Before there was postoperative cognitive dysfunction (POCD), there was “pump head.” The latter is a syndrome of cognitive decline after cardiac surgery that is well recognized by clinicians and feared by patients. There is no doubt it happens. A landmark study 15 yr ago showed that over 40% of patients were cognitively impaired 5 yr after coronary artery bypass graft (CABG) surgery, results subsequently confirmed by others. Pump head is analogous to POCD; it is not dementia, but naturally there is concern one may lead to, or predict, the other. This is where it gets messy. Patients needing CABG surgery have cardiovascular disease, and cardiovascular disease is a well-established risk factor for cognitive impairment and dementia irrespective of surgery. In fact, patients with coronary artery disease managed medically or with a percutaneous procedure have the same prevalence of cognitive decline 6 yr later as those who have on-pump CABG surgery. This suggests that “pump head” is likely a misnomer and that preexisting cardiovascular disease is the main driver of cognitive decline in these patients. But does CABG surgery push patients slowly over the edge to full-blown dementia? That’s the important question addressed by Evered et al. in this issue of Anesthesiology.

Evered et al. asked whether CABG surgery is associated with the development of POCD and dementia, and whether the former is associated with the latter. They recruited patients at a 12-month follow-up appointment from a study designed and powered to evaluate differences in POCD between high- and low-dose fentanyl for cardiac surgery and followed them for another 6.5 yr. They classified preexisting cognitive impairment (PreCI), POCD by a standard test battery, and dementia by the clinical dementia rating scale and review of all cognitive and functional data. The main result paints a relatively bleak picture of long-term cognitive outcome among CABG surgery patients: about one in three patients were demented (30.8%) or had POCD (32.8%) 7.5 yr after surgery. However, while POCD was associated with higher mortality, a result that confirms studies involving noncardiac surgery, it did not predict dementia. In fact, of the subset (N = 113) evaluated for both, only 44% with dementia satisfied criteria for POCD. What did predict dementia was nonsurgery/anaesthesia factors, specifically, peripheral vascular disease and PreCI. Thus, the work of Evered et al. suggests that disease burden and the state of cognition before surgery, not the surgery itself, are the main drivers of cognitive outcome after.

This is a significant contribution because there are no other prospective studies of cognitive outcomes of surgery and anaesthesia that can boast of such long-term follow-up. Still, the results should be interpreted cautiously. First, the control group did not have cardiovascular disease or surgery, and there was no control for coronary artery disease managed without CABG surgery. Consequently, one cannot resolve whether the POCD (and mortality) was related to the surgery or the vascular disease. Also, as noted by the authors, lack of a baseline dementia evaluation makes it difficult to attribute the higher prevalence of dementia in the study group to vascular disease versus surgery. Previous studies indicate that cardiac surgery does not accelerate cognitive decline, but none have used formally diagnosed dementia as the outcome. Second, only 35% of patients from the
original study had follow-up for both POCD and dementia, meaning outcomes were not available for the majority and analyses were based on relatively small numbers in some of the categories. This is reflected in the wide CIs for PreCI and peripheral vascular disease as predictors of dementia, suggesting low validity and reproducibility of the univariable model. The absence of a multivariable analysis is another limitation, as it may have led to different conclusions. Finally, the loss to follow-up is an issue because, in studies of cognition, subjects who fail to complete testing are often those most likely to be impaired. As such, the study of Evered et al. may underestimate the prevalence of POCD, dementia, and the relationship between the two after CABG.

That said, the work of Evered et al. conveys a vital message: preoperative factors, especially cardiovascular disease and baseline cognition, may have more to do with postoperative cognitive outcome than anesthetic or surgical factors. That is not to say anesthesia and surgery are irrelevant, as some studies suggest otherwise,7,9 but rather that the impact of these and other perioperative events on cognition may vary widely depending on the preexisting state of the patient. We already know that preoperative coronary artery, pulmonary, and renal diseases influence postoperative outcomes, but much of the clinical research to date on postoperative cognition, and especially POCD in noncardiac surgery, has grouped patients by age and procedure but little else. As a result, study cohorts might unknowingly be quite heterogeneous with respect to risk variables, making associations between patient factors—anesthesia/surgery variables—and postoperative cognitive decline all the more obscure. Going forward, studies of postoperative cognitive decline should control for preoperative vascular disease, not just after the fact in the data analysis but prospectively by matching groups—including nonsurgical controls—for the presence and severity of such disease. Evered et al.'s observation that PreCI predicts post-CABG dementia is not surprising as, like vascular disease, cognitive impairment is a risk factor for dementia irrespective of surgery. It is also consistent with data from a recent study of twins that demonstrated preoperative cognition is more important than anesthesia or surgery in determining postoperative cognitive function.10 So, as we have argued previously,11 maybe it is time to institute routine preoperative cognitive screening for certain risk groups. Even among elective nonsurgical cancer patients, about 30% of those over 65 yr of age screen positive.12 Whether a brief cognitive screen can provide the information necessary to predict postoperative cognitive complications or outcome, let alone equip us to do something about it, remains to be seen, but the work of Evered et al. suggests it is worth trying.

The study of Evered et al. adds to a growing literature that reframes and shifts the discussion about causation of postoperative cognitive decline from intra- and postoperative variables to the preoperative state of the patient. Although preexisting conditions are unlikely to be the whole story,3-15 this shift is overdue and welcome, for chief among the many things we do not know about cognitive outcomes of surgery is the influence of the patient.

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Competing Interests

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Correspondence

Address correspondence to Dr. Culley: dculley@partners.org; Dr. Crosby: gjcrosby@partners.org

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Phlegmatic Temperamental Education for 1893 Classes by “Masters of Anaesthesia”

Flemish artist Maerten de Vos’ watercraft-seated Phlegmaticus (1583) reminds us that a patient of phlegmatic temperament flows with the cold, wet “phlegm” humor linked by ancient Greeks with elemental water. By 1893 at Chicago’s Post-Graduate School of Anaesthesia (PGSA), professors were tutoring future “Master of the Science of Anaesthesia” candidates to anticipate that a phlegmatic patient’s anesthesia might comprise—what today’s anesthesiologists would characterize as—(1) a swift induction, (2) a risk for heart-depressing anesthetic overdosage (termed “concussion” by the PGSA), and (3) a smooth emergence. PGSA Professor James M. Clyde, D.D.S., M.S.A., taught that phlegmatic patients might not only resist preanesthetic calming but also be vulnerable to an anesthetically “over-charged atmosphere … [which could] paralyze the medulla and cause death” from cardiac “concussion.”

George S. Bause, M.D., M.P.H., Honorary Curator, ASA’s Wood Library-Museum of Anaesthesiology, Schaumburg, Illinois, and Clinical Associate Professor, Case Western Reserve University, Cleveland, Ohio. UJYC@aol.com.