SMART EM Stress Testing Summary

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This is a little bit of a behemoth lecture and summary – but well worth it.

Stress Test: A test to determine whether a patient has myocardial ischemia during tachycardia

- Exercise stress: looking for ECG abnormalities while patient runs on a treadmill
- Stress Echo: echocardiography to look for wall motion abnormalities during tachycardia
- Perfusion imaging ('nuclear stress'): radionucleoide tagged infusion with CT imaging to evaluate myocardium, looking for areas of poor perfusion – can be done at rest or during tachycardia

AHA: *For low to intermediate risk chest pain patients (normal ECG, negative enzymes) exercise stress testing increases sensitivity and negative predictive value for short term death or myocardial infarction. All such patients should therefore have this test performed within 72 hrs, and preferably ASAP*

How did we get here? Evolution of the stress test in general practice:

1. Early (pre-1960s): Case reports and series describing ECG changes in patients during angina, and eventually during induced exertion/tachycardia. (Bousfield, Master x4, Levine, Wood, Littmann)

2. Mid (1960-1990): Studies comparing stress to cath findings, suggest no utility for high or low risk patients (Combs, Weiner, CASS, Harol, Goldman). Possible utility for roughly 50% prob.

- a. Frolicher, 1998: likely most rigorous study of stress test accuracy, ~200 patients with stress and cath, shows sensitivity 45%, specificity 85%.

3. Late (1990-): most influential study is Mark, 1991, the Duke Treadmill Score study. Introduced a scoring system that allowed stress testing in low risk patients to risk stratify patients to <1% likelihood of death, and led to increasingly widespread acceptance of stress testing for this purpose. Statistically, study suggested addition of stress to clinical judgment adds 5% accuracy.

The expansion of stress testing to acute patients:
Markowitz to Schwartz: Stress typically done in hospital 72 hrs after pain following MI. All pts known CAD, thus stress not used for dx but to predict 3-vessel or 'left main' patients for potential CABG. However, stress was still felt unsafe for unstable angina. Swan, Wilcox, and

Butman stress unstable angina patients and conclude stress is safe if done 48-72 hrs after pain free. Subsequently,

1. 1991 Larsen: oft-cited, supported early stress after 'unstable angina'—10% death/MI within 1 month, and stress test at discharge was same result as stress at 1 month. However, 'unstable angina' actually 20% STEMI, 80% +enzymes or EKG. (MI meant exclusively 'Q-wave MI').

2. 1994: AHCPR (early AHRQ), on basis of Larsen recommends outpatient stress within 72 hrs for 'low-risk unstable angina', inpatient stress for higher risk (after 48 hrs pain-free).

3. 1997: AHA guideline recommends stress after 'unstable angina' for CABG/PCI potential. Positive result –> cath; intermediate result◊ additional testing; negative result –> medical therapy. However, no recommendations for low risk chest pain (negative enzymes and EKG).

How did we get to 'rule out stress' for low risk chest pain? Major cultural shift:

1. Fear of litigation and of missing MI

- a. Major articles about MI and malpractice, e.g. Rusnak 1989.
- b. Oft-cited Pope 2000 study – 11,000 ED chest pain pt's all rechecked 48h later with EKGand enzymes: 8% MI and 9% unstable angina overall. 2% of MI patients (n=19) missed, i.e. 0.18% of chest pain population. Cited as a failure to diagnose! Mortality rate identical between missed and identified, however customized 'risk adjustment' hinted atincreased mortality—but statistically insignificant. Pope paper now cited ubiquitously as suggesting high miss rate and increased mortality among misses.

2. With movement toward managed care and capitation in '80s came increasing interest in brief observation protocols to replace costly admissions in those not clearly having ischemia.

3. Increasing social consciousness about heart disease and chest pain, deteriorating primary care infrastructure, lead to increasing low risk chest pain visits.

Does stress rule out short term MI or mortality in low risk chest pain? 10 studies, 2 examples:

1. Gibler 1995 – early 'chest pain unit' study of 1000 pt's with nondiagnostic ECG. Included enzymes, ST monitoring, echo and treadmill stress. 12 MIs, all enzyme positive. 1 pt with negative stress test had MI 3 days later.

2. Amsterdam 2002: ~1000 stable pt's w/ nonspecific ECG's, immediate stress in ED. 5 MI's, all had positive enzymes in ED. 1 pt having an MI pt missed by stress, captured by enzymes.

3. Consistent theme throughout: enzymes identify all contemporaneous MIs, and stress tests do not identify additional patients with short term risk that are missed by enzymes.

What happens to low risk chest pain with invasive testing or imaging? Two examples:

1. Defelipe 2001: 248 randomized to immediate cath vs stress, 0 MI or death in either group. 11% of cath pts receive revascularization procedure vs 4% of stress pts.

2. Goldstein 2007: similar, coronary CT angio vs nuclear stress for low risk chest pain—CT angio quadrupled interventions, but 0 MI or death in either group at 6 months.

Could revascularization benefit low risk chest pain patients with a positive stress?

Invasive vs medical treatment for NSTEMI/ACS literature may be relevant, as it is presumed that patients with negative enzymes and EKGs but positive stress tests are ACS patients:

1. Cochrane review (Honig): No difference in mortality, small MI benefit for invasive therapy among those with positive troponins. However INCREASED mortality if negative troponins.

2. JAMA meta-analysis (O'Donoghue, 2008) – no MI or death benefit, but again trend toward benefit if troponins positive, which means trend toward harm if troponin negative.

Invasive vs medical treatment for 'stable CAD' may also be relevant, as it is possible that these are not true ACS patients but rather stable CAD patients:

1. COURAGE trial (Boden, 2007): significant ischemia or +stress –> if cath showed obstructive CAD, randomized to stent vs medical: no change in MI or mortality, mild symptom reduction

Conclusion: if people with proven obstructive CAD or proven ACS don't benefit from PCI, then low risk chest pain patients are virtually certain not to benefit

Actual risks for ED chest pain (see SMART Chest Pain Risk podcast)

With nonspecifc or normal ECG and 1 neg trop:

- 1. <40 years old: risk for death/MI in 30 days = 1 in 500
- 2. 'Low risk', regardless of age, according to treating emergency physician = 1 in 250
- 3. Being admitted and believed to be 'intermediate' risk = 1 in 125

With two neg troponins

- 1. Hamm 1997 study: if two troponins negative only 1 event in next 30 days. (n=700+, 18% MI's)
- 2. Nabi 2010 study: 1031 patients considered intermediate risk by EP, 8 MIs and 0 deaths, all detected on serial troponins. No death or MI in next 6 months.

Most recent data addressing stress test added to two negative enzymes?

1. Chan, Hollander: (admitted patients), only 20% of pt's got a test: no difference in MI/mortality between pt's with positive or negative stress

2. Myer 2006: ok to delay stress to 72h

3. Scheuermeyer 2012: low risk chest pain population, some stress as outpatients, some no stress at all; 3.5% MI, all diagnosed in the ED on enzymes. No MI/unstable angina in 30 days.

Conclusion: After a two-troponin rule-out, the stress test adds nothing tangible to risk stratification of a low risk chest pain patient. It may, however, lead to unnecessary testing and needless harm.