

Coronary Artery Disease as Clogged Pipes A Misconceptual Model

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A recent advertisement on the back cover of a special health issue of the *New York Times Magazine* section read “Ironical that a plumber came to us to help him remove a clog.”¹ The ad referred to doctors in the cardiac catheterization laboratory as “one kind of pipe specialist,” and noted that the patient in the ad returned to work “just 2 days after having his own pipes cleaned out.” Although the image of coronary arteries as kitchen pipes clogged with fat is simple, familiar, and evocative, it is also wrong.

Conceptual models help us to make sense of a complex world. In both research and clinical practice, our conceptual models inform our decisions about which outcomes to measure, which confounders to adjust for, and which patients are likely to benefit from a particular treatment. For example, the model of cervical cancer as the consequence of infection with certain strains of human papilloma virus has led to the development of a vaccine to prevent cancer. However, when a conceptual model is flawed, as in the case of the clogged pipes analogy, we are supposed to discard it and replace it with a more accurate one. In *Structure of Scientific Revolutions*, Kuhn observed that such paradigm shifts do not occur easily.² Instead, scientists cling tenaciously to the old, accepted model, despite its inability to explain observed phenomena. In medicine, too, misconceptions embedded in an outdated model may linger for years, influencing physicians’ practice and patients’ expectations. Simple models, especially those we have worked for years to get the public to understand, may be the hardest to abandon.

Misconception Number 1: Arterial Narrowing Is the Problem

The clogged pipe analogy of stable coronary heart disease has been particularly difficult to dislodge. This representation had its origins in the 1970s, when researchers observed that the degree of coronary obstruction correlated with subsequent risk of myocardial infarction.³ According to this model, cholesterol plaques in the arterial walls slowly encroach on the lumen, causing silent ischemia first, then angina, and eventually infarction. Diagnosis begins with physiologic stress testing, looking for supply–demand mismatch, and progresses to angiography to find blockages. Treatments based on this theory include both coronary bypass and angioplasty, the latter often explained to patients as a Roto-Rooter or in the case of

the magazine ad, as a Rotablator. Results of such revascularization procedures are visually striking and, in stable disease, may lead to the erroneous conclusion that the plumbing problem has been fixed and the risk of myocardial infarction ameliorated. Indeed, early proponents of coronary artery bypass were so convinced by the angiographic evidence of success that they questioned the need to conduct randomized trials to assess the impact on myocardial infarction or mortality.⁴

Four decades later, we know that the interactions between dietary fat, serum cholesterol, and arterial endothelium are complex and dynamic.⁵ Although high-grade stenoses can cause chronic angina, most cardiac events occur at lesions that appeared mild on previous angiography.⁶ These plaques contain a lipid-rich core covered by a thin fibromatous cap. Inflammatory cells (eg, macrophages and mast cells) within the plaque may become activated by microbes, autoantigens, or inflammatory molecules (activated plaque model). The activated cells secrete cytokines and proteases that weaken the fibrous cap, causing it to erode or rupture. The newly exposed subendothelium and procoagulant factors precipitate platelet aggregation and local thrombus formation, sometimes leading to infarction. Before rupture, these plaques often do not limit flow and may be invisible to angiography and stress tests. They are therefore not amenable to percutaneous coronary intervention (PCI).

To add to the confusion, in the setting of myocardial infarction, the model of an obstructed pipe is accurate, and interventions aimed at eliminating the thrombus, either thrombolytics or angioplasty, can be lifesaving. But for patients with stable disease, local interventions can only relieve symptoms; they cannot prevent future myocardial infarctions. Indeed, at least 12 randomized trials conducted between 1987 and 2007 and involving >5000 patients have found no reduction in myocardial infarction attributable to angioplasty in any of its forms.^{7,8} Despite this overwhelming evidence, the plumbing model, complete with blockages that can be fixed, continues to be used to explain stable coronary disease to patients, who understandably assume that PCI will prevent heart attacks.⁹ Cardiologists also cling to the belief that for patients with stable coronary disease, an open artery is beneficial,¹⁰ and the approach to stable coronary artery disease continues to be a search for ischemia. Not surprisingly, a substantial minority of cardiologists also believe that elective angioplasty and stenting can prevent heart attacks.⁹ These

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beliefs translate into practice: As many as half of all elective PCIs may be inappropriate.¹¹

Misconception Number 2: Fat Clogs Arteries

The plumbing model—in which dietary fat or cholesterol is slowly deposited in arterial walls, leading to blockages—also perpetuates misconceptions about fat consumption. Although atheromatous plaques contain lipids, they are not composed of fat directly from the diet. Low-density lipoprotein is produced primarily in the liver and may infiltrate vascular endothelium, where it can initiate a complex inflammatory response, especially at sites of hemodynamic strain.⁵ This inflammatory response can lead to arterial remodeling, in which plaque growth within the vessel walls is accommodated by outward enlargement of the vessel. In that case, large plaques may not encroach on the lumen and are therefore hidden from angiography. These plaques are particularly dangerous both because they are prone to rupture and because before rupture they do not limit flow and therefore do not induce formation of protective collaterals. High-density lipoprotein removes low-density lipoprotein from peripheral tissues through reverse cholesterol transport to the liver and may have antiinflammatory properties.¹² Although saturated fat increases low-density lipoprotein cholesterol, it also increases high-density lipoprotein, so the net effect on cardiac risk is neutral.

The American Heart Association's (AHA's) early diet recommendations reflected beliefs about fat in the 1980s. They recommended limiting total dietary fat to 30% of calories and saturated fat to 10% on the basis of the caloric density of fat and the association of saturated fat with coronary heart disease across countries.¹³ The interpretation of this evidence was selective and ignored the low rates of heart disease in Crete and among the Inuit, as well as the notorious French Paradox, wherein the French were noted to consume a diet high in both fat and saturated fat yet had low levels of heart disease.^{14,15} From these recommendations, low-fat diets became synonymous with heart-healthy diets and gave birth to a generation of low-fat, high-sugar substitutes.

More recent observational studies do not support the use of low-fat diets. In 1997, the Nurses' Health Study demonstrated that after proper adjustment neither total fat consumption nor saturated fat was associated with heart disease and that polyunsaturated fat was actually protective.¹⁶ Subsequent studies have also found no link between saturated fat and heart disease.^{17,18} In keeping with the new evidence, the AHA changed its diet recommendations, eliminating restrictions on total fat and acknowledging that low-fat diets had adverse effects on high-density lipoprotein cholesterol.¹⁹ But for patients with elevated low-density lipoprotein, they have tightened restrictions on saturated fats and now recommend that consumption not exceed 7% of total calories. Although it may be argued that reducing saturated fat will increase consumption of polyunsaturated fat, in many products, fat is simply replaced by sugar. More recently, the AHA recommended that people limit their intake of sugar, which now appears to contribute to obesity, hypertension, and subsequently coronary heart disease.²⁰ However, patients and many doctors have not gotten this message. The AHA's heart-healthy label still appears on a number of low-fat, high-sugar foods, including fruit juices and sugary cereals,²¹

whereas patients continue to believe that dietary fat, especially the saturated fat found in cheese and bacon, is the cause of heart disease. A recent survey revealed that 73% of patients believed eating fried food increased the risk of heart attack, the same proportion that believed smoking did.²² A Google search of "artery-clogging saturated fat" returns 195 000 hits, many offering advice about a healthy diet.

Promoting Evidence to Doctors and Patients

How do we end the misconceptions? First, institutions like the AHA should be more proactive in educating doctors about the inflammatory disease model and how to communicate it to patients. The 2011 American College of Cardiology Foundation/AHA/Society for Cardiovascular Angiography and Interventions guideline for PCI²³ is a step in the right direction, stating that patients considering PCI "should understand when the procedure is being performed in an attempt to improve symptoms, survival, or both." The guideline points out that "evaluation of 61 trials of PCI conducted over several decades shows that despite improvements in PCI technology and pharmacotherapy, PCI has not been demonstrated to reduce the risk of death or [myocardial infarction] in patients without recent [acute coronary syndrome]" but makes no mention of pathophysiology. Few medical questions have engendered 61 randomized trials, yet the authors of the guideline still rate the Level of Evidence as "B" (limited populations evaluated or data derived from a single randomized trial or nonrandomized studies). One explanation for this persistent skepticism is that clinicians and investigators, working from an outdated conceptual model, have mistakenly focused on improving the technology for keeping open flow-limiting lesions, believing that better stents would eventually yield a mortality benefit in stable disease. The inflammatory disease model makes clear that such attempts are doomed to fail because vulnerable plaques cannot be identified or stented before rupture. Although some will argue that myocardial infarction can also occur at the site of flow-limiting stenoses, mathematical models suggest that these lesions contribute relatively little to modifiable risk.²⁴ This is demonstrated empirically by the lack of survival advantage when bare metal stents are compared with balloon angioplasty or drug-eluting stents are compared with bare metal ones. Nevertheless, additional trials comparing PCI plus optimal medical therapy with optimal medical therapy alone continue. For example, Fractional Flow Reserve Versus Angiography for Multivessel Evaluation 2 (FAME-2) recently compared these 2 therapies in patients with functionally significant stenoses, as measured by fractional flow reserve.²⁵ Again, no statistically significant difference was observed between the study arms in terms of myocardial infarction or mortality. The trial was stopped prematurely because of a reduction in urgent revascularization among patients who underwent PCI, proving that performing PCI in all patients up front eliminates the need to perform PCI in 1 of 6 patients later. In contrast, 3-vessel coronary artery bypass graft surgery, which provides flow around all plaques, has been shown to decrease mortality, as the inflammatory model predicts. Placing the guideline evidence in the context of a conceptual model might offer a way for clinicians to understand and believe the results.

In addition, hospitals and doctors should stop using the old plumbing analogy in advertisements, Web sites, and educational material and when obtaining informed consent for PCI. Clearly, the current consent process in regard to PCI for stable angina is deeply flawed because most patients do not correctly understand the benefits of the procedure they are about to undergo, and many do not have angina.^{9,26} How then might this new model be communicated simply to patients? Doctors could begin by explaining that coronary artery disease, whether diagnosed by angiography or stress testing, is an inflammatory disease in which cholesterol from the blood is deposited in artery walls, causing an inflammatory reaction, like a pimple. When those pimples pop, they cause the blood in the arteries to clot at the site. If the clot closes off the entire artery, that causes a heart attack, and emergent medical attention is required to remove the clot. Thus, for patients who have coronary disease, it is crucial to take steps to reduce the inflammation, including both evidence-based lifestyle changes (smoking cessation, exercise,²⁷ stress reduction,²⁸ and a Mediterranean diet)^{29,30} and taking medications that reduce inflammation and prevent thrombosis (aspirin and statins).³¹ Doctors should state plainly that for preventing heart attacks, these are the only effective measures. If patients have ischemic symptoms (many do not), then they can be told that old plaques, liked scarred old pimples, may partially obstruct arteries and cause symptoms and that these symptoms can be relieved with medications. If the symptoms persist despite maximal medical therapy, patients could be offered PCI to relieve those symptoms.

These steps are likely to encounter opposition, partly because it is difficult to admit that in the past we got it wrong and performed what now appear to have been unnecessary procedures, but also because our current payment system continues to reward interventions based on the old model and cardiac procedures are an important source of hospital revenue. It is unlikely that hospitals will begin to advertise the power of generic medications and lifestyle changes to combat heart disease. Nor will physicians quickly abandon a practice that both supports their income and seems to make sense. Appropriate use criteria, especially if tied to reimbursement, could provide an important impetus to change,³² but only if payers, including the government, are willing to implement them. In the current environment, insurers are unlikely to enforce criteria that prominent interventionalists oppose as not being evidence-based or patient-centric criteria.³³ Perhaps in the coming world of payment reform, physicians within accountable care organizations may choose to adopt appropriateness criteria as a means of keeping their own costs low. In that case, better models should be embraced as a means to more patient-centered, cost-effective care.

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