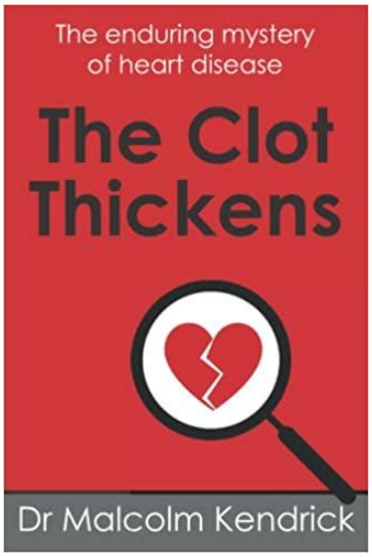
"It's All About Blood Clotting, Not Cholesterol"
a review by Dick Burkhart of

The Clot Thickens - The Enduring Mystery of Heart Disease

By Dr. Malcom Kendrick (2021)



The Scottish cardiologist Malcom Kendrick is back with his trademark wit, and cut-to-the chase erudition, explaining to us in plain English what all that medical jargon actually tells us about heart disease. It's about our arteries and how they get abused in a plethora of ways, leading to strokes, heart attacks, internal bleeding, and worse.

It's all about damage to the artery walls, leading to rapid blood clotting (like a scab) to cover the damage, followed by the growth of a new layer of the outer artery wall (endothelium) to cover the clot while specialized cells break down and dispose of the clot, leaving a calcified remnant. After repeated cycles of damage, clotting, new growth, and disposal, the result is a multi-layered plaque that narrows the artery and may eventually lead to blockage.

Cholesterol only makes a direct appearance as sharp crystals in certain plaques, transported there by red blood cells, not by so-called "bad cholesterol" (transported by LDL = low density lipoprotein). However there is a variation on LDL, called Lipoprotein(a), or Lp(a), which is often found in plaque and mistaken for LDL. It turns out that Lp(a), unlike LDL does not have the purpose of transporting cholesterol in the blood (to cells with LDL receptors).

Instead Lp(a) is related to Vitamin C and to blood clotting. A critical role of Vitamin C is to facilitate the construction of collagen, which strengthens cell walls. Without Vitamin C you get scurvy and may die due to leakage from weakened blood vessels. Except that Lp(a) rushes to the rescue, acting like Vitamin C, by keeping the fibers strong in the blood clot covering the damage, making a tougher layer of protective plaque.

The problem comes when lack of Vitamin C is not the issue – when other things are damaging the artery walls and Lp(a) ends up contributing to a tough multilayered plaque that narrows the artery. Kendrick's conclusion: The problem is that Lp(a) is sometimes drawn to clots even when not needed. LDL is only a minor

problem, and it turns out that LDL lowering drugs like statins have very little effect on Lp(a). This constitutes a huge threat to the trillion dollar statin industry.

The rest of the book digs deeper into the bio-chemical structure of artery walls and how this structure is damaged by cortisol and a host of other things. Cortisol is a steroid hormone that rapidly mobilizes energy resources in response to stress or strain, such as in the "fight or flight" response. Excess energy is stored as body fat, especially as "beer belly". Too much cortisol over longer periods of time leads to insulin resistance, or failure, with insulin being in charge energy storage, such as converting excess glucose into fatty acids.

But the key problem with cortisol is that it is toxic to the outer artery wall – the endothelium and its covering by a cilia-like forest of thin and slippery fibers – the glycocalyx, while also stimulating the production of clotting factors. Cortisol is also one of the "most powerful immunosuppressants known". A consequence is that when cortisol or similar steroids are prescribed to combat autoimmune diseases, cardiovascular disease is a predictable consequence.

Thus mental illness produces heart disease via the production of cortisol in response to the strains of depression and anxiety. That is, it's not just things like smoking and diabetes. Diabetes is another big one in that, like cortisol, it damages the artery wall. But it also reduces the synthesis of nitric oxide, which relaxes and dilates blood vessels, keeping them much healthier. In fact, nitric oxide is often cited as an important factor in cardiovascular health and is recommended as a supplement, along with many vitamins, good exercise, a low carb diet, and plenty of sun and social life.

Kendrick also has a chart (p 256) of which nasty things you need to fix first, according to how much they tend to reduce your life expectancy. That is, this book is written for all with an interest in medical science, especially out-of-date doctors and curious patients. Yet it has practical advice, based on real evidence and understanding, not fads or speculations, for everyone with intimations of mortality.