Update in Hospital Medicine 2011

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Submitted in collaboration with the SGIM Academic Hospitalist Taskforce
Special Thank you:

Anneliese Schleyer
Brian Harte
Dan Steinberg
Michelle Mourad

No conflicts of interest.
Update in Hospital Medicine 2011

- Updated literature since March 2010

Process:
- CME collaborative review of journals
  Including ACP J. Club, J. Watch, etc.
- Four hospitalists ranked articles
  Definitely include, can include, don’t include
- Removed articles covered by others
Update in Hospital Medicine 2011

Chose articles based on 2 criteria:

1) Change, modify, or confirm your practice.
2) Change, modify, or confirm your teaching.

- Hope to not use the words
  Markov model, Kaplan-Meier, Student’s t-test
  Focus on breadth, not depth
  Not all randomized-controlled trials
Update in Hospital Medicine 2011

- Major reviews/short takes
- Chance to ask questions
- Case-based format
Syllabus/Bookkeeping

- Final presentation will be posted

- Can email me for copies:
  sharpeb@medicine.ucsf.edu
Case Presentation

A 62 year-old woman with a history of HTN, CAD, and COPD presented with a three days of shortness of breath and productive cough. She has had four COPD exacerbations this year.

You are told that the patient has been taking metoprolol 50mg twice a day. When discussing the plan, the intern asks, “Do you think the β-blocker might be causing all the exacerbations?”
What is the impact of her long-term β-blocker use on her COPD?

A. It is likely contributing to her recurrent exacerbations.
B. It has no impact on her COPD.
C. It is decreasing her mortality because she has coronary artery disease.
D. It is decreasing exacerbations and decreasing mortality.
E. Who cares. She probably isn’t taking it anyway. I hate my job.
β-blockers & COPD

Question: What is the impact of long-term β-blocker use in patients with COPD?

Design: Observational cohort study, 2230 pts, > 45 years old; known COPD; 45% with CAD/risk factors; 7 yr follow-up, β-blocker use vs. no β-blocker use

### Results

Overall mortality was 31%
COPD exacerbations in 50% of patients

<table>
<thead>
<tr>
<th>β-blocker use</th>
<th>Adjusted HR* (95% CI)</th>
</tr>
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* With propensity scoring
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<tr>
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</tr>
</tbody>
</table>

Outcomes similar in those w/ and w/o CAD

Question: What is the impact of long-term β-blocker use in patients with COPD?

Design: Observational cohort study, 2230 pts, > 45 years old; known COPD

Conclusion: β-blocker use decreased mortality & exacerbations; True for pts without overt cardiovascular disease;

Comment: Retrospective, other confounders? CAD likely under-diagnosed in COPD. Benefit from cardiac & pulmonary effects? β-blockers not harmful in pts with COPD; Don’t stop β-blockers on admission.

What is the impact of her long-term $\beta$-blocker use on her COPD?

A. It is likely contributing to her recurrent exacerbations.
B. It has no impact on her COPD.
C. It is decreasing her mortality because she has coronary artery disease.
D. It is decreasing exacerbations and decreasing mortality.
E. Who cares. She probably isn’t taking it anyway. I hate my job.

*Update in Hospital Medicine*
Case Continued

Based on the history, exam, and testing, she is diagnosed with an exacerbation of her COPD. She is moderately ill and admitted to a floor bed.

The team has decided that in addition to bronchodilators, she should receive systemic corticosteroids. What is the optimal route and dose for the steroids?
What is the optimal route and dose for the corticosteroids?

A. Solumedrol 1 gram IV every 6 hours.
B. Solumedrol 125mg IV every 6 hours.
C. Prednisone 60mg PO once a day.
D. Prednisone 20mg PO once a day.
E. Hey you, Giants fan, why don’t we give her the same dose of steroids Barry Bonds was taking?
Steroids in COPD Exacerbation

**Question:** In COPD exacerbations, what is the optimal dose and route for corticosteroids?

**Design:** Observational cohort study; 79,985 pts with a COPD exacerbation, non-ICU admits; Compared low-dose oral vs. high-dose intravenous steroids;

Low-dose = 20-80mg prednisone/day
High-dose = 120-800mg prednisone/day

Tx failure: ventilation, hosp. death, readmit

## Results

- 92% (71,628) given high-dose intravenous steroids
- Median doses: 60mg vs. 600mg per day

### Low-dose vs High-dose

<table>
<thead>
<tr>
<th></th>
<th>Adjusted Odds Ratio**</th>
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<tbody>
<tr>
<td>Treatment Failure</td>
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**With matching & propensity scoring

### Results

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</tr>
</thead>
<tbody>
<tr>
<td>Treatment Failure</td>
<td>0.93 (0.84-1.02)</td>
</tr>
<tr>
<td>Length of Stay</td>
<td>0.90 (0.88-0.91)</td>
</tr>
<tr>
<td>Costs</td>
<td>0.91 (0.89-0.93)</td>
</tr>
</tbody>
</table>

** With matching & propensity scoring

### Steroids in COPD Exacerbation

**Question:** In COPD exacerbations, what is the optimal dose and route for corticosteroids?

**Design:** Observational cohort study, 79,985 pts with a COPD exac; compared low-dose oral vs. high dose intravenous steroids.

**Conclusion:** Low-dose oral steroids no worse than high dose; in propensity analysis, treatment failure, LOS and cost were lower w/ oral low-dose.

**Comments:** Retrospective, database, confounders, etc. Confirms guidelines & smaller studies; most pts should get low-dose PO steroids.

What is the optimal route and dose for the steroids?

A. Solumedrol 1 gram IV every 6 hours.
B. Solumedrol 125mg IV every 6 hours.
C. Prednisone 60mg PO once a day.
D. Prednisone 20mg PO once a day.
E. Hey you, Giants fan, why don’t we give her the same dose of steroids Barry Bonds was taking?
Case Continued

The team had prescribed prednisone 60mg daily and this is continued.

During the Assessment and Plan, the student inquires, “I read that only 50% of all COPD exacerbations are thought to be bacterial and yet we’re giving her antibiotics. Why is that?”
Why do we give antibiotics to patients to with a COPD exacerbation?

A. Antibiotics improve symptoms but don’t decrease mortality.

B. Antibiotics decrease treatment failure and possibly decrease mortality.

C. Antibiotics should only be given to patients with a COPD exacerbation if they also have pneumonia.

D. Well, you know, I am a bit worried about Pfizer. They only made $8 billion in profits last year.
Antibiotics in COPD Exacerbation

Question: Do antibiotics improve outcomes in pts admitted to the hospital with a COPD exacerbation?

Design: Retrospective cohort study; 84,621 pts w/ a COPD exacerbation, non–ICU admits; Abx given on hosp day 1 or 2 vs. “not treated”

Common abx for COPD exacerbations
Tx failure: ventilation, hosp. death, readmit

Rothberg, et al. JAMA; 2010; 303: 2035.
**Results**

- 79% of pts given at least 2 days of abx
- Hospitals ranged from 65% – 95%
- Quinolones, cephalosporins, macrolides

<table>
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<tr>
<th>Abx vs. Not Treated</th>
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### Abx vs. Not Treated

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<tr>
<td>0.87 (0.82-0.92)</td>
<td></td>
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- Decrease in ventilation, in-hosp mortality, readmission
- Increase in *C difficile* rates (0.19% vs. 0.09%, p=0.001)

** With matching & propensity scoring

Steroids in COPD Exacerbation

Question: Do antibiotics improve outcomes in pts admitted to the hospital with a COPD exacerbation?

Design: Retrospective cohort study; 84,621 pts with a COPD exacerbation, non-ICU admits; Compared abx given on hosp day 1 or 2 vs. “not treated”

Conclusion: Antibiotics associated w/ improved outcomes; in propensity analysis, less tx failure; increase in C diff rates, no other side effects

Comments: Similar methodological issues
Most patients given abx, improves outcomes
Confirms prior meta-analysis - mortality benefit
Pts in hospital w/ COPD exac should get abx

Rothberg, et al. JAMA;2010;303:2035.
Why do we give antibiotics to patients to with a COPD exacerbation?

A. Antibiotics improve symptoms but don’t decrease mortality.
B. Antibiotics decrease treatment failure and possibly decrease mortality.
C. Antibiotics should only be given to patients with a COPD exacerbation if they also have pneumonia.
D. Well, you know, I am a bit worried about Pfizer. They only made $8 billion in profits last year.
In a large database of patients hospitalized with a COPD exacerbation and given antibiotics, macrolides and quinolones had similar treatment failure, although macrolides were associated with less antibiotic-associated diarrhea.

In a prospective, randomized, double-blind trial of 170 patients with a severe COPD exacerbation (ICU), trimethoprim-sulfamethoxazole was equivalent to ciprofloxacin for all clinical outcomes (mortality, LOS, readmission for COPD exacerbation, etc.).

There is not high-quality evidence to help with antibiotic selection. Guidelines recommend broader spectrum for high-risk patients.

Case Continued

With your evidence-based treatment, she improves over the next 3 days. You are preparing her for an appropriate discharge.

In multi-disciplinary rounds, the case manager says, “Are you going to make a PCP appointment? You know, she’ll have a better outcome if she is seen in a timely manner.”
How do you respond to the case manager’s statement about PCP follow-up?

A. It might keep her from getting readmitted.
B. I think that’s only for patients with CHF exacerbations.
C. I think it can reduce medication errors.
D. I don’t think it really makes any difference.
E. Why don’t you make the freakin’ appointment?
PCP Follow-up in COPD

Question: Does early PCP follow-up after a COPD exacerbation impact outcomes?

Design: Retrospective cohort, 62,746 pts w/ COPD exacerbation + known PCP/pulm.; d/c home

Known PCP = ≥ 3 visits/year
Known pulmonologist = ≥ 1 visit/year

## Results

- Total of 66.9% had outpatient visit in 30 days
- Older, African-American, lower SES less f/up
- Large cities, teaching hospitals less f/up

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<tr>
<th>Follow-up vs. none</th>
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Results

- Total of 66.9% had outpatient visit in 30 days
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Readmit for COPD, pneumonia, CHF
No difference for PCP or pulmonologist

PCP Follow-up in COPD

Question: Does early PCP follow-up after a COPD exacerbation impact outcomes?

Design: Retrospective cohort, 62,746 pts w/ COPD exacerbation + known PCP/pulm.; d/c home

Conclusion: Rate of timely PCP =66%; Pts lacking timely PCP f/up had higher ER visits/readmits; some specific pt populations worse;

Comments: Retrospective, other MD f/up?, Quality of care? Clear benefit; just the right thing to do Unclear optimal follow-up; future randomized studies; important to make the appointment.

How do you respond to the case manager’s statement about PCP follow-up?

A. It might keep her from getting readmitted.
B. I think that’s only for patients with CHF exacerbations.
C. I think it can reduce medication errors.
D. I don’t think it really makes any difference.
E. Why don’t you make the freakin’ appointment?
Summary

Definitely

1) Continue β-blockers in patients with COPD.
2) Give antibiotics to patients admitted with a COPD exacerbation.

Consider

1) Treating mild-moderate COPD exacerbations with low-dose oral steroids (60mg/day PO).
2) Scheduling PCP follow-up for COPD exacerbations within 30 days to reduce re-admissions.
Case Presentation

Mid-afternoon your resident runs up to you to share that Mr. Johnson, a 62 year-old man with cellulitis has developed a dense R-sided hemiparesis and aphasia. The nurse states he was completely normal 4 hours ago.

The head CT is negative and he has no contraindications to thrombolysis. What do you do?
What do you do?

A. At 4 hours it is too late to try thrombolysis – all we can do is provide supportive care.

B. We can still give thrombolysis at 4 hours – it may improve his outcome but won’t impact mortality.

C. We can try thrombolysis as it may improve his outcome but it will increase his chance of dying.

D. Stroke . . . Stroke . . . I think I’ve heard of that. Isn’t there a Billy Squire song from the 1970’s called “Stroke?”
Thrombolysis in Acute Stroke

Question: Is it safe and efficacious to administer tPA 3 to 4.5 hours after acute ischemic stroke?

Design: Meta-analysis of RCTs; 8 studies; 3670 pts; Outcomes were Modified Rankin, mortality, hemorrhage;

Included ECASS, ATLANTIS, NINDS, EPITHET
Median time to treatment was ~ 4 hrs in all trials

Thrombolysis in Acute Stroke

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is a positive Odds Ratio of > 1.0 (statistically significant) to achieve a Modified Rankin score of 0-1 from 0 – 270 minutes (4.5 hours) based on the summary from this meta-analysis.

Thrombolysis in Acute Stroke

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is an increase in mortality at around 4.5 hours from symptom onset in patients given alteplase.

Thrombolysis in Acute Stroke

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is a increased risk of parenchymal hemorrhage in patients given alteplase in the setting of acute stroke compared to placebo. The increased bleeding risk is significant after 60 minutes.

Thrombolysis in Acute Stroke

**Question:** Is it safe and efficacious to administer tPA 3 to 4.5 hours after acute ischemic stroke?

**Design:** Meta-analysis of RCTs; 8 studies; 3670 pts; Outcomes were Mod. Rankin, mort, hemorrhage

**Conclusion:** tPA in the 3-4.5 window for eligible patients improves outcomes at 90 days; mortality may go up at 4.5 hours; bleed increase at 1 hour

**Comments:** Different methodologies but well done; Should be considered in the 3-4.5 hr window (NNT = 15)

Supported by updated 2009 AHA guidelines, But - earlier is still better (time is brain)

What do you do?

A. At 4 hours it is too late to try thrombolysis – all we can do is provide supportive care.
B. We can still give thrombolysis at 4 hours – it may improve his outcome and won’t increase mortality.
C. We can try thrombolysis as it may improve his outcome but it will increase his chance of dying.
D. Stroke . . . Stroke . . . I think I’ve heard of that. Isn’t there a Billy Squire song from the 1970’s called “Stroke?”
You decide to administer tPA to the patient. Unfortunately he does not do well and has persistent symptoms, including moderate dysphagia.

Two days later you are rounding with the team and the patient has audible gurgling when you walk in the room.

The medical student asks, “Is he going to get pneumonia from that?”
Short Take: Gurgling and HAP

In a prospective, observational study, 20 patients with audible gurgling were compared to 60 patients without gurgling on the same unit. After controlling for age, dementia, opiates, and stroke, gurgling predicted hospital acquired pneumonia (HAP) with an odds ratio (OR) = 140 (95% CI, 5.6-3,529).

You ensure aspiration precautions are ordered and that he is getting the appropriate diet.

When walking to see the next patient, you notice one of the interns is wearing a freshly laundered short sleeve scrub top and realize this is part of a hospital-wide effort to reduce nosocomial infections.

You ask, “Hey, do you think that thing really reduces the spread of infections like MRSA?”
Short Take: White coat vs. Short sleeves

In a prospective, randomized, controlled trial, by 8 hours, there was no difference in bacterial or MRSA contamination between white coats and newly laundered short-sleeved uniforms.

In fact, after 3 hours of wear, the short-sleeved uniforms had 50% of the bacterial load of the white coats.

Summary

Definitely

1) Wash your hands!
2) Be aware that gurgling in the hospital may increase the risk for hospital-acquired pneumonia.

Consider

1) Treating patients with acute stroke in the 3 to 4.5 hour window with thrombolysis.
2) That your clothes in the hospital rapidly become contaminated with bacteria.
Case Presentation

A 65 year old man with history of HTN, ischemic CM (EF 45%), and cocaine abuse presents with 2 days of DOE and orthopnea.

Home meds: ASA, Metop 25 bid, Lasix 80 daily

CV: tachycardic, regular rhythm, JVP ~10 cm
Resp: bilateral crackles

Your new Sub-I reports that overnight she started therapy with lasix 160 mg IV every 12 hours
Which are true about furosemide in CHF?

A. You will send the patient home faster if you prescribe continuous furosemide
B. You should check a creatinine because that dose probably dinged his kidneys
C. You should check orthostatics because that dose probably bottomed out his blood pressure
D. That dose may help your patient feel better faster but will not change his length of stay
E. Let’s consult Orthopedics and ask.
Question: What are the optimal dose and optimal route for administering furosemide?

Design: Double blind, double dummy RCT; 616 patients with h/o CHF and >1 month use of furosemide 80-240 mg daily; 2x2 factorial design:
- low dose vs high dose furosemide
- bolus Q12 vs. continuous dosing

Low-dose = 1:1 outpatient furosemide dose
High-dose = 2.5:1 outpatient furosemide dose

Outcomes: 1) Global assessment of symptoms
2) Change in creatinine in first 72 hrs

Furosemide % CHF

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is no difference in symptoms out to 70 hours when comparing bolus vs. continuous dosing of furosemide in this patient population.

Furosemide % CHF

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is a slight improvement in symptoms in the high-dose group compared to the low-dose furosemide group in these patients with CHF.

Furosemide % CHF

For copyright reasons, the Figure presented on this page at SGIM cannot be published on-line.

The Figure showed that there is a trend toward a bigger bump in creatinine with the continuous (vs. bolus) dosing and with high-dose (vs low-dose) but this did not achieve statistical significance.

Pts in high dose strategy had larger diuresis & faster improvement in dyspnea

No other differences across a range of secondary end-points

Furosemide and CHF

Question: What are the optimal dose and optimal route for administering furosemide?

Design: Double blind, double dummy RCT; 616 patients with h/o CHF and >1 month use of furosemide 80-240 mg daily; 2x2 factorial design: low dose vs high dose furosemide and Q12 vs. continuous

Conclusion: High vs low dose Lasix & intermittent bolus vs. continuous Rx strategies equivalent global symptoms & renal outcomes

Comment: Secondary end-points suggest high dose strategy may improve dyspnea & diurese patients faster

Which are true about furosemide in CHF?

A. You will send the patient home faster if you prescribe continuous furosemide
B. You should check a creatinine because that dose probably dinged his kidneys
C. You should check orthostatics because that dose probably bottomed out his blood pressure
D. That dose may help your patient feel better faster but will not change his length of stay
E. Let’s consult Orthopedics and ask.
On hospital day 3, you and the resident find your patient coming out of a hallway bathroom followed by a trail of sweet smelling smoke and a burn hole on his gown. He complains of chest pain.

On exam, his pupils are 10 mm, he is mildly confused, pulse is 130 bpm, BP is 180-100 mmHg. ECG shows new ST depressions in lateral leads, cardiac enzymes are drawn.
What do you prescribe next?

A. Aspirin
B. Aspirin, nitroglycerin, and a β-blocker
C. Aspirin and nitroglycerin only
D. Nitroglycerin only
E. IV dilaudid to make it a speedball
β-blockers for cocaine-induced CP

Question: Are β-blockers unsafe for treating chest pain in the setting of recent cocaine use?

Design: Retrospective cohort study; 331 consecutive CP patients with (+) tox screen for cocaine; compared pts receiving β-blockers ED vs those who did not

## Results

- 328 patients admitted from ED with (+) tox screen
  - 151 received β-blocker in ED (46%)
- Pts receiving β-blockers in ED were older, had higher SBP on presentation, and more likely to have h/o HTN
- Metoprolol most commonly prescribed β-blocker
  - IV metoprolol (74%), PO metoprolol (11%), IV labetolol (12%), PO labetolol (2%), PO atenolol (1%), PO propranolol (1%)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>β-blocker in ED</th>
<th>No β-blocker</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>151 pts</td>
<td>177 pts</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>2 (2%)</td>
<td>3 (2%)</td>
<td></td>
</tr>
</tbody>
</table>

Results

No difference for ischemic ECG changes, (+) troponins, or intubation in first 24 hours

- β-blocker group had larger decrease in SBP and less likely to receive vasopressors in first 24 hours

- Of 124 patients discharged on β-blockers:
  - Most on metoprolol
    - 55% metoprolol, 25% atenolol, 15% labetolol, 3% carvedilol

- Adj HR* death if dc’d on β-blocker: 0.29 (95% CI: 0.09-0.98)

* Adjusted for age, sex, race, outpatient ACEI/ARB, outpatient CCB, h/o ESRD, (+) troponin

**β-blockers for cocaine-induced CP**

**Question:** Are β-blockers unsafe for treating chest pain in the setting of recent cocaine use?

**Design:** Retrospective cohort study; 331 consecutive CP patients with (+) tox screen for cocaine; compared pts receiving β-blockers ED vs those who did not

**Conclusion:** No negative outcomes associated with β-blocker Rx for patients with cocaine associated chest pain.

**Comment:** Retrospective study but evidence supports changing current practice

What do you prescribe next?

A. Aspirin
B. Aspirin, nitroglycerin, and a $\beta$-blocker
C. Aspirin and nitroglycerin only
D. Nitroglycerin only
E. IV dilaudid to make it a speedball
Case Continued

Your patient experiences only a mild troponin bump and promises to behave the rest of this hospitalization.

During rounds on hospital day 4, the patient's nurse asks if the patient is going home today. It looks fine, but he is going to change the peripheral IV if the patient stays.

You recognize an opportunity to win some brownie points with the nurses.
## Short Take: When to replace PIVs

A systematic review of 6 RCTs involving 3,455 patients in hospitals and community settings compared routine PIV removal vs. removal only as needed.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Routine Replacement</th>
<th>Clinically Indicated Replacement</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteremia</td>
<td>0.4%</td>
<td>0.2%</td>
<td>NS</td>
</tr>
<tr>
<td>Thrombophlebitis</td>
<td>7.2%</td>
<td>9%</td>
<td>NS</td>
</tr>
<tr>
<td>Cost</td>
<td>+$6</td>
<td>---</td>
<td></td>
</tr>
</tbody>
</table>

Case Continued

With your evidence-based treatment, she improves over the next 2 days.

As your team prepares your patient for discharge, the case manager asks, “Are you going to make a PCP appointment? You know, he’ll have a better outcome if he is seen in a timely manner.”
How do you respond to the case manager’s statement about PCP follow-up?

A. It might keep him from getting rehospitalized
B. Follow up appointments are only helpful for patients with COPD exacerbations
C. It really doesn’t make any difference
D. It reduces medication errors after discharge
E. Snort derisively and ask if that’s what they do at “Mayo”
Early follow-up after CHF hospitalization

Question: Does early PCP follow-up after CHF hospitalization reduce readmission rates?

Design: Retrospective cohort; 30,136 pts at 225 hospitals; compared readmission rates for hospitals in lowest quartile for early follow-up with those in higher quartiles.

Early follow-up = within 7 days

Results

- 38,176 patients discharged from 225 hospitals
- Median early f/u rate=38.3% (maximum 63.7%)
- 18.1% had same discharge and follow-up physician
- Blacks over-represented at hospitals with low early f/u rates

## Results

A table showing the adjusted hazard ratios (HR*) and 95% confidence intervals (CI) for early follow-up rates in quartiles, along with their corresponding P-values:

<table>
<thead>
<tr>
<th>Quartile - % early f/u</th>
<th>Adjusted HR* (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (&lt;32.4%)</td>
<td>1 (reference)</td>
<td>---</td>
</tr>
<tr>
<td>2 (32.4-37.9%)</td>
<td>0.85 (0.78-0.93)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3 (38.3-44.5%)</td>
<td>0.87 (0.78-0.96)</td>
<td>0.005</td>
</tr>
<tr>
<td>4 (&gt;44.5%)</td>
<td>0.91 (0.83-1.00)</td>
<td>0.05</td>
</tr>
</tbody>
</table>

- Adjusted for age, sex, black race, med history, admission lab results, complete d/c instructions, referral to CHF disease management program, year of index hospitalization, index LOS

- No difference for PCP vs Cardiologist follow-up
- No association between early follow-up & mortality

---

Early follow-up after CHF hospitalization

Question: Does early PCP follow-up after CHF hospitalization reduce readmission rates?

Design: Retrospective cohort; 30,136 pts in 225 hospitals; compared readmission rates for hospitals in lowest quartile for early follow-up with those in higher quartiles.

Conclusion: Patients discharged from hospitals with lowest rates of early follow-up had highest rates of rehospitalization.

Comment: Retrospective study but results make intuitive sense. Low early follow-up rates suggest significant challenges for change.

How do you respond to the case manager’s statement about PCP follow-up?

A. It might keep him from getting rehospitalized
B. Follow up appointments are only helpful for patients with COPD exacerbations
C. It really doesn’t make any difference
D. It reduces medication errors after discharge
E. Snort derisively and ask if that’s what they do at “Mayo”
Summary

Definitely

1) Give preference to intermittent, high-dose furosemide strategy for CHF hospitalizations.
2) Advocate for hospital protocols that substitute daily PIV assessment for scheduled PIV change

Consider

1) Treating patients with cocaine-associated chest pain with β-blockers when clinically indicated
2) Scheduling PCP follow-up for CHF exacerbations within 7 days to reduce re-admissions.
Update in Hospital Medicine
A 59 year-old man with a long history of alcohol abuse is admitted to your non-teaching service with severe acute pancreatitis.

On hospital day 4, despite optimal treatment, he continues to have severe pain and cannot take any POs. It seems likely he will be NPO for at least a few more days.

How do you manage his nutrition at this point?
How do you manage nutrition in patients with severe acute pancreatitis?

A. I think you have to go with total parenteral nutrition (TPN) since he is still having pain.
B. I would continue the dextrose in the IVFs for now.
C. I would place a feeding tube and start enteral nutrition.
D. Isn’t there some surgical saying about “don’t mess with the pancreas”? No way I’m feeding this guy.
Question: Is TPN or enteral nutrition better in patients hospitalized with acute pancreatitis?

Design: Systematic review, 8 RCTs, 348 pts; outcomes of mortality, organ failure, infection, others.

- Timing varied – just defined as “early”
- At or below Ligament of Treitz

### Results

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Effect of EN v TPN (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td></td>
</tr>
<tr>
<td>Multi-organ failure*</td>
<td></td>
</tr>
<tr>
<td>Surgical intervention*</td>
<td></td>
</tr>
<tr>
<td>Systemic infection*</td>
<td></td>
</tr>
</tbody>
</table>

* Moderate quality evidence

# Results

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Effect of EN v TPN (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>RR 0.50 (0.28-0.91)</td>
</tr>
<tr>
<td>Multi-organ failure*</td>
<td>RR 0.55 (0.37-0.81)</td>
</tr>
<tr>
<td>Surgical intervention*</td>
<td>RR 0.46 (0.26-0.82)</td>
</tr>
<tr>
<td>Systemic infection*</td>
<td>RR 0.39 (0.23-0.65)</td>
</tr>
</tbody>
</table>

- Trend toward shorter LOS (-2.4 days)
- In severe acute pancreatitis, EN with lower mortality (RR 0.18)

* Moderate quality evidence

**TPN v Enteral Nutrition for Pancreatitis**

**Question:** Is TPN or enteral nutrition better in patients hospitalized with acute pancreatitis?

**Design:** Systematic review, 8 RCTs, 348 pts; outcomes of mortality, organ failure, infection, others.

**Conclusion:** Enteral nutrition seems better than TPN for nearly all clinical outcomes;

**Comments:** Syst-review but only 350 patients Yet, seems enteral nutrition is better, supported by guidelines; Should prob be post-pancreas (IR placed/)

Timing is not clear – if 5-7 expected?

How do you manage nutrition in patients with severe acute pancreatitis?

A. I think you have to go with total parenteral nutrition (TPN) since he is still having pain.
B. I would continue the dextrose in the IVFs for now.
C. I would place a feeding tube and start enteral nutrition.
D. Isn’t there some surgical saying about “don’t mess with the pancreas”? No way I’m feeding this guy.
Case Continued

Two weeks later the patient is finally improved after getting enteral feeding and supportive care.

It turns out this is his fourth admission in the last 6 months and you’re worried about possible readmission. You wonder if he has any of the most common risk factors for readmission.
In a retrospective, observational study at one AMC, the 30-day readmission rate was 17%. In multivariate analysis, black race, inpatient narcotic use or steroids, cancer, renal failure, CHF, and weight loss were associated with readmission.

This adds to a growing body of evidence which helps identify and target patients at high risk of readmission.

It’s been a busy couple of days and you seem to be leaving later and later each day. This non-teaching service is rough . . .

It’s already 4:30pm, and your day is a blur of box checking, answering pages, and fixing printer jams...you haven’t even started the 12 notes you have to write . . .

You ask yourself... just where did the day go?
Just where did the day go?

A. I had 4 family meetings and 2 transitions to comfort care.
B. Care coordination rounds took forever today…
C. Your pager went off 32 times in one hour; you know because you were counting.
D. Do you think it is easy to make sure all of my notes are “high-complexity?”
E. I guess the 2 hours on Facebook could have been better spent.
Hospitalists- Time in Motion

Question: How do hospitalists spend their time and is this affected by patient volumes?

Design: Time motion, observational study, 24 admitting hospitalists for 2 shifts each, non-teaching service, single AMC

- Activities recorded in 1 minute increments
- Multitasking recorded

## Results

<table>
<thead>
<tr>
<th>Main Task Category</th>
<th>Percentage of time</th>
<th>Est # of Time/ shift</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use of Electronic Medical Record</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Communication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct Patient Care</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Professional Development</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Travel</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Personal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Avg 10 hr shift; 13 pt encounters
## Results

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<thead>
<tr>
<th>Main Task Category</th>
<th>Percentage of time</th>
<th>Est # of Time/ shift</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use of Electronic Medical Record</td>
<td>34%</td>
<td>4 hours</td>
</tr>
<tr>
<td>Communication</td>
<td>25.9%</td>
<td>3 hours</td>
</tr>
<tr>
<td>Direct Patient Care</td>
<td>17.4%</td>
<td>2 hours</td>
</tr>
<tr>
<td>Professional Development</td>
<td>6.5%</td>
<td>40 minutes</td>
</tr>
<tr>
<td>Travel</td>
<td>6.2%</td>
<td>40 minutes</td>
</tr>
<tr>
<td>Personal</td>
<td>5.7%</td>
<td>40 minutes</td>
</tr>
</tbody>
</table>

Avg 10 hr shift; 13 pt encounters

## Results

<table>
<thead>
<tr>
<th>Major Category Breakdown</th>
<th>% of Main Category</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EMR</strong></td>
<td></td>
</tr>
<tr>
<td>Documentation</td>
<td></td>
</tr>
<tr>
<td>Orders</td>
<td>58.4%</td>
</tr>
<tr>
<td>Read/reviewing</td>
<td>20.2%</td>
</tr>
<tr>
<td>Other</td>
<td>19.4 %</td>
</tr>
<tr>
<td></td>
<td>2.1 %</td>
</tr>
<tr>
<td><strong>Communication</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Outgoing call</strong></td>
<td></td>
</tr>
<tr>
<td>Face to face</td>
<td>36.9%</td>
</tr>
<tr>
<td>Incoming call</td>
<td>28.1%</td>
</tr>
<tr>
<td>Sending page</td>
<td>14.4%</td>
</tr>
<tr>
<td>Rounds</td>
<td>8.6%</td>
</tr>
<tr>
<td>Receiving page</td>
<td>3.8%</td>
</tr>
<tr>
<td>Email</td>
<td>3.4%</td>
</tr>
<tr>
<td>Reviewing page</td>
<td>2.9%</td>
</tr>
<tr>
<td>Fax</td>
<td>1.8%</td>
</tr>
<tr>
<td></td>
<td>0.1%</td>
</tr>
</tbody>
</table>
Hospitalists: Where did the day go?

Question: How do hospitalists spend their time and is this affected by patient volumes?

Design: Time motion, observational study, 24 admitting hospitalists for 2 shifts each.

Conclusion: Most time spent using the EMR and communicating with other staff.

Time spent in direct care of pts low, but not affected by pt load.

Comments: High burden of documentation and communication central to hospitalist role; Suggests better technology might improve efficiency.

Just where did the day go?

A. I had 4 family meetings and 2 transitions to comfort care.
B. Care coordination rounds took forever today…
C. Your pager went off 32 times in one hour, you know because you were counting.
D. Do you think it is easy to make sure all of my notes are “high-complexity?”
E. I guess the 2 hours on Facebook could have been better spent.

Update in Hospital Medicine
Seven days after discharge, the patient returns with acute shortness of breath, pleuritic chest pain, and hypoxia. The ED calls very early in the work-up and you have a moderate-to-high pre-test probability for pulmonary embolism (He had refused DVT prophylaxis).

You are waiting for the CT scan and wonder about anti-coagulation before the diagnostic test . . .
Should the patient be anti-coagulated before the CT scan for PE?

A. Yes, if there is a high pre-test probability.
B. No, we should wait for the confirmatory test since there is a risk of bleeding.
C. It depends.
D. Hey, wait, CT scan? Where’s the d-dimer? I thought this was an evidence-based talk – I want my d-dimer!
Early v Late Anticoagulation in Acute Pulmonary Embolism (PE)

Question: Does timing of anticoagulation impact outcomes in acute PE?

Design: Retrospective cohort study, 400 adults diagnosed w/ PE in ED, single center; all OK to get anti-coagulation; all got heparin

Outcomes:
• “Early” = heparin in the ED vs. not
• Therapeutic aPTT in 24 hours vs. not

## Results

- 70% of pts got heparin in ED (“early”)
- 85.8% of pts had therapeutic aPTT in 24 hours

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Early</th>
<th>Late</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-day mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>aPTT ≤24</th>
<th>aPTT &gt;24</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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Results

- 70% of pts got heparin in ED (“early”)
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<th>Late</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital mortality</td>
<td>1.4%</td>
<td>6.7%</td>
<td>.009</td>
</tr>
<tr>
<td>30-day mortality</td>
<td>4.4%</td>
<td>15.3%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>aPTT ≤24</th>
<th>aPTT &gt;24</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital mortality</td>
<td>1.5%</td>
<td>5.6%</td>
<td>0.093</td>
</tr>
<tr>
<td>30-day mortality</td>
<td>5.6%</td>
<td>14.8%</td>
<td>0.037</td>
</tr>
</tbody>
</table>

### Results

- Performed multiple logistic regression
- Propensity scoring

<table>
<thead>
<tr>
<th>Heparin in ED vs. Not</th>
<th>Adj. Odds Ratio**</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-day mortality</td>
<td></td>
</tr>
</tbody>
</table>

** With matching & propensity scoring

Smith SB *et al.* *Chest* 2010; 137:1382.
Results

- Performed multiple logistic regression
- Propensity scoring

<table>
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<th>Heparin in ED vs. Not</th>
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<tbody>
<tr>
<td>30-day mortality</td>
<td>0.22 (0.08-0.60)</td>
</tr>
</tbody>
</table>

** With matching & propensity scoring

Early v Late Anticoagulation in PE

Question: Does timing of anticoagulation impact outcomes in acute PE?

Design: Retrospective cohort study, 400 adults w/ PE in ED, single center; all OK to get anti-coagulation

Conclusion: Early + earlier therapeutic anti-coag in newly diag. PE assoc. w/ lower mortality

Comments: Single center, retrospective, confounders, define “early,” heparin, etc. Prior studies showed better outcomes; Expert guidelines suggest treatment for pts w/ high probability while awaiting testing

Should the patient be anti-coagulated before the CT scan for PE?

A. Yes, if there is a high pre-test probability.
B. No, we should wait for the confirmatory test since there is a risk of bleeding.
C. It depends.
D. Hey, wait, CT scan? Where’s the d-dimer? I thought this was an evidence-based talk – I want my d-dimer!
A systematic review including 18 RCTs in 1463 hospitalized post-operative patients (not enough general medicine patients) revealed graduated compression stockings (GCS) decreased VTE risk.

The risk reduction was particularly strong when combined with another prophylaxis method (aspirin, heparin).

- GCS alone: OR 0.35 (95% CI: 0.26-0.47, P<0.001)
- GCS+other: OR 0.25 (0.17-0.36, P<0.001)
Summary

**Definitely**
1) Support high-tech approaches to improving documentation and communication efficiency.
2) Anticoagulate early in acute PE if indicated.
3) Use compression stockings to help prevent DVT in post-operative patients.

**Consider**
1) Using enteral nutrition in acute pancreatitis, especially if severe.
2) Identifying patients who are at high-risk of readmission.
You are rounding with your team and the intern presents a frail 82 year-old man with metastatic colon cancer who presented with shortness of breath and confusion. The team thinks he has pneumonia or a line infection.

His pressure has been dropping over the last few hours despite intravenous fluids and you need to start a pressor to maintain his blood pressure.

The intern asks, “I know we’re trying to be evidence-based so what pressor should we start?”
How do you respond? What pressor should be started in this patient with septic shock?

A. Dopamine.
B. Phenylephrine (Neosynephrine®).
C. Norepinephrine (Levophed® = “Leave-’em-dead”).
D. Vasopressin.
E. Pressors? Seriously? How about another P-word? How about palliative care?
Question: In pts with shock, what is the optimal first-line vasopressor agent, dopamine or norepinephrine?

Design: Multicenter, RCT, 1679 pt with shock; dopamine vs. norepi at standard doses.

- Shock = MAP < 70mmHg despite IVF + hypoperfusion
- Septic shock (62%), Cardiogenic (17%), Hypovolemic (16%)

## Results

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Norepi</th>
<th>Dopamine</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality (28 d)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality (12 mo)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shock resolution</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arrhythmia</td>
<td></td>
<td></td>
<td></td>
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## Results

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<tbody>
<tr>
<td>Mortality (28 d)</td>
<td>48.5%</td>
<td>52.5%</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality (12 mo)</td>
<td>63.0%</td>
<td>65.9%</td>
<td>NS</td>
</tr>
<tr>
<td>Shock resolution</td>
<td>6.0hrs</td>
<td>6.3hrs</td>
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</tr>
<tr>
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<td>63.0%</td>
<td>65.9%</td>
<td>NS</td>
</tr>
<tr>
<td>Shock resolution</td>
<td>6.0hrs</td>
<td>6.3hrs</td>
<td>NS</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>12.4%</td>
<td>24.1%</td>
<td>P&lt;0.001</td>
</tr>
</tbody>
</table>

Arrhythmias were mostly atrial fibrillation.
Cardiogenic shock mortality higher with dopamine.

## Dopamine vs. Norepinephrine

<table>
<thead>
<tr>
<th>Question:</th>
<th>In pts with shock, what is the optimal first-line vasopressor agent, dopamine or norepinephrine?</th>
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</thead>
<tbody>
<tr>
<td>Design:</td>
<td>Multicenter, RCT, 1679 pt with shock; dopamine vs. norepi at standard doses.</td>
</tr>
<tr>
<td>Conclusion:</td>
<td>No diff in 28-day mortality; no difference in other clinical outcomes (clinical, 12mo mort);</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias much more common w/ dopamine; dopamine worse for cardiogenic</td>
</tr>
<tr>
<td>Comments:</td>
<td>Well done, large study, diverse population; Prob should use norepi as first-line agent;</td>
</tr>
<tr>
<td></td>
<td>Work with ED/ICU providers.</td>
</tr>
</tbody>
</table>

How do you respond? What pressor should be started in this patient with septic shock?

A. Dopamine.
B. Phenylephrine (Neosynephrine®).
C. Norepinephrine (Levophed® = “Leave-’em-dead”).
D. Vasopressin.
E. Pressors? Seriously? How about another P-word? How about palliative care?
You recommend starting with norepinephrine.

The patient is ultimately diagnosed with staph aureus bacteremia from pneumonia. Forty-eight hours later, despite vancomycin he remains febrile with positive blood cultures.

When you suggest getting an ID consult, the R3, rolling his eyes, says, “Ok, whatever.”
A. Look, I’ll be honest, I’m just a little uncomfortable managing this case.

B. You know, I think this is a really good case for the ID fellow to see.

C. There is actually some evidence that patients with *S aureus* bacteremia do better with an ID consult.

D. Did you just roll your eyes? Really? I mean, did you just want to kill this patient? Seriously . . . .
How do you respond to the resident?

D. Did you just want to kill this patient? Go right ahead . . . I have to say, I am so sick and tired of you duty hours-trained residents thinking you’re all that. Do you know how hard I worked? We were qzero when I was a resident. Do you hear me? Qzero! We drew our own blood, took our own vitals, did gram stains, walked up hill to work in the snow both ways. You all have it easy with your uptodate, iphone, ipad, ibrains . . . why don’t you just take your cocky self right back to internship until you learn some real medicine . . .
ID Consult in Staph Bacteremia

Question: What is the value of an ID consult in patients with *S. aureus* bacteremia?

Design: Prospective, single center cohort study; 341 pts w/ *S. aureus* bacteremia over 2 years; multivariable analysis;

- MRSA in 54%
- Healthcare-assoc in 57%
- Hospital-acquired in 32%

### Results

- A total of 111 (33%) had an ID consult
- Overall one-year mortality was 41%
- ICU admit, cirrhosis, age assoc w/ mortality

<table>
<thead>
<tr>
<th>ID consult vs. No</th>
<th>Adj. Hazard Ratio**</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-day mortality</td>
<td></td>
</tr>
</tbody>
</table>

* Co-morbidities & degree of illness

Results

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</tr>
</thead>
<tbody>
<tr>
<td>28-day mortality</td>
<td>0.44 (0.22-0.89)</td>
</tr>
</tbody>
</table>

* Co-morbidities & degree of illness

### Results

<table>
<thead>
<tr>
<th>Process</th>
<th>ID Consult</th>
<th>No ID Consult</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct Antibiotics</td>
<td>90%</td>
<td>79%</td>
<td>0.01</td>
</tr>
<tr>
<td>Correct Duration</td>
<td>81%</td>
<td>29%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TEE Performed</td>
<td>34%</td>
<td>8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Retained Focus</td>
<td>13%</td>
<td>15%</td>
<td>0.50</td>
</tr>
</tbody>
</table>

ID Consult in Staph Bacteremia

**Question:** What is the value of an ID consult in patients with *S. aureus* bacteremia?

**Design:** Prospective, single center cohort study; 341 pts w/ *S aureus* bacteremia over 2 years; multivariable analysis;

**Conclusion:** Mortality assoc. w/ staph bacteremia is high; ID consultation lower 28d mort; improved processes of care

**Comments:** Single institution, confounders;

ID consults are good in *S aureus* bacteremia;
three other studies with similar results

Likely should have lower threshold for consult

How do you respond to the resident?

A. Look, I’ll be honest, I’m just a little uncomfortable managing this case.
B. You know, I think this is a really good case for the ID fellow to see.
C. There is actually some evidence that patients with *S aureus* bacteremia do better with an ID consult.
D. Did you just roll your eyes? Really? I mean, did you just want to kill this patient? Seriously . . . .
Case Presentation

Unfortunately, the patient does not do well. Despite all therapies, he progresses to maximum ventilation, renal replacement therapy, and 3 vasopressors in the ICU.

You find his daughter at the bedside and discuss his code status. She wants to know what his chances of survival to home are if he has a cardiopulmonary arrest.
In a large retrospective database of nearly 50,000 patients who had a cardiopulmonary arrest in the ICU (2000-2008), those on pressors were 55% less likely to survive to discharge.

The absolute rate of discharge to home in these patients was 3.9%. Age and ventilation lowered this even further.

You calmly and clearly explain that given his age, underlying metastatic cancer, malnutrition, multi-organ dysfunction, and pressors he is unlikely to survive.

She says, “No way – he’s going to make it. He’s going to pull through.”

You inquire, “Why do you feel so strongly he will survive given how sick he is?”
How might she respond to your question?

A. He’ll make it – he’s strong. He’s a fighter.
B. Well, today he looked right at me and squeezed my hand – he’s wants to live.
C. He’s been this sick before and pulled through – he’ll do it again.
D. I just feel it, you know. I just know he’s going to make it.
E. It was hard to come up with an appropriate “joke” answer for this one. But, if you’re the one person that has to answer E, go right ahead.
Perceptions of Prognosis

Question: What sources of knowledge do surrogate decision makers use to estimate prognosis?

Design: Prospective, structured interviews; single ICU acad med center; 179 surrogate decision makers; day 3-5 of ICU stay

- Written estimates of chance of survival
- “What has made you think this is his/her prognosis?”

Results

- Only 2% (3/179) relied exclusively on physician’s prognosis
- Only 47% reported at least part of their estimates were based on MD prognosis

1) Intrinsic qualities/will to live
2) Physical appearance
3) Unique history of survival
4) Bedside presence improves survival
5) Optimism, intuition, faith

### Perceptions of Prognosis

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<td>Conclusion:</td>
<td>Many factors help establish prognosis: perception of pts strength, physical appearance, own presence, faith/optimism</td>
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<td>Small, qualitative study; Prognosis is complex; our input is limited Help us understand when “they (surrogates) just don’t get it” Consider asking about specifics</td>
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Summary

Definitely

1) Understand how surrogates will view prognosis in their loved ones.

Consider

1) Using norepinephrine as your first-line agent in patients with shock.

2) Infectious diseases consultation in patients with *S aureus* bacteremia.

3) Patients in the ICU on pressors have a low chance of survival if they code.
Update in Hospital Medicine 2011

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Sponsored by the SGIM Academic Hospitalist Taskforce