

Perioperative stress testing: The new high risk paradigm

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INTRODUCTION

For sometime, anesthesiologists have been asked to assist in the assignment of risk for an operative procedure, especially the patient with cardiac disease. For most of these patients, the preoperative assessment involves a decision about getting a stress test and dealing with the results. But there is little data to support this practice, and recent events suggesting that mechanical coronary artery intervention actually increases perioperative risk of MI and death have muddied the perioperative water.

This practice of risk assessment for the surgical patient appears to have been «codified» with the New York Society of Anesthesiologists physical status scale first published by Saklad⁽¹⁾.

As anesthesia and anesthetic techniques have reduced the complication rate from an anesthetic, predicting and preventing perioperative morbidity and mortality from cardiac disease has become the Holy Grail for many anesthesiologists, internists, and cardiologists. It appears that one of the earliest publications for assignment of risk(s) from cardiac disease is the paper published by Goldman et al in 1977⁽²⁾.

Currently, the «working document» for preoperative evaluation of the cardiac patient for non-cardiac surgery (NCS) has become the ACC/AHA guidelines, first published in 1996⁽³⁾ with revisions in 2002 and 2007⁽⁴⁾.

Despite well-accepted guidelines, though, much of medical practice rests on «usual and customary behavior», and using guidelines to change this physician behavior and practice remains difficult. For example, even though our institution has «endorsed» the ACC/AHA guidelines, we continue to find patients who, by virtue of the guidelines, met none of the guideline criteria for testing but underwent testing anyway. And, as will be discussed later, the whole issue of stress testing and intervention to «fix» cardiac artery lesions is now under review for increasing, rather than decreasing, overall risk of death to patients.

Each scenarios (i.e.; should have been tested but was not; did not need the test but was tested anyway, got tested and intervention) is accompanied by financial and medical complication issues that are beyond the scope of this talk. It is important to keep in mind that every medical test and procedure has a potential downside – a significant aspect to the patient with a positive stress test who has a stroke during the follow-up, clean cardiac catheterization. In their Guideline Update for exercise testing, the ACC / AHA quote a rate for myocardial infarction and / or death as a result of exercise testing to be in the 1 per 2,500 tests⁽⁵⁾. And then there is the whole issue of whether or not stress testing accurately predicts which patient will have a perioperative cardiac event. In their prospective study of 457 vascular surgery patients undergoing preoperative dipyridamole-thallium stress testing, Baron et al found that information obtained from this stress testing did not accurately predict adverse cardiac outcomes⁽⁶⁾.

There are three parts to this talk: 1) understanding stress testing; 2) a discussion of the current guidelines for stress testing and follow-up management, and 3) a discussion of the current situation with preoperative intervention.

CARDIAC STRESS TESTING

Cardiac stress tests can be envisioned as a two part procedure. In the first part, some sort of activity / exercise or drug is used to either increase heart rate (i.e.; cause the heart to need more oxygen), or increase blood flow. Important points to remember include:

- 1) Heart rate is the principle determinant of myocardial oxygen demand;
- 2) Unlike most other tissues in the body, the heart extracts nearly all the oxygen from the blood that passes through the myocardium (i.e.; there is little or no reserve); and

3) The only way to meet increased oxygen demand on the part of the myocardium is to increase oxygen delivery (dependent upon cardiac output < stroke volume and heart rate >, oxygen saturation, hemoglobin, and, at extremes, PaO₂).

The various types of «stressors» for stress testing attempt to increase oxygen demand by increasing heart rate, or drugs are used to increase blood flow by decreasing the resistance of the coronary vascular bed (see Figure 1). In tests that increase heart rate in order to provoke myocardial ischemia (see Figure 2), a maximum heart rate for age (called MHRA = 220 – age) and a «target» heart rate (80% MHRA) should be calculated. Any heart rate stress test that does not achieve at least 80% MHRA **is inadequate for the conclusion of «no inducible ischemia»**.

«Stressors» are shown in table I. The most common and most studied stressor is the Bruce Protocol (Table II) for a Treadmill Exercise Stress test. In the Bruce Protocol, a patient

is set onto a treadmill at 1.7 mph (2.5 feet/second) at an incline of 10%. Every three minutes, the treadmill speed is increased, and the treadmill incline is increased by an additional 2%. Each of these «levels» has known amounts of oxygen work associated with it, and most cardiologists will report this oxygen demand. Oxygen demand is reported as «METS», which is the «metabolic oxygen equivalent» of 3.5 mL O₂ / min/kg (one MET is considered the resting oxygen demand).

There is good correlation between New York Heart Association functional status and workload METs (Table III).

The second part of the stress test is the determination of «ischemia» secondary to the stressor. Common modalities for evaluating myocardial ischemia in the setting of a stress test (i.e.; supply versus demand inequality) are shown in table IV. For a treadmill (or other exercise test), the most common and easiest-to-use evaluation for ischemia is the surface electrocardiogram. In order to perform a test using only the surface electrocardiogram for ischemic evaluation, a patient

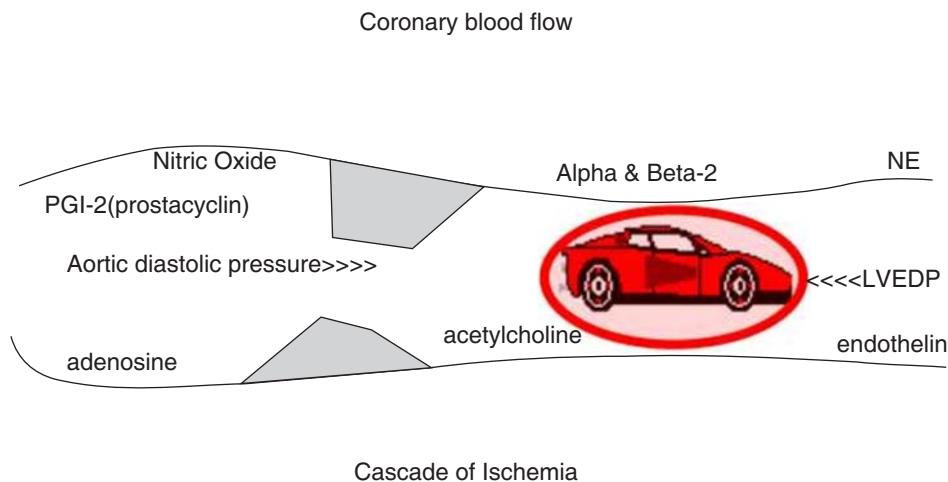


Figure 1. Coronary blood flow (Courtesy Joe Swafford, MD, FACC)

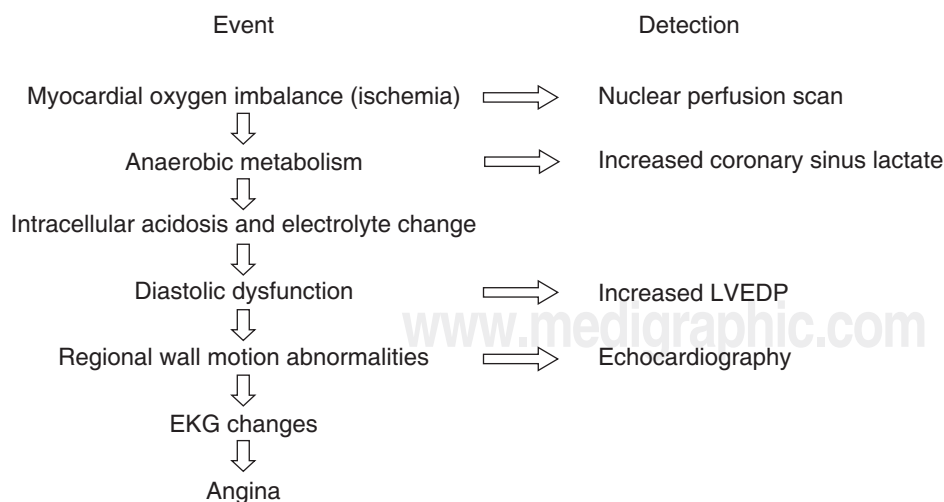


Figure 2. Cascade of myocardial ischemia.

Table I. Cardiac stress test «stressors».

Exercise protocols
Bruce (treadmill) – 1.7 mph, 10° incline at start
Modified Bruce (treadmill) – no incline at start
Other treadmill – most are less strenuous than Bruce
Naughton – 2-3.4 mph, 2 minute stages, low workload differential each stage
McHenry – 2-3.3 mph, 3 minute stages, 3-21% grade
Balke – 3.3 mph, 1 minute stages, incline increases 1% each stage
Bicycle ergometer
Arm grip test
Pharmacologic protocols
Dobutamine (heart rate test) – difficult if patient on beta blocker
Adenosine (vasodilator) – relatively contraindicated with pulmonary disease
Dipyridamole (vasodilator, precursor to adenosine)

Table II. Bruce protocol.

Stage	Speed (mph)	Grade %	Duration (min)	METs	Total time (min)
1	1.7	10	3	4	3
2	2.5	12	3	7	6
3	3.4	14	3	9	9
4	4.2	16	3	14	12
5	5.0	18	3	16	15
6	5.5	20	3	20	18

Table III. Relationship between New York Heart Classification and METs.

Functional class	METs workload on stress test
I (No limitation)	> 7
II (Mild dyspnea on exertion)	4-7
III (Significant dyspnea on exertion)	2-3
IV (Dyspnea with activities of daily living)	1

Table IV. Cardiac stress evaluation modalities.

Surface electrocardiogram
Echocardiography
Transthoracic
Transesophageal
Nuclear imaging
Thallium
Tetrafosmin
Sestamibi

must have a normal resting electrocardiogram without bundle branch block, abnormal ST segments, or abnormal T wave morphology. Other modalities include echocardiography as well as nuclear imaging. Figure 3 shows the guidelines that we use at MD Anderson for patient referral.

Reporting of stress testing has not been standardized, and, often cardiologists take shortcuts. Additionally, there remains no uniform agreement regarding the precise diagnostic criteria for positive or negative exercise stress testing. Also, certain areas of the heart (especially the inferior wall) and certain types of patients (women with large, pendulous breasts; the morbidly obese; or the diabetic) often have «thinned» nuclear uptake without hemodynamically significant coronary artery lesions.

The scribbling «Normal stress test, cleared for surgery» on a prescription pad from a cardiologist (or internist) is inadequate and should not be accepted. At a minimum, every stress test report should include:

- Indication for test
- Type of test and evaluation
- Duration of exercise, if exercise was used (see text, below)
- Heart rate achieved and % of MHRA (if heart rate test)
- Reason for stopping the test
- Patient symptoms, if any
- Interpretation of test
- Ejection fraction, wall motion, and heart volumes (nuclear or echo study)

As with any test, results include both false-negatives (i.e.; test was interpreted as normal but the patient has coronary artery disease) and false-positives (i.e.; test was positive for coronary artery disease but the patient has none). Assuming that 80% MHRA was achieved and the patient exercised more than 6 minutes (into Bruce stage 3), the incidence of false-negatives is LOW. In fact, the longer that a patient exercises on the Bruce Protocol, the lower the incidence of false negative results. Any patient who cannot exercise sufficiently (i.e.; the patient achieves > 80% MHRA before 5 minutes) should be evaluated for cardiomyopathy PRIOR

to an elective case. Women often have significant false-positive exercise treadmill tests, and some sources report this rate to exceed 60%⁽⁷⁾. Additionally, hypotension from dobutamine infusion might significantly «unload» the heart so as to preclude the development of segmental wall motion abnormalities. Thus, some significant lesions could be missed in this instance⁽⁸⁾.

Contraindications to exercise testing generally include: poor exercise conditioning (debilitation), lower extremity peripheral vascular disease, severe hypertension, and significant valvular stenosis. In general, in the presence of an indicated stress test (i.e.: the need to rule out coronary artery disease), one can identify a test modality based upon patient characteristics.

Alternatively, in the absence of a contraindication to intravenous contrast agent (primarily renal insufficiency or history of anaphylaxis), one can, if need be, proceed directly to coronary angiography.

It is important to remember that most of these tests identify the patient with a hemodynamically significant (> 70%) stenosis in an epicardial vessel. Small vessel disease (like that present in diabetics), left ventricular hypertrophy, elevated left ventricular end diastolic pressure or lesions less than 70% can produce unfavorable stress test results without a positive finding at angiography. Thus, the patient with an «abnormal» stress test, but a «normal» angiogram should probably be considered higher risk than the patient with a

normal stress test, especially in the presence of multiple risk factors for coronary artery disease (male sex, advanced age, positive family history, history of hypertension, presence of diabetes, sedentary lifestyle, and diabetes).

Lastly, one should remember that the patient with epicardial vessel lesions in the 50-70% occlusive range could have atherosclerotic plaques which can rupture under stress. These patients are much less likely to have developed significant collateral circulation, and, thus, these patients are more likely to present with sudden death as their evidence of plaque rupture.

INDICATIONS FOR STRESS TESTING

At the MD Anderson Preoperative Consultation Center, we have adopted the ACC / AHA Guidelines for minimal criteria for referral for stress testing. The flowcharts are included as an appendix to this document. From our standpoint, the most complicated issues surrounding these guidelines include:

- 1) The patient with previous exposure to cardiotoxic chemotherapy, since these patients can be markedly deconditioned and have cardiomyopathy unrelated to coronary artery disease;
- 2) The patient with recently discovered diabetes, no evidence of end-organ involvement, but scheduled for a «high risk» operation; and

Nuclear Stress Test Decision Tree

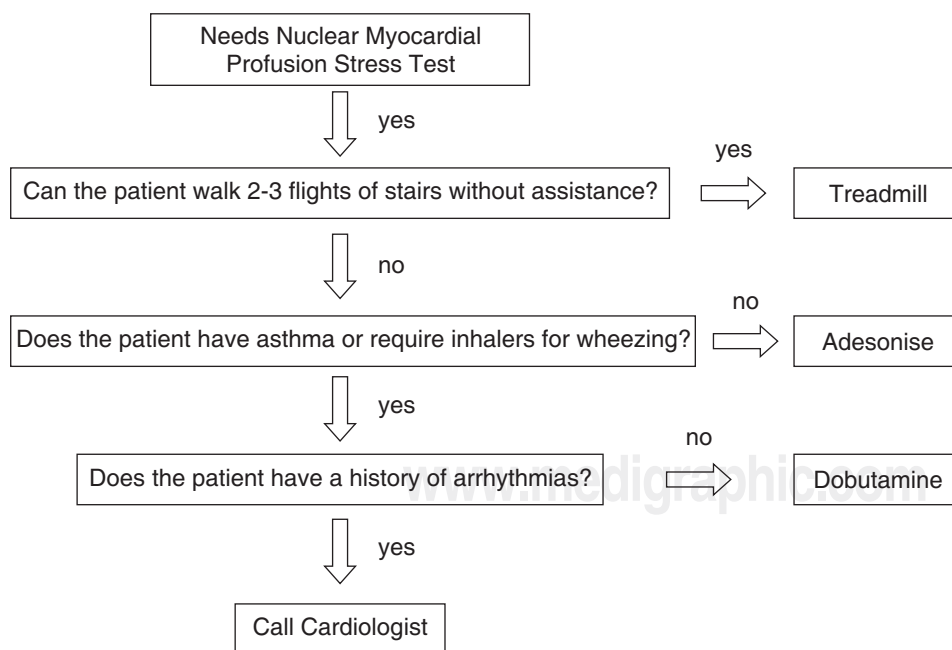


Figure 3: MD Anderson Flowchart for nuclear cardiac stress test orders.

- 3) The patient with recently discovered renal insufficiency, no evidence of end-organ damage, but scheduled for a «high risk» operation.

These guidelines are «expert opinion» and have not been subjected to rigorous, peerreviewed investigation. And there is controversy. Some suggest that use of these guidelines will reduce testing and costs associated with preoperative evaluation without increasing the complication rate⁽⁹⁾. On the other hand, another report suggests that the criteria used for selecting patients for noninvasive testing are too broad and lead to increased testing without benefit⁽¹⁰⁾.

MANAGING THE OUTCOME(S) OF STRESS TESTING

While the determination of epicardial disease utilizing some form of cardiac stress test seems relatively straight-forward, the treatment of patients with a variety of lesions is not. There is considerable disagreement among practitioners regarding the use of medical management, percutaneous coronary intervention, and coronary artery bypass grafting in these patients⁽¹¹⁾.

However, two recent randomized studies shed considerable light on this issue.

In a multicenter study involving 462 Veteran's Administration patients with significant angiographic epicardial lesions (but without unstable coronary disease, left main lesions, significant cardiomyopathy, or aortic stenosis) scheduled for abdominal or infrainguinal vascular surgery, McFalls et al. randomized patients to percutaneous intervention, coronary artery bypass grafting surgery, or medical management prior to surgery⁽¹²⁾. They found no difference in the 30 day rate for myocardial infarction (12% intervention group vs 14% medical management group, $P = 0.37$), and no difference in mortality at 32 months post surgery (22% intervention group vs 23% medical management group, $P = 0.92$). They did find, though, that the vascular surgical event was significantly delayed owing to the intervention when compared to the medical management group. They also noted 10 deaths in the intervention group and 1 death in the medical management group after randomization but prior to the vascular surgery, as well as 21 patients originally assigned to medical management who underwent a coronary artery intervention procedure during the study follow-up period.

In yet another study, Poldermans et al screened 1,880 patients scheduled for elective abdominal / infrainguinal revascularization surgery. They identified 101 patients who showed extensive preoperative ischemia, and randomized 49 to preoperative revascularization. The other 52 were slated for surgery without invasive intervention. Three patients in

the preoperative revascularization group died prior to their vascular surgery, and, at 30 days and 1 year, there was a small survival benefit (not statistically significant) accruing to the non intervention group⁽¹³⁾.

DRUG-ELUTING STENTS, BARE METAL STENTS, AND CORONARY ARTERY BYPASS GRAFTING

This issue is far from settled, though, as these studies were not adequately powered to clearly make the choice between intervention and medical management. Noting that no survival benefit accrued to those patients with a preoperative coronary artery intervention, Moscucci and Eagle wrote in an editorial that providing expert medical perioperative care (beta blockade with bisoprolol⁽¹⁴⁾ or atenolol⁽¹⁵⁾ and administration of statin drugs⁽¹⁶⁾) is superior and less costly than coronary artery intervention for these patients. However, the POISE study, reported to date only in abstract form, suggests that perioperative beta blockade is not for every patient⁽¹⁷⁾.

The preoperative intervention controversy goes way back, and none of these findings should be a surprise. In 1999, Pitt et al published a multicenter study in which 341 patients with stable CAD were randomly assigned to atorvastatin or angioplasty; they found that 21% of patients in the angioplasty group had a post-ischemic event *versus* 13.4% in the atorvastatin group (not statistically different)⁽¹⁸⁾. In 1997, Tu et al compared use of cardiac procedures (angiography, PTCA, and CABG) in Canadian *versus* US elderly patients and found a slightly (but statistically significant) lower 30-day mortality (22.3 *versus* 21.4%) but identical (34.4 *versus* 34.3%) one year mortality, despite a revascularization rate of 2.8 *versus* 21.8%⁽¹⁹⁾.

Completely ignored is a study from Germany, in which 101 patients with documented CAD were randomized to exercise training or PCI; although no deaths were recorded in either group, the exercise group had far fewer ischemic events than the PCI group (12 *versus* 30%)⁽²⁰⁾. Admittedly, though, it is difficult to engage a preoperative patient in 12 months of intensive exercise.

THE NEW HIGH RISK PARADIGM

There remain a number of issues. First, what about the asymptomatic patient who, by ACC criteria, is shunted into the preoperative non-invasive testing schema? Since they are asymptomatic, the only scoring system to determine whether or not any intervention helps them is longevity. At this time, and despite years of performing and studying various forms of mechanical interventions, no survival benefit has ever been shown to accrue (excluding the patient with left main disease) when compared to medical management⁽²¹⁾. In fact, in a recently published study of 2,287 pa-

tients with stable coronary artery disease, PCI conferred no advantage with respect to MI or death⁽²²⁾.

Second, as perioperative physicians, we are now faced with a new dilemma: any patient who presents for surgery with a recently placed coronary artery stent must be considered high risk, especially if they have been undertreated with their aspirin and thienopyridine (clopidogrel or ticlopidine)⁽²³⁻²⁵⁾. And just what constitutes appropriate treatment duration is now in question.

Over the past 7 years, reports of patients who underwent preoperative angioplasty with stent placement and who suffered a perioperative cardiac event have appeared. Kaluza et al reported 7 MIs and 8 deaths in 40 patients who underwent NCS less than 6 weeks after [bare metal] stenting (BMS) of their coronary vascular system⁽²⁶⁾. Vicenzi et al reported a postop MI in a patient treated with a BMS [for an MI] 32 days prior to surgery⁽²⁷⁾. Sharma et al retrospectively reviewed the cases of 47 patients who underwent elective NCS within 90 days of coronary artery stent (BMS) placement. They found that the risk of perioperative death was significantly higher in patients undergoing NCS within 3 weeks of surgery, and it was increased if the thienopyridine was discontinued. They found no difference in perioperative bleeding or transfusion, but this retrospective review had a very small sample size⁽²⁸⁾. Finally, in what appears to be the index case for acute perioperative drug eluting stent (DES) thrombosis, Murphy and Fahy published a case in which a 44 year old woman undergoing gynecologic surgery suffered a postoperative STElevations MI 2 weeks after a sirolimus-eluting stent⁽²⁹⁾.

Current ACC / AHA for post-stent drug therapy state that, at a minimum, patients should be treated with clopidogrel 75 mg and aspirin 325 mg for 1 month after BMS implantation, 3 months after sirolimus DES implantation, 6 months after paclitaxel DES implantation, and ideally, up to 12 months if they are not at high risk for bleeding⁽³⁰⁾. However, antiplatelet therapy often is stopped at the instruction of physicians, dentists, and other healthcare providers who are to perform an invasive or surgical procedure on the patient because of concerns about excessive procedure-related bleeding. Currently, there are no prospective studies of invasive dental procedures on patients taking a thienopyridine alone or in combination with aspirin, and no well-documented cases of clinically significant bleeding after dental procedures, including multiple dental extractions have been published. As a result, in February, 2007, the ACC / AHA issued the following guidelines⁽³¹⁾:

1. Before implantation of a stent, the physician should discuss the need for dual antiplatelet therapy. In patients not expected to comply with 12 months of thienopyridine therapy, whether for economic or other

reasons, strong consideration should be given to avoiding a DES.

2. In patients who are undergoing preparation for percutaneous coronary intervention and are likely to require invasive or surgical procedures within the next 12 months, consideration should be given to implantation of a bare-metal stent or performance of balloon angioplasty with provisional stent implantation instead of the routine use of a DES.
3. A greater effort by healthcare professionals must be made before patient discharge to ensure patients are properly and thoroughly educated about the reasons they are prescribed thienopyridines and the significant risks associated with prematurely discontinuing such therapy.
4. Patients should be specifically instructed before hospital discharge to contact their treating cardiologist before stopping any antiplatelet therapy, even if instructed to stop such therapy by another healthcare provider.
5. Healthcare providers who perform invasive or surgical procedures and are concerned about periprocedural and postprocedural bleeding must be made aware of the potentially catastrophic risks of premature discontinuation of thienopyridine therapy. Such professionals who perform these procedures should contact the patient's cardiologist if issues regarding the patient's antiplatelet therapy are unclear, to discuss optimal patient management strategy.
6. Elective procedures for which there is significant risk of perioperative or postoperative bleeding should be deferred until patients have completed an appropriate course of thienopyridine therapy (12 months after DES implantation if they are not at high risk of bleeding and a minimum of 1 month for bare-metal stent implantation).
7. For patients treated with DES who are to undergo subsequent procedures that mandate discontinuation of thienopyridine therapy, aspirin should be continued if at all possible and the thienopyridine restarted as soon as possible after the procedure because of concerns about late-stent thrombosis.
8. The healthcare industry, insurers, the US Congress, and the pharmaceutical industry should ensure that issues such as drug cost do not cause patients to prematurely discontinue thienopyridine therapy and to thus incur catastrophic cardiovascular complications.

These data, suggesting that preoperative stent placement actually increases the perioperative risk of MI or death, along with the randomized study by McFalls⁽¹²⁾, wherein preoperative mechanical intervention produced no benefit (and perhaps a detriment), should be sufficient to make any reasonable clinician hesitant to send a patient for a preoperative stress test or intervention, other than to prescribe beta

blockers and statins⁽³²⁾. But the drive to «do something» remains strong.

SUMMARY

Understanding cardiac stress test indications and limitations can help the anesthesiologist determine perioperative risk. For high risk patients, the data obtained from this testing might help guide perioperative care. Additionally, review-

ing stress test results might prevent an inappropriately prepared patient from undergoing a surgical intervention that might be better delayed until appropriate drug therapy is commenced.

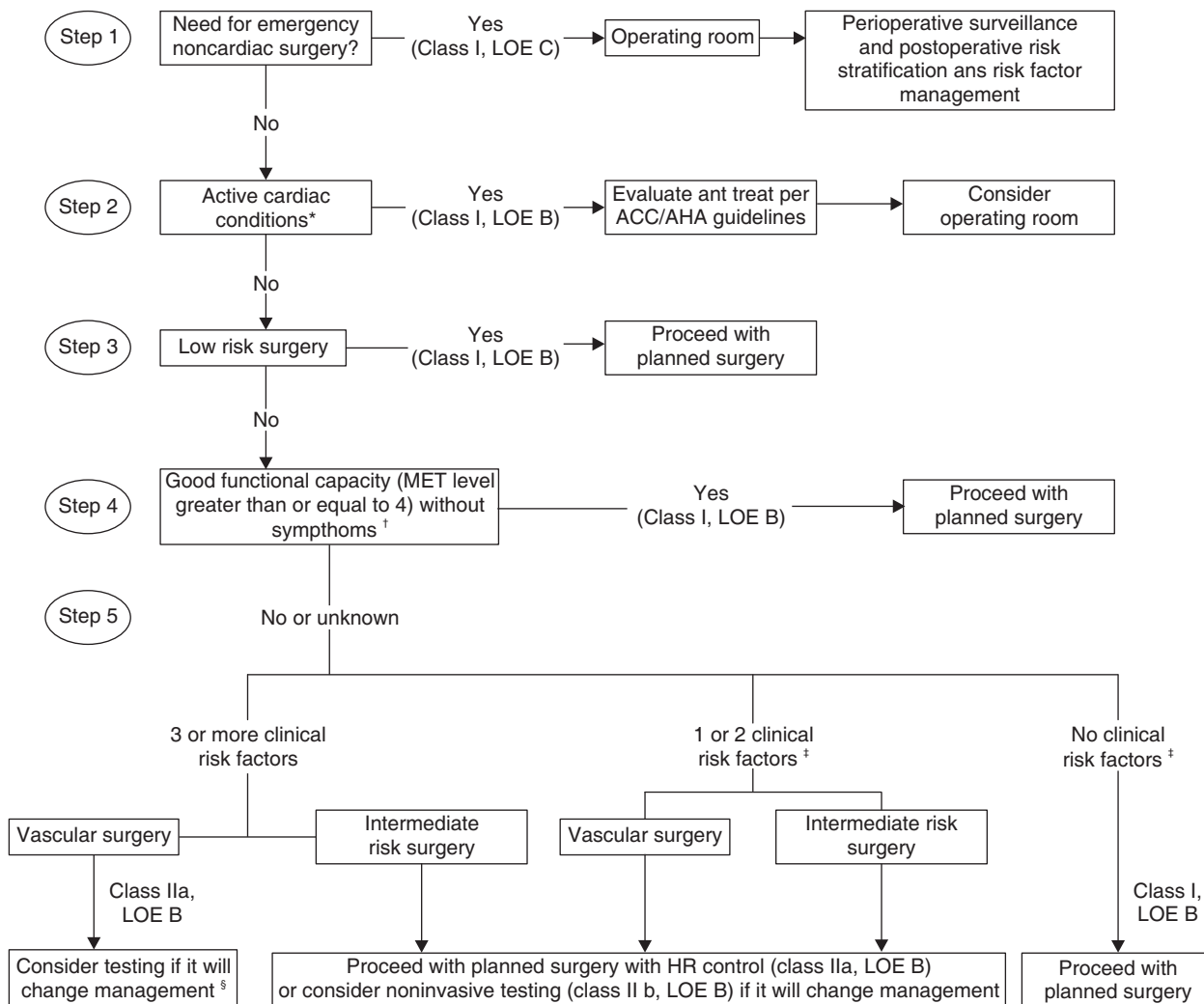
Understanding the literature, which suggests that very few patients (especially if asymptomatic) will benefit from perioperative testing and resultant intervention should lead to reduced testing, reduced intervention, and less delay in getting a patient into the operating room.

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Appendix: Preoperative stress test guidelines from the ACC/AHA.



Cardiac evaluation and care algorithm for noncardiac surgery based on active clinical conditions, known cardiovascular disease, or cardiac risk factors for patients 50 years of age or greater. *See table 2 for active clinical conditions. †See table 3 for estimated MET level equivalent. ‡Clinical risk factors include ischemic heart disease, compensated or prior HF, diabetes mellitus, renal insufficiency, and cerebrovascular disease. §Consider perioperative beta blockade (see Table 11) for populations in which this has been shown to reduce cardiac morbidity/mortality. ACC/AHA indicates American College of Cardiology/American Heart Association; HR, heart rate; LOE, level of evidence; and MET, metabolic equivalent.

Adapted from Fleisher et al, 2007⁽⁴⁾

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Note that testing is indicated only where it will affect outcome or change management.