Role of Epicardial Fat to Buffer Deformation and Vibration in Coronary Arteries

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ABSTRACT

OBJECTIVES: To examine the putative physiologic role of epicardial fat to buffer the coronary arteries and to review the data on deformation and vibration in coronary arteries.

METHODS: OvidSP Medline, Embase, and PubMed were systematically searched. Eligible articles on vibration in arteries and deformation of coronary arteries were assessed.

RESULTS: Coronary arteries are unique because they undergo substantial deformations with twisting, bending, and stretching that are due to cardiac contraction and the tethering of the large coronary arteries to the epicardial surface of the heart. In addition, phasic coronary artery pressure and blood flow are not synchronous producing a high negative stress phase angle between circumferential strain and wall shear. Fluid flow–induced vibrations, a universal finding in conduits transporting fluid with pulsatile flow, have been documented in arteries. Arterial vibration can damage the structure of arterial wall, especially elastin and endothelial cells, leading to alterations in the arterial function. Support for a beneficial mechanical role for epicardial fat is based on the data that wrapping material to the outside of conduits not only reduces the vibration but also decreases their movement. The overall impact of an external wrap is a reduction in the probability of conduit fatigue and failure. Characteristics of the artery, such as shear modulus, are a function of the properties of each layer of the artery. The application of epicardial fat to the adventitia of arteries alters the biophysical characteristics of the artery which is the sum of each layer including the epicardial fat. Vibration, resonance, and deformation energy are lost when they hit the surface of an absorbing material.

CONCLUSIONS: The integration of the biophysics of coronary arteries with knowledge of material damping principles supports a physiologic role for epicardial fat to buffer deformation and vibration in coronary arteries.

KEYWORDS: Epicardial fat, coronary arteries, arterial vibration, biophysics

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Introduction

Defining the existential question for epicardial fat, the adipose tissue on the surface of the heart, which is especially prominent around coronary arteries, leads to the proposal of a number of putative physiologic roles for epicardial fat.[1] These roles include homeostasis in the regulation of fatty acid metabolism for coronary arteries, thermogenesis, protection of the cardiac autonomic ganglia or nerves, as well as the production of hormones and adipokines.[1–3] Epicardial fat has genetic programs for production of substances such as adrenomedullin, adiponectin, and angiotensin-2 that have the potential to alter coronary artery vasoreactivity and possibly modulate the process of atherosclerosis.[4] A potentially very important proposed function of epicardial fat is "to buffer the coronary artery against the torsion induced by the arterial pulse wave and cardiac contraction or to offset rapid changes in the width of the blood vessels with arterial pulse."[1] Epicardial fat was proposed to also "limit the motion of the coronary arteries perhaps reducing the potential extremes of coronary artery velocity."[1] Although most of the (other) roles proposed for epicardial fat were based on biochemical or physiologic data, the mechanical role of buffering the coronary arteries was more speculative and the basic data to support this proposal were not presented. Since then, more data have accumulated to support the adverse effect of epicardial fat on the development of coronary atherosclerosis,[5,6] including the finding that removal of some epicardial fat may mitigate atherosclerosis development.[7] Thus, it has become more important to examine and even justify this potential physiologic role of epicardial fat in the protection of coronary arteries. The basis of the buffering effect of epicardial fat on coronary arteries will be examined based on the known mechanical properties and stresses of the coronary artery and the data on the benefits of a layer of material surrounding a flexible tube carrying pulsatile fluid which is analogous to a coronary artery.

Methods

OvidSP Medline, Embase, and PubMed were systematically searched. The search strategy was vibration or deformation AND coronary arteries or coronary vessels. The inclusion criteria were human studies and those in English. The exclusion criteria were studies on percutaneous coronary intervention or coronary stents and studies of vessels other than coronary arteries, eg, aorta, myocardial deformation rather than coronary artery deformation, and methods of study or modeling

approaches. The initial search identified 286 studies. Eligible articles on vibration in arteries and deformation of coronary arteries were assessed, and articles that met the inclusion and exclusion criteria were identified. The reference lists were scanned for other studies. A total of 24 studies were identified and used. Because of the paucity of data on vibration and coronary arteries, books on flow-induced vibration were consulted.

Results

Mechanical deformation of coronary arteries

During cardiac contraction and relaxation, there is considerable mechanical deformation of coronary arteries. Indeed, coronary arteries have been considered unique because they undergo substantial deformations with twisting, bending, and stretching that are due to cardiac contraction and the tethering of the large coronary arteries to the epicardial surface of the heart.[8] The cyclic change in coronary artery shape not only alters coronary artery curvature but also produces discrete flexion points.[9]

Another unique feature of coronary arteries is that phasic pressure and blood flow are not synchronous as the maximum pressure is in systole and most coronary blood flow occurs during diastole (although there are rare exceptions).[10–12] This unique relationship is responsible for a highly negative stress phase angle between coronary artery circumferential strain and wall shear.[13] The highly negative stress phase angle between circumferential strain and wall shear may play a role in localization of coronary atherosclerosis. Stein et al[14] showed that there is a significant correlation between the mechanical stresses due to cyclic flexion of the artery and the rate of progression of coronary atherosclerosis assessed by serial coronary angiography. They concluded that these mechanical stresses contribute to arterial tissue damage or fatigue leading to accelerated progression of atheromatous plaques.[14] Three-dimensional intravascular imaging of the human coronary artery identifies stress-strain patterns that represent, in part, the level of deformation in plaques.[15] The mechanical stresses of arterial torsion can be modified, to some extent, by the myocardial tissue on one side and epicardial fat on the other side which likely limit the positive remodeling of coronary arteries during the development of the atherosclerotic plaque.[16]

Coronary artery deformation due to myocardial contraction has a significant effect on wall shear rate patterns in the coronary arteries.[17] During cardiac contraction, the radius of curvature of the coronary artery decreases considerably near bifurcation points, and at its maximum, the radius of curvature decreases at a rate of 2.3% per millisecond.[18] The radius of curvature or the angle of cyclic flexion contributes to arterial stress. The geometry of coronary arteries can be characterized by their curvature, torsion, and tortuosity.[19] Arterial wall shear stress increases with an increase in artery curvature and torsion which may promote the development or acceleration of atherosclerosis in arteries.[20]

Vibrations originating from arteries

The capacity of arteries to resonate was apparently first recognized by the physiologist Otto Frank.[21,22] Lin Wang et al[23,24] have recently refocused attention on this topic and contend that one of the functions of the heart is to generate radial oscillatory motion or resonance or vibrations of the arterial system. Fluid flow–induced sound and vibrations are well recognized in physics and have been well characterized mathematically.[25-27] A basic physics principle is that a system of finite dimension will have its own natural frequencies. The arterial system will have natural frequencies dependent on its geometry, physical properties of the vessel wall, constituents of the blood flowing through it, and boundary conditions at junctions.[23] Vessels can also produce subharmonic and chaotic vibrations.[28] Cardiac contraction generates an input force into the arteries that can be decomposed by Fourier series analysis into many harmonic forces of different frequencies.[24] This is analogous to and supported by the observations that pulsatile fluid flow through pipes conveying fluid generate a time-dependent harmonic component superposed on a steady-state harmonic.[29]

The overall principle is that the magnitude of the vibration will be dependent on the vessel wall size, axial tension, vessel characteristics including size, as well as the composition of the fluid in the vessel.[26,29,30] Thus, the type and extent of coronary artery oscillatory displacement are dependent on these factors in coronary arteries which in turn are influenced by coronary artery anatomy. There are differences in diameter, length, and tortuosity between the right coronary artery and left anterior descending and circumflex coronary arteries. The resonance frequency will depend on the displacement (transverse or torsional), elasticity, and length of each of these coronary arteries.[31] Another factor is the presence, degree, and location of arterial stenosis that can excite vibrations with the specific frequencies dependent on the nature of the arterial wall.[32]

Arterial vibration can produce damage to and reductions in the function of the elastin component of the arterial wall.[33,34] Vibration can cause damage in vascular endothelial cells[35] and produce endothelial cell dysfunction.[36]

Reductions in the extent of deformation and vibration: the putative role for epicardial fat based on engineering principles

Attenuation of deformation and vibration has been the subject of intense investigation.[37,38] Damping is the phenomenon by which mechanical energy is dissipated in dynamic systems.[38] The basic principle is that vibration, resonance, and deformation energy are lost when they hit the surface of an absorbing material.[39] It is of special interest in pipes carrying fluid that are potentially harmful and is important to industries transporting fluid, such as oil from underwater sites or across land.[27] Vibration dampening materials and approaches have been developed over many years. It has been known for a long time

that the external application of a material wrapping round a pipe carrying fluid will reduce the vibrations of the pipe.[40] The damping properties of a composite wrap can reduce the sound pressures in the wall of the pipe, reducing the likelihood of fatigue failure.[40] The composition of the wrapping material has been studied and varied to improve the damping properties and includes materials such as carbon fiber–reinforced polymer composites and multiple fluidic flexible matrix composites.[41,42] Each material can be characterized by propagation constants—which identify the amount of wave that will be propagated by the material and an attenuation constant—indicating how much of the wave will be reduced as it travels through the insulating material.[39] Such wrapping decreases the acceleration in the wall of the pipe, reduces the amplification of the oscillations, and in turn decreases the vibrations in the pipe.[40]

Although epicardial fat might appear to be a flimsy wrapping material, adipose tissue has defined tensile strength and "toughness" which are due in large part to the extracellular matrix and collagen network surrounding the adipocytes.[43,44] Vibration dampening material, however, can be as seemingly flimsy as felt or as thin as tape. Vibration dampening materials have been the subject of many patents. The dampening of vibration consists of wrapping the conduit with material as simple as felt (US patent 12/040,870) or adhesive tape (US patent 3,217,832). Over the years, the wrapping material composition has become more complex so as to permit fluids under high-pressure impulses to be transported while minimizing the structural damage to the conduit (US patent 5381834).

The protective benefit of material surrounding a vessel can be explained from an arterial perspective. Lu et al[45] modeled the coronary artery as a two-layered structure—one with the intima and media consisting of vascular smooth muscles, endothelial cells, elastin, and some collagen and the other consisting of the adventitia containing collagen, fibroblasts, and elastin. They found that shear modulus after the application of a transverse force is greater for the adventitia than the intima/media layer, but the artery's biophysical properties are the sum of both layers.[45] Epicardial fat is contiguous with the adventitia of the coronary arteries.[1] The moment of inertia and shear modulus of a vessel is the sum of the products of polar moment of inertia and shear modulus of each layer. Thus, the stress-strain characteristics of the coronary artery will be the sum of all of its layers including the layer applied externally to it, namely, the epicardial fat.

This study suggests an explanation for the controversy surrounding the role of epicardial fat in coronary atherosclerosis. Small or "normal" amounts of epicardial fat can play the physiologic role to buffer the deformations and vibrations in the coronary artery that can accelerate atherosclerosis. In contrast, larger amounts of epicardial fat because of its capacity to secrete hormones and cytokines[2,4] may overcome the beneficial effects to mitigate arterial stress and instead lead to atherosclerosis.

Study limitations

There are no data that directly measured vibration, deformation, and wall stress in vivo in coronary arteries in the presence and absence of different amounts of epicardial fat.

Conclusions

In summary, coronary arteries undergo substantial deformations with twisting, bending, and stretching that are due to cardiac contraction and the tethering of the large coronary arteries to the epicardial surface of the heart. In addition, they are subject to fluid flow–induced arterial vibrations that can damage the structure of arterial wall, especially elastin and endothelial cells. The lessons from pipes and flexible tubes transporting pulsatile fluid are that applying material to the outside of the conduits will not only reduce the vibration but also decrease the movement of the conduit. The overall impact is a reduction in the probability of conduit fatigue and failure. The current epidemic of obesity which is translated into increases in epicardial fat[46] may transform a beneficial mechanical function of epicardial fat into a deleterious biochemical one that accelerates atherosclerosis.[5,6] Integration of the biophysics of coronary arteries with knowledge of material damping principles supports a physiologic role for epicardial fat to buffer deformation and vibration in coronary arteries.

Author Contributions

Simon Rabkin researched the literature and wrote the paper.

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