

BMC survives Joint Commission visit

The Joint Commission's five day unannounced survey in January 2007 validated Boston Medical Center's commitment to providing safe patient care.

Boston Medical Center demonstrated full compliance with all but two of the National Patient Safety Goals. We received a Requirement for Improvement (RFI) in Admission Medication Reconciliation and for using the Joint Commission's identified "Dangerous Abbreviations" in the medical record. Of note, these two National Patient Safety Goals are among the most frequently cited nationwide.

The Medical Center submitted an action plan addressing how we will correct the RFIs. Following acceptance of the plan by the Joint Commission, we will be required to collect and submit data for four consecutive months to the Joint Commission. To be in full compliance, we must achieve 90% compliance.

Keith Lewis, MD Chairman of Anesthesia is leading the effort to eliminate the use of those abbreviations identified by the Joint Commission to be dangerous. Medical record audits are being conducted in all clinical services.

If a dangerous abbreviation is identified during an audit, the writer will receive a personal letter from Dr. Lewis to bring this to his/her attention. If a second infraction is identified, the writer and the Chief/Director/DON will receive a letter.

If a third infraction is identified, the writer will be requested to explain their rationale for continuing to use dangerous abbreviations that could place patients at risk to the Patient Safety/Quality Council.

The Admission Medication Reconciliation process was redesigned following the Joint Commission survey. The new process requires that the MD/PA/RNP complete the medication section on Part I of the Admission History & Physical. It is required that all sections and boxes be completed. The nurse verifies the admission orders against the list documented on the History & Physical. If the nurse identifies any discrepancies the MD/PA/RNP is paged and requested to make any necessary changes within an hour.

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If the nurse remains unable to reconcile the medications after one hour, the attending physician is paged and notified of the incomplete medication reconciliation. A multidisciplinary team was formed to develop a standardized process for medication reconciliation any time a patient is transferred from one location of care to another. This team continues to meet and will develop a process to be piloted in the upcoming weeks.

A Requirement for Improvement was noted in how critical test results are conveyed to the clinician responsible for the care of the patient. A multidisciplinary team is working to standardize the process for calling back critical results and documenting the time from the call back to notification of the clinician responsible for the care of the patient.

The Joint Commission's unannounced survey process validated Boston Medical Center's commitment to patient safety and providing *Exceptional Care Without Exception.*

D Mehegan

THE INPATIENT TIMES

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The Inpatient Times

All the news that makes you more fit to treat!

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The relationship between pharma and physicians:

What do you think?

The relationship between the pharmaceutical industry and physicians has come under increasing scrutiny over the past few years. While this relationship is critical to designing and evaluating new therapies to improve the health of the public, the interactions have not consistently been faithful to the public interest and to the best ideals of medical professionalism. We have an opportunity to refine our guidelines for this relationship - indeed, our failure to confront this relationship with clear and compelling guidelines may damage the image of our profession and our health care institutions. Since a number of leading academic medical centers have met this challenge with new guidelines, I believe we have an important opportunity and responsibility to define our values and how those values should guide our interactions with the pharmaceutical industry. In order to provoke further thought on this issue, I would like to propose a series of questions for your consideration.

As stewards of medical education, what is our responsibility to insure that our educational activities are as free as possible of bias, including bias derived from commercial interest? Most continuing education is sponsored by commercial entities and most trainees have been exposed to marketing efforts of industry. While industry has an important role in discovery and in publicizing discovery, a considerable body of literature suggests that industry-sponsored educational events may be subject to bias in favor of their commercial interests.

Do gifts or meals of any monetary value from the pharmaceutical industry influence the attitudes or prescribing habits of physicians? Surveys of physicians suggest that most physicians believe that their own attitudes or prescribing habits are not influenced, but suspect that those of others are! Is it possible that we are better at recognizing bias in others than in ourselves? Surveys suggest that we are....what do you think?

Do we have an obligation as an academic medical center to assure patients that we act solely in their interest, based on the most objective information available? Surveys of patients are very clear in answering this question affirmatively. The basic precepts of medical professionalism, including our valuing the importance of discovery, objectivity, and commitment to the welfare of patients compel us to be certain that we utilize and disseminate the best information available.

Does use of free medications provided by industry in clinical practice contribute positively to patient care relative to the cost? While provision of free medications has potential to increase access to drugs for patients who cannot otherwise afford them, it has also been shown lead to prescription of higher cost medications and to extensive use of free medications by health care professionals for their personal use. Are these costs justifiable?

In closing, I hope you will consider this issue in the context of our professional values, not those of industry. The pharmaceutical industry has a fiduciary responsibility to its stockholders and plays within the rules of our economic system. I propose that we formulate guidelines that distinguish our profession and our medical center, and look forward to working toward this goal!

D Coleman

What's in a name?

“As long as I am here, I'm calling this place City and that place University hospital,” said the Emergency Department secretary one night, ‘I'm not having any of this Menino or Newton stuff.’ It was one of my first nights working in Boston Medical Center, and it was the beginning of two years of confusion as I tried to figure out just which building was which in this sprawling metropolis that is BMC.

I was here about six months when I realized that HAC/Menino/City hospital were all the one building. However, I was watching *The Departed* last Summer and one of the characters makes reference to Boston City Hospital, I was struck with an immense sense of pride. I expect our facility is one of the only hospitals to be mentioned in an Oscar winning movie.

The problem with East Newton lies not in the nomenclature, but rather in the architecture. It appears to be connected to nearly every building, namely DOB, Preston, School of Medicine and Robinson. However, the difficulty finding the connection between these buildings is the real challenge. It is perhaps easier to find one's way out of a hole in Kirkuk than it is to get from the DOB to Preston 5. And it is always hard to define where Evan ends and where Robinson begins. I get especially confused by the signs reading ‘leaving Evans and entering Robinson’, not least because they seem to be placed half way along corridors, and I never knew I was in Evans to begin with.

However, despite East Newton's multiple connection points, it is unfortunately lacking the ultimate connection. Every resident dreams of a sky corridor being created between HAC and East Newton. It is not a small request understandably. But during the winter months, when the paths are covered with ice and snow, walking between campuses for teaching, clinic or even food is an altogether unpleasant prospect.

Of course there is the urban myth surrounding a tunnel that apparently runs between both campuses. Some say it was part of the Big Dig. Some claim to have even seen it. Rumors abound as to why it is not in common use, the most popular rumor being that it is lined with asbestos and too risky to allow common use.

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I think most likely that it is part of the Big Dig and therefore is either closed most of the time, or undergoing refurbishments.

Just across from Harrison Avenue Emergency Department entrance then, there is the historic Mallory Building which is itself undergoing considerable amount of changes. Barbara McInnis house is moving in there and whether this will lead to another name change is unsure.

Finally then, there are a large number of sizable buildings being constructed between Harrison Avenue and the Freeway. The biggest is by far the new parking lot. However, there are some who argue that the parking lot is not in Boston Medical Center at all, it is so far off campus. I remember the first morning that people started parking there one of my colleagues complained that “the new parking lot is practically in Braintree. There should be a light-rail system connecting it to Boston Medical Center.”

Finally then the most entertaining aspect of the lay out of BMC is the names attached to the streets near the new labs and parking lot. Arguably the most romantic of these is the path leading up the new Bioterrorism lab which is eloquently called Biolab Drive.

It reminds you that the people who work at One Boston Medical Center Place, wherever that is, still have a sense of humor.

J Ryan

**Congratulations to the entire
Inpatient Service at Boston
Medical Center for having given**

1425

**Influenza vaccines to inpatients
this season!**

**That is a 6.7% increase over last
season!**

**Thanks for your hard work on
this!**

New Troponin I is better than ever!

At the end of April, the Chemistry Laboratory will replace its current Troponin I assay with the more sensitive Ultra Troponin I assay (Siemens Medical Solutions Diagnostics). This assay will meet updated precision guidelines recommended by the ESC/ACC which define an increased value of troponin as a measurement exceeding the 99th percentile of a reference control group of non-diseased individuals. Any troponin elevated above the upper limit of the reference population will now be defined as a “myocardial infarction,” reflecting any amount of myocardial necrosis caused by ischemia. Thus previously diagnosed individuals with severe or unstable angina might now be diagnosed as having had a small MI.

This does not imply that all elevated troponins reflect coronary plaque rupture, however. Other conditions resulting in myocardial cell damage can contribute to elevated troponin (e.g. myocarditis, cardiac surgery, heart failure, etc.) and clinical correlation is required in interpreting value. Please obtain Cardiology Consult for suspected ACS or when needed for assistance with interpreting the clinical significance.

With the new Troponin I assay, the reference range will be:

< 0.05 ng/ml = **negative**

≥ 0.05 ng/ml = **abnormal** (suggestive of myocardial injury)

Of note, the new troponin assay will still utilize blood samples drawn in the same heparinized (green top) tube as the old version.

Using troponins in the diagnosis of AMI

This policy has been reviewed and accepted by both Cardiology and Laboratory Medicine:

Patients with suspected AMI should have baseline total Creatine Kinase (CK) and troponin I drawn at the time of presentation (CK-MB should NOT be drawn if a troponin I is ordered). CK-MB should only be ordered for suspected reinfarction i.e., if a patient had prior infarct within previous two weeks.

Only total CK should be used to peak infarctions.

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Once a patient has a critical troponin I value (i.e., >5 ng/ml) the patient *should not* have a repeat troponin within next 10 days. Further troponin ordering in a patient already ruled-in delays obtaining values for patients for whom we are awaiting their initial value thus potentially delaying their treatment.

When the laboratory staff call back the Critical Alert Value (troponin >5 ng/ml), the following comment will be appended to the EMR:

Clinical staff notified that repeat troponin I not necessary unless the value is inconsistent with the clinical findings. Use total CK to quantitate infarction size. The ordering clinician should CANCEL ALL FURTHER TROPONINS ON OBTAINING A TROPONIN ≥ 5NG/ML.

If there are pending Troponin I orders in SCM they MUST be canceled by the ordering clinician, as the laboratory staff are unable to do this.

When ordering Troponin I to rule out myocardial ischemia, the orders should be every 6 hr's for 3 separate lab draws (i.e. drawn at time 0, 6 and 12 hrs). A patient should only have 3 negative troponin tests ordered within a 24-hour period. The Chest Pain, Acute Coronary Syndrome, and ST Elevation MI Pathways can help you manage the lab ordering appropriately.

If there is a question of reinfarction then CK-MB and not Troponin I should be ordered (order CK with CK-MB). Troponin I remains elevated for 7-10 days while CK-MB peaks and returns to baseline within 2 days. If the total CK begins to rise for a second time (not as a continuation of the initial rise), a series of 3 CK-MBs at time intervals of 6 hours can be ordered.

Total CK, CK-MB and troponin I samples should be drawn in a green top (heparinized tube).

*B Magnani
D Whalen*

Don't forget:

Follow-up appointments can be made by your friendly neighborhood Unit Secretary. Just let them know you've placed the order in SCM (at least one day before discharge). You can do this for any patient who may have difficulty making an appointment.

Prealbumin: Exactly what does it tell us?

The use of hepatic proteins as a marker of nutritional status evolved from studies of kwashiorkor (protein malnutrition) and marasmus (protein-calorie malnutrition), long before there was reliable knowledge of the far-reaching systemic effects of the inflammatory process. As knowledge on this subject has expanded, use of these proteins has become increasingly controversial. Albumin, one of the earliest, and most widely utilized, has been shown to be a poor marker of protein status, slightly more reliable as a marker of general nutritional status, and reasonably accurate as a predictive measure of morbidity and mortality. With albumin's reliability in question on several levels, prealbumin (PAb) has become a popular adjunct to assessing nutritional status. It should be noted that PAb is not related to albumin at all, but is better known as transthyretin, highlighting its action in thyroxine transport.

Nomenclature aside, this protein is also notably different from the traditional protein markers of nutritional status in key ways. The body pool is smaller than albumin, making it more sensitive to smaller variations in production and breakdown. This allows variations in PAb to be detected earlier in the disease state. This smaller pool size is complemented by a shorter half-life; albumin half-life is on the order of 18-20 days, whereas PAb is only 2 days, allowing PAb to be much more responsive to acute changes. As a negative-acute phase protein, PAb falls during the acute phase of injury/illness. Limits of how far this measure falls and how quickly are controversial, but utilizing it in conjunction with a positive acute phase protein, such as C-Reactive Protein (CRP), has become a common way to garner useful conclusions. CRP is widely viewed as a reliable measure of the extent of inflammation and corresponding acuity of the clinical status. When CRP begins to fall, the inflammatory response is beginning to subside and the patient is leaving the catabolic state and entering the anabolic state. It is at this point that one can utilize PAb most effectively; as a negative-acute phase protein, it should rise. If this fails to occur, the patient is likely nutritionally compromised and the response or lack thereof can be utilized to judge

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the extent and aggressiveness of nutritional intervention that is warranted.

Further supporting PAb's role in identifying patients at nutritional risk is the finding that PAb is one of the last proteins to be affected by liver disease and many other chronic diseases. Therefore, PAb is a fairly sensitive and useful marker that retains accurate function until fairly late in disease processes and is largely uncomplicated by co-existent diseases. This being said, it is important to assess this measure, as with all measures of nutritional status, in the context of the patient's overall health, as systemic diseases, such as kidney failure will create artificially high PAb readings due to retention.

Utilization of PAb to represent nutritional status in a non-acutely ill population is more controversial. PAb levels among non-acute patients are quite variable and not as tightly correlated with nutritional status. The evidence correlating PAb with protein status is weaker still, reinforcing the belief that PAb's rightful place is in the ICU and as supportive evidence in already established malnutrition to further categorize the extent of malnutrition, and more importantly the aggressiveness of nutritional intervention required. This global assessment is key to proper nutritional management, so don't hesitate to page your floor's Dietitian to assist you in interpreting PAb levels and determining exactly how to manage your patient's nutritional status.

S Weiss

Good communications with your patients' PCPs facilitates good care – both inpatient and outpatient!

Have you spoken with your patients' PCPs recently?

**Remember:
It is important to notify PCPs on admission, discharge, and with any significant event.**

It's just good patient care!

What internists should know about Remeron, Seroquel, and Zyprexa

Remeron - Seroquel - Zyprexa ..., almost sounds like a law firm; but actually these medications are extremely helpful to patients who are hospitalized in the general hospital. The goal of psychiatric consultation/liaison is to treat the symptoms that are interfering with the medical treatment. Thus, these medications have specific symptom based treatment purposes as well.

1. Remeron (mirtazapine). This is an anti-depressant that effects serotonin and norepinephrine. It is an excellent medication to utilize it's side-effects to benefit hospital patients. Remeron's lowest dose available is 15 mg, and should be started at that dose or lower. The side-effects of this medication include sedation and appetite stimulation. Even greater at 7.5 mg. As the dose increases, the side-effects of sedation and appetite stimulation become less. Thus, if you have a patient who cannot sleep and is not eating, think of Remeron at doses of 15 mg or 7.5 mg. A good agent to use in the elderly and/or patients who are in a delirium. As well, even at this low dose, it will help improve depression and anxiety. Remeron also has anti-nausea properties.

Take home points:

At low doses, good for sedation and appetite stimulation.

Always give at bedtime (30 minutes before desired sleep)

2. Seroquel (quetiapine). This is a atypical antipsychotic. If you prescribe it, the patient should have their eyes checked yearly for the development of cataracts. It has a very low likelihood of causing extra pyramidal side effects (EPS) or increased prolactin levels. It commonly causes sedation, orthostatics, weight gain, reduces the clearance of ativan and may enhance the effects of antihypertensives. Usually given once or twice/day. Many patients who do not have a thought

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disorder, but are substance dependence, "like" the effects of this medication due to the sedation and calming effects. It can be given at HS to utilize the side effect of sedation. Doses range from 25 mg - 50 mg QID. Another use is in the agitated and/or delirious patients. A low therapeutic level is best, and this gained by giving the medication BID at 2 pm and again at HS.

Take home points:

Substance dependence patients can benefit from it.

Better effects with a therapeutic level

Avoid PRN dosing only

3. Zyprexa (olanzapine). Another atypical anti-psychotic medication which frequently causes weight gain. With Zyprexa, it is highly associated with abnormalities of glucose metabolism, including new-onset diabetes and spontaneous ketoacidosis, even in patients without pre-existing risk factors. Always check glucose levels before and after starting medication. This also causes sedation, and should be given at HS. Another good medication for the agitated/delirious patients. Better effects are obtained from a low therapeutic level, and not just PRN dosing. You do not want to chase the symptoms but seize them! A dose of 2.5 - 5.0 mg at HS is a preferred starting point.

Take home points:

Check glucose levels

Appropriate for agitation and delirium

Build a level

Give at HS, and avoid only PRN dosing in agitation/delirium

J Chengalis

WAIT!! Just cuz it's not flu season anymore doesn't mean you shouldn't give out *Pneumovax!!* Don't forget to vaccinate your patients who need this shot!

Narrative Medicine 101: Lessons from a story

Who would have thunk it? Reading stories can make us better doctors? According to proponents of the evolving field of narrative medicine, becoming better readers (and writers) can improve communication skills, resulting in better interactions with patients and less burnout for ourselves.

Narrative medicine is “medicine practiced with the narrative competence to recognize, absorb, interpret and be moved by the stories of illness.”¹

As someone who routinely reads the “reflections” pieces in journals before tackling the technical pieces, I was curious to learn more about narrative medicine. Rita Charon, MD is a pioneer in the field and published the first comprehensive text on the subject, *Narrative Medicine: Honoring the Stories of Illness* (Oxford University Press, 2006). Martha Montello, a literature professor who teaches this to Harvard Medical students once a year, gave me my introduction in a recent seminar. And I, in turn, found a group of (relatively) willing BMC residents to help me test out the claims.

On a cold day in February, the well-documented nadir of residents’ morale, we (3 residents and 2 attendings) gathered in a conference room. After a lively discussion of Kafka’s short story, “A Country Doctor,” each resident was asked to write briefly about a difficult patient encounter. The following writing sample demonstrates how writing (or reading) from different perspectives can bring out insights that might help identify, clarify or resolve difficult issues we face in caring for patients.

Over the past month I spent at the VA hospital I routinely had inflammatory interactions with a particular patient’s daughter. The patient was an elderly gentleman who was wasting away – his body riddled with prostate cancer, osteomyelitis, and contractures confining him to bed. He refused to eat and refused interventions such as PEG and NGT that may have improved his nutritional status (he weighed 71 lbs). His daughter was always trying to dictate his treatments and interventions,

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and would often persuade the patient into changing his mind. The patient would make one decision when alone (DNR/ DNI for example) and reverse this decision when his daughter was present. She would often yell at the team saying “He wants what I want. I’m his proxy.” When discussing quality of life measures rather than invasive procedures she would say “There’s no quality of life when you’re dead.” She would go against nurses and doctors orders, and would demand certain medications be prescribed.

In part two of the exercise, residents were asked to write about the same incident from the viewpoint of the person with whom they were having the conflict.

I come to the hospital everyday and religiously sit at my father’s bedside feeding him, nurturing him only for the doctors to tell me on a daily basis that he is dying and there is little they can do. I have cared for many critically ill family members and I feel I know what my father needs. He needs anxiety medications not pain medications. The antibiotics they are giving him are making him sick. He wants the feeding tube; it’s the only way he’ll get better. If you doctors didn’t harass him about the tube he would agree to it. I want him transferred to another hospital where they’ll actually listen to me. He’s my biological father whom I’ve only known for several years, but I’m not ready to lose him now.

As a beginner in the field of narrative medicine, I see this approach as a promising way to remind ourselves of the difficulty and beauty of our calling to listen to and interpret patient stories. The next time we find ourselves in the midst of a conflict about a patient, perhaps a moment of reflecting on the story will provide useful insights.

Thanks to the residents who participated in the seminar, and to Jeffrey King, MD for permission to reprint his writing sample.

C Rich

References:

¹ Charon, R. *Narrative Medicine: Honoring the Stories of Illness*. New York: Oxford University Press, 2006, vii.

Treating Community Acquired MRSA

Epidemiological studies indicate that community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections are now common in many regions throughout the U.S. In one study of community-onset skin and soft tissue infections (SSTIs) caused by *S. aureus*, three-fourths of those cases were due to CA-MRSA and most cases were due to a single clone – CA-MRSA USA 300. In a recent study conducted at eleven university-affiliated emergency departments in 2004, 76% of SSTIs were caused by *S. aureus* and, of those, the prevalence of MRSA ranged from 15 to 74%. At Boston Medical Center, our outpatient rates appear to be at the lower end of this range, but are rising.

CA-MRSA has been associated with a different resistance cassette than that found in most nosocomial MRSA strains. CA-MRSA is often susceptible to non- β -lactam agents such as doxycycline and trimethoprim-sulfamethoxazole (TMP-SMX). Clindamycin susceptibility is also high, while susceptibility to macrolides and fluoroquinolones is more variable. The CA-MRSA strains have been associated with the Panton-Valentine Leukocidin (*pvl*) gene, believed to be a major virulence factor. Although the vast majority of CA-MRSA infections are SSTIs and have excellent prognoses, invasive necrotizing infections constitute a significant minority.

Many studies suggest outcomes of SSTIs are not impacted by empiric antibiotic choices. This is because surgical drainage is the most important therapeutic intervention. However, in a recent retrospective review, therapy was successful for 95% of patients who received empiric therapy with an antibiotic active against CA-MRSA as compared with 87% of those patients who did not. Although dicloxacillin or cephalexin may still be a cost-effective choice based on the data, it is appropriate to cover CA-MRSA for patients with SSTIs, particularly if surrounding cell

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ulitis is present. Doxycycline, TMP-SMX, and clindamycin are all reasonable options. The clinician should be aware, however, that TMP-SMX does not adequately cover group A streptococcus, another common cause of SSTIs.

Necrotizing fasciitis caused by toxin-producing CA-MRSA has been described. Surgical treatment is, again, the primary focus of therapy. However, if CA-MRSA is a consideration in a patient with necrotizing fasciitis, empiric therapy with vancomycin and clindamycin is recommended.

CA-MRSA has also caused life-threatening cases of necrotizing pneumonia. In a report from the CDC, 17 cases of community-acquired pneumonia (CAP) caused by *S. aureus* with 5 deaths were reported during the 2003-04 influenza season. Eighty-eight percent of those cases were due to MRSA. Of available isolates, toxin genes were detected in all; the *pvl* gene alone was detected in 88%. The authors recommend empiric therapy for CA-MRSA during influenza season in communities with a high prevalence of that pathogen. Due to the rising incidence of CA-MRSA pneumonia and its associated risk of necrotizing infection, the BMC Antibiotic Management Team CAP recommendations vary slightly from national guidelines. To provide coverage for pneumococci, *H influenzae*, atypical pathogens such as *Chlamydia*, *Mycoplasma*, and *Legionella*, as well as expand coverage for CA-MRSA, we consider ceftriaxone with doxycycline an excellent empiric regimen. For patients requiring ICU management, we follow national guidelines and coverage of CA-MRSA is accomplished, if indicated, with vancomycin.

CA-MRSA is a relatively new entity. Treatment recommendations are based on previous experience with MSSA and nosocomially-acquired MRSA, *in vitro* data, and limited clinical trials. Much more needs to be studied to determine the best therapy. If patients are responding poorly, the clinician should determine, when applicable, if surgical drainage has been adequate and if antibiotic therapy is optimal. There should be a low threshold to change to intravenous vancomycin therapy; linezolid or daptomycin are other options in the patient failing or intolerant of treatment. As more is learned, more definitive guidelines can be developed.

T Barlam

A few twisted points about *Torsade de Pointes*

The fluoroquinolones have achieved an important role among antibacterials. Different members of this class are active against several gram-positive and gram-negative bacteria and work by blocking bacterial DNA gyrase. This prevents the relaxation of supercoiled DNA, an essential step in normal transcription and replication. They have low toxicity, good side-effect profiles and are broad spectrum agents. Furthermore, they have excellent oral bioavailability. Their attractiveness has also resulted in their overuse with the unfortunate development of resistant organisms. This has been an impetus for the development of newer, more potent fluoroquinolones.

As newer fluoroquinolones become main stream, a rare but potentially catastrophic complication has been noted with more frequency: *torsade de pointes*. Although other antibiotic classes have been known to cause this, notably the macrolides and the azoles, quinolone induced *torsade* is a phenomenon that is less widely known and therefore potentially more dangerous. In fact, *torsade* has been described with just about every fluoroquinolone, but is most common with sparfloxacin, grepafloxacin, gatifloxacin and levofloxacin.

Torsade de pointes (TdP) is a French term that means “twisting of the points”. The term was first coined by Desertenne in a French medical journal in 1966. Electrocardiographically, TdP is irregular, polymorphic and looks like a party streamer. Contrary to popular belief, it may be self-limited and, when short, may be asymptomatic. All TdP is polymorphic VT, but the converse is not true. TdP is polymorphic VT occurring in the setting of QT prolongation.

A prolonged QTc interval (a QT corrected for the heart rate) of > 500 msec or an increase of the QTc by 60 msec from baseline are established risk factors for the occurrence of TdP. The mechanism of most drug-induced (or acquired) QT prolongation (or prolongation of repolarization) is the blockade of the rapid component of the delayed rectifier potassium current.

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The incidence of TdP in the general population is unknown, but has been estimated to be approximately 8.6 cases/10 million individuals. In an inpatient population, where multiple “insults” to repolarization may exist, the risk would be expected to be much higher. The term “repolarization reserve” was introduced by Roden of Vanderbilt University. It describes the adaptation of repolarization to a variety of insults. Thus, the accumulation of multiple risk factors predisposes individuals to developing TdP. In the normal heart, there exists practically no potential for TdP to develop. However, when multiple risk factors accumulate, the repolarization reserve becomes exhausted resulting in electrical instability. The recipe for disaster appears to be the following:

- 1) Clinical or subclinical ion channel mutation (necessary, but not sufficient)
- 2) Underlying cardiac disease (ischemia, CHF, bradycardia, atrial fibrillation)
- 3) Electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia)
- 4) Organ Impairment (renal insufficiency, severe liver disease)
- 5) Co-administration of potassium channel blockers (e.g. amiodarone, haloperidol, tricyclics, azoles, macrolides, antimalarials, etc.).

So how safe are these drugs? The quinolone most likely to cause TdP, sparfloxacin, is no longer available in the US. Ciprofloxacin remains the safest quinolone to date with only 3 case reports in the literature. As for gatifloxacin, levofloxacin and moxifloxacin, these agents are considered interchangeable and considered to be low risk. It should be pointed out that case reports always underestimate the true incidence: in the last three years, the EP consult service has seen approximately four clear cut cases of TdP as a consequence of levofloxacin use, all associated with other risk factors.

In cases like these, the risk of TdP can be reduced by following a few simple strategies:

- a) Avoid the overuse of fluoroquinolones.
- b) Check the QTc on patients on medications known to prolong it.
- c) Identify patients with the risk factors above who are at increased risk of TdP.

P Lelorier

PEG tubes in the setting of advanced dementia: Is it worth it?

Scenario:

Mrs. S is an 86-year old female with a history of coronary artery disease and severe Alzheimer’s dementia. Over the last 3 months she has had multiple admissions for frequent falls, aspiration pneumonia, and urinary tract infections. On this occasion she is brought to the ER due to decreased appetite and worsening dementia. The patient has three sons who are involved in her care and would like to meet with you to discuss goals of care for their mother.

They are in agreement that they would like her to be DNAR/DNI. They have not been able to reach a consensus on placement of a feeding tube (PEG – Percutaneous Endoscopic Gastrostomy). One of her sons states, “I understand that she is close to dying. That doesn’t mean I want to starve her or make her feel uncomfortable. Plus, we might be able to extend her life and prevent these aspiration pneumonias.”

Commentary:

It is the physician’s responsibility to address the family’s concerns and help them make the best decision for their family member. These concerns often include comfort and prolongation of life.

It is critical to remind the family that both they and the physicians need to make decisions for the patient based on knowledge of the patient’s wishes prior to her decompensation. You, as the care provider, can accomplish this by presenting an open-ended question such as, “How did your mom feeling about feeding tubes?” That may trigger memories of conversations in which the patient may have expressed her thoughts on feeding tubes.

Families are often under the impression that they are going to “starve” their family member if they do not give consent for feeding tube placement. Studies have shown that patients

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experience minimal hunger or thirst during this phase of advanced dementia.¹ Their thirst sensation can be satisfied with ice chips and mouth swabs.²

It is important to address the family’s concerns for comfort and prevention of aspiration pneumonias. Though families hope to decrease patients’ discomfort by placing feeding tubes, the opposite can be true as well. There are complications associated with feeding tube placement. These range from skin infection and tube clogging to perforation. A secondary complication of feeding tube placement may be the need to restrain a demented patient to prevent them from pulling the tube. In addition, observational studies have shown that feeding tubes do not prevent aspiration in dementia patients.³

Some families also worry that they are hastening the patient’s demise by refusing to place the PEG tube. If families have this fear, I explain to them that dementia is an incurable and terminal disease. In fact, one-year mortality rates among dementia patients with difficulty eating are not significantly different when placing a feeding tube compared to withholding it.⁴

Conclusion:

After learning about the complications associated with PEG tube placement and the fact that it will not prolong her life, Mrs. S’s family decided not to place a feeding tube. They decided to focus on maintaining her comfort at the end of life.

A Agrawal MD

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A few landmarks in the history of physical diagnosis

17th century B.C.

The Egyptians were one of the first civilizations to systematically document the practice of medicine. The first recognized physician was the Egyptian priest Imhotep, who many consider to be the true father of medicine. The Edwin Smith Papyrus (Seventeenth Century B.C.) and the Georg Ebers Papyrus (Sixteenth Century B.C.) are an instructional system of the diagnosis and practice of medicine, which referred to audible signs of disease within the body. Egyptian methods of diagnosis used information obtained by examination of the patient. These papyri contain astute diagnostic observations. For example, hernias were noted "When you judge a swelling on the surface of the belly...what comes out...caused by coughing." The Wall of Twin Temple of Kom Ombo on Nile, which was the center for medical care in ancient Egypt, has a hieroglyphic relief depicting various medical and surgical instruments.

4th Century B.C.

Hippocrates, the "Father of Medicine," advocated for the search of philosophical and practical instruments to improve medicine in 350 B.C. He discussed a procedure for shaking a patient by the shoulders (succussion) and listening for sounds evoked by the chest. Hippocrates also used the method of applying the ear directly to the chest and found it useful in order to distinguish between the accumulation of water and pus within the chest. Water bubbled like "simmering vinegar."

3rd Century A.D.

Caelus Aurelianus listened to the chest in A.D. 200 by placing his ear in direct contact with it in order to diagnose bronchitis. And Aretaeus of Cappadonia described abdominal sounds in dropsy as being drum like (tympanic).

16th Century A.D.

In the sixteenth century, the renowned surgeon Ambroise Pare noted that "if there is matter or

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other humors in the thorax, one can hear a noise like that of a half filled gurgling bottle."

Physiologist Robert Hooke speculated in the 1700s after listening to the beating heart "who knows, I say, but that it may be possible to discover the Motion of the Internal Parts of Bodies...by the sound they make; that one may discover the Works performed in the several offices and shops of a Man's Body, and thereby discover what Instruments or Engine is out of order."

18th Century A.D.

Joseph Leopold Auenbrugger provided the first comprehensive description of percussion of the chest in his 1761 monograph. He began to employ percussion in 1754 as a physician at the Spanish Hospital in Vienna and attributed his discovery to his boyhood experience of watching his father tapping to determine the fluid level in kegs. Auenbrugger tapped the patients with his fingertips with the hand drawn closed to determine the point where percussion detected an abnormality. He described the sounds as either high pitched, muted or dull. But percussion never received general acceptance. It was Jean Nicholas Corvisart, physician to Napoleon Bonaparte, and teacher of Laennec, who moved percussion into the mainstream of medical practice. Corvisart adapted Auenbrugger's technique by using the plantar surface of his fingers to strike the chest. He published a French translation of Auenbrugger's text in 1808, which was widely read. John Forbes of England translated the text into English in 1824, using original case observations to illustrate the usefulness of percussion.

19th Century A.D.

Collin in his 1824 monograph on respiration devoted a chapter to percussion. He preferred slight tapping with a stethoscope as the best means of producing the percussed sound, perhaps reflecting the fact that he was an assistant to Laennec who used this technique. A.D. Piorry introduced in 1826 the use of a solid piece of

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material, usually ivory, as a pleximeter to improve the quality of sound as a result of tapping the pleximeter placed firmly against the chest (mediate percussion) rather than the chest wall itself (immediate percussion). He adapted the stethoscope to include a pleximeter and published his inventions in 1828. In Germany, Wintrich introduced the first percussion hammer in 1841. By this time percussion had become an accepted diagnostic modality.

Rene T.H. Laennec's teacher Corvisart was accustomed to placing his ear over the cardiac region of the chest to listen to the heart. Many students of Corvisart used the unaided ear to listen to the heart of their patients. Nevertheless, the evolution from listening with the unaided ear (immediate auscultation) to the aided ear (mediate auscultation) awaited Laennec's invention of the stethoscope in 1816. Laennec rolled a quire of paper in sort of a cylinder and applied one end of it to the region of the heart and the other end to his ear, and was pleased to find that he could thereby perceive the action of the heart more clearly and distinctly than by the immediate application of the ear.

Interestingly, Laennec does not mention in the first edition of his text in 1819 the experiment of the famous physicist and physician W.H. Wollaston, who, in 1810, reported using a long notched stick resting on his foot with his ear resting on the other end to count the sounds of muscle contraction in his foot. Initially, Laennec simply called his invention "le cylindre", but later chose the name stethoscope from the Greek words stethos (chest) and scope (to look at). John Forbes, who translated Laennec's text into English in 1821, first applied the Latin word auscultation (to hear) to the practice of medicine.

Laennec described the different sounds produced in the chest cavity by the movement of air, movement of lung tissue, accumulation of lung fluid, reverberation of the voice and beating of the heart. Mediate auscultation with the stethoscope was accepted slowly into medical practice during the remainder of the

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nineteenth century.

20th Century A.D.

By the twentieth century, inspection, palpation, percussion and auscultation became the standard physical diagnostic approach to examining a patient and remains so today. In the end, the stethoscope became the symbol of the learned physician because it enabled doctors to hear the signs of patients' respiratory and circulatory diseases.

21st Century AD

This could well become the age of the electronic stethoscope. Immediate playback simultaneously to multiple listeners is accomplished easily, and this playback can also be performed at reduced speeds without distortion of sound pitch. Simultaneous visual display provides to the student precise identification and localization of specific sounds within the cardiac cycle. With portable handheld devices and wireless communication, audiovisual presentations to groups of any size are possible in both bedside and auditorium settings.

Perhaps most importantly, however, these methods provide a critical vehicle for the teaching of cardiac auscultation—a method that can and should be preserved for future generations.

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