UNDERSTANDING CHEST PAIN

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In loving memory of Henry J. L. Marriott, M.D., F.A.C.C. (1917-2007), mentor, teacher, consultant, brilliant Cardiologist, terrific dancer, loyal friend, and at the end—courageous warrior.

Dr. Marriott once began one of his many lectures with this question: "How many of you have ever had chest pain?" All of us, right? At least that was the case when we first began intense CrossFit workouts. A muscle ache in the vicinity of the ribs or sternum would make you pause with: "What's that?" "Oh yea**—**those last two reps", a *superficial* muscle pain.

HEART PAIN

Chest pain is a whole lot different when it comes from a heart muscle deprived of oxygen. Such pain may or may not involve the chest per se and it even has a different name—"angina". According to reports from those who have experienced it, pain originating in oxygen starved heart muscle (myocardium) can be mild or severe, but it's a deeper more alarming type of pain than the chest muscle twinges or aches experienced by many of us days after or during exercise.

J. Willis Hurst M.D. once published descriptions of angina given by his patients. These colorful accounts reflect the common perception of the sensations felt behind the sternum (substernal), in the back, abdomen, shoulders, either or both arms, chest, or mouth that are described as tightness, heaviness, squeezing, strangling, aching, burning, a weight, numbness, pressure, a suffocating feeling, nausea or a feeling of "heartburn".

Here are some very expressive quotes from patients with angina:

- "A red hot poker"
- "A shoe box in my chest"
- "A toothache"
- "Hot flame in the upper part of my mouth"
- "An elephant on my chest"

- "Jaw pain"
- "Arthritis"
- "A bad feeling in the upper portion of my back"
- "Tracheitis"
- "Sternal whisper" [This one is hard to imagine!]
- "Dryness in my throat produced by effort or emotional stress"
- "Smoke in my chest" [This one too! Would only a smoker use this description?]
- "Someone choking me from behind"

One patient described different degrees of his pain like this:
Severe: "A large fish hook stuck under my jaw and hung up from a scaffold."
Moderate: A small fishhook caught in my lower jaw".
Mild: "A needle and thread being pulled through two lower teeth."

THE PROBLEM

Heart attacks *(myocardial infarctions)* are the result of a total occlusion in a coronary artery, depriving the heart muscle supplied by that artery and its distal (downstream) branches of its blood supply. The area immediately affected by the blocked artery is dead or dying. The muscle surrounding the dead area is injured--still living but not functioning in that it is unable to contract and balloons out with every contraction, and the area surrounding the injured muscle is weak and ischemic (oxygen-deprived).

GLOSSARY
Angina: Pain originating from the heart muscle
Athero-: A buildup of anti-inflammatory and marker cells along with some cholesterol within the walls of an artery
Coronary: Pertaining to the heart
Endothelium: A thin layer of cells lining blood vessels
Myocardial infarction (MI): A condition in which a localized area of myocardium is dying or dead because of insufficient blood supply.
Myocardium: Heart muscle
Sternum: Breast bone

THE CAUSE

Occlusion of a coronary artery is caused by the formation of a plaque in the lining of the artery (the endothelium). This plaque is called "ath-er-o-scler-osis". "Athero" comes from a Greek word meaning "a lump of hardened porridge". *Athersclerosis* is sometimes confused with *arterosclerosis*, which is another Greek word for hardening of the arteries (loss of elasticity) and is one of the causes of high blood pressure.

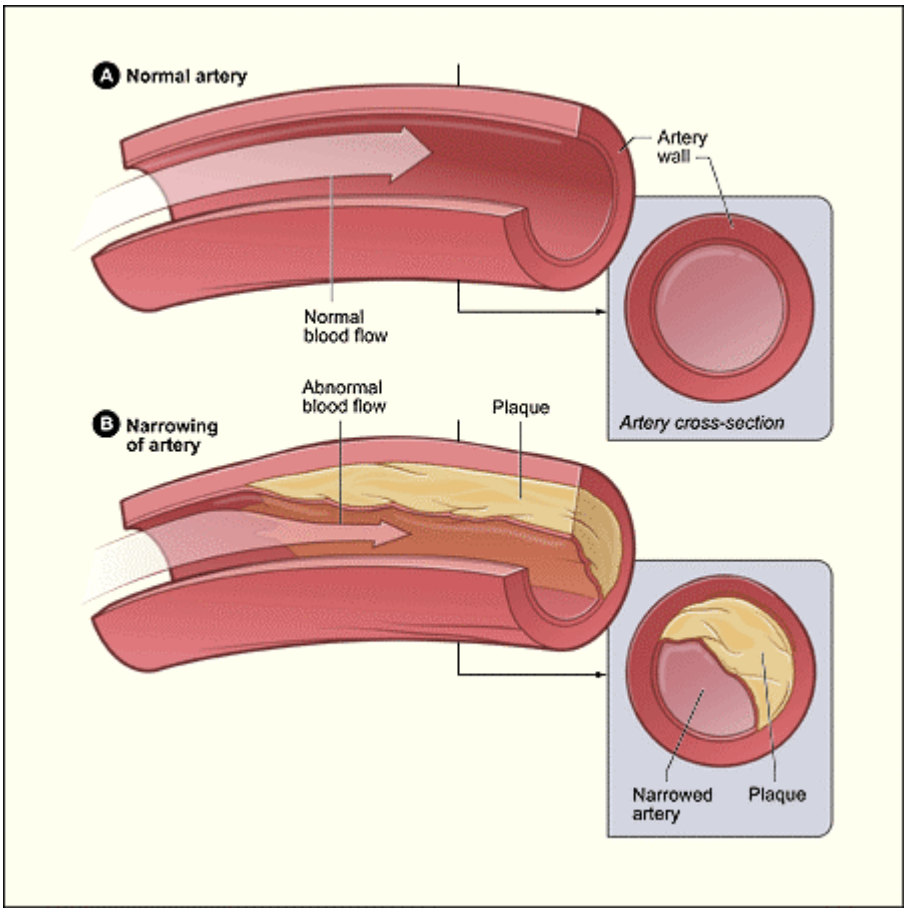
Atherosclerosis is caused by a chronic inflammatory response to an injury or irritation of the lining of the coronary arteries (endothelium). Major factors that result in the injury are high blood sugar,[1] [2] cholesterol, cigarette smoking (active and passive),[3] and some drugs. Another significant risk factor is food prepared in used cooking fat, discussed below under prevention.

PATHOPHYSIOLOGY

As you know, the normal protective response to inflammation anywhere in the body is the accumulation of white blood cells, so too in the walls of coronary arteries. However, in the heart the well-meaning white blood cells do more harm than good because they migrate across the inflamed cellular lining of the artery along with many types of cellular markers for inflammation. Additionally, inflammatory cells (monocytes) circulating in the blood stick to the surface of the plaque like Velcro.

In any other place in the body, such a reaction would be protective. In a coronary artery it becomes a threat, forming a potentially obstructive plaque within and on the endothelium. Thus, most of the plaque is not made up of cholesterol, as we always thought, but rather white blood cells and specific markers for inflammation, as well as cholesterol crystals.[4] [5] The figure below is a very diagrammatic representation of atherosclerosis—an artist's conception.

Atherosclerosis



WHERE DO THE LDLS AND HDLS COME IN?

Low-density lipoproteins (LDL)---are the bad guys in our cholesterol panel---but only if their level is too high. When the LDL blood level is low, LDL can easily pass in and out of the endothelium. However, high levels of LDL no longer have freedom of passage so that they accumulate within the arterial wall, causing more inflammatory reactions. This process is further abetted by low levels of those good guys---high-density lipoproteins (HDL), that would, in sufficient numbers, be up to the task of removing fats and cholesterol from the cells.[6] This is why we shoot for reference ranges of low LDL and high HDL.

Once collected and trapped within the vascular lining, cholesterol crystallizes and expands, tearing and perforating fibrous tissues. Thus, it is thought to have a role in the rupture of the plaque.[7] As you will see, plaque rupture is a critical event in unstable angina.

THREE TYPES OF ANGINA

The three types of angina are stable, unstable, and variant (Prinzmetal's angina), all of which are caused by severe narrowing or occlusion of a coronary artery that results in loss of oxygen to the section of myocardium supplied by the occluded artery. The difference among these three types of angina lies in the mechanism (pathology) of the occlusion.

Stable Angina

Stable angina is precipitated by activity (running, walking, rowing, intense CrossFit workout). Symptoms abate with rest and resume with activity. Usually, such a person is already under a physician's care and has a definitive diagnosis after cardiac catheterization.

Pathology. In stable angina, a growth of atherosclerotic deposits protected by a fibrous cap develops within the wall of the blood vessel, but does not completely occlude blood flow. Anginal attacks can be precipitated by temporary constriction of the already narrowed, but *stable*, blood vessel. The pain experienced has pretty much the same character each time. There is danger that stable angina will become unstable without warning, so it goes without saying that it is too late to start jumping into an intense exercise routine.

Unstable Angina

Unstable angina is an acute coronary syndrome—a medical emergency in which time is of the essence. It is a critical prophecy of an imminent heart attack. Chest pain can sometimes be difficult to categorize. However, in patients with unstable angina there are reliable signs, called Wellens syndrome, when the stenosis is in the proximal left anterior descending coronary artery. I've written a special section at the end of this article for Emergency Dept and Critical Care Physicians and Nurses.

In the U.S. there are at least 1 million hospitalized patients each year admitted with a diagnosis of unstable angina. There are many more who never reach the ER.

Any one of the symptoms listed below is a serious indicator of unstable angina and an impending heart attack, requiring urgent medical attention.

1) Angina that occurs unpredictably at rest (or with minimal exertion), usually lasting >10 min
2) Severe and new onset angina (i.e., within the prior 4–6 weeks)
3) Angina that occurs with a crescendo pattern, i.e., builds gradually, plateaus, and subsides gradually.

Pathology. Other than the symptoms, it is the pathology that differentiates unstable angina from stable angina. Understanding the events that occur within the blood vessel helps one to understand the reason for the symptoms and to remember them.

For a moment, visualize a case of *stable angina* with plaque covered by a protective cap within the wall of a coronary artery. Suddenly, the cap ruptures, allowing blood clots to gather on the disturbed surface of the rupture and block blood flow. This catastrophic event is called *unstable angina*. The sequence of events leading up to total occlusion clearly explains why the unexpected onset of pain may be independent of activity, sudden in appearance, and crescendo in nature, becoming severe as the clot builds and gradually subsiding as the clot shrinks.

An understanding of this mechanism, leaves no doubt that such pain requires immediate medical attention—the occlusion is developing and moving toward total blockage of the blood vessel and a myocardial infarction. It is only a matter of time—seconds, minutes, hours, days, weeks. There is no way of knowing. Total occlusion has been known to happen during transport from the ER to the cardiac catheterization lab.

EMERGENCY RESPONSE TO UNSTABLE ANGINA

- Call 911 if the pain is new to the person, severe, crescendo in character, or lasting more than 10 minutes—all signs of unstable angina, a true emergency.
- Call 911 even if you are uncertain about the symptoms.
- Reassure, but be firm in your opinion that the symptoms require emergency medical attention.
- If this unexpectedly occurs during a workout, do not allow the person to continue with the workout nor to drive home.

PRINZMETAL'S ANGINA (VARIANT ANGINA)

Prinzmetal's angina refers to chest pain caused by a decrease in blood flow because of spontaneous spasm of a coronary artery without significant atherosclerotic plaques. These symptoms happen at rest and often may awaken the person during the night. Obviously, if the mechanism of the angina has been misdiagnosed and there is actually a plaque, that

plaque could rupture and result in total occlusion of the vessel. Therefore, a definitive diagnosis is of great importance.

RECOVERY

It has been proposed that the positive effects of aerobic training on cardiac structure during rehabilitation are neuro-hormonal and that they improve microcirculation within the heart muscle and functional endothelial integrity. This of course results in an enhancement of the blood supply to the injured myocardium and faster healing.

In preconditioned hearts, i.e. hearts that were exercised regularly prior to the coronary occlusion, there are fewer negative effects, in that the damage (infarct) is smaller, arterial inflammation less, hospital stay shorter and the myocardium returns to and retains a strong structure (a.k.a. remodeling).

PREVENTION

Exercise your hearts! At CrossFit we are exercising our hearts every day, creating a sturdy microcirculation and improving the function of our heart muscle—grossly and at the cellular level. You can think of your workouts as a **preconditioning** for your hearts for the rest of your life.[8 9 10 11]

Red wine. Current evidence supports a contribution of red wine to the prevention of cardiovascular disease. [This has been known for years, but it's nice to be reminded of something good.]

Cooking with canola oil and vegetable oils. It is no secret that restaurants use and reuse their big vats of vegetable oil for frying. These oils are unstable when heated. The heat renders the products of this degradation more damaging to the lining of blood vessels. Since endothelial (cell lining) dysfunction is an important early step in the development of atherosclerosis, these components could further increase the formation of plaques.[12] [13]Obviously, it is not only the deep fried foods in "Fast Food" restaurants that cause endothelial damage, but in any restaurant or even at home. In a 2006 study it was confirmed that there is no difference in the acute adverse effect of the ingestion of different oils on the endothelial function. All the oils, either fresh or deep-fried, produced an increase in the triglyceride plasma levels in 10 healthy young volunteers. [14]

Cigarettes. Cigarette smoking is the most important modifiable (potentially reversible) risk factor for atherosclerosis and is associated with endothelial damage in the arteries of otherwise healthy young adults. [15] Avoid second hand smoke.[16] I don't think any of you actually smoke!

Check your BP. As you age, check your blood pressure occasionally and see that it stays within normal limits, with a systolic BP (the top number) at least below 140 mm Hg. Hypertension is preventable with life-style changes or medications.

LDL down; HDL up. Beat your lipid panel into submission. You know how.

Take good care of your cells. The walls of our blood vessels are lined with living cells that constantly interact with each other, the fluid that surrounds them, and the blood that flows over them. SO, let's take care of those cells; feed them well and provide them with a gusty flow of blood--you lucky CrossFitters.

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