Have you ever gone fly-fishing for trout and all you caught were carp? It is a frustrating experience to be sure. Often, the river or lake looked like “fish country” and the conditions and approach made sense, but usually the problem was simple. You were fishing in the wrong place.

This simple metaphor reminds us of the journey we have experienced during the past 25 years with evaluating and treating coronary artery disease in patients undergoing noncardiac surgery. We began the journey by clearly enunciating that heart attacks were a major cause of important and sometimes fatal postoperative outcomes. We derived a number of epidemiological and Bayesian methods for identifying patients most likely to harbor underlying coronary artery disease. We then identified and confirmed that a number of noninvasive techniques could be used to further stratify coronary risk in several clinical risk cohorts, potentially allowing a rational approach to screening patients into low-, moderate-, and high-risk subsets. The water looked right, our approach looked right, so we fished. It made eminent sense that by revascularizing blocked or narrowed coronary arteries, we could reduce the risk of postoperative heart attacks and coronary death. “We were fishing for TROUT (Treatment to Reduce Outcomes in Noncardiac Surgery) but we caught a CARP (Coronary Artery Revascularization Project)! We were fishing in the wrong place.”

In this issue of Circulation: Cardiovascular Quality and Outcomes, Garcia et al provide us with an important secondary analysis of the data from the CARP trial. They convincingly and unequivocally show us that among stable coronary patients having vascular surgery while on excellent medical therapy, coronary artery revascularization has no impact in lowering either perioperative or long-term risk of cardiovascular death or myocardial infarction, regardless of their clinical risk status. We were fishing in the wrong place.

Why doesn’t coronary artery revascularization reduce the risk of perioperative myocardial infarction and death? We believe that there are a number of reasons, many of which were learned through countless clinical trials of patients with both stable and unstable coronary syndromes. First, most heart attacks are caused not by severely stenosed epicardial plaques (the kind one might “fix” before noncardiac surgery) but by disruption or rupture of milder, heavily lipid laden, “vulnerable” plaques that may look remarkably innocent on angiography. When we studied the coronary pathology of patients experiencing a fatal myocardial infarction after noncardiac surgery, we found that the most severe coronary stenosis was usually not the culprit lesion. Had the patient’s physicians offered coronary angioplasty of the most severe coronary stenosis preoperatively, they would have missed the culprit lesion more than half the time! We were fishing in the wrong place. We still do not know how to reliably identify apparently stable but vulnerable plaques.

Second, we have discovered that our gold standard for identifying the severity of coronary artery disease and for planning its therapy is not gold. As studies of coronary flow, vessel remodeling, and coronary flow reserve have come to light, we increasingly recognize that the visual images we see on coronary angiography provide us just one angle of repose on a multifaceted pathophysiology. Coronary arteries can be diffusely diseased and yet “look” remarkably normal on an angiographic image. Coronary disease is much more complex than just epicardial plaque burden and morphologies. Coronary bypass surgery and coronary artery stenting do not prevent thrombosis, vasospasm, decrease in dynamic coronary flow reserve, small-vessel disease, or impossible myocardial demand. We were fishing in the wrong place.

Third, we have increasingly come to know that prophylactic coronary revascularization has a dark side. Let us illustrate the point this way. If you wanted to create a human model for an acute coronary syndrome, how would you do it? First, you would identify patients with stable, easily accessed, segmental coronary plaques. Second, with a balloon or perhaps a small scalpel, you would disrupt the endothelial integrity of the vessel. Then, within days, you would place such patients in a stressful environment. Perhaps you would create a catecholamine overload, raising the heart rate and blood pressure with altered shear stress. Maybe you would create a prothrombotic state. Or, perhaps you would promote inflammation by injecting cytokines or challenge patients with massive infusions of crystalloid. Don’t all of these perturbations happen to some degree when we revascularize coronary artery disease and then proceed to noncardiac surgery? All coronary revascularization procedures carry with them a risk of perioperative myocardial infarction and possibly death. Thus, even if there is some downstream benefit to stenting or...
bypassing the most severe stenoses in a patient, it may be offset by the risk of our “risk-reducing procedure!”

So, where should we fish? The guidelines tell us to identify coronary disease through a careful history, physical examination, and, when appropriate, a selected noninvasive test. CARP tells us that with best medical and preventive therapy—appropriate β-blockade, aspirin as appropriate, and statins both preoperatively and postoperatively—outcomes will be as good as they would be if we also provided prophylactic coronary revascularization. For patients who develop postoperative myocardial ischemia, intensified medical treatment and, when necessary, coronary intervention is usually effective in treating a problem that occurred despite our best medical therapy, not because we perturbed a previously stable situation.

Charcot said that diseases do not change, but we do, as we recognize what was formerly imperceptible. It is time to stop fishing for CARP!

Disclosures
None.

References


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We Were Fishing for TROUT and We Caught a CARP: Musings on Perioperative Management in an Age of Enlightenment
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