OXYMORONIC MEDICINE

A Handbook for Hospitalists and House Staff

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This handbook is intended to help Bridgeport Hospital trainees take better care of their patients. It is not entirely evidence-based medical advice and should complement conventional resource materials.

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INTRODUCTION

Doctors must learn, contextualize and apply an immense quantity of ever-increasing medical science. "Common sense" refers to an innate ability to solve problems; as a good plumber might examine a leak, hypothesize the most likely sources and attack the possibilities sequentially (based on pre-test probabilities) until the problem is solved. Common sense, however, is an oxymoron. It is not common and is undervalued (probably because it includes the word "common").

In the early stages of medical education, students and residents are overwhelmed by the information – medical and logistic – that they must begin to master. Not surprisingly, then, they tend to gravitate to studying facts first. But inevitably they spend more and more of their intellectual efforts making sense of and learning to harness the facts.

While knowledge and good communications skills are essential, the arsenal of extraordinary clinicians includes a hefty dose of common sense. In daily rounds, trainees inevitably realize that solving most patient problems does NOT require extraordinary intelligence or book-knowledge. There is no advanced calculus involved. Most day-to-day problems require a decent understanding of pathophysiology and an organized approach that can be explained in terms that a bright plumber can easily understand. Perhaps the "master" in "master physician," "master plumber" or master-of-any-vocation refers to superlative common sense problem-solving skills coupled with a library of experience. Encyclopedic knowledge may help but it is not necessary. In some doctors, it actually gets in the way.

Since there are many excellent textbooks already in print, this handbook offers something different: a paucity of "facts" and more common sense ways of considering medical problems. Since it reviews most common inpatient problems, it is a roadmap to becoming an excellent houseofficer or hospitalist.

MEDICAL PROBLEM-SOLVING

Data Acquisition

In order to help a patient, you must gather information including history, physical examination and tests. Each step builds on the last. A well-taken history, raises hypotheses with expected examination findings. So we begin the examination focusing on organ systems and/or seeking abnormalities that will support or refute various differential diagnoses. Similarly, diagnostic testing is performed to support or refute principle differential diagnoses derived from the history and physical.

These skills take a life-time to master. You can only improve if you focus on honing them, every day. Yes, you need to learn pathophysiology just to know the right questions to ask. But, the patient will almost always lead you to the "right" answer if you're willing to invest the time to perform a good history and physical. Let the patient tell her story and be on guard that you don't lead them to your conclusions.

How we gather and synthesize data is predicated on: knowledge, experience and problem-solving skills. Information can be gathered in a chaotic fashion – casting a broad net of unrelated questions, hoping to sift out to a correct answer. This is a recipe for frustration and poor care. Alternatively, information can be gathered in a methodical manner; where a well-versed medical observer can know exactly what you're thinking as you gently guide the patient, testing various hypotheses. There are many different constructs for categorizing/gathering medical information. Some attack a problem by thinking about organ systems, others by "kingdoms of disease" (i.e. infectious, inflammatory, neoplastic, traumatic, idiopathic, metabolic, etc.). In the case of medical history-taking, each additional piece of information can be thought of as a "test." From the moment you walk into a room, your mind processes information. You see a patient of a particular age, gender, race and level of comfort/consciousness – all of which affect the pre-test likelihoods of various conditions. You begin with your introduction and the open-ended "what brings you here today?" As the patient's story unfolds, each additional piece of information, changes likelihoods of various conditions in the differential diagnosis. Some patients are able to provide most of the required information with little or no prompting. Others need gentle guidance – as you clarify, filling in the blanks of their history required to test this or that working hypothesis. As a general rule, if a patient complains of pain, the clinician should illicit information about all 7 elements of pain (quality, quantity, duration, position, radiation, exacerbating, alleviating). It is also very important to clarify the temporal "trajectory" and pattern of symptoms. Did they start in a crescendo fashion and continue to the current moment unabated? Did the slowly build over time? Did they wax and wane? When was the last time you felt completely normal? The pace and pattern of symptoms often helps narrow differential diagnoses. Review of systems germane to organs and/or disease processes that might explain the chief complaint should be probed while acquiring the history of present illness (not postponed to be done *en block* during the "review of systems"). By the time you get to the physical examination, you should have gathered enough information to have included the correct diagnosis in your primary (top 5) differential diagnoses, more than 90% of the time. Physical examination findings and laboratories less often reveal a new, entirely unexpected diagnosis *vis a vis* the chief complaint. Rather, more often, the exam and labs confirm or refute hypotheses derived from the complete history.

This level of sophistication can only be achieved if you become a student of Medicine. See as many patients as possible, listen to them carefully, read as much as possible about their likely diagnoses and then watch carefully as more information informs an ultimate diagnosis. Too often, in our fragmented medical system, residents haven't time to follow-up to learn the results of tests and final diagnoses. They complete an intake, order some tests and the encounter becomes a dead-end.

Reassembling the data for write-up/presentation

Interestingly, the process of recounting a patient's history (in written or oral form) mimics the history-taking process. The presenter knows the history, exam, labs and clinical course to date and so has abundant information that is not initially available to the listener or reader. Accordingly, an excellent presentation is one in which the presenter methodically produces and tests various plausible hypotheses – and with each additional piece of information builds a case for the one or two diagnoses he/she thinks is most appropriate based on ALL the information. Keep in mind that the presenter may not always have the right answer and the listener may "detect a signal" that was missed entirely by the presenter. This happens sometimes when a trainee presents to an experienced faculty member. Moreover, the presenter may possess incomplete information because he "didn't know the right questions to ask," erred during data acquisition, or misrepresented facts to coincide with his hypothesis.

However, as one compiles more and more experience, knowledge and facility with medical reasoning, the likelihood of "missing signal" entirely decreases. (It never disappears – so humility is an attribute that both protects patients and prepares us for our inevitable human errors).

Heuristics is the term used to describe this process of discovery – the cognitive gymnastics we use to solve a problem. There are some common pitfalls that should be avoided. "Premature closure" of medical reasoning is a common error. This occurs when, due to our previous experience and knowledge, we insist on a particular diagnosis and ignore information that refutes our working hypotheses. The interim diagnosis should be considered a working hypothesis that must be tested. Each piece of additional information either supports or refutes the working hypothesis until the weight of evidence supports that diagnosis or leads to alternatives. Since Medicine is an inexact science, sometimes the definitive answer can take days, weeks, months or years to develop. Some diseases, lupus for example, may take years to full develop, meeting "diagnostic criteria." Avoiding the common pitfall of premature closure requires that we consistently question the validity of our primary diagnoses until the data unequivocally support a conclusion. Premature closure is an error of ignorance or, worse, arrogance.

Another common error is to ignore Okham's razor also referred to as the "rule of parsimony." Always try to explain a symptom-sign complex with one diagnosis before resorting to multiple co-existent diagnoses.

A good friend in his early 40's recently recounted his medical history. He presented to his internist who referred to a gastroenterologist with hematochezia that had caused iron deficiency anemia. He was scoped from above and below and nothing but hemorrhoids was found. For months, doctors insisted that the bleeding must be caused by occult esophageal reflux disease (he had mild intermittent heartburn, like most of us) and treated him with antacids. "Over and over, I told them that when I had bowel movements, it often turned the water deep red!" Finally, after a year, they listened and realized that bleeding hemorrhoids were the cause. If you take the time to listen to the patient, he will often literally tell you the answer. Labs may confirm a diagnosis, but more often than not the correct diagnosis comes from a careful history and physical.

If you revere and hone your history-taking and physical examination skills, you will begin on the road to becoming a master clinician. Insist that your teachers watch you in the room with patients to help you improve these skills. Ask patients to provide you with feedback on how you might improve. Allow yourself to enjoy being with the patient. It may take a little more time, but it is a great investment in your proficiency and satisfaction.

Also, often-neglected information that frequently informs patient care is the course of disease from the moment a patient enters the healthcare system to when you see them. Scrutinize the medical records of the emergency technicians in the field, in transit and the emergency department. Crucial historical information and pathophysiologic data demonstrate the pace/course of illness gleaned by investing 5-10 minutes to examine these records. Similarly, whenever possible, review outpatient and old inpatient records that sometimes provide information that can expedite and simplify the diagnostic process, and reduce redundant unnecessary testing (and risk for the patient).

WHETHER AND WHERE TO ADMIT

Patients should be admitted to hospital if:

1. Modalities (intravenous fluids/medications, monitoring) that cannot be administered as an outpatient are absolutely required, or,
2. The disease is likely to progress to life-threatening severity despite treatment, or,
3. The disease may progress and the patient isn't reliable enough to come back with worsening and/or is unlikely to comply with treatment, eventuating in a life-threatening situation.

Once the decision is made to admit a patient to hospital, you must determine whether care can be administered safely on the hospital ward, or whether a special unit is required. Most hospitals have two levels of special care units: "step-down" and "intensive care (ICU)." These beds are differentiated from ward beds because they have continuous monitoring of heart rate and pulse oximetry, and higher nurse:patient ratios (either 1:3 or 1:2) that allow nurses to follow other physiologic parameters (e.g. blood pressure, airway, labs, neurologic status) more frequently than can be provided by a nurse with 5-8 patients dispersed in as many hospital rooms. Usually ICU is reserved for patients with cardiopulmonary instability requiring pressors, mechanical ventilation, rapidly evolving neurologic events, or critical acid-base-electrolyte abnormalities that require

very careful correction and threaten life. Step-down beds offer 1:3 or 1:4 nursing and monitoring, but usually do not include patients with acute cardiopulmonary failure. Each hospital has criteria for how to use monitored beds guided by these general principles. Another simple litmus test is to ask yourself where you'd want your mother to receive her care, remembering that ICU's and step-downs have risks (multi-drug resistant pathogens, invasive monitoring etc.) to weigh against the benefits of vigilance/early intervention.

Irrespective, perhaps one of the most important skills that differentiate an outstanding houseofficer from a mediocre or poor houseofficer is ability to detect (and act against) severe acute illness. Patients with immediately life-threatening disorders may be very frightened-appearing or obtunded, but they are rarely calm and non-distressed. Almost always they have signs of catechol excess. The faster the heart rate, the more severe is the stress. Sinus tachycardia (≥100/min) warrants concern. If blood pressure is high with the fast heart rate, consider pain/anxiety, toxins (e.g. cocaine/amphetamine intoxication) and withdrawal syndromes. If blood pressure is normal or low with tachycardia, then either stroke volume (i.e. the heart is sick) or vascular resistance (e.g. sepsis) is low. Both situations are life-threatening and demand a prompt diagnostic and therapeutic approach, with careful monitoring/watchfulness until the storm passes. Respiratory instability is also a sign of life-threatening disease. An adult breathing >30-35 breaths/minute is very ill. Their work of breathing is excessive and likely to cause respiratory fatigue/failure and CP-arrest if not reversed. Those with oxygen saturation less than 95% on 100% non-rebreather facemask are also at risk. Those who have mental status changes and impaired airway protective mechanisms (cough, gag, swallowing) are at particular risk of hospital morbidity and mortality. One sign of the incompetent airway that is often overlooked is audible gurgling of secretions above the glottis during passive breathing or speech. Patients with normal defense mechanisms would expectorate and swallow upper airway secretions. Those who gurgle are at risk of those secretions dripping back down into the lower airway/lung, causing lung injury or pneumonia.

Exemplary physicians don't leave the bedside until either the life-threatening phase of illness has passed OR they actuate safety mechanisms (ICU, step-down, or telemetry, with frequent nursing based on severity).

Admission (and transfer orders) should include the standard ADCVANDISL mneumonic (i.e. A=admit to Dr. X; D=diagnosis; C=condition; V=vitals checked at what frequency and alarm values for call MD; A=allergies; N=nursing orders; D=diet and drugs/medications; I=intravenous fluids; S=special tests/imaging; L=laboratory test orders). Special attention should be paid to ensure that any medications taken at home (or in the area from which the patient is transferred) are continued if appropriate. In addition to being right about which tests to orders and medicines to give, rigorous attention to nursing orders can make the difference between life and death. Hospitals are understaffed and nurses are overworked. If a new patient has even a small potential of instability, consider ordering more frequent nursing checks until the patient demonstrates stability. For potentially unstable admissions, for all ICU/step-down transfers to the wards, and/or for ward patients who have newly become unstable, I insist on the following:

"Nursing checks of vital signs:
Every 2 hours for 6 hours, then,
Every 3 hours for 9 hours, then,
Every 4 hours for 12 hours, then,
Every 6 hours for 18 hours, then per routine (which is usually every 8 hours).
Call doctor for: heart rate <50, >120/min, systolic pressure <90, >160 mmHg, respiratory rate <6, >30/min, temperature <96.5°, >100.9°F."

If there is any potential for hypoxemia, I request checks of pulse oximetry ever 6-12 hours and ask to be called for <92% saturation. This approach "bridges" potentially unstable patients through the first 2 days on the wards – to ensure that they are checked at frequent intervals for a sufficient duration to reduce the likelihood of a missed decompensation. While ward nurses don't like the very frequent checks, the extra work is not continued indefinitely and most will bow to this simple safety, patient-first approach.

Although the terms "evidence-based care pathways" and "practice guidelines" are demonized by some clinicians, if they are constructed carefully, using available evidence, they should be actuated. Numerous evidence-based patient safety interventions can be integrated in "order

sets" (e.g. head of bed 30 degrees, DVT prophylaxis, etc.). The advent of computerized medical records and order-entry will only increase availability of pathways and queues to apply evidence to help patients. When local and national experts have taken the time to prepare care pathways, it is arrogant to believe "you know better" especially if you haven't taken the time to examine the literature carefully *and* critically. Most importantly, not even the most experienced clinician remembers every nuance of care; pathways are a way to provide reminders of the evidence so "the little details" aren't missed. Trainees should still understand the rationale for each step/therapy in care pathways. In the end, such tools promote the quality of care, expedite patients' recovery, reduce iatrogenesis, and minimize unnecessary testing.

Additionally, every day and especially during transfers and discharges, we should examine medication lists to: a. ensure that only medications that are absolutely required are being administered, b. consider drug-drug interactions, c. consider drug-organ interactions. The process of reconciling medications is also very important. For example, when patients are admitted to hospital, as many of their home medications should be continued as is safe/feasible. When patients are discharged, compare and "reconcile" pre-admission to hospital medications. It makes no sense to discharge a patient on a new ACE-inhibitor if he/she has been taking (and has supplies of) another similar but not identical drug. It also makes no sense to discharge patients on medications that require frequent dosing if you know they are non-compliant and/or unable to comply. If they can't afford the medications you've prescribed, they simply won't fill the prescription and may rebound to hospital with serious complications because of your poor forethought.

Prescribing the correct medication must include measures to enhance patients' compliance. Taking the time to explain medications, (the why's and how's), may enlist the patient's efforts in his/her self-care and provide you with important information about logistic problems that could prevent therapeutic success. This sounds like social science, but being a good doctor requires taking the time to maximize the likelihood of success when the patient leaves the hospital (see below). Moreover, this elevates the discussion to one of true informed consent. It is the most ethical model for practicing medicine and, by requiring more time spent communicating, increases satisfaction and connection (of both patient and doctor).

ROUTINE PRACTICES THAT IMPROVE OUTCOMES AND RESPECT PATIENTS

The following measures have been recommended by the Agency for Healthcare Research and Quality (AHRQ; "Making Healthcare Safer;" http://www.ahrq.gov/clinic/ptsafety/pdf/front.pdf). This federal agency examines the medical evidence to prescribe practices that improve patient outcomes. However, they have no enforcement power, so many of their excellent recommendations are not routinely followed. Overwhelming evidence suggests that large numbers of patients (upwards of 100,000/year) die in hospitals each year because of medical errors (i.e. committed mistakes) AND failure to apply simple evidence-based methods of prevention (i.e. mistakes of omission).

Hand-washing

Even in excellent hospitals, physicians and staff fail to wash their hands, religiously, before touching patients. The primary reason for the high prevalence of infections with multiple drug resistant bacteria (e.g. *Staphylococcus, Enterococcus* and *Pseudomonas*) and *Clostridium difficile* is insufficient hand-washing. A purist might argue that the initial genesis of these microbes is overuse of antibiotics (which is true). But the reason for the unacceptably high prevalence results from transmission by caregivers. I teach my trainees that they are the "vectors" of nosocomial infection. To liken ourselves to mosquitoes or ticks is an apt metaphor, as the scourge of nosocomial infection is caused by our carelessness.

Thromboprophylaxis

One of the great mysteries of hospital medicine and perhaps the strongest indictment of physicians' neglect of quality/safety (i.e. "the performance gap" coined by the Institute of Medicine) is failure to thromboprophylax all at-risk hospitalized patients. Data and consensus guidelines have been available for more than two decades. Yet at many hospitals, the rate of proper thromboprophylaxis is less than 50% (of those reporting – the rates are likely even less in hospitals unwilling to monitor and/or publish their results). The list of indications for thromboprophylaxis is long (cancer, severe lung disease, MI, stroke, major surgical – especially orthopedic – procedures, etc.). Since most patients admitted to hospital are non-ambulatory and many have evidence-based indications, thromoboprophylaxis must become a "default treatment" i.e. everyone gets it unless the patient is ambulatory. While there may be medical contraindications to heparin (e.g. intracranial

hemorrhage, heparin-induced thrombocytopenia), there are practically none to pneumatic compression boots. Until hospitals create a default approach to thromboprophylaxis, with or without computerized order entry, physicians must take responsibility. *Thromboembolic death of a patient who has not been prophylaxed is inexcusable.* And if doing the right thing isn't enough, courts in several jurisdictions have awarded damages in excess of $1 million to harmed plaintiffs when this measure is overlooked by caregivers.

Informed Consent

In the U.S., the guiding medical ethical principle is respect of patients' autonomy. Informed consent is a process that *ends* with signing a paper (the consent form is the least important component). It involves explaining to a patient who has capacity (or to surrogate decision-makers when the patient is incapable of understanding) the nature of a treatment, it's risks, benefits and alternatives. Any clinician can determine whether a patient has capacity to make healthcare decisions. Capacity includes a patient's ability to choose and communicate, understand risks/benefits/alternatives, offer reasoning for choices and make choices that are consistent with their values. In clinical practice, hospitalized patients sign a general consent-to-treat that is extrapolated to cover routine therapies; it would be logistically unfeasible to obtain informed consent for every single treatment (e.g. imagine having to get consent for every potassium replacement). However, some treatments and all invasive procedures have sufficient risks that they must be formally consented and documented in the medical record. As a general rule, for informed consent (and any time you need to know whether a patient understands what you've explained) you should use "play-back" i.e. after you're done, ask the patient to tell it back to you in their own words. This allows you to recognize misunderstandings and to explain again if the patient doesn't get it. Irrespective, except in life-threatening emergencies, seeking informed consent is **not** optional.

Informed consent may be even more important when prescribing therapies. Place yourself for a moment in the role of patient. Will you be more likely to comply with a prescribed medication if the doctor says "just do it" or if he explains why its important with risks, benefits and alternatives? Informed consent is the predicate for the ethical practice of medicine and it is likely to maximize the likelihood of patients' compliance (if other factors, like expense etc., allow them to comply).

Along these lines, patients also have the right to choose the nature of their deaths. Many physicians say they're either too busy or too uncomfortable to have frank discussions with patients. Instead many patients receive aggressive life-sustaining therapies like CPR and mechanical ventilation by default – never having had the opportunity to opt-in or –out. The Patient Self-Determination Act requires hospitals only to ask whether patients have advance directives, and does not require caregivers to ask hospitalized patients what life-sustaining measures they do or don't want. Nowadays, if a patient is ill enough to be hospitalized, he/she is usually ill enough for their heart or lungs to fail. It may be inconvenient – and take time – but it is the only way to respect patients' autonomy.

Perioperative beta-blockade

Patients with precedent coronary disease or diabetes may benefit from administration of pre-operative beta-blockers to reduce the likelihood that perioperative catechol surges will cause myocardial infarction. The evidence isn't overwhelming, but it is a form of prophylaxis with few complications. Current data suggest that outcomes are only improved if beta-blockers are titrated, in high-risk individuals, to achieve a heart rate of 55-70/min and avoiding hypotension. Future studies should help determine precisely which populations benefit most.

Tight glucose control

One prospective, randomized study demonstrated that critically ill (surgical) patients benefit markedly from tight control of glucose (80-110 mg/dl) with insulin drips. A second study using this protocol in medical critically ill patients did not demonstrate improved mortality (though patients tended to do better). Although the benefits may be less in wards patients and the hypothesis has not yet been studied, it is not unreasonable to actuate protocols to more carefully control hyperglycemia in these patients. It is not practical to insulin drip ward patients (requires significant resources – nursing, laboratory, etc.) in the absence of strong evidence. Nonetheless, physicians should treat hyperglycemia more aggressively in hospitalized patients. A goal of 110-150 mg/dl is reasonable, albeit not evidence-based. Most experts now agree that "sliding scale insulin" a time-honored tradition is not supported by evidence. Basal-bolus treatment i.e. long-acting insulin once or twice daily and pre-prandial short-acting insulin, has become a *de facto* standard of care. In general, when insulin requirements are not known or in flux due to stress/steroids/poor oral intake etc., short-acting insulin can be delivered by a sliding scale. But once the variables determining glycemia stabilize,

a portion of the total daily insulin requirement (roughly 1/3) should be given as long-acting insulin with the remainder is given as short-acting peri-prandial doses. Patients' normal convalescent doses of insulin can be used as a foundation for estimating acute-care needs, taking into consideration glycemic effectors (more short-acting is often required if a patient is eating, stressed and/or receiving corticosteroids; less is often required while a patient is receiving little/no nutrition).

Precautions for the elderly

As chairman of my hospital's mortality review, I had the opportunity to investigate and understand common reasons for cardiopulmonary (CP-) arrest. Although ACLS training dwells on primary arrhythmic reasons for CP-arrest, respiratory reasons may be just as common. Amongst respiratory reasons, airway insufficiency is a leading cause. Many patients, especially the elderly, those with neurologic disease (especially CVA) and those receiving narcotics may have varying degrees of airway insufficiency. Airway insufficiency means that airway reflexes (glottic function, gag, cough etc.) that normally keep saliva and food from dripping over the glottis into the lungs are attenuated or absent. This can be heard as a gurgling sound (of secretions above and/or below the glottis that normal patients would expectorate) as the patient breathes or talks. Micro- and/or macro-aspiration promote hypoxemia and increased work of breathing, which, if severe, may cause critical acidemia and respiratory or cardiac arrest. Prevention includes maintaining the head of the patient's bed at 30° whenever there is a question regarding airway competence, minimizing medications that attenuate airway reflexes (i.e. narcotics) and performing swallowing evaluations before orally feeding whenever micro- or macro-aspiration are suspected.

Just as aspiration is a major problem of the hospitalized elderly, so too is delirium. Hospitals are perilous for the fragile elderly. If the medical problem that brought them to hospital isn't enough, the omni-present risk of hospital infection, medications (with their own side-effects, plus medication- and organ-interactions), falls and the simple change of environment (often without their eyeglasses and hearing aides), conspire to cause delirium. Unless the causes are reversed rapidly, delirium can persist and is associated with poor outcomes. Prevention reduces the prevalence of delirium and mortality of patients. It is particularly important to minimize medications. Moreover when it is absolutely necessary to add a new medication, interactions with other medications and effects of organ dysfunctions on metabolism must be considered.

The Yale Delirium Prevention trial (N Engl J Med 1999; 340: 669-76) demonstrated 5% fewer cases of delirium when caregivers provided the following to at-risk patients: frequent orientation/activities, earliest possible mobilization, minimal use of psychoactive medications, assurance of adequate (night-time) sleep, eyeglasses/hearing aides (as needed) and sufficient hydration. Another trial (J Am Geriatri Soc 2001;49: 516-22) demonstrated that geriatric consultation with special attention to adequate brain perfusion, fluid/electrolyte balance, pain control, minimal psychoactive medication use, bowel and bladder function, nutrition, early mobilization, and environmental stimulation reduced the frequency of delirium by 18%. There is no excuse not to actuate these simple counter-measures – or to consult a geriatrician to do so. If my parents get admitted to hospital, I will insist on daily suggestions from a geriatrician. They are particularly attentive to prevention and adept at geriatric pharmacology.

However, one thing is clear: many elderly are drugged into a state of passivity. These medications have profound side-effects and newer tranquilizers – that were FDA approved for schizophrenia but NOT for behavior modification of the elderly – have contributed to numerous unintended consequences including weight gain, insulin resistance/diabetes, aspiration/pneumonia, respiratory failure/need for an artificial airway and possibly obstructive sleep apnea. Many of these medications are impacted by renal and hepatic dysfunction and by drug-drug interactions that vary considerably in the elderly. So even if a dose is OK on one day, it may be a gross overdose when the patient become dehydrated or gets a new medication. Medications should be the last resort in treatment of agitation/delirium and when organic reasons are addressed all efforts should be made to reduce or discontinue these powerful and potentially harmful agents. Frequent drug holidays should be mandatory once risky levels of agitation have been controlled with medications (otherwise there is no way to know whether reversal of organic precipitants is sufficient long-term treatment and whether drug levels are building to dangerous levels).

Physical and Occupational Therapy

There is now abundant and ever-increasing data to suggest that bedrest causes proteolysis of muscles; so-called disuse atrophy. This process is expedited substantially by infections and other cytokine-driven illnesses (i.e. the vast majority of med-surg patients). There is also

abundant evidence that physical therapy has a powerful impact on patients' outcomes. For example, amongst critically ill ventilated patients, daily physical therapy (all the way up to and including walking patients with their ventilators) allowed more than twice as many to go home vs. to a nursing home. Similarly, amongst the hospitalized elderly, outcomes were substantially improved when patients received ambulation therapy in the first 48 hours. There is rarely a reason for a patient to remain at strict bedrest; and we – doctors – must order and insist that (most) patients are gotten up to the chair and receive regular (at least thrice weekly) physical therapy. It makes no sense to save a patient from an organic illness only to unnecessarily doom them to a nursing home or home where they cannot function (or worse, fall to break their hip).

Nutrition

While there isn't a single randomized double blind study to suggest that starving patients is superior to feeding them, you also wouldn't jump out of an airplane without a parachute. Unfed patients become catabolic which impedes healing. Calculate caloric requirements using widely available formulae (e.g. Harris Benedict) – and if you're not good at math, simply send 2000-2500 kcal to the bedside each day. Don't assume the patient eats the food – calorie counts should be ordered unless trays are emptied. Use the prealbumin, understanding it's limitations in acute disease (e.g. false normal with steroids), to index sufficient nutrition and anabolism.

Testing

Daily laboratories are unnecessary for the majority of patients and contribute to morbidity in the form of iatrogenic anemia. Consider using pediatric specimen tubes to reduce the required volume of phlebotomy and if you carefully consider whether a test is really needed, you'll likely find that you can reduce dramatically the number of unnecessary tests in your patients. Similarly, chest radiographs are often over-ordered. Except when used to ensure that hardware is appropriately placed, chest radiographs should be ordered only when a physiologic change (e.g. worsening hypoxemia or work of breathing) warrants.

Also, far too many patients have central lines placed for the convenience of caregivers rather than because other, safer, peripheral access is not possible. While very frequent blood draws (every 1-6 hours), need for intravascular pressure monitoring (during shock) and resuscitation of shock are reasonable indications, too often these risky devices are placed for convenience and left in for far too long. A way to "self-remind" is to include the day-count for all intravascular catheters and list the indication for that day in daily progress notes.

Sign-outs

Nowadays inpatient medical care is fragmented. Patients are often followed by many doctors, some who know little or nothing about them because of "on-call" systems. Accordingly, patients are vulnerable to errors associated with poor sign-outs or to in-action (the "let's wait until the patient's main doctor returns before acting" syndrome) that can promote harm. Since studies have demonstrated the odds of adverse events during cross-coverage are 5-6 times those during coverage by patients' primary doctors, the AHRQ lists detailed sign-outs as one method to promote patient safety. They cite a study in which a computerized system of sign-outs eliminated the excess odds of cross-coverage-related adverse events. *Common sense* dictates that all physicians caring for hospitalized patients should be thoroughly familiar with their problems and acute illnesses. The fact that we needed scientific evidence to prove it is telling. Yet sign-outs are NOT routine in some practices.

While not technically an issue of formal sign-outs – verbal communication is exceedingly important in Medicine. When something is really important you should both write an order AND contact the person who will actuate the order to ensure they know early of what your patient needs. Consultants should be contacted on the phone; you should be prepared to provide a brief description of the patient's course and the explicit clinical questions you're asking him/her to address. Similarly, when a life-death or time-sensitive order is written, you should speak with appropriate personnel (nurse, pharmacist, laboratory technician) to give them a "heads up." Too many important therapies are given too late because doctors simply write an order and leave, assuming that it will be effected rapidly. When something needs to be done rapidly, use your muscles of phonation and telephonic devices to ensure your patient gets what he needs, when he needs it.

Documentation and Handwriting

One of the most baffling facets of American Medicine is the illegibility of many medical notes and orders. There is little question that unintelligible notes lend little guidance to other clinicians who depend upon the medical record to provide care. Illegible orders are a common cause of adverse events. Since physicians are perfectly capable of writing neatly if they want, there can be only one explanation: that illegible

hand-writing is a mark of laziness and disregard for patient safety. The advent of computerized medical records may obviate some of the risks to patients – but it's not clear why physicians haven't required, of each other, that our written communications be decipherable.

Once you've committed to writing legibly, also see to it that the medical record is a complete narrative of a patient's progress during hospitalization. A detailed history & physical and daily progress notes/management plans that consider differential diagnosis, diagnostic plan and therapeutic plan are an absolute minimum requirement. However, the record should be reviewed at least twice a day to consider suggestions of other clinicians. Major decompensations should be described carefully in the hospital record in separate timed/dated updates. Such decompensations include: hemodynamic instability (e.g. high or low pressures or heart rates), respiratory compromise (e.g. tachypnea>30/min, increasing oxygen requirement to maintain oxygen saturations), neurologic events (e.g. seizures, new neurologic deficits or fluctuating levels of consciousness), new symptoms or signs (e.g. abdominal pain, nausea, vomiting, chest pain, etc.) or hypo-/hyperthermia. The patient should be interviewed and a new SOAP (subjective/objective/assessment/plan) note should be written for the new problem. The primary physician of record should be informed and have the opportunity to help formulate a diagnostic/therapeutic plan. In medical school, we were taught that "if it isn't documented it didn't happen." While due diligence may be done in the absence of documentation, the patient and those who review the record after you deserve a narrative description of inter-current events. From a medical-legal standpoint, good documentation demonstrates diligence and allows reviewers to understand exactly what you were thinking and doing. This honest and ethical approach also obviates the shortcomings of recall bias.

Transfers

When patients must leave care-units for tests, they are at particular risk of adverse events. There are mechanical risks (e.g. falls). Most routine transfer aides and radiology technicians are not trained to react to subtle signs of patient decompensation, and often radiology departments are distant from principal care areas and ill-equipped for cardiopulmonary resuscitation. Administration of medications, oxygen and fluids can be inadvertently interrupted – sometimes with life-threatening results. Patients may "disappear" into CT or MRI machines, where cardiopulmonary decompensation is not noticed until a full arrest. Accordingly, the onus is on the physician to ensure that patients "going on a road-trip" are sufficiently stable to go without additional safeguards. Unfortunately many hospitals do not have "fail-safe systems" to recognize mishaps before they lead to significant patient harm. So, in general, patients who are very tachypneic (>30/min) or ventilator-requiring, tachycardic (>120/min), hypotensive or pressor-requiring, and/or with significant psychomotor agitation should be accompanied by a nurse and/or physician for "road-trips." Continuous monitoring (pulse oximetry for respiratory instability and/or cardiac monitoring for cardiovascular instability) should be ordered if it is not a routine hospital safety net default for patients. Patients with mental status changes should never be left unattended, as they can fall to floor with severe consequences. Patients with significant work of breathing and/or coma should be considered for endotracheal intubation for airway protection during CT's or MRI's unless a physician is prepared to observe carefully and intervene. While all of this is only common sense, it is not routinely implemented.

Renal prophylaxis

Acute renal failure during hospitalization is often preventable and associated with increased mortality. Hypovolemia and under-resuscitated shock causes ATN. Prompt recognition and aggressive volume resuscitation (see next chapter) reduces the magnitude of perfusion-related renal insults. Nephrotoxic medications (e.g. aminoglycosides, acyclovir, amphotericin) should be used cautiously and only when necessary in patients with pre-existent or acute renal dysfunction. When nephrotoxic medications are absolutely necessary, hypovolemia – which potentiates toxicity – must be avoided. Contrast-induced nephropathy is also a potentially preventable complication. In patients with renal disease (acute or chronic) alternate methods of securing diagnoses should be used whenever possible. If radiographs requiring intravenous contrast are absolutely required, patients who are hypovolemic should receive volume expansion with 250-500 ml of normal saline just before administration of contrast. Finally, there is increasing evidence that the administration of n-acetyl-cysteine (NAC) before contrast (preferably several doses whenever possible) reduces nephrotoxicity and *may* attenuate mortality. Orally administered NAC has few complications, so the "up-side benefits" warrant strong consideration whenever nephrotoxic contrast agents are necessary.

Palliative Care

Palliative care is not just for the dying. It is a discipline whose purpose is to explore with patients their care predilections (understanding that choices may change with time, so it is an on-going dialogue) AND provide symptom relief beyond treatment of underlying illnesses. Discussions with patients and families regarding these issues require a special expertise and time that most busy practitioners don't have. Symptom management – especially of pain – is similarly complex and requires additional training and patience. In my opinion, every patient with end-stage heart or lung disease, end-stage dementia, later stages of (and certainly "terminal") cancer or who is leaving intensive care after prolonged illness should be followed by palliative care, to provide ongoing end-of-life counsel and to assist in symptom management.

The "Extras"

Simply addressing patients' organic problems is a daunting challenge. It requires knowledge, perseverance and excellent problem-solving skills. However, truly exemplary care addresses other "peripheral," but equally important, issues simultaneously. First, acute illness often impacts patients' physical fitness, and in some cases prevents patients from "simple disposition to home." They may require visiting nurses or "meals on wheel," outpatient physical therapy or home oxygen to "bridge" them to wellness. Accordingly, whenever the acute illness is likely to erode patients' physical or physiologic reserve, it is worthwhile beginning physical therapy early during hospitalization. There is ever-increasing data to support that the investment in physical therapy, as tolerated, throughout hospitalization expedites patients' overall rehabilitation.

Disposition planners – case managers and/or social workers – should be consulted early during hospitalization to begin the very time-consuming process of creating a discharge "soft-landing" for patients. By involving these colleagues early, they can follow the evolution of the patients' illnesses and begin putting into place interventions at home, to facilitate ongoing recovery or apply for transfer to rehabilitative or skilled nursing facilities. If these plans are not begun early during hospitalization, supports may not be ready when the patient is ready for discharge, thereby prolonging his/her stay and risk (through another day of exposure to iatrogenic complications). While these professionals may help facilitate "soft-landings" for patients after discharge, ***doctors*** remain primarily responsible for ensuring that plans are comprehensive and well-conceived.

Acute illness often has psychosocial ramifications for patients. Understanding the patient's view and the impact of disease on his/her life can help clinicians to fashion long-term solutions that are more likely to contribute to long-term wellness. For example, illness may cause depression. It may impact the relationship of a patient with loved ones. It may affect one's livelihood. While we may not be experts in "psychosocial issues," we owe it to our patients to explore these areas and provide personal or consultative support when appropriate.

Often we will send the patient out with a list of 10 or more medications to take, with nary a thought about the patient's ability to comply with the regimen or even afford the medications. Success of any therapeutic plan is highly dependent upon the degree to which patients understand their illness and therapies, and are invested in compliance. In this age of "rapid hospital throughput," we spend insufficient time attending to these details and thereby neglect our patients. We spend huge amounts of time, energy and money fixing their acute problems, but sometimes drop the ball laying the groundwork for them to continue recovery and remain well.

CARDIOVASCULAR PROBLEMS

About tachycardia

Tachycardia (≥100/min) is extraordinarily common in hospitalized patients – so common, that residents often overlook its importance. Tachycardia *always* means something; most often either abnormal cardiac conduction and/or excessive circulating catecholamines. A regular, wide complex tachycardia usually means ventricular tachycardia resulting from injured or scarred myocardium, electrolyte deficiencies, hypoxemia or catechol excess (or combinations thereof). Narrow complex tachycardias are most often caused by excess catechols. Consider first pain, anxiety, withdrawal syndromes and CHF when tachycardia is accompanied by hypertension. Consider first infection, CHF, pre-shock/shock and pulmonary embolus when tachycardia is accompanied by normotension or hypotension. Irrespective, tachycardia should always be explained since it could signal a problem that requires specific therapy to treat the underlying cause.

Approach to the patient with shock

Shock may be defined as insufficient substrate delivery by the circulatory system to meet global demands. The result is tissue injury, usually attributable to tissue hypoxia (septic shock may be a notable exception) resulting in end-organ dysfunction if shock persists. A useful mantra is that during shock cells die and if enough cells die, organs die and if enough organs die, the patient dies. It is difficult for trainees to appreciate that dawdling during resuscitation of shock promotes morbidity and mortality.

The bedside approach proposed by Jesse Hall and Larry Wood (University of Chicago) empowers clinicians to attenuate mortality from shock by rapidly formulating a hypothesis and beginning aggressive resuscitation. Low blood pressure (usually systolic less than 90 mmHg) is the clinical sign that brings shock to the attention of caregivers. Oliguria, mental status changes, hypo- or hyperthermia and sometimes signs of hypoperfusion (cold, mottled extremities) are other seminal findings that may precede or accompany overt hypotension. However, tachycardia and a low normal blood pressure may be a sign of early shock. A blood pressure of <90 mmHg does not necessarily mean shock (e.g. young healthy women) and a blood pressure of >90 mmHg does not preclude it (e.g. an elderly vasculopath who ordinarily has a blood pressure of 190 mmHg and drops to 100 mmHg rapidly). With these caveats in mind, hypotension is the most common manifestation that prompts recognition and resuscitative efforts.

Any patient with shock requires immediate attention to the "ABC's" that is "airway, breathing and circulation." Since patients with shock are often under-perfusing their brains they may lose airway reflexes and be at risk of aspiration. The threshold for endotracheal intubation for airway protection must be low (any gurgling, gagging, large retained oral secretions, snoring/upper obstruction requires placement of an artificial airway). Insofar as shock causes metabolic acidosis, it increases the work of breathing while the diaphragms are not receiving adequate supply, so fatigue and respiratory arrest leading to a deadly double acidosis is a major risk. So endotracheal intubation and ventilation for excessive work of breathing is indicated (don't wait for a blood gas if the patient with shock is breathing above 35/min for more than 10-15 minutes and the underlying cause is not rapidly reversible). Once the patient is intubated, great care must be taken to avoid dynamic hyperinflation that can impede venous return and contribute to hypotension (bagging no faster than 30/min and once on the ventilator use a square inspiratory wave at 60 L/min). A rhythm strip or ECG is indicated in most patients with shock to ensure that arrhythmia requiring CPR (see below) is not present. Simultaneously, we attend to the "C" for circulation.

The U of C algorithm for circulatory resuscitation (see Figure 1 next page) applies, at the bedside, the basic physiologic relationship: blood pressure=cardiac output times vascular resistance. We cannot "lay hands on" the vascular resistance, but we can examine for cardiac output. So the first question to ask when assessing a patient with shock is: ***"Is it high output or low output hypotension."*** This can be determined very rapidly just by examining the extremities of the patient: **warm with bounding pulse and brisk capillary refill is high output hypotension (i.e. sepsis) until proven otherwise**. Bedside management consists of: 1. Administration of crystalloid, wide open until urine output increases, CVP is 10-12 cmH2O or the patient develops signs of congestion (basilar crackles and/or decrements of the PaO2/FiO2 ratio), 2. Immediate administration of antibiotics for the most likely sources of infection, the most likely causative agents (based on site and patient's origin i.e. home vs. recently hospitalized/ institutionalized), and the patient's immuno-competence (i.e. broader coverage is required with increasing immuno-incompetence), and, 3. Identification and drainage of loculated foci of infection (drain abscesses, remove or drain behind stones etc.). For severe sepsis, septic shock and even simple sepsis in an immuno-incompetent patient, the onus is on the clinician to "rule out" a drainable focus of infection. Administration of activated protein C in non-bleeding patients with severe sepsis (APACHE>24) has been shown in one study to attenuate mortality. Since relative adrenal insufficiency may complicate severe sepsis and secondary adrenal insufficiency is quite common in the general population, replacement of the adrenal axis with hydrocortisone 80 mg thrice daily is reasonable when hypotension requires vasopressors to maintain SBP>90 mmHg. An ACTH stimulation test can be performed before administering hydrocortisone, or for very unstable patients the first dose of steroid can be dexamethasone, which does not affect the ACTH stimulation test results. Hydrocortisone can be stopped if the axis tests normal.

Diseases other than sepsis (e.g. bowel ischemia, pancreatitis, thermal injuries, active inflammatory disease, acute on chronic liver failure) that cause systemic inflammatory response syndrome (SIRS) may affect the circulation in a similar fashion – through cytokine effects of nitric

oxide-mediated vasodilation – but are much less common. Other very rare causes of high output hypotension include wet beri beri, Paget's Disease, AV malformations, and chronic salicylate use.

If the patient is in a low-output state, the next question to ask is: ***"Is the heart full or empty."*** Heart overfilled low-output hypotension is cardiogenic shock. Since there are two sides of the heart, examine them separately. When the left heart is overfilled, patients have signs of pulmonary edema often with coincident jugular venous distension. The most common causes of acute left heart failure are acute coronary syndrome (see approach below), arrhythmia, and valve dysfunction in that order of frequency. The approach to myocardial infarction is discussed below – but fluid must be administered more judiciously when left ventricular MI with shock is accompanied by pulmonary edema. If the lungs are clear, but the right heart is over-filled (i.e. increased jugular vein pressure) there are three main possibilities: right-sided myocardial infarction, pulmonary embolus and tamponade. These conditions benefit from fluid resuscitation – though overfilling of pulmonary embolus can exacerbate right heart ischemia.

If the patient is in a low-output, heart-empty state, hypovolemic shock requires prompt attention to possible sites of major bleeding (GI track, retroperitoneum, groin, thorax) in case a surgical or other invasive intervention is required. Therapy includes securing 2-3, short, large bore (≤18 gauge) intravenous catheters and administration of crystalloid (normal saline) "wide open," with the assistance of medical students and/or interns whose triceps are well suited to squeezing the bags as they observe proper resuscitation. Coagulapathies should be corrected: FFP for elevated PT or PTT and platelet transfusions for thrombocytopenia and/or when the patient is taking a medication like aspirin that blocks platelet function. Alternative diagnoses include post-intubation hypotension (occurring in roughly 30% of patients), adrenal insufficiency/failure, anaphylaxis and neurogenic shock that result from spinal cord injuries.

Nearly all forms of shock, save left heart infarction with severe pulmonary edema, benefit from bolus ("wide open") resuscitation with crystalloid until either the shock is reversed or the patient develops a decreasing PaO2/FiO2 ratio. Another approach is to administer "boluses" (i.e. normal saline wide open) in 250-1000 ml aliquots until the shock reverses. Even subtle reductions of oxygenation can signal leaking into the lung, which can limit fluid resuscitation. I am a bit more judicious in fluid administration if the patient has severe pneumonia and/or lung-shunt-related hypoxemia (seen as whiteness on the chest radiograph) before resuscitation because in most cases, shunt will worsen with aggressive fluid resuscitation. In these situations, I begin inotropes – usually starting with dopamine - earlier (after 500 ml to 1 L administered). Unless the patient responds to 1-2 liters of crystalloid infusion and/or the patient remains hypotensive despite "wide open" fluids, dopamine is an elegant medication for initial resuscitation. In lower doses (up to 10 mcg/kg/min), beta-agonist effects predominate increasing cardiac output through its inotropic and chronotropic effects. As doses increase, above 10 mcg/kg/min, increasing alpha-agonist effect squeezes the blood vessels. It should be used cautiously, if at all, for an empty system especially when hypovolemia is accompanied by ventricular hypertrophy since tachycardia can worsen shock in an underfilled system. Keep in mind that relationship of heart rate and stroke volume is an upside-down-U-shaped curve. Stroke volume and cardiac output increases with heart rate to a point, but then starts to decrease when diastolic filling time is insufficient. The "heart rate of diminishing returns" can be substantially lower for patients with diastolic (e.g. hypertrophs) or systolic dysfunction. Since many patients have heart dysfunction, they do not tolerate excessive tachycardias. Some vasoactive agents (epinephrine>dopamine>norepinephrine>dobutatmine) are positive chronotropes. If bedside personnel are not very careful, they may exceed the "best" heart rate after which blood pressures can paradoxically fall. I have reviewed cases when the response to worsening hypotension was to increase one of these medications and well-meaning personnel were unwittingly promoting shock. As a general rule, there is little to be gained from kicking the heart to rates much above 120/minute, and in some patients (e.g. extreme hypertrophs and those with critical mitral or aortic stenosis) >100/min may be excessive.

The bedside approach described above (i.e. high output or low output?, then, heart full/heart empty?) allows diagnosis within minutes followed by an immediate, aggressive hypothesis-driven trial of therapy. However, in 20-30% of cases after 30-60 minutes, the patient is not improving. In such cases, a mixed form of shock should be considered. Echocardiography is a very powerful noninvasive tool in these situations. A vigorously contracting heart, that empties to end-systolic obliteration signals hypovolemia and rules out ventricular dysfunction as the sole reason for shock. Segmental wall motion abnormalities, coupled with ECG changes, suggest that coronary ischemia is contributing. Valvular failure is usually evident if present. A thin, dilated right ventricle with paradoxical septal motion suggests acute right heart syndrome (pulmonary

embolus). Tamponade can also be diagnosed. In the past, pulmonary artery catheterization, to measure right-sided pressures and to infer left-sided pressures and cardiac output, was commonly used. However, this invasive therapy does not improve outcomes and is seldom indicated now. Instead, examination of arterial pulse waveforms provides a reliable means of predicting patients' preload responsiveness. Large respiratory-phasic variability (i.e. pulsus paradoxus) >15 mmHg of systolic pressure or pulse pressure predicts that the patient will benefit from additional volume infusions if not otherwise contraindicated by increasing hypoxemia. Unfortunately, the utility of this finding ONLY applies to mechanically ventilated patients who are breathing comfortably and synchronously with the ventilator.

Although one study suggests that filling to a CVP=10-12 mmHg and using inotropes and blood to address mixed venous hypoxemia may attenuate mortality in septic shock, oximetric central lines are expensive and may not be necessary. We usually resuscitate with femoral vein lines (to avoid a pneumothorax that might further promote shock/death) to a CVP of 8-10 mmHg (10-12 mmHg in ventilated patients). When femoral vein catheters are used they should be removed after no more than 48 hours or when the patient stabilizes. Although there are some caveats and this is a bit arbitrary, it is probably the rapidity of the resuscitation that is most important and attaining this moderate right-sided filling probably precludes gross hypovolemia for most patients. Arbitrary transfusions are not appropriate. But when a patient is exsanguinating, blood (with or without FFP/platelets) is used as preferred volume. Note that as a general rule it is better to administer too much than too little fluid – underperfused organs promote multiple organ failure and death while congestive heart failure is more easily treatable. Since my housestaff has begun a "surgical" approach to resuscitation, shock-related multiple organ failure is extremely uncommon relative to similar hospitals without this approach. The U of C approach saves lives.

Approach to the patient with acute coronary syndrome (ACS)

Myocardial ischemia results when insufficient oxygen is delivered to cardiomyocytes to meet their metabolic demands. The cells are damaged, but not killed. Myocardial infarction (MI) describes when either insufficient or no oxygen is delivered to the myocytes for sufficient time that their membranes become leaky and the cells die. Although myocytes may be thus injured beyond a partially closed vessel (i.e. "demand" ischemia or infarction), MI results more commonly from rupture of or thrombus formation on a previously non-critical plaque. The World Health Organization definition is simple i.e. two of the following:

1. Symptoms: Chest pain or pressure, epigastric discomfort, new shortness of breath at rest, shoulder heaviness or pain, jaw pain, left arm pain. Men experience classic "substernal chest pressure" more often than women who may experience ACS without chest pain and, instead with shortness of breath or other symptoms listed above.
2. Electrocardiographic evidence of injury including ST segment elevations or depressions in two contiguous leads, and/or T-wave abnormalities (new changes of T-wave vector and/or biphasic T-waves compared to previous or to normal ECG when previous are unavailable).
3. Enzymatic evidence of myocyte membrane leak – Either increased troponins (6-8 hours following the onset of symptoms) or increased absolute creatine phosphokinase (CPK) with increased MB band (≥5%) and simultaneous rise and/or fall of both. N.B.: Enzymes are most useful when patients have ACS i.e. the syndrome. Their utility as a marker of an acute epicardial coronary event decreases when patients are asymptomatic. Elevated troponins almost always connote damaged myocardium. When patients present with elevated troponins and renal failure (creatinine>2.0 mg/dl), it is impossible to time when membrane injury/leak occurred unless sequential levels increase. Troponins don't rise *because* of renal failure alone, rather renal failure affects troponin kinetics/clearance. In patients with renal failure and an elevated troponin, sequential troponinins and/or CPK's can be used to determine whether myocyte injury has occurred in the previous 6-12 hours or is evolving in hospital. When troponins are negative on admission, there is no need for serial CPK's unless rhabdomyolysis is suspected. A second troponin, drawn 6-8 hours later, will determine *post hoc* whether the syndrome is accompanied by myocyte membrane damage.

Perhaps the most common error is to NOT treat while waiting for confirmatory tests. An MI is literally shock of cardiomyocytes. Cells are dying with time and all efforts should be made to minimize cell death, thereby limiting patients' morbidity and mortality, until the diagnosis is "ruled

out." As a resident, my CCU attending's mantra was "time is muscle" and if it's your mother's heart muscle that's dying, you would want aggressive treatment while waiting to confirm the diagnosis.

Diagnosis of myocardial cell damage/leak is confirmed by sequential collection of enzymes (at 6-8 hour intervals) and electrocardiograms on admission, at or before one hour, as a patient's symptoms change, and at 6-8 hour intervals until there are no interval changes i.e. "evolution." A very useful practice is to document the grade of the patient's symptoms (e.g. 5/10 chest pain) and the systolic blood pressure on the top of each ECG as it is obtained. This allows caregivers to integrate symptoms, double product (heart rate times systolic blood pressure indexes myocardial oxygen demand) and ECG changes.

The cornerstones of management are to: a. Re-open a closed or partially closed vessel, and, b. Reduce myocardial oxygen consumption. So, notify an invasive cardiologist early, so that he/she can grease the wheels of the cath lab. For ST-segment elevation MI, thrombolysis can be considered if rapid catheterization is not possible and not otherwise contraindicated. Reopening the closed vessel should be achieved as rapidly as possible and this is a measure of the quality of Cardiology programs and hospitals.

Irrespective, every patient who isn't hemorrhaging should chew an aspirin immediately upon recognition of ACS. Enough supplemental oxygen should be administered to maintain pulse oximetry saturations of 98-100%. Small amounts of morphine (1-3 mg) and nitroglycerin (sublingual, topical or intravenous) can be given to reduce pain, which reduces myocyte-damaging catechol excess. Patients should remain at bedrest and thromboprophylaxis is imperative. Myocardial oxygen consumption should be driven down pharmacologically. Since heart rate is the greatest contributor to O2-demand, beta-blockers should be considered in hypertensive tachycardic patients to titrate the heart rate to 50-60/minute whenever possible (i.e. so long as the systolic pressure remains>100 mmHg). However, recent data from a very large study ("COMMIT") suggested that too much beta-blocker can reduce benefits in the first day by causing iatrogenic hypotension. Accordingly, beta-blockade must be performed very carefully, avoiding hypotension during the first 24 hours. An elegant method to achieve acute beta-blockade is to administer 2.5-5.0 mg of metoprolol every 5-10 minutes until the goal heart rate of 50-60/min is reached or the systolic blood pressure drops below 110 mmHg. It can then be continued orally at doses of 12.5-25 mg every 8 hours, with holding parameters for hypotension and bradycardia. If patients present with severe congestive heart failure, beta-blockers must be used more carefully, usually after some preload reduction (with nitrates/furosemide), but they still should be added, even in patients with coexistent obstructive lung disease. Only in patients with cardiogenic shock or high-degree heart block are beta-blockers withheld. Aspirin and beta-blockers are the medications that have the largest incremental benefit in reducing mortality from MI. If the systolic blood pressure remains more than 100 mmHg after titrating the heart rate down to 60/minute, then add either a "test-dose" of captopril (6.25 mg orally) followed by 12.5 mg thrice daily or simply start with 12.5 mg thrice daily. Then if there's still excess (systolic>100 mmHg) blood pressure left over, write an order to titrate the systolic pressure to 100 mg with a nitroglycerin drip. Minute-to-minute control of blood pressure requires an ICU or step-down bed; whereas many uncomplicated MI's or "rule outs" with a low clinical suspicion can be managed in a well-staffed, (heart-) monitored unit. In the COMMIT study, the addition of clopidogrel to aspirin (starting from hospital admission) was found to benefit patients with MI. The addition of cholesterol-lowering medications, most often statins, should also be initiated before hospital discharge.

There is an increasing appreciation that the pathophysiology of coronary disease in many women differs from the "typical" paradigm (i.e. of a dynamic, occluding plaque) of most men. Many women experience cardiac ischemia/infarction without substernal chest pressure. "Atypical" symptoms (e.g. isolated shortness of breath, epigastric discomfort, neck/jaw/shoulder/back discomfort, or nausea) are more common in women presenting with ACS and "angiogram-negative" coronary disease is more prevalent. Some of these women have macrovascular "smooth" plaques and others have small vessel dysfunction that is not readily apparent on conventional angiograms. Some early data suggest that statins, beta-blockers and tricyclic antidepressants may attenuate morbidity in these patients, but this is currently an area of very active research.

Another form of myocardial syndrome that can occasionally mimic MI is Takatsubo cardiomyopathy in which catechols (due to stress or catecholaminergic acute illnesses) cause chest pain, acute "outpouching" of the left ventricular apex and leaked troponins, all with patent coronary arteries. Treatment leads to complete reversal and includes beta-blockers and management of conditions that increase catechols.

Approach to the patient with congestive heart failure

Congestive heart failure (CHF) is amongst the most common diseases that precipitate the need for hospital admission. The most common cause of CHF is left heart failure from left ventricular dysfunction, valvular dysfunction or arrhythmias. Left ventricular dysfunction can be qualified as principally diastolic (normal ejection fraction and systolic function) or systolic (which is often accompanied by diastolic dysfunction). Echocardiography has become the principle method used to confirm the clinical diagnosis and explore pathogenesis.

The most common causes of purely diastolic dysfunction include hypertension, aortic stenosis and obesity (with or without obstructive sleep apnea), while idiopathic hypertrophic cardiomyopathy (IHSS) and infiltrative cardiomyopathies (e.g. sarcoid, amyloid) are more rare. Diastolic dysfunction also *precedes* systolic dysfunction during coronary ischemia. In general, patients with diastolic dysfunction require higher than normal filling pressures to empty and do poorly with excessive tachycardia (i.e. they require longer diastolic filling times to prime their noncompliant ventricle that are precluded by heart rates >>100/min). Common causes of systolic dysfunction include coronary ischemia/myocardial infarction (usually with segmental wall-motion abnormalities), alcoholic cardiomyopathy (diffusely dilated heart), and end-stage dilation due to chronic hypertension and/or valvular disease (e.g. aortic stenosis/regurgitation or mitral regurgitation). Although murmurs and arrhythmias may be apparent on presentation, they may also occur intermittently to cause CHF, and disappear by the time of presentation (i.e. ischemia-related valvular dysfunction or paroxysmal tachyarrhythmias). The cause should be defined for all patients presenting with acute heart failure.

Right heart failure (i.e. with elevated jugular venous pressure and peripheral edema) is most commonly caused by left heart failure. However, causes of pure right heart failure (cor pulmonale) with normal left heart and clear lungs on examination include: chronic lung and/or chest-wall disease (e.g. obesity and kyphoscoliosis), pulmonary vascular disease including pulmonary embolism and obstructive sleep apnea.

The most common symptoms of CHF are shortness of breath, cough, exercise intolerance, orthopnea and paroxysmal nocturnal dyspnea. The most common signs include those of catechol excess: tachypnea, tachycardia, hypertension (except when systolic dysfunction is extreme). Adventitious breath sounds vary with the severity of the disease. Wheezes without crackles may occur in early left heart failure (with high pulmonary vein pressures and before flooding of the lung). It is vital to remember that "all that wheezes is not asthma" as many patients with chronic obstructive lung disease have unrecognized heart failure as the reason for their wheezing. Crackles in the lung bases connote mild CHF and the higher up crackles are heard, the more severe is the failure. Heart sounds may include an S3 when there is diastolic or systolic dysfunction while an S4 is usually present with diastolic dysfunction (often from hypertension). Valvular diseases can be detected by auscultation of murmurs, while arrhythmias are usually obvious by clinical examination (irregular heartbeat) and/or electrocardiogram.

As mentioned above, the type (left, right or both) and mechanism of CHF should be defined to promote proper management. Most patients with CHF from left ventricular dysfunction, valvular dysfunction and arrhythmias benefit from oxygen (to return saturations to >90%), diuretics (furosemide 20-100 mg depending upon kidney function) and nitrates. Greater care must be taken during preload reduction of patients with pure right heart failure (or aortic stenosis), because sudden reductions in filling pressure occasionally precipitate severe hypotension. When patients are hypertensive with left ventricular failure and creatinine clearance>40 mg/min, angiotensin converting enzyme inhibitors (or angiotensin receptor blockers) can be started during hospitalization and have been shown to improve long-term survival and quality of life. If renal function is considerably impaired, hydralazine and nitrates is another combination that improves long-term outcomes and has mortality benefit above and beyond beta-blockers and ACE inhibition in African Americans. Beta-blockers have also been shown to impact long-term mortality from CHF. Carvedilol or other beta-blockers should used in patients with left ventricular systolic dysfunction and CHF. Aldosterone antagonists (spironolactone and eplerenone) improve mortality in both chronic CHF and LV dysfunction after acute myocardial infarction, while digoxin has shown only benefit in reducing the frequency of hospitalization for CHF. Potassium levels should be monitored in patients receiving both spironolactone and renin-angiotensin blockers.

If cardiac ischemia contributes to CHF, the patient should be ruled out for MI (see above) and receive an aspirin and beta-blocker once oxygenation is improved (O2 saturation>90% on 40-50% facemask; see management of ACS above).

Arrhythmias may cause or result from CHF – often it is difficult to tell. Irrespective, timely management is essential. Atrial fibrillation/flutter is commonly associated with CHF and requires rate control. Digoxin, which takes 12 hours to begin blocking A-V conduction,

is time-honored, but beta-blocker (e.g. metoprolol) or calcium channel blocker (e.g. diltiazem) are more potent negative chronotropes that can be carefully titrated to slow AV-conduction without causing hypotension.

When left ventricular failure is accompanied by hypotension and/or poor end-organ perfusion (e.g. increasing azotemia), 2-3 days of "dobutamine tune up" may be used to promote clearance of pulmonary edema and promote more favorable hemodynamics. Although the predominant salutatory effects are realized during the infusion, some patients experience prolonged symptomatic relief even after cessation of the drip.

Treatment of pure right heart failure includes administration of oxygen and identification/treatment of reversible causes (e.g. PE, bronchospasm, tracheobronchitis, pneumonia, right ventricular infarction). In the initial 6-12 hours, preload reducing agents (diuretics, nitrates and narcotics) must be used cautiously, if at all, since the right ventricle may be, initially, preload sensitive. Note that RV afterload reduction with oxygen administration promotes increased venous return. Patients with co-existent left ventricular diastolic dysfunction often develop pulmonary edema *as a complication* (roughly 24-48 hours after admission) if diuresis is insufficient. Note that oxygen should be administered cautiously in patients with chronic respiratory acidosis. Roughly 10% of patients with chronic hypercapnia will develop hyperoxia-induced acute hypercapnia. If this iatrogenic reason for hypercapnia is not recognized patients may be unnecessary intubated. Unlike true hypercapnic respiratory failure when patients are breathing rapidly shallowly and are uncomfortable, hyperoxia- (and narcotic-) induced hypercapnia causes the patient to be comfortable or even somnolent. It can be reversed or prevented by titrating the fraction of inspired oxygen to maintain the oxygen saturation around 90-92% in this group of patients.

A "quick" primer on hypertension

This section will emphasize management of patients with primary hypertension (as secondary hypertension requires appropriate work-up to treat underlying causes). Many studies have now concluded that the long-term goals of blood pressure management should be a systolic pressure of <120 mmHg and diastolic <80 mmHg. Although blood pressure must be reduced slowly and carefully in patients with severe chronic hypertension and cerebrovascular accident or with malignant hypertension, most patients tolerate treatment to outpatient goals over 2-3 days of hospital care. Current evidence suggests that diuretics should be considered for nearly all patients (hydrochlorathiazide or chlorthalidone each day with potassium replacement therapy). However, since most patients do NOT reach targets with diuretic alone, the choice of a second agent will depend heavily on patients' demographics and coexistent diseases. The general rule for treating hypertension is to maximize one drug before adding another (to maximize the likelihood of compliance). Occasionally dose-related side-effects may also provide a "ceiling" for a given agent. Nowadays, the plethora of medications and evidence provides clinicians with the opportunity to customize and maximize benefits for patients. For example, hypertension in African-Americans responds better to calcium channel antagonists than to ACE-inhibitors or beta-blockers. Those with co-existent coronary disease, especially following MI, should receive beta-blocker which has a dramatic effect on reducing long-term mortality. Those with diabetes should receive an ACE-inhibitor (especially with microalbuminuria, for renal-protective effects). Those with diastolic dysfunction benefit from negative lusitropes/chronotropes (e.g. carvedilol, metoprolol, diltiazem). Those with systolic dysfunction benefit from ACE-inhibitors, shown to improve outcomes of patients with CHF. In general, beta-blockers increase insulin resistance, and have an unfavorable effect on blood lipids and may increase bronchospasm, but should not be withheld in patients who have had a MI or mild-moderate COPD. Since most patients have combinations of various diseases listed above, clinicians can customize anti-hypertensive regimes taking into consideration demographics and comorbidities, choosing medications that confer the greatest benefits and least complications. It is also important to choose a regimen with which the patient can be compliant (and can afford!) AND to counsel the patient about: their disease, why it is important to treat (with concrete examples), complications they might expect from their medications etc.. *Smoking cessation and weight loss should be pursued simultaneously and persistently.* Smoking and obesity account for so much excess cardiovascular disease, suffering and cost to society, that doctors are obliged to perseverate. Prescribing the right medication is relatively easy, while promoting healthy behaviors can be frustrating. But perhaps patients have more to gain by modifying their behaviors – techniques that clinicians must learn and deploy.

Approach to the patient with pulmonary embolus

Pulmonary embolus (PE) could also be grouped among pulmonary disorders, but the pathogenesis of hypoxemia in PE (i.e. reduced cardiac output causing reducing mixed venous oxygen returning to low V-Q units) and it's extreme manifestation of right heart failure also make it a circulatory problem. More than 90% of pulmonary emboli come from the large veins of the upper legs. Deep vein thrombosis (DVT) and PE are a continuum termed thromboembolic disease. Risks for thromboembolic disease include bed-bound for more than 24 hours, long plane trips, cancer, severe lung disease, stroke, congestive heart failure, MI, critical illness/mechanical ventilation, previous thromboembolus, inherited thrombophilias and surgery (especially for hip fracture). Dyspnea is almost always present, but other symptoms include pleuritic chest pain or tightness, cough and hemoptysis. Signs include tachycardia, tachypnea, fever (especially with infarction), and reduced lung volumes on examination due to splinting. The chest radiograph is most commonly normal but in order of decreasing frequency, small lung volumes, plate-like atelectasis, wedge-like infiltrates (infarction), segmental oligemia (Westermark's sign), and pleural-based densities (Hampton's hump) may also be present. The electrocardiogram commonly has a sinus tachycardia but *may* also occasionally exhibit atrial tachyarryhthmias, right axis deviation, right bundle branch block, p-pulmonale, and S1-Q3-T3. The Wells clinical prediction rules for DVT and PE should be applied (http://www.aafp.org/afp/20040615/2829.html). Historically, the diagnosis of PE was secured with ventilation-perfusion imaging demonstrating "high-probability" abnormalities. Low and intermediate probability scans are indeterminate, and pre-test probability is used to determine whether to proceed with additional testing. In such circumstances, duplex ultrasound of the legs (which should be done in any work-up) and d-dimer ELISA assays may be used in the work-up, keeping in mind that positive d-dimers are only useful for outpatients with the syndrome and low Wells score. The evolving standard-of-care is to screen outpatients with low pretest likelihood with highly sensitive/specific d-dimer; a negative test coupled with a low pretest probability (Wells score) makes PE very unlikely. For those with moderate or high pretest probability, CT angiography is the diagnostic method of choice unless the patient is at risk of contrast nephropathy (then use leg ultrasound or VQ scan).

Delayed therapy is associated with worse outcomes. Anticoagulants should be started immediately (especially when confirmatory tests will take more than 30 minutes to obtain), unless the pretest likelihood of DVT/PE is very low and/or there is a contraindication to anticoagulation. Treatment of DVT and PE is the same: full-dose anticoagulation with either unfractionated heparin (to INR 2-3), low-molecular weight heparin (LMWH) or fondaparinux, with initiation of coumadin after the first 24-48 hours. LMWH and fondaparinux should not be used in patients with renal failure (especially creat clearance<40 ml/min). There is good evidence that DVT not complicated by PE can be treated with LMWH and coumadin in the outpatient setting. Thrombolysis is reserved for those without contraindications (serious bleeding) who are either in shock and/or who have right ventricular failure demonstrated by echocardiogram as thin-walled, RV dilation and paradoxical motion of the septum. Most patients with massive PE will also benefit from careful volume resuscitation to improve RV filling, keeping in mind that over-resuscitation promotes RV ischemia and worsening RV stroke volume. Animal studies suggest that dopamine is a reasonable first-line inotrope-pressor for PE, and norepinephrine is reserved for those unresponsive to dopamine. Although there are no definitive data, inferior vena cava interruption (filter) can be considered when embolism recurs despite therapeutic anticoagulation (i.e. "a therapeutic INR") or pulmonary embolism itself is of sufficient severity or is superimposed on pre-existent cardiopulmonary disease so that further emboli risk long-term cardiopulmonary insufficiency (or death). Interruption is also indicated if there is an absolute contraindication to anticoagulation (e.g. major hemorrhage).

PULMONARY PROBLEMS

About tachypnea

Like tachycardia, tachypnea (especially≥20/min) always means something and may be the earliest, single sign of a life-threatening problem. Tachypnea may result from pain, anxiety, sepsis, pulmonary embolus or congestive heart failure, in which case the blood gas usually demonstrates respiratory alkalosis. Increased CO2 production (usually from systemic inflammatory response syndrome) and metabolic acidosis also cause tachypnea. Finally tachypnea may result from acute, chronic or acute-on-chronic lung disease. Tachypnea should always be explained (consider whether it is the earliest sign of a serious syndrome) and underlying reversible causes should be defined and treated.

Respiratory insufficiency/failure

There are two types of respiratory failure: hypoxemic and hypercapnic. Hypoxemia almost always results from ventilation-perfusion (V-Q) mismatch i.e. poorly ventilated regions (either airway disease or distal airspace flooding/collapse) with normal blood flow to those regions. V-Q mismatch that corrects with supplemental oxygen is predominantly airway disease (bronchospasm, secretions) whereas that which does not correct with high concentrations (e.g. 100% supplemental oxygen) is shunt (ventilation=0; normal perfusion). Severe shunt may cause insufficient oxygenation (O2sat<90% and PaO2<60 mmHg) that necessitates mechanical ventilation. Anatomic shunt is caused by flooding or collapsing of alveoli (whereas physiologic shunt – which is more uncommon - can occur with atrial septal defects, patent foramen, AV malformations of the lung). Alveoli may flood with pus (pneumonia), blood (hemorrhage) or fluid (CHF, acute lung injury i.e. ALI). They may collapse due to loss of reflexes (sighs, cough, yawn), upper airway obstruction with collapse distally, and distal airspace inflammation, as with pneumonia and ALI. Positive pressure ventilation (PPV) improves oxygenation by reducing atelectasis which contributes to most shunt-causing processes. It also reduces preload thereby reducing pulmonary edemagenesis (to the greatest extent in patients with cardiogenic pulmonary edema). Note however that PPV (including positive end-expiratory pressure) also can negatively impact oxygenation if they reduce venous return and cardiac output or increase physiologic shunt (by alveolar hypertension exceeding capillary pressures thereby "forcing" blood to poorly ventilated segments – more commonly in patients with pneumonia than ARDS).

The degree of V-Q mismatch is also affected by hypoxic pulmonary vasoconstriction (HPV) i.e. mechanisms in the lung to vasoconstrict poorly ventilated areas and vasodilate well-ventilated areas. Local (pneumonia, PE) and systemic (sepsis) processes in which nitric oxide is pathologically produced attenuate HPV and accentuate hypoxemia, often well in excess of the anatomic shunt fraction.

Less common contributors to hypoxemia (in order of frequency) include low cardiac output or hemoglobin (in the setting of V-Q mismatch), hypoventilation, and low barometric pressure (at high altitudes). Note that the last two reasons present with a normal aleveolar-arterial (A-a) oxygen gradient where P_AO2=barometric pressure times the fraction of inspired oxygen minus the PCO2 divided by the respiratory quotient (0.8 when we consume 1/3 fat, 1/3 carbohydrate, 1/3 protein), and P_aO2 is measured. The normal A-a gradient is <12 mmHg. A common misunderstanding is that "diffusion block" contributes to hypoxemia which may be the case in world class athletes (during optimal output and very fast pulmonary transit times) but is seldom applicable to ill patients.

Except for congestive heart failure in which facemask continuous positive pressure (CPAP=5-10 cmH2O) may prevent the need for invasive ventilation, patients with lung injury, hemorrhage and pneumonia that is refractory to 100% inspired oxygen should receive endotracheal intubation and positive pressure ventilation. Positive end-expiratory pressure (PEEP) is used to prevent end-expiratory atelectasis and higher doses (>5 cmH2O) are usually required for patients with ALI/ARDS and lung hemorrhage (see Figure 4,5).

Hypercapnic respiratory failure results when respiratory neuromuscular capacity (defined as a force generated by the neuromuscular circuit: brain, afferent neurons, junction, respiratory myocytes of diaphragm and other respiratory muscles) is insufficient to meet loads of breathing. The loads include mechanical resistive-pressure work of breathing, elastic-pressure work of breathing and the "metabolic" requirements (CO2 production and wasted dead space ventilation) which requires more mechanical work (larger minute ventilation) to maintain eucapnia. ***In most cases, hypercapnic failure results from overloading though insufficiency of the neuromuscular pump can also contribute***. For example, respiratory muscles may be negatively impacted by sepsis, malnutrition, electrolyte deficiencies, thyroid dysfunction, medications (e.g. cortiocosteroids), which are common in ill patients but are seldom the sole cause of respiratory failure. Primary pump failure (with completely normal loads) is uncommon, but results from narcotic overdose, polio, ALS, Guillian Barre, phrenic nerve damage, and myasthenia. In these situations, measured loads are not often grossly elevated and patients manifest weakness in other non-respiratory muscles.

As mentioned above, there are three types of hypercapnic failure:

1. Acute – pH<7.36, pCO2>40 mmHg, bicarbonate≤24 meQ/L - loads are of sufficient magnitude to overwhelm an initially normal neuromuscular circuit OR in which a neurologic cause of pump failure progresses rapidly. These patients are usually distressed with rapid shallow breathing until they near respiratory arrest OR in some primary neurologic-related failure.

2. Chronic – pH=7.32-7.36, pCO2>40 mmHg, bicarbonate>24 meQ/L - a restrictive load like obesity or obstructive load like COPD gradually erodes respiratory muscle reserve and eventually outstrips capacity but slowly enough to allow metabolic compensation of a rising bicarbonate.
3. Acute on chronic – pH<7.32, pCO2>>40 mmHg, bicarbonate>24 meQ/L - an acute increase in load and/or decrement of capacity is superimposed on a system that is already functioning near its maximum output, thus leading to abrupt increments of hypercapnia with insufficient time for renal compensation. These patients are distressed and breathing rapidly and shallowly.
4. Iatrogenic hypercapnia (not respiratory failure) – pH<7.32, pCO2>>40 mmHg, bicarbonate>24 meQ/L, PO2 usually >70 mmHg; due to too much inspired oxygen or administration of respiratory depressants (narcotics). Unlike acute or acute on chronic hypercapnic RF, these patients are not uncomfortable, often somnolent and breathing (generally) <20/min. Fixes with reduced inspired oxygen to yield oxygen saturation around 90% or administration of reversal agents (e.g. flumazenil for benzo overdose, narcan for opiod overdose).

COPD patients with acute on chronic hypercapnic RF due to bronchospasm benefit from non-invasive positive pressure ventilation (NIPPV) started at low levels (e.g. 7 cmH2O inspiratory and 5 cmH2O PEEP) and increased (2-4 cmH2O increments of inspiratory pressure until the patient is comfortable or 20 cmH2O is reached). It should be used more cautiously if the acute illness is pneumonia, because NIPPV impedes expectoration. The use of NIPPV for patients with restrictive acute on chronic hypercapnic RF (e.g. obesity or kyphoscoliosis) is not well-studied, but is worth trying if patients do not have immediate indications for endotracheal intubation (see below).

In general, NIPPV must be attempted cautiously and clinicians must remain at the bedside until the patient improves. Signs of improvement include reduced tachycardia/tachypnea (and there is blood gas evidence to confirm safety). Signs of failure include increasing subjective distress, increased tachypnea, hemodynamic instability and/or altered mentation all of which contraindicate use of this modality. ***No blood gas (save PaO2<60 mmHg on 100% inspired oxygen) absolutely mandates intubation and invasive ventilation. However, excess work of breathing, even without blood gas evidence of hypercapnia, mental status changes and hypotension are indications for endotracheal intubation and positive pressure ventilation.*** Nobody can fault you for intubating in such circumstances, but waiting too long can contribute to avoidable cardiopulmonary arrest. Novices and experts alike should err on the side of securing the airway in such cases (if the patient has not refused invasive ventilation understanding its risks, benefits and alternatives). You can always extubate someone after rapidly repairing them. You cannot raise the dead.

Nonetheless, endotracheal intubation and initiation of positive pressure ventilation is itself a life-threatening procedure. Often patients who require endotracheal intubation (for airway protection or true respiratory failure) have been working hard to breathe and the relaxing medications (e.g. benzos, narcotics) used to facilitate intubation are catecholytic; venodilation results in venous pooling (reduced venous return and therefore cardiac output) that is dependent upon the size of the patient and the amount of administered medication. Second, the transition from negative pressure (unassisted breathing in which zero is ordinarily the highest pressure in the thorax at end-expiration) to positive pressure breathing engenders an increase in mean intrathoracic pressure which is transmitted to the right atrium. Any first-year medical student will recall that venous return is proportional to mean systemic pressure minus right atrial pressure. During intubation and positive pressure breathing, Pms goes down and Pra goes up, so venous return often drops dramatically. More than 30% of patients develop transient hypovolemic hypotension – and some will have a full cardiac arrest requiring CPR. Additionally, when muscle relaxants are used to intubate patients they often go from breathing over 30/minute down to at whatever the respiratory therapist chooses to bag them which potentiates respiratory acidosis if too slow. Since post-intubation shock is usually preventable, consider volume loading at-risk patients (the obese, those with low blood pressures even before intubation and those who are very catechol-driven), minimize use of medications whenever possible and stand-by with vasoactive medications/fluids in case “the bottom falls out.” Ask for bagging at 25-30/min when the patient has been tachypneic prior to intubation and start at similar rates on the ventilator until blood gases determine PCO2 and Ph. An ABG (or VBG when ABG is not obtainable) should be drawn within 10 minutes of endotracheal intubation. An ounce of prevention is worth a pound of cure.

Pneumonia

The most common pulmonary admitting diagnosis is pneumonia. Many patients with mild pneumonia can be treated safely as outpatients. The following require hospitalization: 1. Hypoxemia on room air, 2. Severe sepsis, 3. Immuno-incompetence, with or without advanced age, 4. Clinical worsening despite a trial of outpatient therapy, and, 5. A non-compliant or undependable patient who may not come back rapidly if the trial of therapy fails.

Pneumonia comes in two flavors: community-acquired (CAP) and healthcare associated (HCAP). This distinction is not arbitrary. More than 95% of pneumonia occurs when upper respiratory pathogens move from the upper to the lower airways. Accordingly, the most common causes of CAP caused by the pathogens most often infecting the upper respiratory tracts of outpatients who have not been institutionalized include*: Pneumococcus, H. influenza, Klebsiella, Legionella, Mycoplasma*. The upper airways of those who are in and out of healthcare facilities (hospitals, nursing homes, dialysis units) are exposed and colonized to community pathogens AND those that occur mainly in healthcare facilities. Thus, HCAP adds the possibility of *Staphylococcus* (especially oxacillin-resistant S*taph*), *Pseudomonas*, and other virulent Gram-negative rods (e.g. *Acinetobacter, E. coli*). Pneumonia occurs less frequently in otherwise well people who possess robust neuro-mechanical (e.g. cough/gag/swallow/mucociliary escalator) and host defenses (e.g. mucosal immunity) to keep upper aerodigestive bacteria out of our tracheobronchial tree. More often pneumonia results when those defenses fail: viral infections or tobacco smoke denude respiratory epithelial defenses, medications/drugs/alcohol/stroke attenuate mechanical reflexes to prevent transgression of bacteria across the glottis, a variety of illnesses impact host cellular/immune-clearance mechanisms.

Pneumonia is a clinical diagnosis that includes a history of cough with or without sputum production, shortness of breath, fever, leukocytosis/penia and an infiltrate on chest radiograph. Most previously healthy patients have most manifestations, but elderly or immuno-compromised patients may lack some elements. However an infiltrate on chest radiograph must eventually be present – I say eventually because the infiltrate may not arise until after the patient is fluid resuscitated. Except for atypical (viral, *Mycoplasma, Chlamydia*) pneumonias which may present as diffuse interstitial infiltrates, most bacterial and fungal pneumonias present with focal infiltrates. Note however that if pneumonia is of sufficient severity, acute lung injury (ALI/ARDS) involving all lung regions may result (so a focal infiltrate can give way to confluent 4-quadrant airspace infiltrates). Note that while ARDS may appear homogenous on radiograph, it is actually heterogeneous with more collapsed/flooded lung in gravitationally dependent lung regions.

Sputum may be diagnostically helpful in some situations. If sputum contains few epithelial cells, numerous white cells and a predominant/abundant gram-stain form, so long as the patient is immuno-competent and not too ill, it is not unreasonable to treat narrowly. But in general and when the sputum does not provide an obvious answer and/or the patient presents with severe sepsis, initial administration of broad-spectrum antibiotics is indicated. Currently, extended generation cephalosporins (e.g. ceftriaxone) with a macrolide (e.g. azithromycin) to cover atypical co-pathogens are widely used therapies for patients with moderately severe CAP (i.e. requiring wards but not ICU). New urine assays for *Pneumococcus* antigen may also be a means of promoting earlier, safe use of narrow coverage when the sputum is equivocal.

For treatment of HCAP, strong consideration should be given to include anti-*Pseudomona*s/anti-*MRSA* antibiotics, especially in the immuno-incompetent patients, until sputum Gram stain and/or cultures allow narrowing of coverage. Blood cultures should be drawn on all patients with pneumonia. Although relatively uncommon, when blood grows an organism in the setting of pneumonia it allows definitive confirmation (whereas definitive diagnosis by sputum is the exception). Treatment of immuno-compromised patients with pneumonia, especially those with organ transplantation and those receiving immunosuppressive medications requires consideration of additional pathogens ("opportunistic bugs") that are specific to each diathesis (see discussion of sepsis below). In such patients, early diagnostic fiberoptic bronchoscopy should be considered, either initially or if a trial of empiric therapy fails to yield clinical improvement after 48-72 hours.

Hypervolemia should be avoided in patients with pneumonia insofar as leak into lungs promotes greater hypoxemia. Supplemental oxygen should be administered to ensure an O2-saturation>95% except in those with chronic hypercapnia when a goal of 90-92% may prevent iatrogenic hypercapnia (since some patients with chronic hypercapnia will *comfortably* – the distinguishing feature - retain CO2 if oxygenation is greater than 92%). In general, patients should get out of bed as soon as they are able and can be changed from intravenous to oral antibiotics when they

have defervesced for 12-24 hours. They can be discharged when they remain afebrile and do not require home O2. However, they should receive instructions to call their outpatient physician if they experience increasing fevers or dyspnea. Occasionally patients may require discharge with home oxygen, especially if they have precedent lung disease, but ongoing oxygen requirements in a patient without previous history should prompt consternation – usually signaling that pneumonia has not resolved sufficiently for safe discharge. Appropriate management includes administration of *Pneumococcal* (in naïve patients) and *Influenza* vaccines before hospital discharge when appropriate.

Aspiration pneumonia/pneumonitis deserves special comment. When a patient has an observed aspiration, it is useful to distinguish whether aspiration was of saliva/upper airway secretions, food or vomit. The implications are different for each. The classic teaching is NOT to treat an observed aspiration until he "declares himself." One of several patterns is common:

a. The patient has a clear chest radiograph initially and on follow-up and has only wheezing on examination suggesting that the aspirated material only irritated the airways.
b. The patient has either one discreet or a diffuse infiltrate, so-called "aspiration pneumonitis." On follow-up, many such infiltrates spontaneously resolve as protective mechanisms in the distal airspaces process and clear the aspirated material and any capillary leak abates. If the aspirated substance (e.g. stomach acid) is very noxious, it may promote a diffuse inflammatory process in the lungs leading to non-cardiogenic pulmonary edema/ARDS. If there are sufficient numbers of bacteria in the aspirated material it may set up a nidus of bacterial, often mixed oral flora/anaerobic, pneumonia.

If the patient is very immunocompromised and/or very hypoxemic initially, consider empiric coverage for community-acquired aspiration (for those in hospital less than 24 hours) with a drug like ampicillin-sulbactam, or for institutionally-acquired aspiration (for those in hospital for >48 hours before aspirating or who have been in/out of a dialysis unit, hospital or nursing home) with a drug like piperacillin-tazobactam. Otherwise, the classic teaching is to wait and watch for the classic signs/symptoms of pneumonia. If the patient develops an infiltrate, productive cough and fever, then coverage of immunocompetent hosts is appropriate.

Asthma/COPD

The work of breathing is analogous to moving gas in an out of a straw attached to a balloon (see Figure 2). Gas moves through the conducting airways where no gas is exchanged AND expands the lung and chest wall, much like loading a spring, from functional residual capacity to inflation of a tidal volume. Thus the total tidal work of breathing includes both resistive-pressure work (that is equal to flow times resistance) and elastic-pressure work (that is equal to the elastance times the tidal volume). In obstructive lung disease (asthma and COPD), the airways become inflamed and/or spastic – the straw is pinched. This increases the resistive-pressure work of breathing. Additionally, since expiration is a passive process (analogous to releasing the stored potential energy of a spring) and the airway resistance is elevated, gas remains "trapped" in the lungs at end-expiration, so each subsequent breath is "stacked" on part of the previous trapped gas volume. This is a dynamic process, i.e. occurs over several breaths and so is termed "dynamic hyperinflation." Hyperinflation renders the system stiffer, thereby increasing the elastic-pressure work of breathing as well (see Figure 2).

Asthma

Asthma is a reversible inflammatory disease of the airways of children and young-middle-aged adults. Dyspnea and cough may develop over minutes to days, based on the provocation and the patient's sensitivity. Precipitants of asthma include viral infection, tobacco smoke, air pollution, dust mites, cold air and exercise. If dyspnea is severe, patients present to the hospital emergency department (i.e. if they don't collapse in the field from sudden asphyxic asthma). Signs of severe asthma include tachycardia, tachypnea, inability to speak in full sentences, prolonged exhalation, wheezing (in very extreme cases, a paucity of breath sounds due to very little air movement), and pulsus paradoxus>15 mmHg. I find asking the patient to either count or repeat a sentence as many times as possible with a single breath helps index dyspnea (of any cause). Peak flows should be considered part of the physical examination and can be compared to the patient's baseline or to nomograms (<40% predicted is very severe, 40-60% is moderately severe).

In the ED, they should receive oxygen, to keep O2 saturations >92% and 3 "back-to-back" nebulized beta-agonist treatments (or continuous treatment for an hour). More than 2/3 of patients will improve subjectively and objectively (FEV1 or peak flow>60% predicted)

allowing discharge with a tapering dose of oral corticosteroid, beta-agonist inhaler used for "rescue therapy" (i.e. as needed up to every 4-6 hours) and a follow-up appointment with their outpatient doctor. Most of the remaining 1/3 of patients will not benefit from many more treatments and will require hospital admission. These patients benefit from intravenous corticosteroids (methylprednisilone 60-125 mg every 6 hours) and ongoing beta-agonist aerosols (either nebulized therapy or metered-dose inhaler with a spacer) every 2-4 hours initially, spaced to every 4-6 hours as symptoms allow. Ipratropium aerosols may also improve ED outcomes of patients with acute asthma, though the incremental benefit is relatively modest. Patients can be discharged when they have reached the ED discharge criteria discussed above and with similar discharge care plans. In general, patients can be taught to self-manage their asthma, measuring their peak flows and symptoms, increasing inhaled steroids or advancing to oral steroids when necessary based on home-treatment algorithms. Beta-agonist therapy is generally rescue therapy i.e. used symptomatically.

COPD

Chronic obstructive pulmonary disease ("COPD") is not a single disease, but rather includes chronic bronchitis or bronchiectasis, reactive airways, and emphysema. Patients may have varying amounts of each diathesis, which may also vary within a given patient over time. The mechanisms by which obstruction cause increased work of breathing are similar to asthma (described above). In addition, patients with emphysema have reduced elastic recoil of the lung parenchyma which may cause small airway collapse during exhalation, thereby promoting dynamic hyperinflation.

Treatment depends upon the underlying "exacerbating" condition. If the patient has signs and symptoms of tracheobronchitis (cough and increasing amounts of purulent secretions with a clear chest radiograph), oxygen supplementation to maintain saturations of 90-94%, antibiotics (doxycycline) and bronchodilation (with short-acting beta-agonist and ipratropium) is appropriate management. Too much oxygen (saturations>92%) can cause iatrogenic acute hypercapnia that is manifest by lethargy and comfortable breathing in some patients with chronic hypercapnia. Although less than 20% of patients are "oxygen-sensitive," every month I find 1-2 patients who have been intubated for lethargy, comfortable breathing and hyperoxia-induced hypercapnia that could have been reversed by simply reducing the fraction of inspired oxygen to achieve an O2 saturation of 90-92%. Antibiotics are <u>not</u> required in the absence of signs/symptoms of infection. Insofar as this population of patients is more at risk for pneumonia, congestive heart failure (the wheezes of which may mimic pulmonogenic bronchospasm), myocardial infarction and pulmonary embolism, these common disorders should also be considered in COPD patients presenting with acutely worsening dyspnea with or without cough.

For patients who reach discharge criteria, recent "GOLD" guidelines suggest therapies based on severity of illness. Home oxygen to maintain O2sat>90% during rest/exercise/sleep improves outcomes. Long-acting cholinergic aerosols (tiotropium), combined long-acting beta-agonist/steroid aerosols and pulmonary rehabilitation are likely of benefit in some groups of patients.

In extreme form, these patients may have chronic hypercapnic respiratory failure – in which case the loads of breathing have increased sufficiently that there is no longer respiratory muscle reserve to deal with an increment of load (e.g. exercise, infection, acute bronchospasm, CHF, pneumonia etc.). These patients live with an acidemic pH (7.32-7.36), pCO2>40 mmHg and a bicarbonate>24 meQ/L (the "footprint" of chronic renal compensation for respiratory acidosis). Any increment in their respiratory load or decrement of neuromuscular capacity results in "acute on chronic" failure epitomized by a pH usually ≤7.32, pCO2>>40 mmHg and bicarbonate >24 meQ/L. Patients with obesity and kyphoscoliosis (i.e. chronic restrictive disease) are similar in exhibiting these paradigms of hypercapnia (i.e. chronic and acute on chronic). The goal is: to define the diatheses that caused the chronic and acute components, and to treat the reversible acute causes. In reality, we seldom identify and treat only one reason for the acute component – never placing "all our eggs in one basket." Instead, we exercise the Casablanca principle, "round up the usual suspects" (i.e. bronchospasm, infection, congestive failure, thromboembolic disease), treating several of the most likely to increase the likelihood of actuating treatment to reverse the true culprit(s). It is no sin not to know precisely which acute diathesis caused the exacerbation. Since every day spent on the ventilator is tangible risk, empiric therapy (while waiting for confirmatory tests) is not inappropriate.

As mentioned above, a trial of NIPPV is appropriate for most patients with acute on chronic hypercapnic respiratory failure who don't have indications (mental status changes, shock, abundant purulent secretions) for endotracheal intubation. This modality is especially effective in patients with acute on chronic hypercapnia with COPD and an exacerbation of bronchospasm. When it is used, NIPPV should be started at relatively low pressures (inspiratory 7 cmH2O and expiratory 5 cmH2O) and the inspiratory pressures can be slowly increased as the patient becomes more comfortable (decreased respiratory rate, heart rate, blood pressure) with the machine. An arterial blood gas should be obtained to confirm reduced PCO2 within 30 minutes of initiating NIPPV. This modality should always be initiated in a monitored (ED, step-down or ICU) setting as these patients have respiratory failure and may require endotracheal intubation if NIPPV fails. And a trained clinician (doctor, nurse or respiratory therapist) MUST remain at the bedside until the patient declares himself as a NIPPV success or failure, requiring endotracheal intubation.

Acute lung injury/ARDS

Acute lung injury (ALI) can be caused by numerous diseases and is defined as diffuse lung infiltrates and hypoxemia (PaO2/FiO2<300). Acute respiratory distress syndrome (ARDS) is reserved for patients with the most severe ALI/hypoxemia (PaO2/FiO2<200). Sepsis is, by far, the most common cause of ALI/ARDS. However a multitude of insults including shock, trauma, transfusions, medications, inhaled/injected toxins, and inflammatory disorders may also cause lung injury. ALI/ARDS is an inflammatory disease of the pulmonary endothelium and epithelium leading to noncardiogenic pulmonary edema AND atelectasis. The most common presenting symptom is dyspnea. Common signs include tachypnea, tachycardia and lung crackles. Treatment hinges on two important concepts: 1. Remove or control the inciting cause as soon as possible, and, 2. Meticulously avoid proinflammatory insults of the lung (infection, transfusions, prolonged shock, ventilatory-induced lung injury; see Figure 3 next page). Patients with ALI/ARDS do not benefit and may be harmed by a trial of non-invasive mechanical ventilation. Accordingly, when these patients approach critical oxygenation (<90-92% saturation while breathing 100% facemask oxygen), intubation and mechanical ventilation are indicated.

The current data suggests that if not used properly, the ventilator promotes ALI (ventilator induced lung injury; VILI). In a prospective, double-blind randomized trial of patients with ALI/ARDS, the use of 6 ml/kg initial tidal volume, then titrating to a plateau airway pressure of ≤30 cmH2O, yielded significantly lower mortality compared to ventilation with 12 ml/kg then titrating to a plateau pressure of ≤50 cmH2O. The current evidence does not dictate an optimal strategy of employing positive end expiratory pressure (PEEP). However, I use the least PEEP required to maintain an O2sat of ≥90% on an FiO2 of ≤60% to minimize theoretical harms associated with oxygen and PEEP therapy. This "least PEEP" strategy requires that the tidal volume be reduced for each increment of PEEP to keep the plateau pressure less than 30 cmH2O (see Fig 4 next page). If more than 20-25 cmH2O PEEP is required, I am willing to live with >60% inspired oxygen for 12-24 hours since hypoxic pulmonary vasoconstriction, slow recruitment with PEEP and improvement of the underlying condition is likely to allow decreasing the FiO2 with time. I give only so much fluid as is absolutely necessary to keep the kidneys perfused (because any administered fluid also leaks into the lung) and will occasionally use "recruitment maneuvers" (i.e. static inflation of the lung) to transiently improve oxygenation. Ventilating in the prone position for 6-12 hours, alternating with recumbent ventilation is rarely required and since it does not impact mortality, I use it only when high PEEP has failed to allow a reduction of FiO2 to 60% or marginal oxygenation despite 100% FiO2, i.e. it is "salvage therapy." I strongly recommend that an experienced intensivist assist directly when >15 cmH2O of PEEP, recruitment maneuvers or proning are required. The key is to be patient and aggressively eliminate pro-inflammatory insults that propagate the lesion, facets that are sometimes neglected.

Hints for routine mechanical ventilation

There are abundant data that ventilators can kill patients. They can cause macrobarotrauma (pneumothorax which, with positive pressure ventilation, often leads to tension pneumothorax) and microbarotrauma (VILI). By increasing intrathoracic pressure they also gate venous return and if misused can promote shock. Accordingly, it is very important to choose safe ventilator settings until an expert (intensivist or pulmonologist) can "fine-tune" the machine.

In general it is wise to begin with assist control (which ensures the patient the preset number of breaths and any additional breaths he wants, if he is able to trigger the ventilator). If care is taken to ensure patient-ventilator synchrony by titrating settings to comfort and adding

sedatives AND pain medications as needed, this mode will promote a restful patient, while you identify and fix what has broken in the patient. Start with a respiratory rate of 10-12/min EXCEPT when patients have been muscle relaxed and cannot trigger breaths in which case you must approximate their respiratory rate before intubation (up to a maximum of 30 breaths/minute – higher values promote hyperinflation and can be dangerous). In general, it is safest for novices to initiate ventilation with a "square" (constant) flow of around 60 L/min of 8 ml/kg. This may need to be lower in patients with ARDS and those with severe obstructive lung disease. Irrespective, when the patient is comfortable and synchronous, go back to the ventilator and choose a tidal volume that yields a plateau (static) airway pressure of 20-25 cmH2O. This will avoid both atelectasis AND barotrauma. **Never** allow the plateau pressures to remain consistently above 30 cmH2O unless approved by an experienced intensive care doctor. (Exceptions for this plateau rule include severe chest wall elastance as with massive ascites, obesity or kyphoscoliosis, severe pre-existent lung fibrosis, and need for very high PEEP>15-18 cmH2O, but in such cases an experienced intensivist is needed). A high peak pressure is usually not dangerous unless it is accompanied by a high plateau pressure (above 30 cmH2O). But be aware that when peak airway pressures are high and alarm limits are not set properly, tidal volumes may be truncated contributing to hypoventilation (i.e. high pressure limits must be turned up which is OK so long as the plateau airway pressure remains <30 cmH2O with the full tidal volume). Figure 5 (next page) (also presents a reasonable, pathophysiology-based approach to patients with very severe hypoxemia.

When patients "high pressure alarm," there are several approaches. First ensure that the airway is clear of secretions and the patient is not biting the tube. This manifests as a high peak-plateau gradient. Second, ensure that the patient is synchronous with the ventilator – not coughing, bucking, or fighting the ventilator. If so, sometimes changing the flow regimes and/or the tidal volumes helps to promote comfort. Use bolus sedatives and/or narcotics as a last resort if the patient seems uncomfortable and simple changes of the ventilator don't help. Also ensure that the endotracheal tube has not slipped into the right mainstem by listening to both lung fields. This manifests as an increase in both the peak and plateau in parallel. Note however, that pneumothorax may also manifest as lack of breath sounds on one side with similar mechanics. Think about getting a chest radiograph and calling for assistance early if pressures remain high.

Think of your ventilated patients as "broken" – they won't fix quickly unless you define what's broken and fix it. If they have hypercapnic respiratory failure (intubated for rapid shallow breathing and/or supportive blood gas), define and repair neuromuscular incapacity and elevated loads (see above). If they have hypoxemic respiratory failure, treat the underlying cause. If they've been intubated for airway protection, fix the neurologic problem that precipitated airway incompetence.

Sedatives should be given only as needed with infrequent exceptions. Analgesics (low-dose opiods) should be given around the clock since critical illness and instrumentation are uncomfortable. Irrespective, patients should be allowed to awaken fully at least once each day, unless they have contraindications (e.g. very high PEEP requirements, prone ventilation). Provide thromboprophylaxis, aspiration precautions (head of bed 30 degrees) and ulcer prophylaxis (e.g. H2-blocker or proton pump inhibitor).

When patients are hemodynamically stable with a PaO2/FiO2>120 allow them ***daily*** trials of spontaneous breathing (SBT) of 30-60 minutes. When they pass (are comfortable, breathing <30/min on less than 7 cmH2O positive pressure, without large increments of blood pressure and heart rate) and can expectorate, they are ready for a trial of extubation. Doctors aren't very good at predicting when a patient will be successful. The only way to know is to perform daily SBTs when they meet the above criteria. Weaning i.e. gradual reduction of the support is unnecessary and harmful.

Integration of cardiopulmonary pathophysiology

Over the centuries, man has developed cardiopulmonary reserve as an adaptive advantage: healthy adults can increase cardiac output (at least 2-3-fold) and minute ventilation (in excess of 20-fold!) significantly for fight/flight, exercise or pathophysiologic stresses. Decrements of cardiac function and/or pulmonary function are common in hospitalized patients. The greater the level of dysfunction, the more "reserve" is eroded, the less stress or exercise is required before patients reach cardiopulmonary limitation. The most common symptom of reduced cardiopulmonary reserve is dyspnea on exertion and when combinations of cardiac and pulmonary dysfunction are sufficient, dyspnea at rest. Good clinicians will ask patients how much exercise it takes before a patient must stop due to dyspnea. This is commonly quantified as distance

walked (and the 6-minute walk test is commonly used as a formal test with reference standards), flights of stairs climbed etc. before shortness of breath forces the patient to stop. It doesn't help distinguish whether limitation is due to heart and/or pulmonary dysfunction, but it does allow us to index severity and monitor responses to therapy.

It is our job to:

a. Quantify the relative contributions of reduced cardiac and pulmonary reserve (PFTs and echocardiography are powerful tools),
b. Treat what is reversible (e.g. cardiac revascularization, preload/afterload/rate control; pulmonary bronchodilation, oxygen when necessary etc.),
c. Limit progression (e.g. control of lipids, aspirin), and,
d. Use exercise/rehabilitation protocols to maximize peripheral energy utilization by muscles.

Patients can also be taught to help with their own management – to recognize when they gain weight with CHF so they can self-administer additional doses of furosemide, recognize early bronchitis or wheezing to take rescue bronchodilators, or increase the dose of inhaled steroids. Increasingly, patients will be taught to become proactive participants in promoting their own wellness, hopefully, collaborating with physicians to prevent acute illness that becomes so severe as to require hospitalization.

INFECTIOUS DISEASE

About fever

Fever (especially≥101.5°F) is quite common in hospitalized patients. Since infection/sepsis is by far the most common reason for fever and the leading cause of mortality of hospitalized patients, it always deserves an explanation. Other causes include allergic and drug reactions, hyperthyroidism and non-infectious causes of systemic inflammatory response syndrome (SIRS). The approach to sepsis is highlighted below, but non-infectious causes should be considered, especially when no site of infection can be identified. Note also that patients with resolving infection continue to "spike fevers" for days following appropriate antibiotics. So long as the maximal temperature is decreasing (with the white count and other indices of patient well-being), therapies are likely effective. All fevers DO NOT require antibiotics. However, antibiotics should not be with-held awaiting positive cultures if the likelihood of a virulent pathogen is high, the patient is immunocompromised or the patient has other accompanying signs of severe sepsis (see below). Antibiotics can always be stopped if microbiologic lab results do not support the diagnosis of infection whereas organs injured from severe sepsis may not recover (and mortality is 30-40%).

Approach to the Patient with Sepsis

Infection is the leading cause of death in hospitalized patients. It accounts for a sizeable number of initial admissions and is a very common
iatrogenic complication. Accordingly, mastery of this process may be amongst the most important skills ascertained by trainees. Sepsis is the response of a host to an organism and/or its toxins. The host response includes tachycardia, tachypnea (with respiratory alkalosis), thermodysregulation (high or low body temperature). If sepsis is severe, end-organs may be affected including the kidney (oliguria), the brain (encephalopathy), the lungs (hypoxemia), the hemostatic system (DIC, thrombocytopenia) and general dysmetabolism (lactic acidosis). The blood vessels become leaky and relaxed (by nitric oxide synthesis) causing effective hypovolemia and in its most severe form, the heart is unable to keep up (due to insufficient preload and vascular tone) leading to hypotension. Perhaps the most important aspect to reducing mortality is to identify sepsis early – to prevent simple sepsis (<10% mortality) from proceeding to severe sepsis (40%) to septic shock (>50%). Early recognition and treatment is the key to stopping the progression. It is important to understand that an immuno-incompetent host can proceed rapidly from simple sepsis to death even with a "wimpy organism." Conversely, immunocompetent patients can die quickly with a virulent pathogen (e.g. *Meningococcus*, *Ebolavirus*, etc.).

Thus constant vigilance for this extremely common disease WILL attenuate mortality. Unexplained tachycardia, tachypnea, respiratory alkalosis, fever, or hypothermia could be the earliest sign of sepsis, which should be considered and circumvented. The clinician should "size up"

the immunocompetence of the host, the virulence of the likely pathogens in the likely putative sites of infection (see Figure 6 next page) and formulate an aggressive empiric strategy since strong evidence supports that mortality increases with delayed treatment and overly narrow antibiosis. Bedside management consists of: 1. Early (e.g. in the emergency department) administration of crystalloid (normal saline) until urine output increases or the patient develops signs of congestion (basilar crackles and/or decrements of the PaO2/FiO2 ratio), 2. Administration of immediate antibiotics based on the most likely sources of infection, the most likely causative agents (based on site and patient's origin i.e. home vs. recently hospitalized/institutionalized), and the patient's immuno-competence (more broadly with increasing immuno-incompetence), and, 3. Identification and drainage of loculated foci of infection (drain abscesses, remove or bypass stones etc.). There is increasing evidence that EARLY administration of antibiotics and fluid resuscitation to refill the empty system attenuate mortality. Especially with severe sepsis and/or immuno-incompetent patients with early sepsis, antibiotics should not be delayed for more than a few minutes to gather cultures. Administration of activated protein C in non-bleeding patients with severe sepsis has been shown in one study to attenuate mortality. Replacement of the adrenal axis with hydrocortisone 80 mg thrice daily is also appropriate when hypotension is unresponsive to 2-3 L of fluid or requires use of vasopressors. Cortisol is required to maintain vascular (especially venous) tone. An alternative is to administer a dose of administer a dose of dexamethasone followed by an ACTH-stimulation test; administering hydrocortisone if the axis is insufficient. Since the normal "maximally stressed" cortisol level is unknown but likely to be >>30, I generally treat empirically for cortisol<30 in patients with shock reasoning that most previously healthy patients make levels much more than 30 when they're in shock. Note that this hypothesis has never been tested.

One important comment on use of antibiotics: Although empiric antibiotics may be required initially, it is imperative that once causative microbes are identified medications are switched to those with the most narrow coverage possible for the pathogen (based on sensitivity reports). This reduces the likelihood that you will contribute to development of multiple drug resistance of pathogens in your patient and in your hospital. Chaotic and thoughtless overuse of broad spectrum antibiotics have contributed to the scourge of multiple-drug resistant microbes that make hospitals increasingly unsafe for ill patients and the only means of addressing the problem is for all clinicians to use antibiotics responsibly.

FLUIDS/ELECTROLYTES/KIDNEY

Fluid choices

Hospitalized patients may require intravenous fluid administration if they cannot take sufficient fluids and salt by mouth. There are few patients admitted to the hospital medical-surgical services who don't require intravenous access. Central access is overused at most hospitals. The choice and rate of fluid administration varies. In general, if a patient is euvolemic, with normal sodium and cannot take oral liquids/food, maintenance fluids with D5½ normal saline at 75-100 ml/H is reasonable. If the patient is hypovolemic, normal saline should be administered until euvolemia is reached (increased urine output and absence of orthostatic blood pressure changes), then substituted with maintenance fluids. The rate of the initial volume resuscitation is determined by the severity of hypovolemia. If the patient is in shock (see above), saline is administered "wide open" (as fast as possible through as many available wide-bore catheters as possible) until shock is corrected or the patient crackles (i.e. shows early signs of congestion with crackles and diminished oxygenation). Orthostasis without shock can also be treated with an initial bolus of fluids (500-1000 ml at a time) until euvolemia is achieved. Intravenous fluids should be changed to oral/gut-administered as soon as possible.

The "flood" and "ebb" of acute illness

Although some patients present with edematous states and attention turns to define and reverse the pathogenesis while diuresing as tolerated, many acute illnesses require initial resuscitation. Fluids are administered to ensure a sufficient delivery of nutrients to vital organs – usually titrated to urine output when the kidneys remain functional. This is particularly true in resuscitation of shock states but holds for less severe disease as well. After administration, fluid starts in the intravascular space. But frequently illness is accompanied by either local or generalized capillary leak due to inflammatory events that increase microvascular permeability. Moreover, acute and chronic illnesses engender reduced intravascular albumin that is exacerbated by intravascular expansion/dilution. Additionally, when we "overshoot" i.e. transiently overfill the system (which is better than underperfusing vital organs), intravascular hydrostatic pressure may also increase. Starling's law of fluid fluxes

dictates that these common facets of resuscitation promote edemagenesis. This phenomenon is most common in critically ill patients, but also occurs in very ill ward patients hospitalized in acute or chronic care facilities for long periods.

The opposite is true during recovery from acute illness when microvascular impermeability returns and fluid returns to the microvasculature and then back to the central circulation. It is not uncommon to find critically ill patients with cumulative fluid balances of over 20 L, most of which will be mobilized during recovery (what goes in must come out). Patients with left ventricular dysfunction commonly develop pulmonary edema during this "ebb" phase if clinicians aren't vigilant to induce a diuresis. Although intravascular hypoalbuminemia (from acute illness, dilution and malnutrition) initially impedes mobilization of fluid, attention to nutrition (to cause albumin synthesis, measured by the prealbumin) and diuresis leads to increasing concentrations of albumin and increasing flux of fluid back into the vasculature. How then to gauge both resuscitation and retrieval?

Except when there is abundant blood in the gut or significant catabolism (e.g. steroid treatment), **the blood urea nitrogen (BUN) is the body's barometer**. When the kidney receives insufficient blood flow, the BUN rises faster than the creatinine. While resuscitation is guided by urine output, fluid mobilization during recovery can be gauged by BUN velocity. If BUN rises more than 3-4 mEq/L and the creatinine changes minimally, it most often signals that the vasculature is insufficiently filled (or that heart function is decreased). Accordingly, I urge my trainees to report fluid inputs/outputs followed by the corresponding change of BUN which allows us to chart the next day's plans (i.e. slow down diuresis if O>I caused a significant increment of BUN, continue diuresis when it hasn't).

Renal failure/acute kidney injury

Acute renal failure results when the kidneys are acutely injured to the point when they can no longer entirely remove toxins (waste) and regulate salt/water balance. Most often, acute renal failure is accompanied by a rising creatinine (and BUN) and reduced urine output. Oliguric renal failure refers to production of less than 400 ml/D of urine (approximately the minimum volume required to clear waste under normal conditions), while anuric renal failure refers to absence of urine output. Since the term renal failure has been used to describe a spectrum of kidney failure ranging from mild pre-renal azotemia that is readily reversible, to rapidly developing anuric failure requiring dialysis, investigators are currently devising precise definitions and staging systems for "acute kidney injury." Irrespective, the classic teaching is to parse the pathogenesis of renal failure as pre-renal, renal and post-renal causes.

Pre-renal azotemia

Pre-renal failure means that failure has resulted because the glomeruli of the kidney are not receiving enough blood flow. Accordingly, this form of renal failure can occur when the cardiac output is reduced. Hypovolemic prerenal azotemia occurs when venous return is insufficient – either the tank is leaking and/or the tank (usually the venous capacitance vessels) has expanded through vasodilation. Cardiogenic prerenal azotemia occurs when the heart has sufficient preload but either the right or left pump is failing (e.g. arrhythmias, ventricular dysfunction, valvular failure, pulmonary embolus). Finally, there may be sufficient volume in the system and a good cardiac output, but blood is not getting to the glomeruli. This can occur due to fixed obstruction of the afferent vessels of the kidney, transient drug effects (e.g. NSAIDs or ACE-inhibitors), or to pathologic vasodilation of other organs that "steal" blood from the kidneys (e.g. sepsis, cirrhosis).

Renal azotemia

"Renal" causes of acute renal failure are schematized by parts of the nephron. First, the glomeruli may be diseased by either chronic diseases (e.g. hypertension, diabetes) or by inflammatory events (glomerulonephritis; GN). The tubules can be damaged by toxins (e.g. radio-contrast, rhabdomyolysis, medications) or during low-flow states (i.e. prolonged prerenal events, especially shock). These tubular injuries are termed acute tubular necrosis (ATN). The interstitium can become inflamed (e.g. interstitial nephritis – IN - due to antibiotics or non-steroidal anti-inflammatory agents).

Post-renal azotemia

Post-renal causes refer to obstruction of urine flow out of the kidneys due to obstruction (e.g. papillary necrosis, ureteral obstruction, bladder dysfunction or obstruction, prostate disease).

The diagnostic algorithm centers on these distinctions. Post-renal causes are ruled out by placement of a urinary catheter (high post-void residuals suggest obstruction at the level of the bladder) and renal-system ultrasonography. Renal causes are examined by microscopic examination of the urinary sediment (glomerulonephritis has dysmorphic red cells and/or red cell casts, ATN has tubular "muddy brown casts," and IN has abundant white blood cells). A fractional excretion of sodium in the absence of recent furosemide administration, is used to screen for a prerenal cause or contribution (a urine sodium/serum sodium * serum creatinine/urine creatinine < 1% suggests that reduced flow is contributory). In cases of GN, a kidney biopsy may be needed to ensure a diagnosis before commencing potentially morbid cytotoxic anti-inflammatory therapies.

If there is no urinary obstruction (i.e. low post-void residual and ultrasound without hydro-), the urinalysis is bland and the patient is making reasonable amounts (>30 ml/H) of urine, it is often reasonable to reverse prerenal causes (i.e. volume expand patients with hypovolemia, tune the heart for congestive heart failure) and observe urine output and BUN/creatinine. If azotemia improves, then no further work-up may be required in many cases. Obstruction above the level of the bladder (hydronephrosis and/or hydroureter) requires a urology consultation for urgent drainage. Abnormal urinalysis requires closer microscopic evaluation of the urinary sediment and appropriate work-up as suggested above in the section on "Renal azotemia."

The temporal course of renal failure depends on several factors: 1. The nature, magnitude and duration of the inciting injury, 2. The precedent function of the kidneys, and, 3. Avoidance of additional insults during the maintenance or recovery phases.

Figure 7 offers several possible courses – but fully defining #1 and #2 above lend the best insight into which pattern any particular patient will follow. Patients with normal kidneys have abundant reserve, whereas patients with chronic diseases that have destroyed nephrons, require a lesser injury to develop "acute on chronic renal failure." So an acute injury may eliminate sufficient numbers of additional nephrons to render the kidneys in permanent failure or in long-term insufficiency (i.e. enough to clear some of the waste, but not down to normal levels).

Classically, following ATN, patients may go through an anuric or oliguric (urine<400 ml/day) phase that lasts days. Those that recover will spontaneously convert to a non-oliguric or even polyuric phase. It is crucial NOT TO INSULT THE KIDNEYS AGAIN DURING RECOVERY (see Figure 7). Insofar as the recovering kidney is not optimally regulating intravascular volume, these patients are at risk of developing hypovolemia and a pre-renal insult during recovery. Similarly great care should be taken to eliminate all nephrotoxins during recovery.

Patients with renal failure often develop hyperkalemia that should be treated if >>5.0 meQ/L (see below) and potassium loads should be avoided. In mild failure, a non-gap metabolic acidosis (RTA) develops, but in moderate to severe failure an anion-gap metabolic acidosis is common. Both can be treated with bicarbonate administered by gut or vein. Many medications that are excreted by the kidneys must be reduced (proportional to the creatinine clearance) to avoid toxicity.

Hyponatremia

The approach to hyponatremia should be "second nature" to every 4th year medical student – it may be the most lectured-about topic in Medicine. Since sodium levels of less than 120 meQ/L can be life-threatening (coma, seizures), hyponatremia requires a thoughtful and deliberate approach to diagnosis and correction. The first step is to determine the intravascular volume status of the patient i.e. skin turgor, mucous membrane hydration, orthostatic blood pressure changes, jugular venous pressure, peripheral edema. Hypervolemic hyponatremia is due to CHF, cirrhosis and nephrotic syndrome, and responds nicely to gradual diuresis with furosemide (20-60 mg depending upon creatinine clearance). Hypovolemic hyponatremia most commonly results from over-diuresis and/or hydrochlorothiazide effects which can be corrected with normal saline (initial bolus to correct orthostasis, then 75-100 ml/H) and potassium repletion.

The most potentially dangerous form of hyponatremia is euvolemic hyponatremia (differential diagnosis: SIADH, hypothyroidism, hypoadrenalism, polydypsia). Since correction of SIADH-related hyponatremia with normal saline may *lower* the sodium, extreme care must be taken in the initial hours of management to ensure that sodium is increasing and at the appropriate rate.

While sodium levels less than 120-125 meQ/L can be life-threatening, so too can overly rapid correction. Women, whose sodium dropped gradually are most at risk of a disastrous syndrome "pontine myelinolysis" which can cause locked-in syndrome – a patient who is awake but cannot move. While pocket hand-books suggest various approaches, the concept that one-formula fits all is not credible. Every patient varies with time as to how much ADH, aldosterone, etc. they have circulating. Accordingly, the key to correction is to begin fluid infusions and monitor the serum sodium every 2 hours until the rate of correction can be determined (i.e. constant rate of correction over 3-4 measurements). The goal is for the sodium to increase at 0.5 meQ/H and never faster than 1 meQ/H. Hypervolemic hyponatremia will respond readily to furosemide and seldom leads to overcorrection. Hypovolemic hyponatremia responds to normal saline and also rarely causes pontine myelinolysis. It is not unreasonable to initiate correction of euvolemic hyponatremia with normal saline (75-100 ml/H) when there are no neurologic signs/symptoms and hypertonic saline (50-75 ml/H) with neurologic abnormalities. Irrespective of how care is initiated, sodium levels should be checked every two hours to ensure the proper rate of correction. Once the velocity of change is determined, the frequency of testing can be decreased. Furosemide can enhance free water excretion when the rate of correction is insufficient with crystalloid. When the sodium has reached 130 meQ/L, fluids can be discontinued and free water restriction should be continued for the duration, when SIADH persists as the cause.

Hypernatremia

Although diabetes insipidus can cause hypernatremia in brain-injured patients (marked by huge output of urine that looks like water) or due to medications (e.g. lithium-induced nephrogenic DI), the most common cause is simple free water depletion. Since intravascular hypovolemia often accompanies severe hypernatremia, repletion of the intravascular space with normal saline at a brisk rate (boluses of 500-1000 ml) until replete should be followed by free water replacement, either by gut and/or intravenously (with D5W or 1/2NS) if the gut is unusable. As with hyponatremia, the correction should proceed at <1 meQ/L/H to avoid formation of "idiogenic osms" that can precipitate brain edema.

Hypokalemia

The most common cause of hypokalemia is diuretics and the preferred route of correction is potassium administered by gut. While potassium can be given up to 20 meQ by vein over 60 minutes, the intravenous route need only be used in emergencies and/or when the gut cannot be used. Except when the gut is unusable, intravenous potassium is a bridge to suppress arrhythmias until oral potassium can be absorbed. Remember that correction of hypomagnesemia is required to avoid kaliuresis that impedes potassium repletion.

Hyperkalemia

Most commonly due to renal failure, over-supplementation, tissue break-down (e.g. rhabdomyolysis), and organic acidosis (e.g. DKA), hyperkalemia can cause cardiac arrest if extreme. In general, potassium levels above 6 meQ/L are dangerous and any level above 5-5.5 meQ/L warrants an electrocardiogram to check for cardiotoxicity (P wave abnormalities, peaked T waves, newly widened QRS). Signs of cardiotoxicity require administration of cardioprotective intravenous calcium (-gluconate or –chloride), measures to shift potassium into cells (albuterol 10 mg aerosolized over 20 minutes; 5 U intravenous insulin with 1 amp of D10W, 1 amp of sodium bicarbonate) and measures to dialyze potassium from the body (kayexalate for the gut, with or without hemodialysis if the kidneys have failed). In extreme cases (e.g. QRS prolongation and/or pre-sine-wave changes), all of these measures are used. In less severe cases, calcium (IV) and kayexalate (PO or PR) can be coupled with 1-2 K-shifting therapies to yield a safe potassium level.

ENDOCRINOLOGY

Diabetes

Diabetic ketoacidosis (DKA) results when a diabetic fails to self-administer sufficient insulin and/or experiences an acute physiologic stress (e.g. infection, MI, hypovolemia). Patients present with anion gap metabolic acidosis and ketonuria. Ketonemia may not be detectable in untreated DKA because the serum test does not detect the prevalent ketone moeity (hydroxybutyrate) in the early phase of the illness. Treatment is very simple: Volume expand with crystalloid. Start with initial boluses of 500-1000 ml until good urine output is achieved. Continue with 250-500 ml/H of normal saline to maintain euvolemia and administer insulin; classically a single bolus of 5-10 U (0.1/kg) followed by a continuous infusion of 5-10 (0.1/kg) U/H. Hourly fingerstick glucose measurements are used to determine when (i.e. a level of 250 mg/dl) to add glucose to

the infused fluids. Continuous insulin infusion continues until ketosis clears. Since closure of the anion gap (to 10-12 meQ) is a surrogate marker of cleared ketones – electrolytes must be drawn every 2-3 hours. Hypokalemia and hypophosphatemia are common in later phases of correction, so phosphorus levels must be measured starting after 2-4 hours of treatment. When the anion gap normalizes, a dose of subcutaneous insulin 3-5 U is administered as the insulin infusion is weaned over 30-60 minutes. Glucose-containing intravenous fluid is administered until the patient is able to eat. Subsequent insulin requirements can be determined by sliding scale and/or use of the basal-bolus-correction strategy (BBC; see below).

The second acute emergency for diabetics is hyperosmolar non-ketotic coma (HONC). Management is the same except that continuous insulin infusion proceeds until the fingerstick glucose reaches 200 mg/dl at which point continuous insulin can be substituted with subcutaneous as for DKA.

Routine management of glucose levels of hospitalized patients with diabetes and/or stress-related hyperglycemia is an area of active flux in 2006. One randomized study suggested that critically ill surgery patients benefit from continuous infusions of insulin and frequent fingersticks to maintain glucose in the 80-110 mg/dl range. A second study by the same authors, using the same methods performed in medical critical care patients failed to demonstrate mortality benefits. Although many institutions have adopted this approach, it is logistically difficult and should be confirmed in future studies. Additionally, the concept of very tight glucose management has already been extrapolated to care of ward patients, without data. Since insulin infusion requires very frequent fingersticks and patient discomfort, it is not appropriate to subject ward patients to this strategy without data. Historically, a "sliding scale" of regular insulin was written to address fingerstick values obtained every 4-6 hours until acute stressors (that promote hyperglycemia), diet and corticosteroid doses stabilize allowing a gradual increase in long-acting insulin, to reduce the need for sliding scale coverage. No single sliding scale fits all patients (who differ with regard to their insulin resistance, insulin production and counter-regulatory milieu), but Figure 8 (see next page) presents a starting sliding scale that my housestaff often uses with reasonable results. Some patients with insulin resistance may require more and some who are super-sensitive to insulin, less.

While sliding scales may be helpful to quantify total daily insulin requirements, the "basal, bolus, correction" (BBC) strategy is used for hospitalized patients whose acute stress/steroid dose/feeding is stable and after determining the total daily insulin requirements. So within a day or two, nearly all patients should have started basal-bolus dosing. The "basal" refers to administering roughly ½ of the daily insulin requirements as twice daily NPH or once daily glargine insulin. The "bolus" refers to a preplanned amount of short-acting insulin administered with meals (usually 40-50% of the daily requirement in divided doses administered just before meals). The "correction" refers to administering extra short-acting insulin (using a sliding-scale) for preprandial hyperglycemia (measured by finger-stick). Reasonable targets for ward patients are values of 100-150 mg/dl. However, routinely allowing glucose values >150 mg/dl is not advisable in light of the available data. In previously diagnosed insulin-requiring diabetics, it is not unreasonable to use their normal outpatient dosing *if they are eating normally*. Acute stress, medications and fluctuations in diet may affect insulin requirements, so great care must be taken to measure pre-prandial and before-sleep finger-sticks to adjust insulin as needed.

Adrenal insufficiency

Adrenal insufficiency is a potentially deadly disease. Since many patients take steroids for inflammatory diseases ranging from connective tissue disease to asthma/COPD, the prevalence of secondary adrenal insufficiency in the general population is relatively high. Accordingly, any patient who has taken any corticosteroid (inhaled or oral) for more than a month, should be considered for replacement doses of hydrocortisone during acute illness to prevent adrenal crisis. When a patient with a disease for which steroids is usual treatment presents with altered mentation, the diagnosis of adrenal insufficiency should be entertained. Note that hypoglycemia, hyponatremia and hypokalemia are helpful but seldom present. There is minimal risk in empiric replacement of the adrenal axis with stress doses (80-100 mg hydrocortisone every 8 hours) while awaiting random and/or cosyntropin stimulation tests, whereas failure to do so in such a patient can lead to shock and death. Hospital-acquired "relative" adrenal insufficiency due to shock, DIC or severe acute illness may also result in the need for replacement doses of corticosteroids.

GASTROINTESTINAL

Gastrointestinal hemorrhage

Gastrointestinal hemorrhage is a relatively common reason for and complication of hospitalization. Upper gastrointestinal bleeding manifests as hematemesis and/or melena, whereas lower bleeding (below the ligament of Treitz) presents with maroon stool or hematakezia. Placement of a nasogastric tube to rule out bleeding in the stomach is a universal step. The approach to resuscitation is very simple: place 2-3 large-bore, short-length intravenous catheters and treat with crystalloid until hypovolemia is corrected. If patients have orthostatic or static hypotension, 500-1000 ml of normal saline should be administered as boluses until signs are corrected. It is generally wise to type and cross patients in case transfusions are required, but blood products should NOT be administered unless there are clinical indications: e.g. exsanguinating hemorrhage, tachycardia at rest despite correction of hypovolemia or severe coronary disease and hematocrit<30% (a soft recommendation predicated on Level II evidence, which becomes firmer if the electrocardiogram demonstrates ischemia or the patient has angina). Platelet transfusions should be considered for brisk bleeders who have received aspirin or other NSAIDS in the previous 24-48 hours, or in patients who are exsanguinating (therefore requiring the equivalent of whole blood). Fresh frozen plasma is also required for severe hemorrhage and those with prothrombin time>15 s. A gastroenterologist can help with upper GI hemorrhage (therapeutic endoscopy/hemostasis), whereas severe lower GI bleeding requires radiologic idenfication of the source (via tagged red cell scan and/or angiography with embolization or vasoactive therapy). It is generally wise to inform a surgeon, especially in brisk GI bleeding, since the need for >5 units of packed cells in a 24 hour period is a general threshold for surgical interventions with acute hemorrhage.

There is some data to suggest that patients with prolonged hypotension, mental status changes, high APACHE II score, and visible vessel on endoscopy are at high risk and may warrant admission to an intensive care unit for monitoring and management until stable. Irrespective of where patients with GI bleed are admitted, they should be monitored carefully for signs of hypovolemia (orthostatic checks every 3-4 hours until clearly stable OR by continuous CVP in particularly unstable patients). Frequent checks of the hematocrit (every 4-6 hours) may be required until it has stabilized (adjusted for transfusions – where each transfused unit should raise the hematocrit by 3%). Good trainees will report whether patients are sufficiently resuscitated based on orthostatic blood pressure measurements or CVP AND sequential hematocrits, including the number of units of packed red cells transfused in a given time interval (e.g. “the hematocrit was 30 at 4 AM yesterday, the patient has received one unit of packed cells since and it was 32 at 4 AM today”). When a patient has had a stable hematocrit for more than 12-24 hours and/or the hematocrit has increased 3%/unit transfused, then bleeding has stopped. Trainees often mistake ongoing blood in the stool as ongoing bleeding. In general, patients continue to pass old blood for several days after they’ve stopped bleeding.

Finally, it is not uncommon for hospitalized patients to experience a drop in the hematocrit. If there are no overt signs of bleeding (i.e. hematemesis, melena, hematakezia), a peripherally drawn hematocrit should be measured to ensure that the decrease is not factitious (e.g. drawn above a line infusing saline or from a central line with insufficient discard of fluid-diluted blood). Similarly, drops of less than 3-4% may be associated with dilution of infused fluids (when O>I by 1+ liters of fluid) and repeated phlebotomy. If the drop is associated with instability, a site of bleeding should be ascertained. Bleeding into the thighs (from IV sticks), thorax (rarely) and retroperitoneum occur occasionally. Hemolysis is also a rare cause of decreasing hematocrit. Bleeding into the gut is most common – and the goal is to identify overt blood NOT occult (i.e. “OB”) positivity. Gastric sampling and examination of stools (sometimes requiring digital exam) is indicated in the work-up for dropping hematocrit in the hospital.

Pancreatitis

The most common causes of pancreatitis are biliary track stones and alcoholism. Patients present with epigastric abdominal pain, nausea and vomiting. Diagnosis is made through the typical history and confirmatory labs (an elevated serum amylase/lipase). Severity can be judged using the APACHE II or Ranson’s scoring system, but in general patients with tachycardia and/or severe hypovolemia are likely to require resuscitation and monitoring for multiple organ dysfunction/failure. Common duct stones should be ruled out early by biliary ultrasound so percutaneous, endoscopic or surgical drainage can be performed if needed. Although CT’s with thin slices of the pancreas are often performed to

obtain a baseline, they seldom impact therapy in the early phases of disease. In severe pancreatitis, especially with necrosis, there is some evidence that administration of broad-spectrum antibiotics (e.g. imipenem) improves outcomes.

Hepatitis

Alcoholic (and other toxic) and viral hepatitis are most common. Patients present with nausea and/or jaundice. Most cases are self-limited. Abstinence from alcohol usually leads to improvement in cases of alcoholic hepatitis. Some cases of toxic (most commonly acetaminophen) hepatitis or infectious hepatitis (B more commonly than A) may lead to "fulminant hepatic failure" occurring over weeks to a few months and includes brain dysfunction (cerebral edema is the most common reason for death). These patients may die without liver transplantation. Also, acute on chronic injury can also cause fulminant failure e.g. a patient with cirrhosis with superimposed acetaminophen toxicity. Accordingly, n-acetyl-cysteine should be considered for patients presenting with acute liver failure if acetaminophen levels cannot be checked rapidly.

The Acute Abdomen

All internists should be able to recognize and know differential diagnoses for the acute abdomen. The classic, brief text by Cope is a must-read for all clinicians. Acute severe abdominal pain usually occurs because a hollow viscus has either become inflamed/ischemic or overdistended. Without frank perforation, these illnesses (including appendicitis, bowel obstruction, diverticulosis, cholecystitis, biliary obstruction and bowel ischemia) cause localized pain, tenderness and, sometimes, guarding. When a hollow viscus ruptures into the free peritoneal space, one of two things happens: a piece of omentum plugs the hole and/or compartmentalizes the leak, or, the entire visceral and parietal peritoneum becomes inflamed causing diffuse pain, tenderness and rebound. The acute abdomen is a surgical emergency, so it is incumbent on internist to recognize clinical signs/symptoms and consult surgeons early to reduce the likelihood of morbidity and mortality.

HEMATOLOGY

Anemia

The approach to the patient with anemia is very simple. First, determine the degree of appropriate bone marrow response by computing the corrected reticulocyte count=observed reticulocyte count*observed hematocrit/ideal hematocrit. A corrected reticulocyte count>2% suggests hemolysis. Blood smears should be examined for schistocytes (intravascular hemolysis). Coomb's tests, serum haptoglobin and urobilinogen are adjunctive tests for hemolysis.

A corrected reticulocyte count less than 2% suggests a hypoproliferative anemia, while >2% (normoproliferative) suggests either a normal response to blood loss or hemolysis. Hypoproliferative anemias can be characterized by the size of the red cells (MCV). Macrocytic, hypoproliferative anemias are due to either folate or B-12 deficiencies, or myelodysplastic syndrome. Microcytic, hypoproliferative anemias are most commonly due to iron deficiency from chronic blood loss (in menstruating women, GI bleeding in everyone else). Normocytic, hypoproliferative anemias are most commonly caused by chronic diseases, including inflammatory diseases (e.g. RA, SLE, TA), chronic infections, chronic renal failure (with creatinine>2.0 mg/dl), acute severe illness (over weeks) or endocrinopathies (e.g. hypothyroidism). It is NOT appropriate to order every test for every anemia. Start with a reticulocyte count, check the MCV of the hemogram and then order appropriate confirmatory tests for each category above. Not uncommonly, trainees will order iron studies and B-12/folate before checking the reticulocyte count or taking note of the red cell size. Lazy medicine costs the healthcare system money and the patient blood. However, just as commonly anemia is ignored because it is so common in hospitalized patients; and so it goes uncharacterized and untreated.

There is no data to suggest that transfusions of red cells to anemic (critically ill) patients enhance outcomes. Packed red cells lose oxygen-carrying capacity with time and may be immunosuppressive. Accordingly, transfusions are reserved for patients with life threatening hemorrhage, symptomatic anemia (chest pain, shortness of breath) and severe coronary artery disease with hematocrit<30% (a soft but respected indication). The evidence-based indications for erythropoietin are now an area of controversy (owing to its overuse and some provocative data suggesting it can cause harm when over-used). It should be used sparingly if at all until its safety is more carefully examined.

Thrombocytopenia

The classic teaching is that platelets decrease because of insufficient production or destruction/sequestration. By far, the most common mechanism of thrombocytopenia in hospitalized patients is destruction or sequestration. Those with sequestration from hypersplenism usually present with thrombocytopenia rather than developing it *de no*vo in hospital. Patients presenting with normal platelet counts that then drop usually have disseminated intravascular coagulopathy (DIC). Tests that support the diagnosis include elevated prothrombin and thrombin times, and fibrin degradation products. Sepsis-related coagulopathy, that doesn't make criteria for DIC but in which platelets are consumed, and drug reactions are also common causes of thrmobocytopenia. As many as 1-2% of medical patients will develop antibodies to platelets after receiving heparin. Heparin-related thrombocytopenia (HIT) is worth mentioning, because it may be accompanied by life- and limb-threatening venous or arterial thrombosis. In general, if platelet counts drop in hospitalized patients and DIC or sepsis is not evident, heparin (even flushes) should be stopped. DVT prophylaxis should be changed to a non-heparin method (e.g. pneumatic compression) and patients should be observed carefully for signs of thrombosis (low threshold to ultrasound legs) until HIT is ruled out. If thrombotic, unfractionated-heparin-related HIT is diagnosed, non-heparin anticoagulants (e.g. hiruden, agatroban) are preferred even though the frequency of HIT is much lower with low molecular weight heparin.

Disseminated Intravascular Coagulation (DIC)

DIC is commonly caused by sepsis and other causes of injury (trauma, burns, SIRS). It is diagnosed by a dropping platelet count, elevated PT and/or PTT, elevated fibrin degradation products or d-dimer and prolonged thrombin time. DIC is treated by treating the underlying causes. When hemorrhage accompanies DIC, FFP is used to replace factors, with the knowledge that to some degree this "fuels" the consumption. Occasionally, patients clot with DIC and require low dose anticoagulation. When clotting occurs with concurrent hemorrhage simultaneous FFP and low-dose anticoagulant infusions are titrated to increase the platelet count as a sign of success. In my opinion, this should not be attempted without the aid of a hematologist (whenever possible).

NEUROLOGY

Cerebral vascular accident

Cerebral vascular accident (CVA) is another very common reason for admission to hospital. The abrupt onset of neurologic deficits, even if transient (e.g. transient ischemic attack; TIA), warrants a careful work-up. Patients who have deficits at the time of presentation, should be considered for thrombolysis if hemorrhage can be excluded, if there are no other contraindications and tissue plasminogen activator can be administered within 3 hours of the onset of symptoms. Otherwise, care of CVA is supportive. CT scans are performed to assess the degree of injury, though in early/small strokes, the CT can be normal on admission (and MRI is required to identify early stroke). Note also that posterior circulation strokes, that are particularly catastrophic may not be apparent on a CT – an MRI is necessary to diagnosis non-hemorrhagic CVA, especially if thrombolysis is considered. If hemorrhage is detected, coagulopathy should be reversed and platelet transfusions considered for patients on chronic aspirin therapy. Intracerebral hemorrhage warrants a call to a neurosurgeon (e.g. to clip a leaking aneurysm), though surgical evacuation is rarely performed for nontraumatic brain hemorrhage. Supportive management of CVA includes: an aspirin each day for non-hemorrhagic, non-embolic stroke, full-dose anticoagulation for non-hemorrhagic, embolic stroke, aspiration precautions (head of bed 30° and nothing by mouth if mentation/swallowing are affected), and nursing neurologic checks every 2-4 hours for the first 72 hours (maximal edema). Although good evidence is lacking, in the first several days, systolic blood pressure should be allowed "to be what it is" within reasonable limits (110-180 mmHg) and treated only carefully to avoid rapid increases or reductions.

When CVA results from "malignant hypertension" (usually with very high systolic pressures and signs of other end-organ hypertensive injury e.g. hematuria/proteinuria, congestive heart failure, hemolysis), blood pressure must be reduced carefully with an intravenous agent (nitroprusside, labetolol), usually no faster than 10 mmHg every 1 hour until reaching a diastolic of 110 mmHg, holding for 6-12 hours, then proceeding to 140/90 mmHg over the next 12 hours. Patients who demonstrate signs of aspiration (i.e. gurgling voice/breath sounds, oxygen desaturations) should be considered for endotracheal intubation for airway protection. In general, maneuvers to reduce intracranial pressure (e.g. mannitol and therapeutic hyperventilation) are **not** useful in CVA (and could be harmful). Occasionally with profound intracranial hypertension

when all else is failing, these can be tried to "temporize" but are seldom successful. All patients with CVA should receive thromboprophylaxis – subcutaneous heparin is usually avoided in patients with hemorrhage, but pneumatic compression boots can still be used until the risks of additional bleeding subside. Also, there are now some anecdotal successes with hemicraniectomy for large strokes (and even hemorrhage) accompanied by intracranial hypertension. But this is extreme salvage therapy that is rarely available or appropriate.

In general, the pathogenesis of CVA should be determined. Doppler ultrasound of the carotid arteries should be performed to rule out the possibility of symptomatic, surgically remediable stenosis. Echocardiography is used to examine for structural heart disease. Patients with atrial fibrillation and/or cardiac-derived embolic disease will require long-term anticoagulation with full-dose heparin (either immediately in ischemic or 7-10 days following a hemorrhage stroke). If the size of a non-hemorrhagic stroke is very large, full-dose anticoagulation may be postponed for 5-7 days to reduce the likelihood of promoting bleeding into the stroke. Echocardiography to rule out clot-predisposing chamber dilation or aneurysms, visible left-sided thrombi and/or intracardiac shunt (that may predispose to paradoxical embolism) is reasonable, at least for work up of the first CVA.

Coma

Coma simply means global dysfunction and unconsciousness due to neurologic injury. Patients may or may not have focal neurologic deficits depending on causes (e.g. trauma versus anoxic brain injury versus infectious versus space-occupying lesion). The classic text by Plum and Posner is worth reviewing as it's descriptions of coma and the rostral-caudal progression (i.e. how space-occupying lesions can progress towards death) is timeless and a must-read for all Medicine trainees.

Anoxic brain injury is a special case scenario that bears comment. Any patient who has had a cardiac arrest or prolonged, severe shock is at risk of anoxic brain injury, which may cause coma in its early phases. The general rule of thumb is that the longer coma persists without improvement following anoxic injury, the less likely is the chance of long-term recovery. In general, if a patient's deep coma has not improved in the first 72 hours, the prognosis is very grim. A large majority of patients with persistent, non-improving coma at 72 hours have a high likelihood of going on to persistent vegetative state if they survive at all. Patients should be supported and medications that affect the central nervous system should be used sparingly or not at all if possible (so as not to contribute to neurologic dysfunction). There is now sufficient evidence to support cooling patients after cardiopulmonary arrest for 24 hours resulting in enhanced neurologic outcomes. This is done in ICUs under carefully controlled conditions, monitoring and mechanical ventilation since both cooling and rewarming includes a number of risks.

Delirium

Perhaps one of the most common nosocomial complications, especially of elderly patients is delirium. Almost always, delirium results in the fragile elderly due to the things we do to them in hospital. Medications, sensory deprivation (not providing needed hearing aids and eyeglasses), nosocomial infections (often because we don't wash our hands), instrumentation (Foley catheters especially) and untreated pain are preventable and may contribute to delirium. The lazy doctor's approach is to drug the agitated delirious patient into a stupor, thereby promoting their injury. Since delirium is very difficult to treat once it's begun, an ounce of prevention is the ONLY proven means of reducing this complication that carries a high morbidity and mortality! See section above on routine prevention that lists proactive measures to reduce the frequency of this serious complication.

The mini-mental status exam

There is NO excuse for failing to document quantitatively the mental status of admitted elderly patients. Some present with dementia that may not have been detected. Some develop encephalopathy during hospitalization. Serial tests may quantify changes and when/if patients return to baseline.

APPROACH TO . . .

There are a number of clinical problems that occur with sufficient frequency in hospitalized patients that houseofficers and hospitalists must master the approach early in their careers. Irrespective of the problem, it is imperative to attend to the "ABC's" first. Resuscitation refers to rapid assessment and remedies to cardiopulmonary issues that are likely to eventuate in (cardiopulmonary) arrest if left untreated. Recent data suggest that "rescue teams" that are summoned early to the bedsides of patients with cardiopulmonary changes may improve patients' outcomes. This section will clarify what belongs in a rescue evaluation.

A is for Airway: Is the patient's airway safe? This question is answered by determining whether the patient's neurologic status and airway protective reflexes are intact sufficiently to expectorate the amount of produced sputum. If secretions can be heard, with or without the stethoscope, clattering above the patient's glottis with each breath or with speech, the airway is in jeopardy. Countermeasures must be taken ranging from head of bed to 30 degrees, nasotracheal or oral suctioning, or in severe cases, endotracheal intubation for airway protection. Patients with insufficient airways may need monitoring/care in a special care unit (where they are checked more frequently and personnel are more proficient with airway problems). Also, patients with insufficient airway protective mechanisms should not be fed by mouth without a formal swallowing evaluation (and even if they pass, if you are still concerned, feeding by mouth should proceed very cautiously – perhaps under direct observation).

B is for Breathing: Is the patient breathing too fast or too slow? In general, patients breathing <8/min are doing so because they are obtunded or are approaching respiratory arrest (after a period of prolonged tachypnea). Respiratory rates much above 30/min and especially those >35/min are very problematic because if something isn't remedied quickly, patients will fatigue and suffer respiratory arrest. Young, otherwise healthy patients will be able to tolerate tachypnea for longer durations. However the frail elderly and/or patients with multiple co-morbidities will fatigue more rapidly and such patients should be unloaded with mechanical ventilation (non-invasive if simple COPD exacerbation, invasive if other reasons) unless there is a rapidly treatable reason for the tachypnea.

C is for Circulation: The heart rate and blood pressure should be measured upon entering the room (and while simultaneously gauging the "A" and "B" above). Blood pressure<90 mmHg, especially when accompanied by mental status changes and oliguria, is indicative of shock and requires immediate aggressive resuscitation (see chapter on shock above).

In addition to the assessment/management of the ABC's, each problem will warrant specific examinations and diagnostic work-ups. It is always prudent to read the nurse's notes from the hours prior to your arrival and to interview the bedside nurse who may often provide crucial information. Documentation of the evaluation should always include a dated/timed "SOAP" note. The "S" is for subjective description of the patient. The "O" should include careful measurements of the vital signs, basic mental status and gross neurologic status (e.g. "moves all 4 extremities" unless the call is for head trauma/fall or new neurologic signs that warrant a complete neuro exam), respiratory and heart examinations. The "A" for assessment and "P" for plan should include a list of the differential diagnoses, diagnostic plan and interim therapeutic plan for each problem. When a trainee conducts such an assessment, he should contact the physician of record, discuss the findings and assessment and agree upon a plan of care. This conversation should be documented in the note.

It is ALWAYS prudent to consider whether something – some medicine, some procedure, some treatment – we've done to the patient is causing the problem. Iatrogenic complications are startlingly frequent and one of the leading causes of unexpected hospital morbidity and mortality. Always review patients' medication lists – clinical problems are often caused by medications. Most often such complications are caused by a medicine recently added that the patient had not previously received. But even medications patients have taken for a long time without complications, can cause problems when metabolism, due to liver or kidney dysfunction, change during acute illness.

As the trainee or hospitalist assesses the ABC's he/she is simultaneously speaking with the patient to clarify directives for management. If the patient is capable, you must determine:

Does this patient want any treatments or would he prefer comfort-care only (see below, under end-of-life issues)?

If the patient wants to pursue treatments-for-cure, will he allow a trial of endotracheal intubation, understanding its risks and benefits?

If the patient has a cardiac arrest, does he want to "die a natural death" (i.e. without a trial of CPR) or for personnel to try CPR? Informed consent requires that the patient understand what the procedure entails (intubation, pressing the chest, electric shocks to chest, medications) and, just as important that fewer than 20% leave hospital, many to nursing homes, and often worse off than they were as a result of organ damage during arrest. When the patient is able to have this discussion, clinicians must not defer to surrogates. When patients lack capacity, and "code status" has not been discussed, surrogates (appointed health care proxy, spouse, children, parents, siblings in that order of preference) should be contacted to clarify the patient's wishes regarding these issues. So too should the primary care attending, who may possess insight into her patient's wishes and may want to attend the moment of crisis. Yet, it is as important to respect a patient's autonomy as to provide treatments for organic problems. And care for comfort is still care. The approach to cardiopulmonary failure i.e. shock and respiratory failure, are covered above in the Cardiovascular and Pulmonary sections of this book. I here cover common complaints which house doctors are called to assess.

It is also worth stating here that if a patient opts out of CPR and/or intubation it does NOT mean that they do not want care. In fact, such a directive does not even preclude care in an ICU or stepdown unit, since greater monitoring and more frequent nursing intervention is a feature of such units that may impact outcomes. In other words, refusal of CPR or mechanical ventilation does not preclude all other restorative care efforts – it simply means that the patient does not want these two procedures.

Shortness of Breath

One of the most common symptoms that bring patients to doctor's attention (either for initial presentation or after hospital admission) is dyspnea. The pathogenesis of dyspnea is complicated but can be parsed into several major categories including lung, heart and metabolic in that order of frequency. Dyspnea resulting from lung disease relates to increased work of breathing. There are two mechanical forms of work performed during breathing i.e. resistive pressure work (=flow*resistance) and elastic pressure work (=stiffness*tidal volume). Processes that reduce cross-sectional diameter of the airways i.e. increase resistance, include mucus, bronchospasm – asthma/COPD - and upper airway tumors/edema/inflammation. Processes that increase system stiffness can occur in the lung parenchyma (e.g. edema, pneumonia, large effusions, atelectasis, pneumonitides, sarcoidosis, etc.) or the thorax (e.g. obesity, kyphoscoliosis, large ascites). Heart failure can cause dyspnea by causing cardiogenic pulmonary edema (thereby contributing to stiffness and hypoxia-related dyspnea) and increased airway resistance, so-call "cardiac asthma." Right heart failure may be associated with dyspnea, but is most often caused by left heart failure or lung diseases (described above). Metabolic reasons for or contributors to dyspnea include excess CO2 production (as occurs in exercise, sepsis, systemic inflammatory response syndrome, hyperthyroidism), hypoxia (see lung diseases above, and pulmonary vascular diseases including pulmonary embolism), metabolic acidoses and severe anemia.

As always, a careful history is essential to decipher whether dyspnea is caused by problems of the heart, lungs or both. Rapid-onset shortness of breath is most often due to flash pulmonary edema (in patients with risks or history of ischemic heart disease, CHF, valvular dysfunction and/or hypervolemia), bronchospasm (in patients with a history of reactive airway disease; asthma or COPD), or acute atelectasis due to mucus plugging (especially in debilitated patients with abundant secretions and/or weak cough). Pulmonary embolus (PE) is also a consideration, especially when the patient has risk factors, has not received thromboprophylaxis and/or dyspnea is accompanied by pleuritic chest pain. Massive central PE presents with sub-sternal pain/pressure, whereas most emboli are usually smaller and present with pleuritic (sharp and exacerbated by deep inspiration) chest pain, often lateral to the nipple line. The pre-test probability of PE increases when patients have been immobilized for long periods (including recent long travel), have cancer or are hospitalized for MI, severe lung disease, surgery, hip fracture or stroke.

While there are other reasons, these 4 comprise a lion's-share of cases of acute dyspnea in hospitalized patients. Anxiety/panic are diagnoses of exclusion. After completing assessment of the "ABC's" and obtaining a quick history, the severity of the dyspnea and examination of the heart and lungs are essential. The severity of dyspnea can be gauged by the degree of tachypnea (see above) and the patient's ability to speak in sentences. Some ask the patient to take a deep breath and count until they have to take another breath; after 100-200 such patients, the

clinician has created his own experiential severity indexing system. Others ask patients to repeat a sentence as many times as possible with one breath.

Examination of the lungs is perhaps the most important step. Wheezing without crackles suggests either cardiac asthma (especially if the patient has a history of coronary or valvular disease, or CHF) or primary bronchial asthma (especially if the patient has a history of COPD or asthma). When dyspnea and wheezing are accompanied by substernal chest pressure, new shoulder pain, neck/jaw pain, or typical left arm radiation, cardiac ischemia must be ruled out. Remember that women experience cardiac ischemia with "atypical" symptoms (sometimes with only nausea or epigastric discomfort) more frequently than men. If wheezing is accompanied by bilateral crackles (bases>mid-zone>apices), the likelihood of cardiac-related dyspnea also rises. Absence of breath sounds in a lung zone suggests atelectasis. A third heart sound supports heart failure accounting for dyspnea. Splinted breathing supports pulmonary embolism, though chest wall pain (e.g. rib fractures) can cause splinting and atelectasis. Absence of breath sounds unilaterally or in a large zone can occur with pneumothorax or lobar atelectasis.

In patients with altered mentation and/or reduced airway protection reflexes, micro- and macro-aspiration are very common causes of bronchospasm and/or acute hypoxemia from plugging (atelectasis) or flooding (i.e. "aspiration pneumonitis").

Most causes of acute dyspnea also cause hypoxemia; pulse oximetry is always indicated and a confirmatory arterial blood gas should follow if room air saturations<95% are confirmed. Hypoxemia due to airway disease (bronchospasm) and small PE respond to modest doses (usually FiO2<40%) of supplemental oxygen, whereas segmental atelectasis, significant pulmonary edema due to CHF and large pulmonary emboli often require >40% inspired oxygen to correct hypoxemia.

An electrocardiogram should be performed to compare to previous – presence of new ECG evidence of ischemia in a newly wheezing patient without precedent lung disease is suggestive of CHF due to ischemia. New signs of right heart strain (new right bundle branch block, new right axis, or S1Q3T3) support but are not pathogneumonic for the diagnosis of acute right heart syndrome and PE.

Dyspneic patients should also have an arterial blood gas analysis and chest radiograph. A clear chest radiograph in a patient with dyspnea and hypoxemia is supportive of pulmonary embolus (wheezing not prominent) or asthma (wheezing prominent). New interstitial infiltrates, cardiomegaly, cephalization and other signs of congestion support a diagnosis of CHF. Absence of lung markings on one side, distal to a thin white stripe suggests pneumothorax.

Hypoxemia should always be corrected – NO PATIENT IS AN ANAEROBE. Too often, clinicians have suggested that "the patient lives at home with a saturation of 82%." This is nonsense. The patient is dying slowly (of right heart failure) at home if their saturation is much below 90%. Hypoxemia promotes end-organ dysfunction and must be reversed. Patients with chronic hypercapnia may require more careful titration of inspired oxygen to avoid hyperoxia-induced hypercapnia (O2 saturations 90-92%), but these are a minority of such patients.

Whenever it is unclear whether a patient has cardiac or bronchial asthma, it is wise not to give large doses of aerosolized beta-agonists since these medications can exacerbate cardiac ischemia. Ipratropium bromide can be given safely when significant doubt remains. On the other hand, if cardiac asthma is suggested, use of preload reducers and anti-ischemic medications (aspirin, nitroglycerin, morphine sulfate, beta-blockers) should be considered. While the "prevailing wisdom" was to avoid beta-blockers in patients with COPD, the data are quite clear that even patients with moderate COPD who are having an acute MI benefit from beta-blockade. Greater care should be taken during administration by watching carefully for worsening bronchospasm/increased work of breathing. Whenever PE is suspected and the risk of major bleeding (especially intracranial) is not excessive, full-dose anticoagulation should be started until thromboembolic disease (CT and leg studies) has been ruled out. Obviously patients who are actively bleeding - with overt blood, not occult blood – are not good candidates for anticoagulation and vena cava interruption may be required if PE is confirmed. Anticoagulation also treats cardiac ischemia and is not inherently harmful to patients with bronchospasm or atelectasis (unless the patient is bleeding).

If atelectasis is suspected ask the patient to cough and deep breath, and if oxygenation improves in front of your eyes, you've proven that lung derecruitment is contributory. Positive pressure ventilation may be useful as a diagnostic and therapeutic test for atelectasis (provided secretions are not excessive) and cardiogenic pulmonary edema, both of which improve (dyspnea and hypoxemia decrease) with positive pressure

ventilation (PPV; inspiratory pressures of 10 cmH2O and PEEP of 5 cmH2O with bi-level PPV and inspiratory pressures of 30-35 cmH2O given by intermittent positive pressure breathing devices) that can be applied by facemask at the bedside.

Finally, if a patient's dyspnea and tachypnea is severe and work of breathing is excessively labored, you cannot be faulted for treating with mechanical ventilation (non-invasive bi-level PPV when COPD or cardiogenic pulmonary edema; invasive for all other causes). The intensivist can always liberate the patient from the machine if she arrives to find that the machine is not necessary . . . but she cannot raise the dead. The pulmonary chapter details a simple, safe formula for starting mechanical ventilation. Since the mechanical ventilator can kill the unstable patient, you MUST understand how to initiate it safely (i.e. assist control, tidal volume 8 ml/kg to start then titrated to plateau airway pressure of 20-25 cmH2O assuring sufficient expiratory time – e.g. I:E=1:5 - to avoid auto-PEEP which can develop in the tachypneic and/or bronchospastic patient). You MUST NOT leave the bedside of patients with severe dyspnea until it resolves or you transfer them to a monitored setting (ICU or step-down) where they can be watched minute-to-minute. Continuous pulse oximetry is required to ensure oxygen saturation>90% and an arterial blood gas should be drawn within 15 minutes of starting mechanical ventilation to confirm that gas exchange is improving.

Chest Pain

Houseofficers and hospitalists are often called to assess chest pain. History is very important. After ensuring safe ABC's, the 7 features of pain should be elicited, since prototypical syndromes may allow expeditious delivery of time-sensitive therapies like thrombolysis, anticoagulation or mechanical coronary interventions. The classic teaching is that chest pain may be caused by diseases of the chest wall (muscle, cartilage, bone), heart, pericardium, aorta, lungs, pleura and GI track. Since any pain can cause tachycardia and hypertension, these are not specific for any particular genesis of pain.

Chest wall pain – is usually caused by trauma and palpation in the area of pain elicits tenderness. However, the specificity of this finding is not 100% since pleural pain (as with PE/infarction) can also cause chest wall point tenderness. Since severe chest pain has potentially life-threatening implications, it should not be attributed to chest wall diatheses unless obvious (e.g. a dermatome of zoster) and/or more ominous causes are ruled out.

Heart pain – Risk factors for coronary disease include hypertension, history of MI in a first-degree relative before the age of 50 y, smoking, diabetes, male sex, and hypercholesterolemia. While some women experience ischemic heart pain without macrovascular coronary atherosclerosis, a majority of patients will have one or several risk factors. Ischemic heart pain is referred to the "6-dermatomes." Heart pain is most often described as a pressure, located sub-sternally (in men). It is much less frequently described as sharp or pleuritic (i.e. a point of pain that is brought on or worsened with deep breaths). This pressure or heaviness can radiate to or involve only the epigastrium, shoulders, neck/jaw, or left arm. Ischemic heart pain, especially when it signals infarction, may be accompanied by shortness of breath (due to diastolic dysfunction of the heart often manifest as wheezing with or without bilateral crackles). Women experience substernal chest pressure less commonly than men – heart ischemia and infarction more often involves one or several of these other symptoms or signs. Electrocardiographic evidence of ischemia or infarction is required in concert with chest pain to make an immediate diagnosis since cardiac enzymes take between 6 (as with troponins) and 12 (as with CPK) hours to increase to confirm the diagnosis. Sometimes symptoms and ECG are so obvious that treatment begins even before enzymatic confirmation (remember that enzymes may take hours before they increase and "time is muscle"). Echocardiographic segmental wall-motion abnormalities are suggestive (especially if previously normal). Management of acute coronary syndrome is detailed above, but involves simultaneously reducing myocardial oxygen demand (with morphine sulfate to reduce pain and catechols, beta-blocker to heart rate of 60/min, captopril and nitroglycerin to a systolic blood pressure of 100 mmHg) and increasing supply (with supplemental oxygen, aspirin, anticoagulants, thrombolysis vs. percutaneous angioplasty vs. CABG in that order of aggression). Since the most aggressive and successful of these interventions requires assessment by a cardiologist and/or activation of a special team (i.e. cath suite or operating room personnel), it is appropriate to contact a cardiologist whenever evolving MI is suspected.

Risk factors for pulmonary embolus (PE) include previous thromboemboli, prolonged immobilization (bedrest or travel), cancer, and hospitalization for MI, stroke, surgery, hip fracture or advanced lung disease. Pulmonary embolus has two presentations. Large central PE

presents with substernal chest pain/pressure that is not dissimilar to MI and is accompanied by significant dyspnea, hypoxemia, tachycardia and, in extreme cases, hypotension. Smaller PE's typically present with dyspnea and pleuritic chest pain lateral to the nipple line. The chest radiograph is usually normal but may show plate-like atelectasis, small lung volumes due to splinting, wedge-like infiltrate (infarction), lateral peri-diaphragmatic round infiltrate (Hampton's hump), or segmental oligemia (Westermark's sign). Electrocardiogram most often shows tachycardia, but may have new right axis, right bundle branch block or S1Q3T3. An increase in A-a gradient is present in a large majority of cases as is a mild respiratory alkalosis on arterial blood gas. Anticoagulation (with heparin unless contraindicated) should be started as soon as PE or MI are considered, and dissecting aorta is unlikely. Diagnosis is confirmed with a CT angiogram of the lungs and venous studies of the legs since >90% of PE originate from the legs. Echocardiography demonstrating a thin, dilated right ventricle with paradoxical motion of the septum is supportive of the diagnosis.

Pericardial pain is usually described as sharp in nature and located substernally and/or left of midline, with radiation to back or shoulders. It may be relieved by sitting upright or leaning forward. When it is suspected clinically, electrocardiogram is very useful in securing a diagnosis. There are many features of pericarditis on ECG, but ST (concave up) segment elevations in more than one vascular distribution (e.g. inferiorly and anteriorly) coupled with reciprocal depressions in aVR, and PR depression are supportive findings. Echocardiography may show an effusion and is diagnostic of the relatively rare cases of pericarditis that lead to tamponade. Anticoagulants should not be administered if pericarditis is considered in the differential diagnosis since it may cause conversion to hemorrhagic pericardial effusion.

Aortic dissection is typified by hypertension, tachycardia and substernal "tearing" chest pain that radiates to the back or shoulders. When the history is typical, the diagnosis is easy to make (and is verified by either CT angiography, MRI, or echocardiogram – whichever can be done most quickly and safely). But more often, the presentation is not so obvious. Occasionally, dissections may "stutter" (come and go) or present with syncope or neurologic deficit (if dissection across carotid) or chest pain more typical of angina (if dissection includes a coronary osteum). Maintaining a high index of suspicion in a hypertensive vasculopath can be life-saving, since immediate medical treatment requires beta-blockade to a heart rate of 60/min, afterload reduction (to systolic pressure of 100-110 mmHg) with intravenous labetolol or nitroprusside and an early emergent call to the cardiothoracic surgeon to assess for surgical repair. Anticoagulants should NOT be administered if dissection is considered, since hemorrhage into the intimal flap can promote extension.

Gastro-esophageal reflux and other esophageal pain can be referred to the chest wall. This pain is often burning in nature, though it can also present as heaviness (e.g. with esophageal spasm) that is difficult to distinguish from ischemic heart pain. It may come on when patients are supine and be accompanied by a sour taste in the mouth during reflux episodes. Other than mediastinitis, in which the diagnosis is not difficult (i.e. following instrumentation with an endoscope or rarely after violent wretching), esophageal pain is not an emergency and clinicians must ensure that heart ischemia, PE or dissection – i.e. emergencies requiring time-sensitive, life-saving therapies – are not causative.

Mental status changes

Houseofficers are frequently called to assess patients for acute changes in their global neurologic status. In addition to the ABC's a thorough neurologic evaluation is necessary. If new focal neurologic deficits are present, then acute cerebrovascular disease, most often stroke, is the cause (see discussion in Neurology section for diagnosis and management). However, perhaps more common in hospitalized patient is delirium, which is typified by confusion, inattention, agitation (though there is a quiescent form of delirium as well), and fluctuating level of consciousness. A thorough assessment for causes of delirium, especially a careful review of new medications and search for infections and metabolic abnormalities, should follow (see detailed section on the Yale Delirium Prevention trial above).

A number of metabolic abnormalities can contribute to altered mentation including acidemia, hypoxemia, electrolyte abnormalities, renal failure and sepsis, that should always be considered in patients with nonfocal neurologic examinations and acute global changes of mentation. A review of all medications administered in the past 24 hours often reveals a culprit. Iatrogenesis should be considered in the differential diagnoses for any problem.

Medications to treat agitation should be the last resort. Pain can cause delirium and if suspected should be treated with acetaminophen and narcotics only if absolutely necessary. Too often we add yet another medication-toxin that adds fuel to "cascade iatrogenesis." When the patient's safety is at risk, the use of subcutaneous haloperidol for acute delirium, starting at 0.5-1.0 mg every 4 hours and adding 1-2 more mg with each dose until the patient is safe can be considered. Haloperidol should NOT be given on a "PRN" basis – to work properly it should be given around the clock and in general a geriatrician should assess the patient within 24 hours to more rigorously rule out other reversible causes of delirium. Since new, expensive anti-psychotics have never been proven superior to haloperidol, they should be used with great caution, unless a patient has QT prolongation when haloperidol is contraindicated (but take note that many other antipsychotics also prolong QT). But good doctors will use medications only as a last resort and after they have carefully considered other possible means of reducing patients' agitation. Similarly, restraints should not be used unless absolutely necessary to prevent the patient from harming himself (and never over the phone without a full, documented assessment).

Opioids, benzodiazepines, and anti-psychotics should not be given parenterally in a non-monitored setting. These medications are unpredictable – no physician can look at a narcotic-naïve patient and know what their response will be. Other patients who are at particular risk are the obese (especially those with obstructive sleep apnea), those with severe lung disease and small and/or frail elderly patients. When parenteral narcotics are used (preferably in an ICU, ED or step-down unit), vital signs should be monitored carefully and regularly for the first 10-30 minutes following administration. Hypotension and hypopnea are life-threatening risks. When treating narcotic- or benzo-naïve or other high-risk patients, begin with a test dose of 0.5-1.0 mg (morphine for pain, benzo for anxiety/agitation, haloperidol for non-alcohol-related delirium). Err on the side of the smaller 0.5 mg test dose whenever possible. You can always give more, but reversal agents (e.g. narcan and flumazenil) are not usually available at the bedside and rapid reversal may be required in some life-threatening situations (e.g. hypotension or respiratory compromise). If the patient tolerates the test dose but remains agitated or in pain, additional doses of 0.5-1.0 mg can be given every 5-10 minutes (10-30 minutes if IM or SQ are used since onset of action will be delayed) until pain or agitation are reduced. Remember that benzos and haloperidol do not treat pain, which sometimes contributes to agitation/delirium. I prefer trying non-narcotic pain relievers like acetaminophen before resorting to narcotics for pain, and when I must resort to narcotics, low-dose oral agents are often effective. Take care not to overshoot – if the symptoms/signs are improving err on the side of holding subsequent doses until the peak effect of previous doses is reached. Patients should be observed carefully for signs of developing airway incompetence, as cumulative doses "catch up" over the ensuing hours or days. Not infrequently, a sizeable quantity can be required to calm a patient, but as drug levels build over several days, drug-drug and drug-organ interactions may lead to dangerously high levels and obtundation. Countless patients are harmed with cavalier use of these simultaneously dangerous but useful (if used properly) medications.

Withdrawal syndromes are commonly encountered by house physicians. Patients who consume more than a few drinks every day, must be observed carefully for signs of withdrawal which include delirium, tachycardia, tachypnea, hypertension, dilated pupils, sweatiness and tremulousness. The extreme of alcohol withdrawal, delirium tremens ("DTs") is life-threatening because treatment requires administration of benzos which alter airway competence increasing the risk of aspiration. I favor use of a longer-acting agent (chlordiazepoxide has been time-honored but low doses should be used – like 25-50 mg every 8-12 hours) to reduce the amplitude of swings from extreme agitation to excessive sedation that may occur if larger doses of short-acting benzo (lorazepam) are used. Then "PRN" smaller doses (e.g. 1-2 mg) of lorazepam can be used to reduce agitation that breaks through the basal treatment with chlordiazepoxide. Beta-blockers and/or clonidine have also been shown to reduce the effects of the catechol storm that is DTs.

Withdrawal from narcotics is also an important problem of hospitalized patients. If patients have been taking opiods, for pain or illicitly, for more than a few weeks, they may experience life-threatening withdrawal (with many of the same symptoms and signs of alcohol withdrawal). Therapeutic doses of opiods are used for pain (estimating hospital needs from the dose the patient uses at home) and methadone when the patient desires to stop using illicit drugs. Much larger than "conventional" doses may be required in habituated and/or addicted patients. But great caution must be exercised not to over- or under-dose such patients. An experienced pain/palliative care specialist (or oncologist when chronic pain is cancer-related) can be of great assistance to reach this balance most rapidly. "PCAs" i.e. patient-actuated doses, are an elegant solution since

patients control their own pain management and will not self-administer when sleeping comfortably and cannot exceed a pre-determined rate. Even with PCAs, great care must be taken to ensure that the programmed doses of morphine do not cause excessive respiratory or hemodynamic depression, but are sufficient to attenuate pain. Novices should seek advice of experienced clinicians until facile with these technical issues.

Fevers

While tachycardia and tachypnea are also common early manifestations of sepsis, fever is the most common early sign that brings infection to the attention of the house doctor. A detailed review of the approach to the patient with sepsis is highlighted above and in Figure 6. Since sepsis is the leading cause of death in hospitalized patients, fever must be considered related to infection until proven otherwise and deserves an aggressive pre-emptive work-up (with history, exam, chest radiograph, body fluid gram stains/cultures) and, if appropriate, empiric therapy while awaiting microbiologic work-up.

However, fevers can also be non-infectious. While a diagnosis of exclusion, non-infectious fevers can be caused by: systemic inflammatory response syndromes (e.g. chemical/aspiration pneumonitis, pancreatitis, burns, allergies, ischemic/infarcted/inflamed bowel), medications (either allergy, idiopathic or due to cytokine stimulation in the case of mediator-therapies), atelectasis (usually a low-grade fever), large hematomas, hemolysis, and thrombotic thrombocytopenic purpura (TTP) just to name a few.

Gastrointestinal complaints and the acute abdomen

Nausea and vomiting are common complaints in hospitalized patients. Infectious gastroenteritis is a rare complication of hospitalization, so medications and neurologic reasons (and even cardiac ischemia) should be considered. Assessment of severe abdominal pain is a skill that all good house doctors should master. And for this, I will waste no space or your time here because a book written more than 80 years ago does this elegantly and completely. *Cope's Early Diagnosis of the Acute Abdomen* is a classic that it takes only a few hours to read and is so rich in detail and powerful bedside skills that it should be mandatory reading for every internist and surgeon.

Falls

Falls are too common in hospitals. Debilitated patients may attempt to ambulate. The delirious may jump out of bed. The house doctor MUST ensure that the patient has suffered no injuries with an emphasis on ruling out intracranial hemorrhage. A thorough history and examination of the scalp, and musculoskeletal and neurologic systems must be documented. If there is any sign of head trauma or new neurologic deficits and/or the patient has any risks for major intracranial bleeding (i.e. anticoagulation), a CT of the head should be performed. Otherwise, imaging and assessment should be *ad hoc* based on examination (e.g. long bones, hips etc.).

Restraints should be used only as a last resort if a patient is a risk to himself. All efforts should be made to define the causes of mental status changes and reverse them. Some prophylactic measures e.g. hip guards, have been used to prevent fractures in high risk individuals, but they are not of proven benefit. Sometimes a bedside sitter is required to ensure a patient's safety, but it is money worth investing if the only choices are use of an ICU bed (for an otherwise stable patient) or leaving the patient unattended an at risk. Beds with nets are also used, but while they may prevent the patient from jumping out of bed, they inhibit care and interactions that could be important for re-orienting the patient.

INVASIVE PROCEDURES

Cardiopulmonary resuscitation (CPR)

Although every medicine-surgery resident-in-training is certified in CPR (Advanced Cardiac Life Support; ACLS), the algorithms learned in the ACLS course, in my opinion, do not maximize patients' chances of survival. When you consider that less than 20% of patients who suffer in-hospital cardiac arrest leave the hospital alive, you realize there is room for improvement. While memorizing the treatment algorithms for the various cardiac arrhythmias is a bare minimum, there are several other common sense tips that may be useful.

Firstly, at any CP arrest, there MUST be only one team leader. That person should identify themselves upon entering the room, ensure that the patient does not have a "do not resuscitate" order and IMMEDIATELY begin: a. Assigning essential responsibilities to team-members, and, b. Diagnosing the etiology of the arrest to actuate therapy. Most codes can be run with 6-7 individuals – all who are not assigned responsibilities should be excused to make room for those performing the resuscitation. Upon entering the room, the team leader should identify himself, ensure that chest compressions are being performed and whether they are creating a palpable pulse. Compressions at 100/min should not stop except to feel briefly, intermittently for a spontaneous pulse. The patient should be placed on a bed-board with minimal interruption of compressions. I like to station one individual whose sole responsibility is to keep their hand on the carotid or femoral region for a pulse. The team leader should insist that electrocardiographic patches are placed immediately and that the defibrillator be in automatic mode until he/she is ready to fully assess rhythm and give needed shocks (then it can be taken out of automatic). Ventricular fibrillation (VF), pulseless ventricular tachycardia (VT) and pulseless supraventricular tachycardia (SVT) should be shocked with at least 100 J initially (asynchronized for VF, synchronized for VT and SVT). Strong evidence suggests that early defibrillation improves outcomes following primary cardiac death (and is even more important than airway/ventilation/ oxygenation concerns). Routine ACLS algorithms can be followed subsequently. Attention to the "ABC's" is essential. Ensure that someone is effectively mask-bagging the patient with 100% oxygen until an artificial airway can be secured; initial hyperventilation to 20-25/min may be OK if the patient has been down a while to help reverse acidosis, but then take care not to hyperventilate patients as this can worsen outcomes. Early in CPR, the team leader should ask the room of team-members: "who saw the patient last and what did they look like." Another individual can be assigned to look at the hospital notes just before to examine for descriptive or laboratory clues. For example, patients who experience significant respiratory distress in the minutes or hours prior to arrest, may have primary respiratory failure leading to cardiac arrest. Typically these patients experience tachycardia, giving way to bradycardia and asystole when the mechanism is respiratory-only. Not infrequently such patients reverse (i.e. convert asystole to bradycardia to tachycardia) by hyperventilation with 100% oxygen ± atropine ± epinephrine. On the other hand, complaints of precedent chest pain raise the possibilities of MI or PE. A patient who was completely fine and experienced sudden cardiac death is most likely to have primary heart arrhythmia (with or without cardiac ischemia) though PE can also present in this manner. For example, if I learn that the patient had a hip fracture repaired 3 days before and had severe new-onset shortness of breath with chest pain before coding, pulmonary embolus, irrespective of the heart rhythm would rise to the top of the differential diagnosis. I'd be inclined to thrombolyse if initial routine ACLS is unsuccessful. High or low potassium, acidosis or hypoxemia before the arrest may provide important information that could affect treatments. If a patient had renal failure and hyperkalemia earlier, I'd be more inclined to try a push of bicarbonate, epinephrine, and insulin/D50 if initial ACLS failed. If you are suspicious that the patient is acidemic, then hyperventilating may be essential to restoring a stable circulation and/or for medications to be helpful, but ensure respiratory rate no more than 15-20 breaths/min (more can induce dynamic hyperinflation, reduced venous return and alkalemia). While administration of bicarbonate has fallen out of favor (as it may worsen intracellular acidosis), occasionally it allows transient improvement in pH vital to restoring cardiac and vascular reactivity. The goal is to restore a heart rhythm long enough to allow you to figure out the genesis of the arrest. What they don't teach you in ACLS i.e. looking at CP arrest like a mystery or riddle that needs to be solved, may be as important as actuating the algorithms. Good CPR is much more than simply going through the motions of an algorithm; to maximize survival it requires concurrent problem-solving to understanding the pathogenesis/mechanism of the CPA AND team leadership/coordination. There is now very strong evidence that the quality of teamwork impacts outcomes, so it is imperative that the leader assign tasks, thinks aloud (so that others can offer ideas/corrections as needed) and is multi-tasking in a manner that inspires confidence of the team.

If electrical activity has returned anew and/or an intervention (medication or shock) is administered, chest compressions should be stopped briefly and the person assigned to monitor the pulse should be queried whether there is a spontaneous pulse. If so, check the blood pressure and if low, administer fluids and pressors (norepinephrine or dopamine by continuous infusion) until the blood pressure is sufficient (>80 mmHg). Patients should be transferred to an ICU as soon as they've had a stable circulation for more than a few minutes (to avoid having to resuscitate *en route* in the hallways).

In my opinion, inpatients should have the option to forgo CPR and mechanical ventilation. The only way to determine whether patients would accept the risks, benefits and alternatives of CPR is to ask them. If someone is sick enough to be admitted to the medical-surgical hospital wards, he/she is sick enough to have a CP arrest. So all patients should receive information about CPR and have the opportunity to "opt out."

Rescuing the hemodynamically unstable patient

In 2007, national organizations required that hospitals create early response teams to address cardiopulmonary instability of hospitalized patients. Team leaders should simultaneously assess the "ABCs," begin a trial of hypothesis-driven treatments to stabilize the patient and simultaneously determine the patient's predilections about invasive therapies (i.e. CPR and endotracheal intubation). Figure 10 outlines a simple algorithm for this "procedure" while Figure 11 provides more explicit guidelines for stabilizing the patient who does not have arrhythmic (i.e. CPR-requiring) hemodynamic instability.

Medical procedures

Many texts and manuals have been devoted to procedures including intravenous catheter insertions (both in small and central veins), thoracentesis, paracentesis, lumbar puncture, arterial catheterization, bladder catheterization, nasogastric tube placement. So rather than do a mediocre job with something that's already been done, I will share the following.

First, you owe it to any patient (or his surrogates when the patient is incapacitated) to explain procedures, risks, benefits and alternatives and allow them the opportunity to refuse. Dr. Martin Tobin and I created a document for the American Thoracic Society to explain critical care and common invasive procedures in laypersons' language. The procedure information sheets can be down-loaded from the internet (http://thoracic.org/sections/clinical-information/critical-care/patient-information/index.html) and will complement your explanations during the informed consent process. The only time it is permissible to disrespect patients by not obtaining (and documenting) their understanding and consent (or refusal), is during life-threatening emergencies. Second, FAR TOO MANY (UNPROVEN) INVASIVE THERAPIES ARE USED IN AMERICAN HOSPITALS. In many hospitals, consent is neither requested nor obtained and many of the "monitoring techniques" (i.e. urinary catheters, arterial catheters, central venous catheters) have little or no evidence to support their efficacy and plenty of evidence to demonstrate their multiple complications. When I trained, nearly half of patients received pulmonary artery catheterizations (PAC) because we physicians followed an ideology and not science. Subsequently, studies of PAC demonstrated no benefit (or worse). This is certainly the case with many other "routine practices" that residents are taught to perform without thinking. Simply consider: if it was your mother, would you want her to get that central line, a-line or Foley? If your answer is yes, then proceed, but be honest with yourself and take it out when it is no longer necessary.

Meticulous attention should ALWAYS be paid to maintain proper sterile precautions. Even in emergencies, skin can be prepared for invasive procedures – but when time permits, both skin preparation AND creation/respect (with mandatory cap/gown/mask) of sterile fields should be observed. Recent data suggest that using a check list before beginning procedures (ensure coagulopathies/platelet defects addressed, verify patient/body part for procedure/consent, prep skin with cyclohexidine in normal 3-swab fashion, scrub, cap/gown/glove, drape with large sterile field, perform procedure with meticulous attention to sterile technique, document procedure with all necessary elements in record, complications/fluid test results when appropriate). A tutorial for invasive procedures is provided by the New England Journal of Medicine and indexed nicely on the Cornell Medical College Website (http://www.med.cornell.edu/education/curriculum/third/med_inv_pro.html)

Finally, unless in extreme emergency, **NOBODY** should perform their first procedure without an already-certified individual present to supervise. Every novice is entitled to two, maybe three attempts, after which the supervisor should mercifully intervene. Experts should even consider using technical aids (for example ultrasound-guidance) when initial attempts fail. I've been to the bedside of too many patients who looked like "pin cushions" because an intern or resident didn't appreciate that "it wasn't gonna happen." It is no defeat to fail – even the best may fail a procedure. It is unacceptable to keep trying at the excessive risk or discomfort of the patient.

Family meetings

Regular meetings with the patient and family members (if the patient agrees), helps keep everyone "on the same page." If you use clear language and are unambiguous about goals and care plans, family meetings ensure that patients'/families' expectations remain consistent with your expectations. You should update the competent patient **daily** about his/her progress and discuss with others only with his permission. When

patients lack capacity, regular family meetings (at the frequency requested by the family) ensure that care is consistent with what the patient would have wanted.

HELPING PATIENTS DIE

Nowadays, only the sickest patients are admitted to hospital. So death is quite common. Since we physicians are mortal (though we may like to think differently) and may be no more comfortable with the idea of death than patients, we must learn to help patients die dignified deaths when medical therapies are unlikely to return them to a quality of life they can accept. Unfortunately, even the most experienced clinicians may disagree about when continued attempts at care-for-cure become futile. We must recognize that this "threshold" is somewhat arbitrary and varies based on our medical knowledge, experience and comfort with death. Irrespective, if we are to serve patients, it is imperative that we become as facile with this "procedure" as we are with fixing the patient's sodium. We must also recognize that it is NOT defeat when we've done our best, ensured that other experts agree and the patient cannot be saved. While care-for-cure may be our goal, when patients are dying despite our efforts, we must become facile transitioning to care-for-comfort-unto-death; in other words a dignified death.

Trainees are often in a difficult position because they may not always have the requisite medical knowledge to know when care-for-cure is futile, and only extends the patient's death and suffering. Moreover, in most cases residents have not had longitudinal relationships with their inpatients. Accordingly, residents should rarely, if ever, approach a patient to explain that they are dying without first discussing with experienced attending physicians who know the patient.

Moreover, there is definitely a right way and wrong way to have these discussions. There are many websites and books to help doctors improve their skills, but the general elements are pretty straightforward. First when the patient has capacity i.e. can make his/her own decisions, the discussion should always occur with the patient. Although family members can be invited into the room if the patient agrees, respect of autonomy requires that the patient provide informed consent for his care plan. Whether speaking to the patient or loved ones, it is usually best to ask them to explain what they understand. This allows you the opportunity to gauge what they know already and the level of explanation (syntax, vocabulary, concepts etc.) that you'll need to use for them to understand your message. It is very important to understand patients' expectations about the acute illness AND their values about what quality of life is acceptable to them. While you can never know prognosis and durations for certain (and it is wise to use words to this effect), the possible and likely clinical courses, including disposition and quality of life, should be explained so that they can reconcile it with their values.

If the likelihood of success as they define it is very low, then, in broad strokes, explain how the current illness has effected the pre-existent health status to make care-for-cure highly unlikely ("never say never" always leaving room for hope). Explain that while in hospital, if the heart stops i.e. the patient dies, "CPR" (i.e. compressions, shocks, and a breathing tube) is used to restart the heart. It is successful in less than 20% of cases and even if initially successful may cause damage to vital organs. Then ask: "If you (or your loved one) were to pass on do you want (or would he want) CPR?" Similarly explain mechanical ventilation i.e. "if he should have difficulty breathing a tube placed into the lungs and attached machine might be used to do the breathing until a repair is performed, but with no guarantee of success." Then ask: "If you (or your loved one) were to have such difficulty breathing and couldn't communicate would you (he) want mechanical ventilation?" NEVER ask anyone: "Do you want us to do everything?" This is a common error of neophytes that promotes only one answer: "of course I want you to do everything." <u>Patients and loved ones need to understand that if they begin to suffer and choose NOT to receive life-sustaining therapies that care continues; they will be kept comfortable with medications.</u> Morphine and anxiolytics can be titrated to eliminate patients' dyspnea, pain, and anxiety. Our orders must be explicit allowing bedside nurses large latitude to provide as much of these medications as required – not so much as to promote death, but as much as is needed to ensure a painless, peaceful death.

Additionally, remember that if a patient has decided against CPR and mechanical ventilation and is failing non-invasive therapies for shortness of breath, it is NOT acceptable to allow that person to asphyxiate. They should be transitioned to comfort-care and loved ones (when appropriate) should be notified in case they wish to be present when the patient dies.

When the patient lacks capacity i.e. cannot understand and/or make rational decisions on his own behalf, the discussion occurs with "surrogate" decision-makers (individual identified in a written advance directive, spouse, parent, children, distant relatives in that order of priority). But the goal is not to determine what they want but rather to examine what the patient would want in this situation (that is carefully explained to them with your best guess of prognosis). NEVER ask: "What do you want us to do," but rather: "What would he want us to do." This helps to relieve guilt while simultaneously respecting the patient, keeping the focus on him. Surrogates don't always know what a patient would want, but often you are left with them to infer, sometimes with advance directives, how to proceed. In Connecticut, advance directives only address terminal or permanently unconscious conditions, so they are very narrow. Since certainty regarding "terminal" is rare, when patients lose capacity, surrogates are best situated to infer what the patient would want. In general, the goal is to obtain unanimity of loved ones before changing a patient's code status or withdrawing/withholding life-sustaining modalities. This is almost always achievable and if there is discord, repeated family meetings and enlisting the help of the hospital ethics committee can help resolve disagreements.

Figure 9 (see next page) shows a simple algorithm for end-of-life decision-making in acute, chronic and acute-on-chronic scenarios. Whenever the patient possesses capacity, physicians should engage him (not surrogates) in end-of-life decision-making. The most appropriate time
to create advance directives and gauge patients' predilections about end-of-life issues, life-sustaining therapies and acceptable quality of life is BEFORE they become acutely ill. That is not to say that advance directives (AD) should ever substitute for a fresh discussion with a capable, acutely ill patient. Rather if the patient becomes so ill that they lack capacity, ADs and previous discussions aid surrogates (either appointed through the AD or loved ones in the absence of directives) in making decisions that are consistent with patients' wishes. Prospective discussions with patients (and surrogates, when patients are incapable) should always consider what the patient would want . . . with regards to both quantity and acceptable quality of life.

Importantly, hospital "rescue team" personnel are well situated to help patients who are in extremis to die peaceful, dignified deaths. Upon arrival to a struggling patient's room, the houseofficer or hospitalist should: rapidly assess the genesis of the patient's suffering while simultaneously determining from the patient, if possible, whether they wish limits to the treatments used to address the situation. If the patient has chosen "no intubation, no CPR," then the physician has a responsibility to ensure this remains the capable patient's instructions, all the while ensuring the patient that medications will be provided to ease the discomfort irrespective. Patients should be given a choice of whether to undergo or forgo a brief trial of medical, non-invasive therapies before proceeding to comfort care (morphine and/or anxiolytics titrated to comfort) or immediate care for comfort unto death. For those who choose, a prompt trial of therapy should follow until the patient "declares himself" a responder or non-responder. Clinicians should not leave the bedside until the patient either improves or management is "transitioned" to care for comfort. Indeed too many patients who are "no codes" experience unnecessary suffering because clinicians persist in a prolonged trial of non-invasive therapies. Patients' primary attending physicians should be notified of the events, so they are both informed and have the option to attend the deaths of their patients if they wish. To date, all of the research emphasis regarding rescue teams has been on preventing codes and unnecessary ICU care. However, I suspect that these teams can play an equally important role in helping patients die without excessive suffering, in a manner that is consistent with their end-of-life choices.

A word about care-for-comfort. Some have referred to it as "withdrawal of care" which is a misnomer. Care proceeds just as vigorously and carefully as care-for-cure, but the goal changes from restoration of health and function acceptable to the patient to death without suffering. If oxygen makes the patient more comfortable, then it should be administered, but with the understanding that it may prolong dying. The mainstay of care-for-comfort is opioids, which reduce pain and dyspnea, and benzodiazepines, which reduce anxiety. Skilled nurses in ICU's or hospice units should manage administration of medications – enough to ensure that symptoms are eliminated and not so much as to promote cardiopulmonary depression. In reality, these medications may hasten some deaths. But that is not the intent; and they should not be used to promote a precipitous death. Since patients respond variably to narcotics (for some a milligram causes a deep coma and for others 10 milligrams has little effect), this is an art more than science. To provide the skilled nurse with sufficient latitude the order should read something like:

Morphine sulfate 2 mg intravenously every 2 minutes as needed (or by continuous infusion up to 40 mg/hour) until no pain and shortness of breath AND lorazepam 2 mg intravenously every 5 minutes as needed until no anxiety. Excellent nurses have been taught to reduce the doses if the patient becomes comatose and respirations decrease to less than 8-10/min (after all, such a patient is not likely suffering from dyspnea) so as not to promote medication-precipitated death.

TIPS FOR TRAINING

Know what you're supposed to know

The best houseofficer knows all of the facts about his/her patient. Within 24 hours he/she has scoured previous medical records, called other facilities for results (when important data resides elsewhere) and confirmed the accuracy of previous diagnoses e.g. PFT's when a patient is "labeled" with lung disease, stress test/ECHO for heart disease etc. **All** abnormalities (on history, physical exam and labs) should be identified and explained with working hypotheses as to pathogenesis in the context of the patient's pre-existing diseases. That's why it is imperative to validate previous diagnoses. Trainees are often overwhelmed with work and so may not take this very important step. Countless times, I've read medical records that label patients with "COPD"," that has been copied from previous records but never validated with PFT evidence. This is a lazy and potentially harmful approach. All it takes to break that cycle is for one good house-officer to interrogate the evidence for old diagnoses and document corrections in the past medical history. While houseofficers may not be expected to make every new diagnosis without the aid of more experienced attendings, there is little excuse for not mastering the past and current facts about patients.

A particularly efficient way of keeping track of old and new diagnoses is to attach a cumulative problem list to the inside cover of each patient's chart. Diagnoses, dates and methods of diagnosis should be listed.

Always think ahead

The best houseofficers are always thinking ahead. For every problem there is a differential diagnosis, diagnostic plan and tentative therapeutic plan until evidence arrives to refute or finalize the diagnosis and plan for each. Thinking about potential complications of underlying diseases or therapies so that "an ounce of prevention" can be put in place, is the mark of an excellent doctor. Exemplary doctors consider the effects of immobility, poor nutrition and convalescence from the acute illness to craft plans, starting on the first day, to *prevent* the likelihood these prolong hospital stay and recovery. Discharge planning starts on Day 1, not the day before discharge. Even if patients can go home, they may need services that take time to arrange. Even arranging for a ride home can impede discharge if you haven't planned ahead and communicated clearly with the patient. Excellent houseofficers consider these nuances of discharge and transfer plans, and complete paperwork the day before. So if all goes according to plan, orders/scripts can simply be signed and actuated, rather than stopping the day to attend to paperwork that could have been completed the night before.

Medical education

In the past 5-10 years, medical education in the United States has been revolutionized by the Accreditation Council for Graduate Medical Education (ACGME). Since residents are trained with public money, the ACGME reasoned that taxpayers deserve some accountability. Before 2000, the system emphasized inculcation of medical knowledge but neglected other elements of medical excellence. The ACGME was charged with creating a new, more accountable system. Residents and patients have been the beneficiaries. Beyond insisting that exhausted trainees are a threat to themselves and patients (thus the 80-hour work ceiling), the ACGME mandated that training programs teach and evaluate 6 domains of "competence:"

1. Medical knowledge,
2. (Quality of) patient care,
3. Professionalism,
4. Communication skills (with patients and other healthcare personnel),
5. Practice-based learning (use of information and one's previous experience to help patients), and,

6. System-based learning (use of healthcare system resources to help patients).

The problem is that even the largest medical faculty and the finest residency does not allow trainees to master all 6 competencies. Excellent training, instead, prepares you to identify the domains of performance and self-improvement vocabulary/skill-set that you will spend a lifetime improving. Think of training as the foundation of your house. Build a strong foundation, become a life-long learner and the final product will be as good as it can be. Trainees should use the very brief period – of practicing with a net - to aggressively and proactively seek education. They should read in texts or reviews about their patients' problems. In general, randomly reading from a medical text is a low-yield procedure, whereas using current patients' problems as an opportunity to read is a powerful method of remembering through association and experience. They should ask both attendings and senior houseofficers questions when they don't understand something. No question is stupid if failure to ask it might harm a patient. *Residents are students* and deserve a strong education.

I urge residents to take every opportunity they can to invite attendings or more senior houseofficers to watch them with patients. The federal government pays for this education and trainees should take advantage of the opportunity to seek feedback. Observed sessions are a powerful means of eliciting objective constructive criticism. Similarly, ask for suggestions on a regular basis from your teachers; it will only make you better. "What 2-3 things did I do well today and what 2-3 things can I improve." Don't take constructive criticism personally – nearly all attendings offer it as a means to help you become a better doctor (not to make you feel bad, which would be counterproductive). Once you're out in practice, it's too late, because there are no formal mechanisms to assess performance in this manner**.** Also, patients are often willing to offer constructive criticism if asked: "How can I become a better doctor?" If the goal is to become as good as you can be, then humility is necessary, blended with a readiness to accept criticism and make changes for the good of patients.

Educating oneself also requires more than reading a text or reviews which are good starting points for interns, but do NOT suffice for advanced residents. You only train once (but you'll continue learning for a lifetime) and you should learn WHY Medicine is practiced as it is now. That requires accessing the primary medical studies that inform various clinical treatments. This approach starts you on a long road of medical learning. Much of what you learn now will change as new evidence becomes available. But if you don't "build your house" on a strong foundation, learning how to read the medical literature critically, you will be left behind as Medicine advances. Learn the right skills (i.e. practice-based learning) now, and you'll never stop learning.

Life-long pursuit of your medical education, to best serve patients, is a very important professional responsibility. And state licensing boards (and insurers) require increasingly proof of such activities. Residency is only the beginning of medical education, since the half-life of many medical practices is only 10-20 years. Most good training programs will teach you how to interpret published papers. But the goal should be to become a skeptical and discriminating consumer of the medical literature. The first step as you read any study is to determine the central hypothesis – and its relevance to your practice (or to what you know about a topic). Second examine the methods carefully. Were methods robust and appropriate to address the hypothesis? Were statistical analyses appropriate? As you examine results, examine the omnipresent "Table 1" carefully. What patients are being described and, if randomized, were patients in compared groups similar for features that might affect the outcomes of interest? Were sample sizes sufficient (i.e. was the study sufficiently powered) to detect a difference if one actually existed? Then examine the outcomes of interest and validity – did the researchers take steps to minimize systematic errors that could have affected the observed result? If you come to the conclusion that the methods are robust and results are valid, the next question is the degree to which the results can be generalized. What patients and under what conditions do the results apply? At the end of the day, you must determine how any given scientific study will affect your practice; how it fits in to other published studies. There is no golden rule here. It will depend upon the size of the study, it's validity, it's generalizability and the likelihood that future studies will be done to confirm the observed results. As a general rule, we should seldom allow one study to change established practices that were previously based on sound science. We should await confirmation in a second cohort, preferably at different study centers. There are too many instances in which results published in very reputable journals are refuted in later research; sometimes harming patients treated in the interim if physicians have prematurely "jumped onboard." First, the nature of scientific inquiry is that sometimes observed events happen by chance (a P value of 0.05 still leaves a 5% chance that the result was explained by random

chance). But, also, a litany of conflicts of interests (financial and scientific) has been illuminated in the past several years. So even when a study yields statistically significant results, it is not unreasonable to be skeptical and await confirmation before changing practice.

Most major medical societies publish expert and/or consensus guidelines that attempt to provide unbiased, rigorous syntheses of how available data should guide practice. Only a minority of practitioners knows the data and can analyze it sufficiently to refute the findings of reputable experts who use robust methods of data analysis. Unfortunately, even some of these expert panels are affected by conflicts of interest (COI) and "consumers" of guidelines should examine what steps are taken to "manage" panelists' COI. Then practitioners can confidently use guidelines and consensus statements to help them make sense of the available data.

Medical professionalism may be the most neglected competency. The American College of Physicians has defined this virtue in it's landmark *Medical Professionalism in the New Millennium: A Physician Charter* (Ann Intern Med 2002). It describes professionalism as a social contract to: a. pursue the patient's welfare, b. respect patients' autonomy, c. defend social justice, d. commit to remain competent, e. remain honest with patients, f. maintain patient confidentiality, g. maintain appropriate relationships with patients, h. commit to improving patient care and access, i. commit to a just distribution of resources, j. commit to guard against conflicts-of-interest that would imperil patient care, k. commit to advancing scientific knowledge, l. work collaboratively and engage in self-regulatory activities. While this is a daunting list, it is clear that physicians have failed to uphold some of these principles in recent decades, thereby eroding the prestige of our profession. If trainees commit themselves to understanding and doing their best to embrace these tenets, they are likely to wear the moniker of "doctor" proudly and reflect the best face of Medicine.

Another neglected competency that is of ever-increasing importance in healthcare is systems-based learning/practice. It includes using the medical system to best serve your patients and those more globally. For example there are numerous systems (everything from some free medications for the uninsured, to meals on wheels, to visiting nurses, to different levels of skilled nursing facilities, to hospice) to assist patients on the road to wellness after discharge. We must muster all resources to maximize their health goals and to support wellness more holistically (see section below on Cultural Competence). Systems-based practice also involves the recognition that it is in your patients' best interest a. to receive timely, evidence-based therapies (e.g. harnessing care pathways when appropriate) for underlying acute and chronic illnesses, and, b. to prepare the foundations for their discharge including physical therapy, discharge planning, etc. so they can go home as soon as possible (to avoid iatrogenic complications and continue recovery with supports that you install there). It also involves "stewardship" of limited resources - the realization that not only is it not in your patients' best interest to remain unnecessarily in hospital, but it penalizes those waiting for beds in the ED AND the hospital which must bear the entire cost of unnecessary days (many insurers do not pay "per-day" but rather per diagnosis). That's not to say we should discharge patients who aren't ready - who might "bounce-back;" but we can likely do a lot better.

Care pathways and evidence-based guidelines

Doctors are a peculiar bunch. We care deeply about our patients. At the same time, we are fiercely individualistic and prideful. The history of American Medicine has been one in which trained, licensed physicians have been able to practice medicine as they've seen fit with little or no oversight regarding quality. Unfortunately, many therapies, detailed in this book, help patients but are not administered routinely. The IOM has referred to this as a Quality Chasm; and researchers have suggested that not only don't we administer evidence-based therapies reliably, but – perhaps even more important – we don't examine ways to ensure that patients will continue to take them; we instruct, but we don't partner with patients to define and then pursue their health goals. Over the past 10-20 years, insurers have demanded increasingly that we apply best evidence to serve patients. In some hospitals, doctors have embraced evidence-based guidelines and care pathways designed to make care easier and more efficient, applying evidence to improve their patients' outcomes. However, others have seen these measures as an incursion – a reduction of their autonomy. Like all humans, change is hard for doctors. *However, it is our professional and ethical obligation to apply evidence to help our patients*. Irrespective of whether we like it or not, care pathways and evidence-based guidelines should be applied routinely. We cannot pretend to know more than experts. Some who refuse to use protocols argue that "my patients are different than those in the studies." While guidelines and protocols are *not* meant to be applied rigidly and in every situation, this is more often than not, a bogus argument. Deviation from evidence-

based therapies and care-plans should be the exception and there should be good reason. In the end, insurance companies will dictate this – we simply won't be paid unless we use "best practices" (and we shouldn't). I urge trainees to embrace care plans and evidence-based guidelines. They are the way to the future. Understand them thoroughly so you can recognize the exceptional cases when they're not applicable, but use them routinely otherwise! In the end, they will make your life easier and simplify your acclimation to the future.

But then realize that the initial prescription is only the beginning. The patient needs to understands the risks, benefits and alternatives – so that they can "own" their healthcare. Affecting their behaviors, and ensuring that treatment (and complications thereof) is consistent with their values is self-defeating. For example, if you prescribe beta-blocker after MI and the patient stops taking it because he can't afford it or it affects his sex life, then you've checked off the box for providing an evidence-based prescription, but failed to serve the patient. The real art of Medicine is getting the patient to understand and partner with you to help keep them well. And that comes through time, trust, cultural competence and interpersonal relationships that you can spend a life-time refining.

Errors

It is best for doctors to accept at an early stage that they are not perfect and expect to make errors. If our errors are used as opportunities to educate ourselves and our peers (so their patients don't experience the same), then this "fact of life" can do good. Unfortunately, most errors go unrecognized because doctors aren't monitoring their cases carefully for errors, don't recognize errors, or they recognize errors but are too embarrassed or frightened to report them. Civil aviation has a robust system of error-analysis (and risk-free, self-reporting), intended to prevent similar future mishaps. Strangely far more patients die of errors than of airplane crashes and, yet, there is no similar system in American Medicine. While fear of malpractice suits may have impeded this, it turns out that the best way to avoid litigation is NOT to hide a mistake. Rather, own up to it and apologize to the patient. Many states have adopted laws that protect the apology – it can't be used in malpractice cases as an admission of guilt in some jurisdictions. Moreover, local leaders can use the error to educate others and create systems to safeguard from similar future mistakes. Errors-reporting should be considered part of our professional responsibilities.

Patient Advocacy and Choosing Medications

Perhaps the most important component of medical professionalism is strenuous patient advocacy. Over the past several years, it has become evident that pharmaceutical companies have used attractive young representatives to ply us with smiles and sandwiches. Sometimes the gifts extend to expensive dinners (in the guise of a CME lecture) or honoraria for "consulting" (irrespective of one's area of expertise). They leave new, expensive medications, banking on the fact that you'll send your patients out with free "trial samples," realizing that once the patient and you are satisfied with the result, you will NOT swap out to a cheaper, equally effective alternative. The result has been over-prescription of very expensive medications and medical devices. While Merck will be punished for it's transgressions, doctors wrote prescriptions for Vioxx™ for years without evidence demonstrating superior efficacy AND with some, albeit anecdotal, data suggesting higher cardiovascular risks. We can claim that we were misled – but we allowed ourselves to be led. Vioxx™ is only one of, now, countless examples. Ask yourself: Why would drug companies invest the money in detailing/promotion if there weren't huge returns? Drug companies are not philanthropic organizations – they strive to provide returns to shareholders (and have done so mightily). Do reps enjoy the endless "kissing up?" When was the last time you saw an unattractive drug rep? Why is that? We mustn't take at face value what we are told by pharmaceutical representatives. At the same time, I can only admire an industry that has so elegantly manipulated us, who purport to be reasonably intelligent. They've made monkeys of us and with each revelation I am more ashamed of what we've become as a profession. We owe it to our patients to ignore the "sales pitch" and stay atop the evidence to best serve our patients. We must examine the data and national guidelines for management ourselves and vigorously defend our patients. Our patients will benefit and the money squandered on unnecessary expensive drugs might be spent instead to fight AIDS or insure our uninsured.

CONCLUSIONS

Humanity is a requisite to enjoying Medicine

Physicians are besieged. The training is grueling. Keeping up with latest developments once you've been trained is daunting. Student loans are monumental. Bureaucrats devote their careers to eroding your practice autonomy. A small number of patients are waiting for an error that will become your malpractice nightmare. Salaries are flat or even decreasing. How then can you maintain your love of the patient? It is difficult, but we need to "keep our eye on the ball" and there are some simple methods that can aid in keeping in touch (literally) with why you chose Medicine in the first place.

Spend *more* time with patients and their families. As reimbursement decreases, many physicians find themselves spending less time with more patients to keep their salary from decreasing. This is a recipe for alienation (of the physician from his "true self" for the pursuit of capital). If you've ever been on the receiving end, you know that patients and families are *frightened*. If you treat the patient as a bundle of organs, you'll receive back very little in return. If you talk with them, understand their fears and help to allay their psychic as well as their organic pain, you will be paid hundred-fold. Medicine will remain worth the personal investment. If you fail to do so, there is no salary that can reimburse your personal sacrifices. A recent article published in *JAMA* suggests that method acting can promote the connection. Why method-act when you can enjoy the real thing? You simply need to invest yourself. Time in the room AND actually touching the patient, as you communicate, are powerful ways to make the empathic connection. You might make a little less money. But to steal from pop-culture advertising: your gratification in allaying the pain of another human being, in their most vulnerable and fearful moment is "priceless."

Acknowledge (that we are) vectors of iatrogenesis

Accept the thinly veiled message of the Institute of Medicine that we, healthcare providers, cause human suffering through our simple neglect. We don't wash our hands between patients. We don't communicate with patients sufficiently to help them understand their illness to enhance their compliance. We fail to administer life-saving therapies that have been definitively proven to benefit our patients. We write illegible and/or incomprehensible notes and orders that do no good or worse. We avoid identifying our errors so that our colleagues and their patients may benefit from our mistakes. Not because we're stupid. Not because we don't know any better. These are indictments of modern Medicine predicated on neglect and in a few cases arrogance. Physicians must realize that every therapeutic choice (or missed opportunity for therapy) has consequences; sometimes complications that contribute to the morbidity and/or mortality of our patients. You don't see the extra cardiac myocytes that died because you forgot to give your patient with MI an aspirin and beta-blocker, but they did nonetheless. You did harm that, if brought to your attention, would be both mortifying and antithetical to why you chose Medicine. The ethical practice of medicine, which I equate to "professionalism," requires that we: 1. Remain abreast of standards-of-care, 2. Apply them, with humanity, at the bedside, and, 3. Constantly reflect on the degree to which our chosen management strategy has harmed the patients (to address the harmed individual and to promote systems that reduce the likelihood of the same in future patients), 4. Treat the patient as we would want to be treated.

Although errors due to lack of knowledge occur occasionally, errors of medical reasoning are more common. Studies have demonstrated that one of the most common medical errors results when clinicians "prematurely close" on a diagnosis i.e. they insist on a given diagnosis without complete information and/or ignore contradictory information that is inconsistent with their hypothesis. Just as we should be vigilant for the degree to which our therapies harm patients, the greatest weapon against the pit-fall of "premature closure" is humility and self-awareness.

General counsel

Human being is more complex than any of us can fathom completely, and ultimately, we're all "in the same boat." Maintain your humility! Patients sense immediately if you think you're better than they. They know that no man or woman is perfect. They seek your advised opinion and your medical best, and if you acknowledge prospectively that there are few certainties, you will find far more success in your interactions (and less risk). If they sense that you're in the trenches with them, they will be reassured and you will be gratified by the team spirit. Consider whether you would want the same thing for your mother, a litmus test that I find very useful (and sometimes very revealing).

Finally, maintain your interests outside Medicine. Your ability to maximally serve your patients is, in part, predicated on your happiness, which requires a rich full life outside Medicine. Our profession is based on love of our fellow man – which is served by our own spiritual health.

If you cultivate both your human connections with patients and your outside-Medicine life, you will not lose faith and revel in the gifts of Medicine.

ADDENDUM

CULTURAL COMPETENCE

The term cultural competence has been used to describe two related but distinct issues in medical care: 1. The level of understanding and skills that doctors apply in interactions with people of widely varying cultural, religious, racial and ethnic backgrounds, and, 2. The level of understanding and mastery of medical cultural logistics that are distinct to the U.S. The United States is called a great "melting pot" for a reason. People from all countries, of all ethnicities, and all religions live in reasonable harmony owing to unique individual liberties coupled with relative "tolerance of the other." The U.S. is by no means a perfect place. While racism, sexism and other bigotries exist in American society, the legal and cultural framework – predicated on egalitarianism – has allowed gradually increasing tolerance for features that are persecuted in other societies. That's not to say that the U.S. has eradicated bigotry, but rather the overall trend is for both cultural tolerance and increasing legal intolerance of bigots. Nonetheless, this melting pot is imperfect and despite lofty intentions, the U.S. system falls short often in healthcare. A seminal work "Multicultural Medicine and Health Disparities," written by the Institute of Medicine, highlights that minority groups – especially Blacks, women and Hispanics – have received less equal care than Whites. While some of these differences in healthcare outcomes may relate to unequal access to healthcare and intrinsic distrust of minorities of "the system," mounting evidence suggests that problem runs deeper: that we doctors provide less apt care to those who are different. This is only human nature, but, nonetheless, violates core principles of medical ethics.

It's difficult to empathize with others whose physical looks, belief-systems or life-styles we don't understand or understand but disagree with. But as doctors we vowed to treat all equally and to the best of our ability. It is as much our professional responsibility to better understand "the other," to more effectively practice our craft, as it is to learn the technical aspects of Medicine. These ideals are codified in the Charter of Medical Professionalism – the modern Hippocratic Oath (see above).

Meanwhile, the practice of Medicine has become increasingly stressful. Many U.S.-trained doctors begin practice with huge debts (often well in excess of $100,000) and all physicians are befuddled by large bureaucratic burdens that impede effectiveness, erode autonomy and make this profession less rewarding than it once was. The number of U.S. citizens choosing to "serve" the population in lower-paying, primary care specialties has dwindled, leaving vacancies that can only be filled by foreign-trained clinicians. Roughly half of all new internists graduate from non-U.S. medical schools. Many international graduates (IMGs) have completed advanced clinical training, advance degrees and research. They often start-over because of the promise of the U.S. system: the newest, most effective therapies in one of the most rigorous and robust medical training systems in the world. And U.S. citizens are the beneficiaries, because resident-physicians provide care for patients without private health insurance (underwritten by the U.S. government by Medicaid – for the poor – and Medicare – for the elderly). Many choose to stay to practice and contribute to this society. The challenges of residency for international graduates are a much-ignored topic. U.S. graduates, most often born and nurtured in the States, have had at least some exposure to pan-culturalism and the American values briefly discussed above. They have spent 4 years "learning the ropes" in the American medical system; everything from how to communicate with minorities more efficiently, to how to present (verbally) and document medical care in the uniquely American manner. They know how to "get around" in the hospital and outpatient venues because they've spent 2-4 years, learning the logistics. They also already know how to live well outside the hospital; everything from how to buy groceries, to how to get around. Every July 1, thousands of IMGs, often with little or no experience in the American medical system are "thrown into the pool" without prior swimming lessons. They need to learn the same Medicine that any U.S.-trained resident does, but they're starting from the deep hole that is varying degrees of cultural incompetence. The following is intended to assist them with the transition. But to solve a problem, we must openly admit that there is a problem – then focus on it and defeat it.

The goal of this section is to review why it is important to focus attention on cultural competence, both in the traditional sense (i.e. care of multi-cultural patients), and, in the practical sense (i.e. how to catch up your skills in getting things done in the U.S. medical system). Indeed many IMGs struggle reconciling their values and cultural norms with those of U.S. society. It is imperative that Medicine trainees understand that their values and culture need not be lost. ***But at the same time, when we put on our white coats to care for patients, we subjugate our own value judgments to understand the patients' and to help them receive the best available care***. Ultimately, IMGs can catch up, but only if they invest some of their sizeable talents and intellectual energy on these tasks starting on the first day of internship.

Finally, this is discussion is much more than Manthousian caprice. There is a movement afoot in the U.S. to require robust educational programs and caregiver performance of cultural competence. On January 1, 2012, the Joint Commission, which accredits hospitals in the U.S., will require that hospitals meet explicit, rigorous standards for the cultural competence (and its exercise) for all hospitals and caregivers therein. Note that this complements brilliantly communication, patient care, professionalism and systems-based practice competencies that are 4 of 6 domains of the U.S. medical educational system.

1.Cultural Competence "a"

A quick crash course:

Race and Ethnicity – While this country was established by White Anglo-Saxons, it soon began to draw multiple White "minorities" that gradually staked a claim a piece of the American pie. Irish, Italians, Greeks, Jews, and more recently Asians and Hispanics from both North and South America were/are among the multitude of "minorities" who slowly melted into the pot. African Americans are a notable exception, because many of their ancestors did not come to the U.S. voluntarily, but rather as slaves. While slavery was abolished in the late 19th century (care of the bloodiest war in American history, against ourselves), overt and ugly racism persisted until the mid-60's. While it has become less prevalent in the 21st century, its effects are still felt. There are still many White Americans who possess conscious or unconscious bigotries toward African Americans (the current politically correct term for Black Americans who also include Africans, Caribbeans and South Americans who emigrated voluntarily). To this day, Black Americans remain under-represented in politics, business and the professions. They have worse healthcare outcomes and often receive inferior education as a group. While there has been gradual improvement, anyone who suggests that Blacks have attained social equality is fooling themselves. Some prominent leaders in the Black community have blamed the break-down of family structure, especially of inner-city, Black families. Barack Obama pointed out that there are too many Black families with absent fathers, and too many Black children drawn into gang violence and away from affirming, responsible positions in society. Many African Americans remain angry – for the history of slavery and oppression, and for the lesser opportunities our society has afforded. Some argue that after so many generations, slavery should not be blamed, that part of the problem is failure of Blacks to take responsibility for their own families, economic status and social problems. It's an easy argument if you're not on the receiving end of discrimination and the hopelessness it may engender.

Hispanics feom Mexico and South America are the fastest growing ethnic group in the U.S. Lured by economic opportunities for their families that are not as easily attainable in their home countries, they come seeking stability and a better life for their children. Spanish is not spoken commonly in Africa, Asia and eastern Europe – the origins of many IMGs. However, many Hispanic immigrants speak very little English. So to be effective, non-Spanish-speaking IMGs will need to learn more about the cultural mores and language of Hispanics.

Depending upon their country of origin, some IMGs will not have been exposed to African Americans, Hispanics and the multitude of other ethnic peoples who comprise the fabric of America. They will see "the other" – often with a distinctive English vernacular or accent,

distinctive culture but sometimes immersed in a sea of social stressors – and the first inclination might be to not even attempt understanding. Learning the technical aspects of Medicine is hard enough, without having to learn about so many cultures.

Without empathy, the doctor-patient relationship is set adrift and the possibility to reverse the disgraceful medical inequalities highlighted by the IOM wanes. Nonetheless, there are specific cultural mores that may greatly impact your dialogue and effectiveness with patients. For example, some cultures are paternalistic and the "man of the family" is very much involved in medical decision-making for other family members. If the patient asks for it, there is nothing wrong with honoring these customs (so long as they do not constrain the autonomy of the patient). Some families believe that God will determine their loved one's fate irrespective of and despite medical technology. With time and experience you will acquire more and more understanding of culture-specific viewpoints. But developing cultural competence requires conscious effort and focus; first understanding that by gathering greater proficiency in this arena you'll be a better doctor for your patients – you'll be providing "patient-centered" care which is far more rewarding than simply mastering technicalities of medicine (i.e. treating the patient holistically rather than as a bundle of organs).

While this short chapter can't help you understand all of the complexities of any group's history (and lumping all people of a given race or ethnicity into any one plight is itself wrong-headed), there are some important tools to use until you get caught up. In fact, you should spend the remainder of your life working to become "culturally competent" just as you will continually learn technical Medicine. Human suffering does not discriminate. There is a common human condition and irrespective of race, life-style or ethnicity, the patient experiences joy and sorrow, pain and pleasure, just as you do. What we have in common is far greater than our differences and as a doctor you can help maximize the joy and pleasure by treating the "whole person," trying to understand them, as you apply both science and art. To do so, you must exert yourself and pay special attention when you encounter a patient from a background that you don't fully understand. Formal diversity training – provided by this and other training programs – will assist in launching your development in this area. But like development in the competencies, it should be a life-long journey.

When language is a barrier – you're not sure what they're saying or you're worried they can't decipher what you're saying - the safest means to help them is to use a universal translator. Many hospitals have 3-way phones that can contact an interpreter of any language. Some offer interpreters via television, where the patient sees and speaks with the interpreter. Hospital personnel are OK if they've been certified for language proficiency. Family members should not be used as primary or sole interpreters, especially if the patient hasn't given permission for you to share with them or if the topic of discussion has very important implications for their health.

Values vary considerably among cultures, countries, and religions. Liberty and egalitarianism – whereby all citizens are treated equally by the law and government, irrespective of their background - are bed-rock principles of the U.S. We honor our fellow citizens' right of self-determination (autonomy) i.e. to make their own decisions so long as they don't infringe on the rights of others. Doctors don't make decisions for patients in the U.S. We provide them with our best medical opinion of what's going on, the options for management, and ensure the patient understands and can consider in the context of their values. Then we do our best to support patients' choices whenever those choices don't infringe on our own ethical principles. This is referred to as a "shared decision-making model." Respect of the individual also extends to the right of privacy. So we protect patients' confidentiality, never sharing their history with other healthcare providers where laypersons might hear and protecting their names, and other "identifiers" from anyone who is not providing medical care. This is called patient confidentiality and is legally protected. These and many other core concepts are included in the Charter of Medical Professionalism created by the American College of Physicians.

Religion and spirituality are also important aspects of human being, that can go neglected during medical care. Religion and spirituality are overlapping but not the same; you can be spiritual but not religious and religious without being spiritual. Religion is self-identification in a group that offers some explanation for the world (usually God) and includes practices/customs, some of which (e.g. blessing of the ill, last rites) may be very important to patients during illness. Spirituality is self-identification with belief in a greater power (whether it is God, physical laws, and/or belief that we exist irrespective of our bodies). Physicians owe it to their patients to determine whether a patient's healing will require more than 21st century medicine; simply ask: "Do you have any religious or spiritual beliefs or practices that we can support during your

hospitalization?" Even if you are not the content expert who will provide that support, you can call on pastoral care, a patient's own personal pastor or congregants to assist. Not infrequently, especially in end-of-life discussions, direct involvement of clergy can reduce suffering and help patients make difficult decisions in the shared decision-making model described above.

2.Cultural Competence "b"

This may be a Manthousism, because in the medical literature cultural competence refers to mainly part "a" above. However, after 18 years teaching in a hospital whose Medicine residents are predominantly IMGs, I've recognized some patterns that are not well studied or publicized. We've been extraordinarily lucky to recruit amongst the most talented IMGs. Most have residency-level training before they arrive. Many have PhDs or MPHs in prestigious U.S. universities. Many have published in peer-reviewed journals. They score at the top decile on all their tests. Yet they arrive "in a deep hole" because they've most often had very limited exposure to our culture, to American patients and to American medical logistics. Its ridiculous on its face to suggest that someone can spend the first 25 years of their life in one country - with its own norms and values – and simply arrive here and be culturally competent. Even most American medical graduates are not culturally competent. Unlike many other countries, the U.S. is a vast "melting pot" of races, ethnicities, religions, values and norms. Very basic tenets of interviewing patients and American social norms are not emphasized at many international schools (as much as in American schools). The deficiencies in non-medical cultural competency are discussed above. Few international graduates have mastered medical (and non-medical) logistics as well as their U.S. counterparts. And you can't blame them. U.S. students have most often grown up in the U.S. and spent 4 years in med school learning the system. But this "hole" that IMGs find themselves in is difficult to dig out of and impedes the velocity at which they can be trained medically in the 6 competencies. They have to expend far more effort to get simple things done until they understand the system and rounds with their teachers get bogged down because communication is sub-optimal (if it takes 15 minutes to get the story instead of 5, there's 10 less minutes for teaching).

Before launching into medical logistics, it is also worth noting that because Primary Care is not a specialty in many countries, most very talented, type-A, IMGs come to the U.S. seeking sub-specialty training. As baby-boomers in the U.S. age, there won't be enough Primary Care doctors to care for our population. While cardiology and gastroenterology may seem glamorous and, at present, are very lucrative, Primary Care is, in my opinion, the most intellectually challenging and noble specialty in Medicine. Since market forces have, to present, ruled in the U.S. forces of supply and demand will drive up salaries of Primary Care physicians. Bureaucratic nonsense will be reduced, because the market will demand it to draw more talented trainees into Primary Care. I'll be very surprised if the current system does not "invert" – Primary Care will necessarily become the premium specialty.

Medical culture in the U.S.

Role as a trainee

The role of a trainee (student or resident) in the U.S. system is unique. They are NOT the legally responsible physician of record, but are expected to know their patients, devise management plans and actuate them AFTER the attending physician's review and approval. Some medical systems are very top-down . . . trainees round with the professor and learn by osmosis; being told what to do and learning by observing the attending's practice patterns. This is a recipe for failure in the U.S. system because all trainees – even students - are expected to first offer their synthesis, before the attending speaks. While no important care plans should be actuated without the attending's ratification, the resident should have devised it, with only minor modifications required from the attending. This model works spectacularly, because then the trainee gets to practice thinking aloud "with a safety net." In our experience as a faculty, this model – where the trainees "take ownership" and insist on presenting the case and what they want to do, using the attending as a backboard/safety mechanism – is the most difficult medical cultural feature for IMGs to adopt. U.S. grads get it, because this is the prime directive in American schools. But many IMGs have difficulty adapting to this system.

Accordingly, trainees are expected to be very proactive (not passive) about their education. They should ask more senior residents and/or attendings every day "what am I doing well and what can I improve." Constructive criticism is the mechanism for self-improvement . . . the more you get, the better doctor you'll become. Too often, trainees are worried that constructive criticism is a "bad thing." Constructive criticism is rarely harmful and is meant to help you define strengths and weaknesses. It is true that faculty need to learn how to offer constructive criticism . . . well, constructively. But the easiest thing for a teacher to do is to let poor performance slide by without intervening. It is a sign of dedication to patients and to your education that the attending offers constructive criticism: to help you identify your strengths and weaknesses so you can serve your patients better. It is a great gift, viewed in this light. Can constructive criticism be "used against you?" Almost never. There are some trainees who either have difficulty with some of the medical competencies and/or who refuse to use constructive criticism to improve their performance. Even then the medical system is avidly protects physicians' rights. Vigorous and well-designed "individualized education plans" can be deployed to help you if you're not able to improve performance without additional assistance. Such efforts don't negatively impact on your career; in fact, they make you a better doctor and if you work hard and overcome deficiencies, it is a very positive reflection of your character and dedication to excellence for the sake of patients.

And this is "where the rubber meets the road." Medicine is increasingly employing the 360 degree evaluation to provide physicians with constructive criticism. So you won't just be evaluated by your attending physician, but by all the other members of the healthcare team with whom you must work fluidly to provide best-patient-care. So in our program, trainees are evaluated by attendings, nurses, case managers, patients, peers, and students. They even evaluate themselves to examine whether their self-assessment matches up with how they're viewed by others (i.e. do they have insight). Self-awareness is the road to self-improvement.

Communication

There are standards of communication that are unique to the U.S. system. Until you master these, patients are poorly served and you're poorly served because you're squandering precious time that could be spent providing better care and learning more Medicine.

Verbal skills –

There are two skills that every intern must master rapidly (if they haven't arrived with these): the full presentation that includes all elements (for conferences and more formal rounds) and the 5-minute presentation for presenting to attendings, consultants etc. hitting only the highlights. This is an art-form that takes a long time to master. The complete presentation includes all details in the following order:

Chief Complaint in patients own words and who gave the history

History of present illness – beginning with a sentence that summarizes (and frames) the important aspects of the patient's presentation including age, sex, race, most germane past history that is contributing to the current problem and/or co-incident diseases that affect Bayesian reasoning regarding the CC and the complaint (including timing, magnitude; e.g. "This is a 78 year old African American lady with a history of hypertension and dementia who presents with two days of increasing shortness of breath, cough productive of yellow sputum and fever to 103 degrees."). Then the entire chronicle relevant to the history of present illness, including the 7 elements of pain (when pain is the complaint), the tempo and severity, and pertinent positives and negatives from review of systems that are relevant to the differential diagnosis of the chief complaint.

Past medical history includes a list of all hospitalizations, surgeries, documentation of various illnesses (e.g. COPD diagnosed by FEV1 40% in 2005), medications (generic name with dose and frequency).

Social history including occupation, ADLs (in all elderly or disabled patients), smoking history, illicit drug history, alcohol history.

Family history of first-degree relatives with minimum exploration of cardiovascular risk factors (HTN, lipids, diabetes), cancer, lung disease, cerebral vascular disease and other germane inherited illnesses.

Review of Systems – Including pertinent positive and negatives from every system that wasn't mentioned in the HPI (general/constitutional, skin, head/neck, neuro, musculoskeletal, lungs, heart, GI, GU, neuro, psych).

Physical exam must include a general description of the patient followed by vital signs measured by you: heart rate, respiratory rate, blood pressure, temperature (nurse's recording OK), your computation of BMI, pulse oximetry saturation.

Head and neck exam (mandatory, even if simply "WNL=normal")
Chest exam (mandatory)
Heart exam (mandatory)
Abdominal exam (mandatory)
Neuro exam (mandatory with full mini mental score if any ?dementia but otherwise general statement of mentation, always list power of 4 extremities, cranial nerves, gross sensation, reflexes as a minimum)
Skin exam (mandatory)
Additional systems as appropriate to the HPI or setting of presentation. Remember that you don't need to mention more fancy tests like egophony or straight-leg raise for every patient. But when they're germane, those pertinent positives or negatives are mentioned in the relative system.
Then list laboratories/tests (OK to say "normal except for . . . ")
Summary statement of everything presented so far: "So this is a 78 year old lady with hypertension and dementia who presents with community acquired pneumonia."
Then for each problem or organ system you should review the following in order of importance: problem, differential diagnosis, diagnostic plan, interim therapeutic plan.
When you become really good at the long presentation it should take no more than 10 minutes.

The 5-minute presentation covers a subset of the full presentation. Generally speaking the HPI must be in nearly the same detail, the PMH, SH, FH and ROS cover only the pertinent positives and negatives (otherwise say "non-contributory"). The physical exam ALWAYS must state the mandatory components listed above plus any additional systems or pertinent examination techniques that inform the diagnosis. Similarly present only pertinent positives and negatives from the labs/tests. And spend most of your time showing that you've identified the important problems and have good differential diagnoses with an interim most likely diagnosis, diagnostic plan and therapeutic plan.

Daily verbal presentations of follow-up should include a rapid run through the SOAP format (subjective, objective, assessment/plan for each problem). Subjective: tell the attending whether there are any new complaints. Objective: tell the attending the vitals, chest/abdomen/neuro plus pertinent systems, any new abnormal positive or negative labs/tests then where you stand for each problem as to diagnostic plan and responses to therapies.

Medicine reconciliation is required on hospital admission, intra-hospital transfer and discharge. You must consciously assess and document what the patient was on before the transition, what the patient was on after the transition and ensure that nothing is missed or duplicated. Most hospitals have special paper forms and/or computer forms to actuate this.
Medical Orders – Most hospitals are moving toward computerized order entry. You will learn during orientation how to use the system, but time and practice will increase your facility. Whenever there is any doubt, ask for help (don't simply choose something from the menu that doesn't look right just because it's the only thing you can find). Evidence-based order sets should be used whenever they are available because they've been created by local and/or national experts. You should understand the rationale behind the order sets and protocols (often available with good CPOE systems) and how the algorithms work. You may occasionally need to customize an order set for a particular patient, which you should do with senior staff's assistance, but in general you should use order sets and protocols when available.

<u>Written communication</u> – Note that the government requires all of the elements listed in the long verbal presentation written in the medical record! Get used to it, make it automatic so you don't have to think about it for the rest of your career. Subsequent daily notes use the SOAP format described above, but much more detail should be included than in daily rounds. Someone should be able to pick up the record and understand where every problem stands. Moreover, all notes written by doctors, nurses, nutritionists etc. should be acknowledged and commented on in the SOAP note. Disposition planning should be an item in the problem list every couple days and then every day leading up to discharge. Nutrition and psychosocial coping with disease issues should be regular components of the "A/P" part of SOAP notes.

Dress

Physicians should dress in professional attire. Published studies show that patients have far more confidence in their doctors if they wear professional clothing: trousers/shirt-tie/white coat for men, slacks/dress/blouse/white coat for women. Blue jeans should be avoided for work. The white coat is the essential element that seems to facilitate patients' confidence. Neck ties should always remain tucked if they are worn (else wear a collared shirt, buttoned high) since they've been shown to transmit hospital pathogens. Whenever you're interacting with patients or families these should be the ground rules. You work hard to learn and practice medicine. To undermine your effectiveness because of your clothing or personal hygiene would be a shame. Moreover, you represent our profession and the hospital you work in (see below).

Courtesy/"Hospitality"

While many physicians find it distasteful, we (and other healthcare workers) are the face of institutions. We can represent the system well or we can undermine it. Across America, increasing attention is being paid to borrow ideas from the hospitality industry – to ensure that proper courtesies are paid to patients. To some degree, patients are guests in our place of work, and we owe them common courtesies, if not more.

Always knock before you enter (and ask to come in), reintroduce yourself until you're absolutely sure that the patient knows who you are (patients often forget our names the first couple visits). Shake their hand or give a gentle touch and ask them "how are you doing today?" Patients complain that we sometimes conduct our rounds and conversations occur between physicians without including them. Talking about someone in front of them would be rude in any culture. But to do so with a patient is particularly inappropriate. If you want to make a point to other clinicians, tell the patient "I'm going to teach them for a moment" but if you do so, take care not to intimate anything that the patient doesn't understand because they may fear the worst. Consider holding the patients hands or a gentle touch (that will help you and them to feel more connected). And always end with "do you have any questions I can answer . . .is there anything else I can do for you before we depart?" While very simple, make these (or something similar) part of your routine. You won't even think about it, but it will make a world of difference to the vulnerable, often fearful, person in the bed or on the examining table.

There are even particular phrases you can learn that apply these principles (and consider making them part of your automatic repertoire). When breaking bad news rather than making yourself the focus (e.g. "I'm sorry that . . . "), consider "I wish I could offer better news." Instead of "smoking is bad for you," consider first, "Could you tell me how you view the effects of smoking on your health." After you've given the patient opportunity to explain in their own words, consider "It sounds like you're telling me . . . do I have all that right?" A number of these phrases are listed in a series published in the Annals of Internal Medicine called "Medical Writings." While it may sound a bit "touchy feely," there are certainly effective and ineffective methods of communication and we owe our patients to at least consider implementing best practices.

At the end of the day, one simple rule always applies: treat patients with the same courtesy and respect that you would want to be treated with if you were in the bed. If you do so, you're doing your best. If it's brought to your attention that your best isn't good enough, then you owe it to yourself and to patients to ask for feedback whenever possible, until your performance (and perceptions of your performance) improves.

Personnel who are part of the team

The U.S. medical system functions on a "team of equals" principle. Everyone on the team, from doctors to janitors are required to facilitate best patient care so everyone has a role to play and everyone deserves equal respect.

Nurses – are often amongst the most knowledgeable about your patients. They often have crucial information and insights about patients' problems. So it behooves you to respect them, ask them for updates regularly (and for whether they have any ideas about improving care).

Discharge planners/case managers – you should converse with them regularly starting from the first 24-48 hours of admission to plot out a soft landing for your patients. Sometimes patients need special services to be arranged that take time . . . best to start working on this from the first day so when the patient is ready to leave, all of the components to maximize chance of discharge success are in place. There are a huge array of outpatient services that can be arranged for patients including: nursing homes for those who can't care for themselves and/or who require a bridge

until they can care for themselves, rehabilitation hospitals, meals delivered to patients who can't cook for themselves, visiting nurses to assist with everything from daily routine care to specialized care. Outpatient physical therapy, nutritionists, psychologists, social workers, occupational therapists etc. can be part of the patient's discharge plan to promote their long-term wellness. There is a very large menu of Geriatrics outpatient services including elder-care (home or institutional daycare for elders), assisted living facilities and home doctors' visits. You will learn much more about these during your Geriatrics rotations, but disposition planning – harnessing and coordinating all the available resources available and applicable – is especially important in elders, as is addressing their end-of-life proclivities (and helping them to arrange accordingly).
Nutritionists – While they may assist with calorie counts and formal recommendations, always feed your patients a minimum of 2000 kcal/day and do calorie counts if you don't think they're finishing their meals.
Physical/Occupational therapy – Aside from your order to get patients out of bed as soon as is medically indicated, physical therapists should be called upon early to assist in keeping patients' range of motion and strength intact. Occupational therapists use techniques to maximize patients' functional outcomes (so they can do tasks like cooking etc.)
Pastoral care - Some patients are religious and pastoral support is an important element of the wellness plan.
These personnel shouldn't do the thinking for you. Rather you should participate proactively with each of them to afford your patients the best care available.

The Medical System

As of July 2008, the U.S. system remains one dominated by market forces. Those who are gainfully employed, usually obtain their healthcare insurance through and share cost with their employers. There are a number of private insurance companies which provide varying levels of coverage – obviously the more items covered, the more coverage costs. As healthcare costs have risen dramatically, more and more employers are struggling to provide these benefits for their employees. Employees themselves have to pay more of their salary to get the coverage and the "deductibles" i.e. the amount they have to pay out-of-pocket each time they see a doctor or buy a medication are ever-increasing. This system is reaching a breaking point and so is very likely to change in the next decade.

The elderly (>65 y) and the poor receive government healthcare called Medicare and Medicaid respectively. Since these programs are the single largest purchaser of healthcare services, they drive prices and over the past several years, they've been cutting costs. So doctors and hospitals are being paid less than in the past, because otherwise the system will collapse. Trainees generally care for the poor (and some elderly), who can control the "mix" of patients in their practice, opt to limit the numbers of patients with poorly-paying insurance. These are often "vulnerable" populations with more substance abuse and less compliance with healthcare directions and follow-up. They are not representative of patients most physicians will care for after they've completed training, though future healthcare reform may change that.

In the outpatient setting, many hospital clinics provide care on a "capitated" basis, i.e. payment of $X to care for Y number of patients each year, irrespective of their burden of healthcare problems. Medicare is a more fee-for-service system, whereby doctors bill for procedures and services (usually stratified as high, medium or low complexity for any given visit). In the inpatient system, hospitals are paid a set amount of money for any given admission diagnosis. So every unnecessary test comes out of the hospital's bottom line. Only tests that are required should be ordered for the immediate acute illness should be pursued (others can be obtained in outpatient follow-up) and patients should be discharged (for their own safety) as soon as possible. Doctors get fee-for-service for their inpatient activities. There are very specific requirements of documentation to be paid at the low, medium or high complexity level of care that you must learn to practice ethically and within the bounds of the law. A good residency will help you understand these (and some are mentioned above in the communication section).

Non-medical logistics

While it may seem odd, it shouldn't. IMGs have to learn how to get along outside the hospital as well.

Finances – You should apply for a social security number immediately upon getting to the U.S. You cannot be paid without this and it is vital to applying for credit cards etc. Consider opening a bank account with checking and a debit card (allowing you to draw directly from your account for purchases). You can also request that your employer "direct deposit" your paychecks into the account by providing a canceled check. Note also that some hospitals have "credit unions" that will provide loans to employees for cars etc. but this is not true of all hospitals.

Housing – Most residents choose to rent apartments, but some will buy a home during training. Real estate agents can be contacted through the yellow pages of the phone book or via Internet for any locale to assist, usually for a fee. Some residency programs provide temporary low-rent housing (until trainees can find more long-term accommodations). Note that most landlords require 2-3 months of rent as a deposit to begin renting.

While you can get a land-line phone if you provide proof of employment and an address, you will require a credit history to obtain a cell phone. Consider bringing a pre-paid cell phone from home to use until you can acquire a credit history sufficient to get a cell (if you really must have one).

Transportation – Some cities have excellent transportation systems so that owning a car isn't necessary. However, the U.S. is a large country without well-developed mass transportation in many locales. So many residents will need to buy a car. To do so, you need credit (or cash). Many credit cards require that you obtain a social security number obtained by registering as with the U.S. government (internet application). To get credit, you need either a credit history and/or to obtain a credit card (which vetts credit history). Membership in the American College of Physicians has allowed many trainees to obtain credit cards which then allow them to establish a credit history. If you pay at least the minimum amount every month without missing payments, you can build a credit history that allows your optimal credit limit to grow (usually starting less than $10,000 but can increase to over $20,000).

Also if you wish to get your U.S. drivers license soon after arrival, you will need to make an appointment with the local Department of Motor Vehicles months in advance. Your international drivers license is only good for a couple months and insurance rates are very high without a U.S. driver's license (yes, you must buy insurance to drive!). I am told that attending a driving school is very helpful and oftentimes they will help arrange an expedited appointment for the license, but these private arrangements must be made well in advance.

Food – While product-specific markets are common in some parts of the world, the most common place Americans buy their food is super-markets. Many chains exist and with time you'll find one where your needs and expectations match the products and prices.

Medications/Personal items – While most supermarkets and super-stores (e.g. Walmart carries everything from food, to tires, to appliances to low-priced medications) include such products, pharmacies (like CVS or Osco) are also used commonly. As a trainee, you'll receive your healthcare through your employer (hospital) which will provide you with "tiers" of coverage. Comprehensive coverage costs more but there is less risk if you acquire an illness. Usually the employer pays a lion's share and each month part of your salary contributes to your health plan. Note that the first month of coverage in some programs is not provided (you must ask whether your new employer's plan does/doesn't so you can arrange for your previous coverage to get you through the first month). Most medical services and medications require a "co-pay" i.e. you pay anywhere from $10 to over $1000 depending upon the medication or hospital service and what your plan stipulates. So it is important to read the fine print of your policy.

Schools – American schools are extremely heterogeneous. Some are excellent. Some are poor. If you have children, do your research on the web. Performances of school districts for communities are available on the internet. Trainees with families with school age children should choose where they live carefully after researching schools.

Personal Hygiene – This may seem self-evident, but there are significant differences in hygiene practices in different cultures. In general, doctors are expected to shower daily, wash/comb hair, apply deodorant or anti-perspirant daily, brush teeth or manage halitosis, shave or keep facial hair

groomed etc. Clothing (including lab coats) should be meticulously clean. In the last 15 years, we've had several misunderstandings of IMGs whose customs were otherwise. They felt badly that no one told them sooner, what was expected.

Personal "Space" and pot pourri – While therapeutic touch can be important in the doctor-patient relationship, colleagues and allied healthcare professionals demonstrate careful courtesies to one another – as equals in the healthcare delivery system. Americans generally require 2-3 feet of "personal space" and physical touch should be restricted to the formalities (e.g. hand-shake) unless it is clear that the person requires less space. Public displays of strong emotions, especially anger, are not acceptable and will undermine effectiveness. Intimate personal relationships must remain outside the hospital and are never appropriate with patients or those you are supervising.

Activities with colleagues – Medical training is a very intense and formative period of our lives. To be most successfully, you must develop strong support systems. Since you are sharing this adventure with your fellow trainees, it is only natural to "lean on each other." Accordingly, it is time extremely well invested, to spend some of your "down-time" outside the hospital with classmates. It builds camaraderie, team spirit and a nearby emotional support system.

This section of Oxymoronic will grow with time as more resources become available and as trainees provide me with feed-back about information that can be added to help them circumnavigate towards cultural competence.

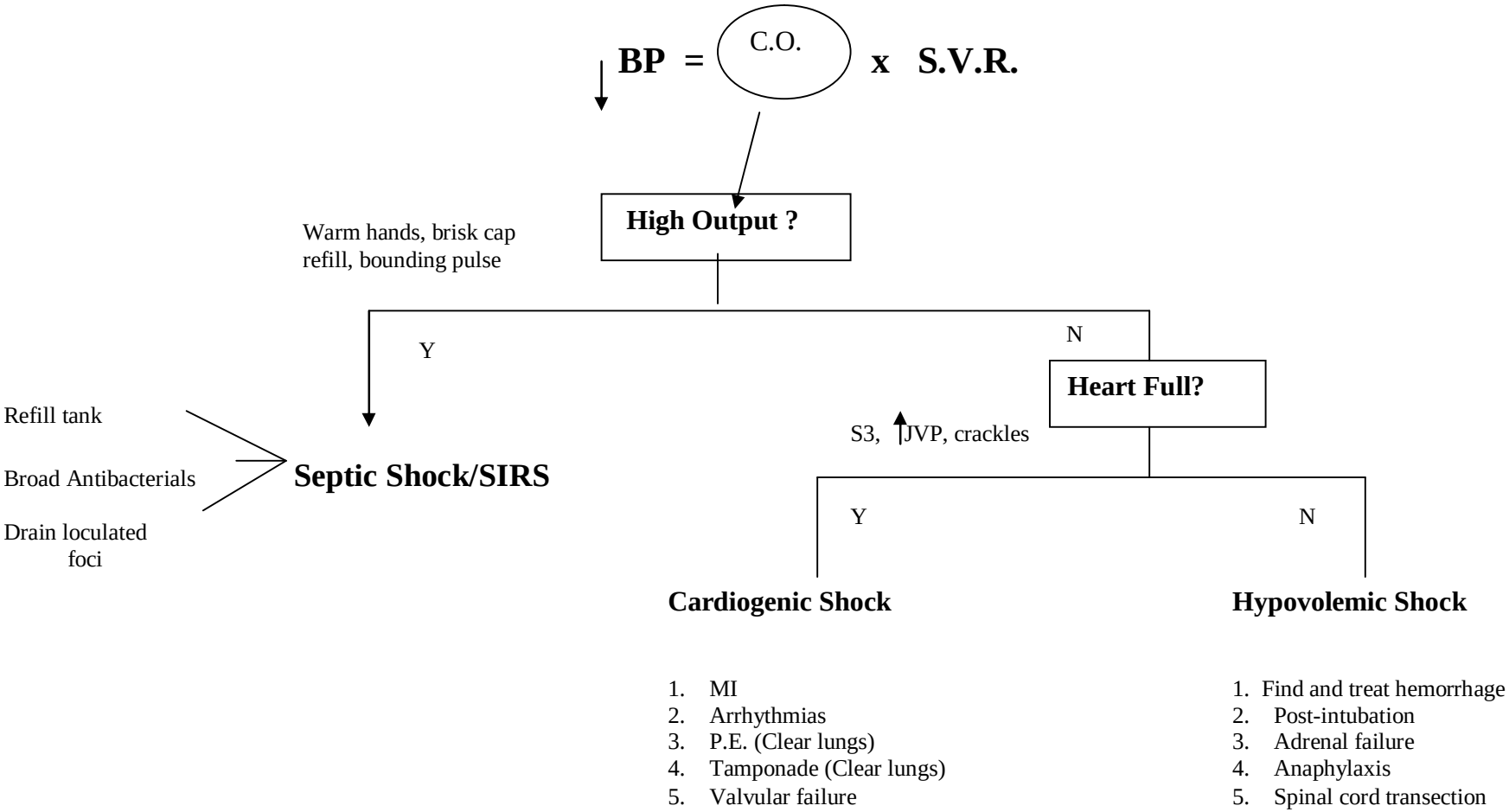

Figure 1 – *University of Chicago bedside approach to the patient with shock.* Shock requires rapid diagnosis and treatment to avoid cell death, organ death and patient death. This algorithm takes no more than 2-3 minutes and empowers the clinician to diagnose and begin management at an appropriate pace.

Figure 2 – *Schematic for mechanics of breathing and dynamic hyperinflation*

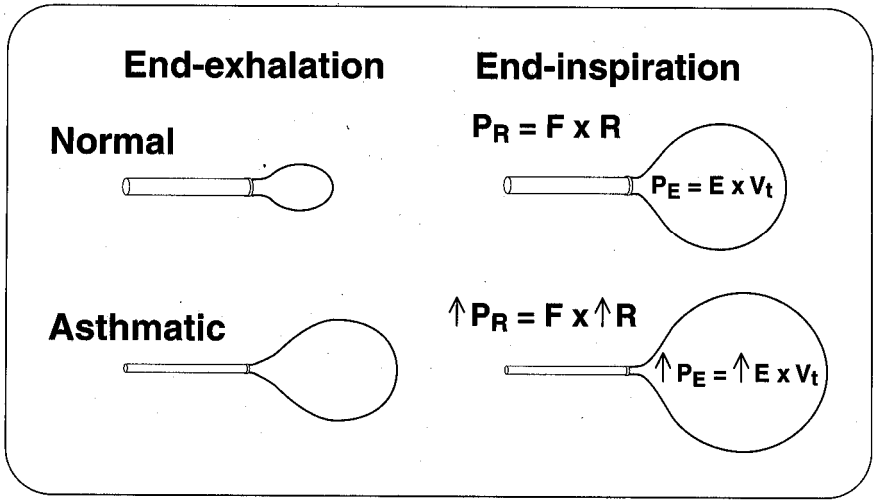

Figure 1—Diagram of the effects of asthma on the work of breathing. During an acute exacerbation of asthma, airway resistance increases, thereby increasing the flow-resistive-pressure work of breathing. High airway resistance also impedes exhalation, so some "trapped gas" is left in the lungs at the initiation of the next inspiration (termed "dynamic hyperinflation"). Subsequent breaths are stacked on the trapped-gas volume, so the lungs (the balloon) begin to hyperinflate. Much as it becomes more difficult to blow up a balloon that is already filled with air, the elastic-pressure work of breathing increases during an acute exacerbation of asthma as a result of dynamic hyperinflation.

Figure 3 – *Cartoon model for the "multiple hits" concept of acute lung injury.*

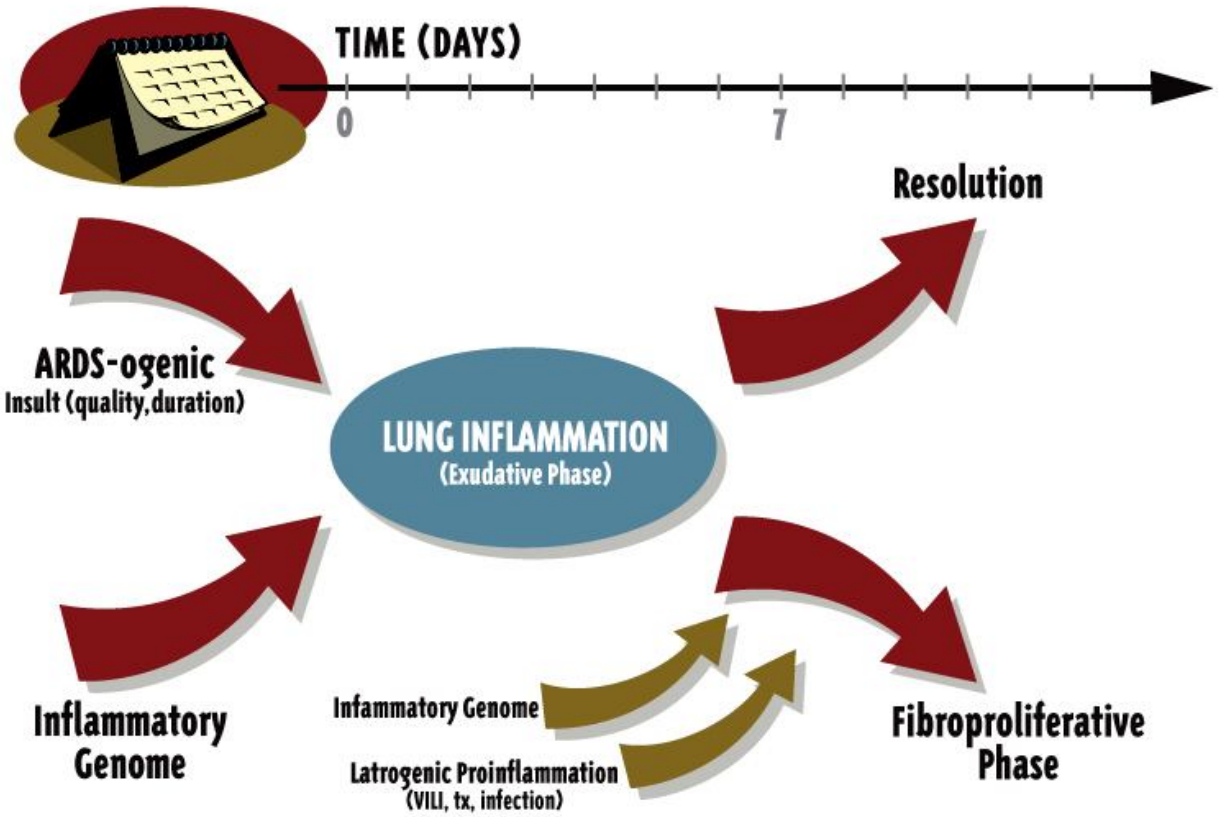

Figure 4 *A Simple Bedside Approach to Stabilize Patients with ARDS* - *All patients begin on 100% inspired oxygen. As PEEP is increased, the fraction of inspired oxygen can be decreased as permitted – usually in decrements of 10% - to keep O2saturation>90%, until reaching safe oxygenation on 60% inspired oxygen. **During recovery: As patients improve, PEEP can be reduced slowly (no more than 1 cmH2O/hour since lung de-recruits quickly and recruits slowly). As PEEP is reduced, airway pressures will fall and when when P_{plat}<<25 cmH2O, increments of tidal volume may be required to prevent sustained atelectasis. (CHF=congestive heart failure, HPV=hypoxic pulmonary vasoconstriction).

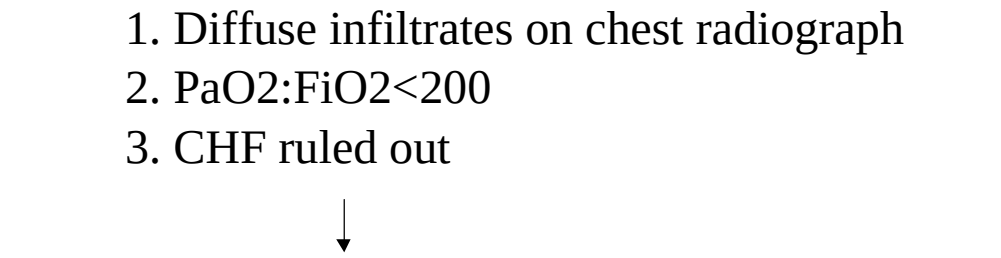

Figure 5 – *Approach the patient with severe hypoxemia.*

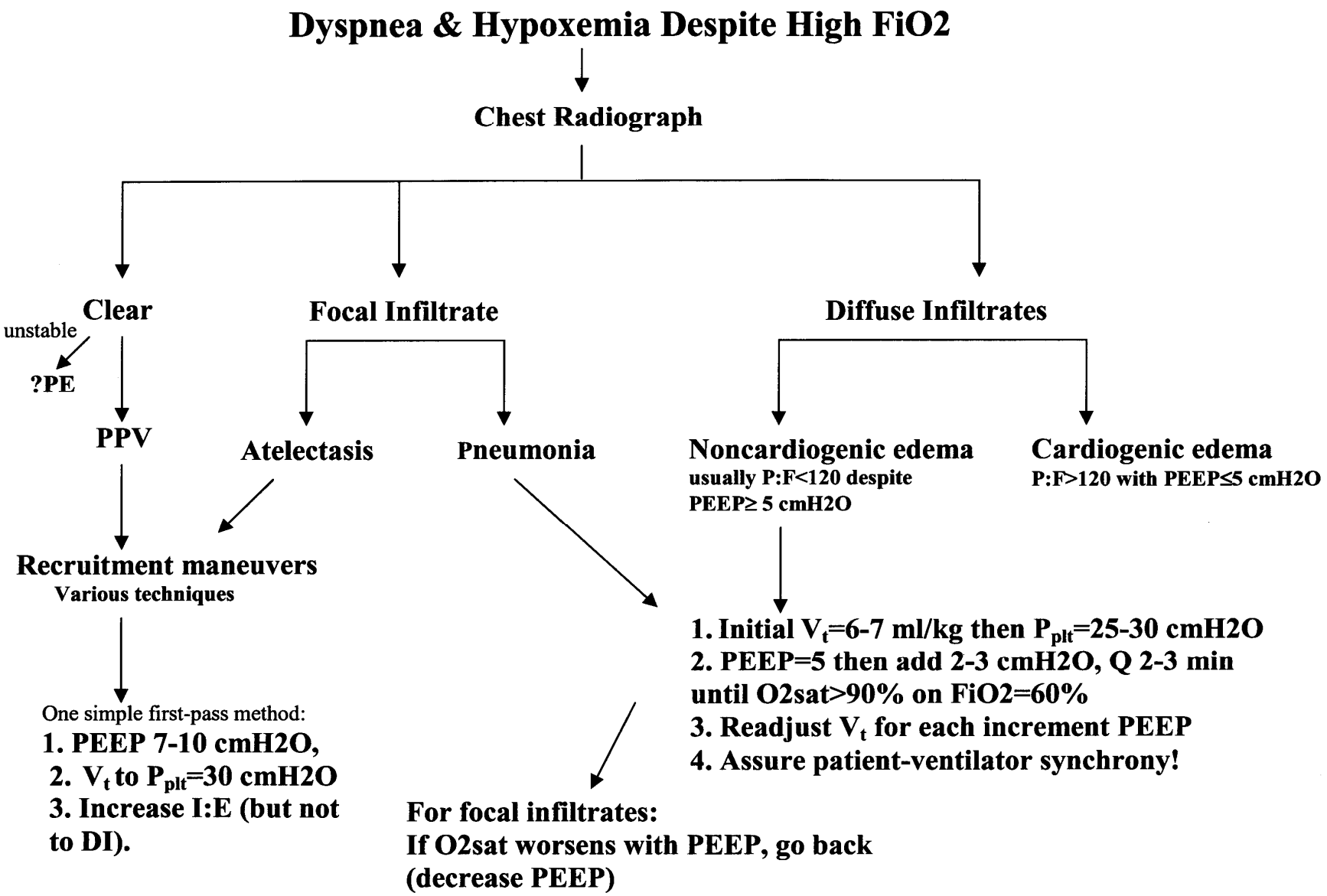

Figure 6 - *Sepsis is the response of the host to an organism and/or its toxins* - Therefore the severity of the response and the rate at which it progresses is dependent upon the immunocompetence of the host and the virulence of the microbe. Event the healthiest patient can be killed rapidly by *Meningococcus* or *Ebola,* while hosts with multiple immuno-deficiencies may die rapidly of septic shock and multiple organ failure from less virulent pathogens.

Host Factors

Microbe Factors

1. Neutrophil #/fnxn
2. Lymphocyte #/fnxn
3. Immuno-suppressing meds
4. Renal failure
5. Diabetes
6. Cirrhosis
7. Malnutrition
8. COPD
9. Host mechanical defenses
10. Obesity
11. Chronic inflammation

1. Microbial virulence
2. Inoculum

Figure 7 – *Cartoon schematizing the course of renal failure.* The particular course will be predicated on: a. the nature, duration and severity of the initial insult, b. the patient's precedent renal function ("reserve"), and, c. whether the kidney is reinsulted during the recovery phase.

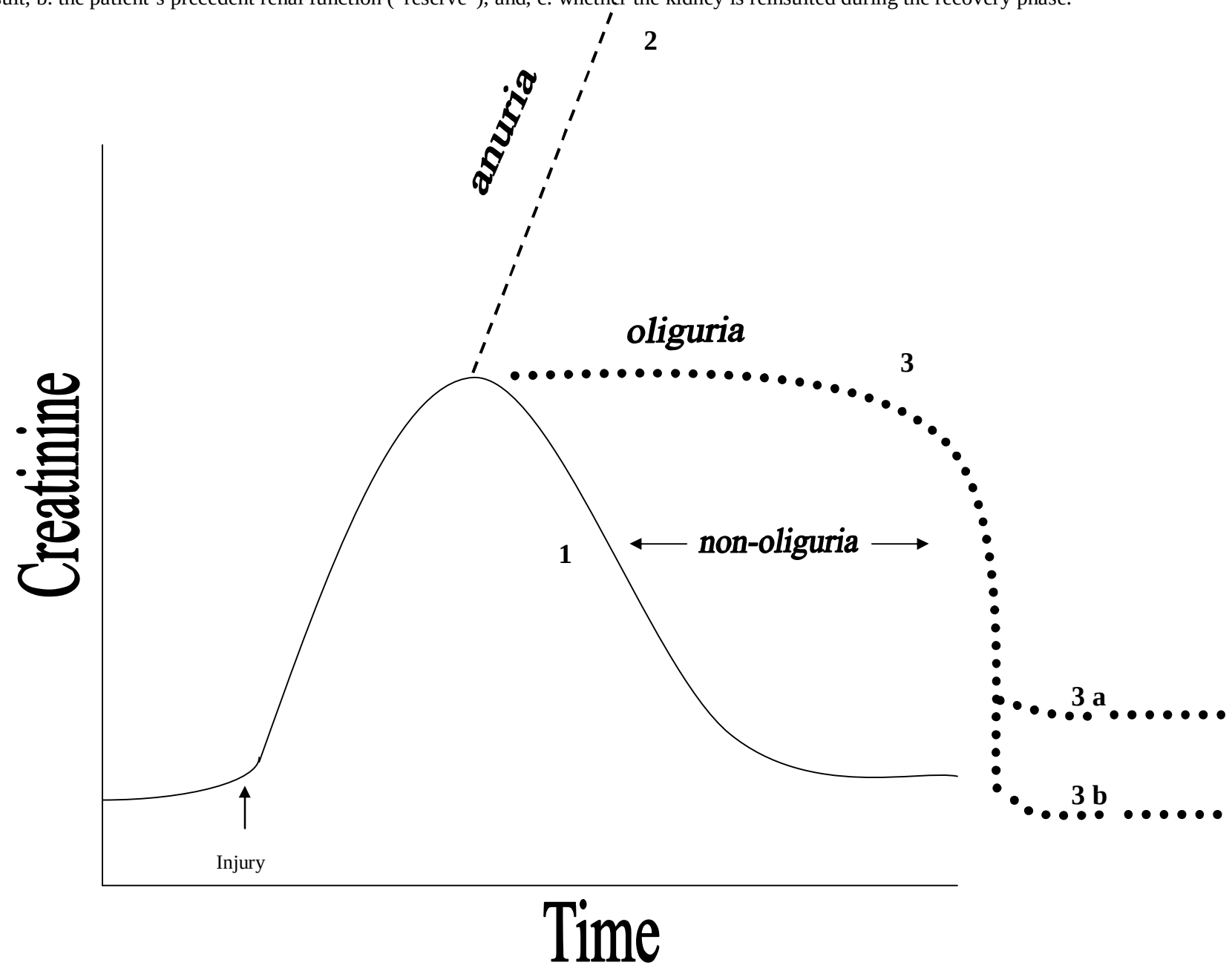

Figure 8 A sample "s*liding scale" for insulin coverage*

FS (meQ/L)	Subcutaneous Insulin (U)
150-200	2
201-250	4
251-300	6
301-350	8
350-400	10
>400	Call MD

Figure 9 – An ABC approach to rescuing the patient with cardiopulmonary instability while determining the goals of therapy based on the patient's predilections.

Respond to calls in person or assign replacement to arrive within 15 minutes

1. Simultaneously check ABCs + CP/gross neurologic exam; ask for immediate therapies (e.g. O_2, nebulized therapies, furosemide)
2. Determine patient's predilections for invasive therapies including:
   a. Monitoring + invasive therapies in ICU
   b. Endotracheal intubation
   c. CPR

   Considering patients' predilections, chronic burden of disease, acute hospital course and likelihood of successful restorative care:

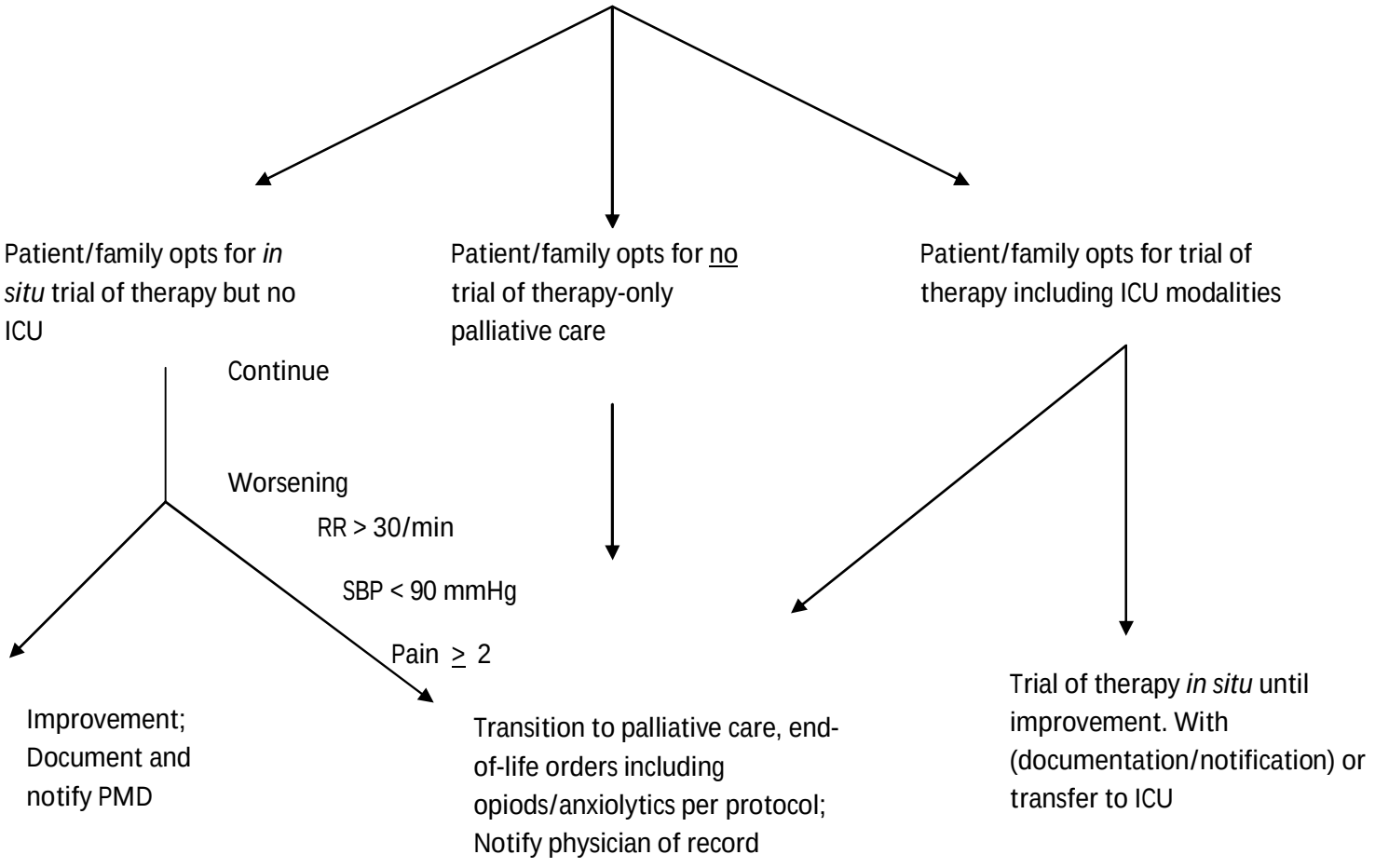

Figure 10 – A physiology-based algorithm for rescuing a patient with hemodynamic instability when arrhythmia requiring cardioversion/CPR is not the cause. Note that such patients often have incompetent airways which require endotracheal intubation for airway protection. They may also have an increased work of breathing that should be reversed rapidly with resuscitation else intubate and ventilate until instability is repaired. See Figure 10 for integration of ABCs.

SBP<90 mmHg and sinus HR>110/min with normotension