obesity,¹⁵⁸⁻¹⁶¹ and lack of exercise¹⁶²⁻¹⁶⁶ contribute to endothelial dysfunction and the subsequent development of atherosclerosis.

Other significant artery-damaging factors are high-normal levels of glucose, insulin, iron, homocysteine,¹⁶⁻²⁴ and fibrinogen,²⁵⁻²⁸ and any level of C-reactive protein²⁹⁻³⁶ that is higher than optimal.

Homocysteine is particularly dangerous because it can induce the initial atherosclerotic injury to the endothelium, then facilitate the oxidation of the fat and LDL that accumulate beneath the damaged endothelium, and finally contribute to the abnormal accumulation of blood components around the atherosclerotic plaque.

Fibrinogen is a clotting factor that accumulates at the site of the endothelial lesion. Fibrinogen contributes to plaque buildup and

can participate in the arterial blockage after an unstable atherosclerotic plaque ruptures.

Glucose at high-normal levels may accelerate the glycation process that causes arterial stiffening, while high-normal fasting insulin inflicts direct damage to the endothelium. High levels of iron promote oxidation of LDL in the damaged endothelium, while low levels of testosterone (in men) appear to interfere with normal endothelial function.

C-reactive protein is an inflammatory marker and directly damages the endothelium. Chronic inflammation, as evidenced by persistent high levels of C-reactive protein, not only creates initial injuries to the endothelium, but also accelerates the progression of existing atherosclerotic lesions.

In response to a large number of published studies, enlightened people are taking charge of the health of their arteries. They are eating better, exercising regularly, and undergoing regular blood testing to identify the specific drugs, hormones, and dietary supplements they need to reduce their atherosclerotic risk factors.

The News Media Can Endanger Your Arteries

At the age of 46, Bill Clinton became the third-youngest man to be elected president of the US. Clinton projected an energetic image and was seen as someone who took physical fitness seriously, despite his admittedly poor diet.

We do not want any of our members to become victims of news media hype about what may have caused Clinton's arteries to clog. We suspect the former president's heart problems were due to many of the atherogenic factors discussed in this editorial. Sad to say, most cardiologists are not even familiar with the multiple heart-attack risk factors that were long ago identified by the Life Extension Foundation.

In this issue of Life Extension, we examine the pros and cons of statin drugs and provide rational strategies for using these drugs if natural approaches fail. We know that many Life Extension members with elevated LDL or cholesterol levels refuse to take statin drugs because of concern about side effects. The good news is that a patented dietary supplement has been developed that has shown remarkable effects in reducing LDL and cholesterol without side effects. For members who have excess LDL or cholesterol, this new supplement could help lower these artery-clogging factors to safe levels.



For longer life,

A handwritten signature in black ink, appearing to read 'W Faloon'. The signature is fluid and cursive.

William Faloon

FDA Permits New Fish Oil Health Claim

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