MOUNT SINAI CONFERENCE



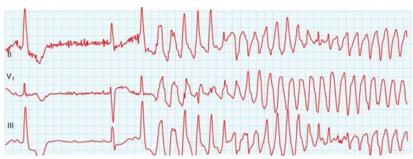
Documentation in the ED, Pt. 1: Billing - Dr. Ashton

In the first of 4 lectures in collaboration with Dr. Pour on ED **documentation**, Dr. Ashton led a boisterous discussion on upcoming changes in how Level of Service (LOS) will be determined. No more click boxes? Not quite; we're still keeping them for medico-legal purposes (for now?), but **as of 1/1/2023, LOS will be determined solely by the documentation of Medical Decision Making (MDM).**

This makes it important to document acknoledgement of positive results that affect decision making (ED course @MS/comment->ED note @ EHC) and to document conversations with consultants (with epic order) and family/NH staff/etc for collateral. Procedure notes continue to alert coders to procedureal billing. Attendings will continue to document critical care time. Check your email: Dr. Pour sent an attachment from Dr. Schimmel of MSH costs.

Cardiology Conference with a Twist - Dr. Shah

Dr. Shah kicked off conference with a complicated case of acquired Torsades de Pointes in a patient who initially presented with seizure.



This patient had significant electrolyte abnormalities, and after normalizing electroyltes and giving magnesium continued to have TdP, most likely 2/2 QT prolonging ED/inpatient medications including azithromycin, levetiracetam, famotidine, and suboxone in addition to his prescribed methadone. This was the springboard into a discussion of Torsades triggers, diagnosis, and management.

Important takeaways included:

- Torsades comes in two varieties: Pause-dependent (most common, as in our case) and Adrenergic-dependent. EKG helps distinguish them and they are treated differently, so diagnosis is important.
- Shock if unstable.
- If TdP is Persistent, plan for TVP with overdrive pacing @ 100 BPM. If pause-dependent, consider isopreoterenol or dopamine or temporization. Avoid beta-blockers if adrenergic-dependent.
- Avoid stacking QT prolonging medications. check med list and EKG, use crediblemeds.org

Junior Small Group

Junior residents were split into three groups and reviewed cardiology cases. Through the (physical but by no means emotional) temporary divider they were heard to have a raucous time and, per report, learned intricacies of the defibrillator/monitor/pacer device (AKA zoll) in addition to other tasty cardiologic tidbits.

Senior Small Group

Dr. Lucazs Cygan (NYP Brooklyn) led the senior residents in a less raucus, but rousing game of find evidence (link) to convince your cardiology colleagues to do what you believe is best for the patient. His discussion armed residents with evidence relevant to 4 scenarios:

1. Patient with active exertional chest pain, normal EKG, **positive** trop. Cards states NSTEMI doesn't need immediate cath. TIMACS trial: In 3031 patients with ACS; showed no difference in death, MI, or stroke between early (≤ 24 hours, median 14) vs. late (≥36 hours, median 51). cath. However TIMACS excluded patients with persistent chest pain and did not investigate immediate cath. The **VERDICT** trial looked at "very early" cath and found no overall benefit, **But** in

- **Treat** with Magnesium (acts as CCB) bolus + infusion, replete electrolytes, stop QT prolonging medications.
- HR affects QTc. Use nomogram to determing TdP risk.

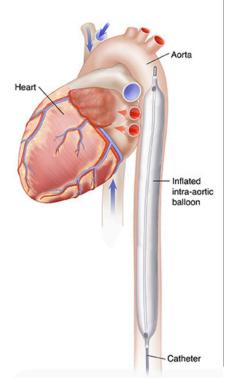
Cardiology Conference Part II: The Intra-Aortic Balloon Pump - Dr. Groden

Dr. Groden started by clarifying that we should address him as "Phill" (2 ells), "Phillip," "Dr. Groden," but not "Grodey" or "Dr. Phil". He also implored us to <u>vote</u> in the important upcoming election.



He presented a CPORT case, reviewing occlusive EKGs and lesion localization before spending the bulk of his talk discussing the intra- aortic balloon pump (IABP). IABP is indecated as a bridge to more definitive devie (i.e. LVAD, Impella) or surgical repair in patients with cardiogenic shock, who are pre/post cath, or who have an occlusive myocardial infarction. The device is placed with seldinger technique, then connected to a monior. It decreases oxygen demand in the heart and improves cardiac output, but has unclear mortality benefit.

Patients with the device in are uncomfortable, sometimes in pain from the device and must like still in bed. Compications include iatrogenic dissection/ ischemia, balloon leaks, bleeding and patients require frequent neruovascular checks, renal perfusion monitoring, checks for blood in the line (balloon leak), wound checks. The device has a mortality benefit in the setting of thrombolytics for occlusive MI and Dr. expects these devices to be more common, expecially in hospitals which must transfer to a cath-capable center.

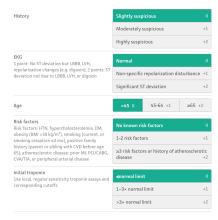


both trials the subgroup analysis of the highest risk patients (GRACE score >140) did find benefit to early cath. Also the outcomes measured (subsequent MI, death and stroke) do not account for the likelihood that if the patient is actively losing myocardium, time to intervention affects quality of life, which was not measured.

- 2. Patient with concerning syptoms but **EKG that does** not meet classical STEMI criteria: literature (i.e. Steven Smith) is moving from STEMI nomenclaure to Occlusive Myocardial Infarction (OMI) because **STEMI** paradigm misses 25-30% of occuluded patients. OMI findings of STEMI equivalents include Sgarbossa criteria (LBBB/Vpaced), anterior QS waves, subtle inferior OMI, Posterior OMI, subtle LAD occulsion, de Winter T waves.
- 3. Patient with recent Coronary CTA, now with angina, refusing troponin. Current AHA guidelines give "warranty period" for cardiac testing (CCTA, stress test, but only once ACS is ruled out. Patient needs troponin.
- 4. Patient with Out of Hospital Cardiac Arrest (OHCA), now

Cardiac Risk: Beyond HEART- Dr. Mukherji

Visiting from Northwell NSLIJ, Dr. Mukherji led an interactive discussion on chest pain, helping us focus our clinical acumen on the most important parts of the chest pain workup, specifically highlighting the HEART score as a subjective, operator dependent, measure that can be appropriately



and inappropriately appied. He states that the adoption of the HEART score did not change practice statistics, that the same numbers of patients are still being admitted, though perhaps these are a slightly different population.

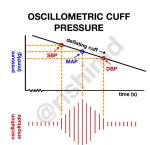
He highlighted the history as the most importat part of our workup, showing that exertional, radiating, pressure associated with nausea, vomiting, diaphoresis that is similar to prior MI is most concerning. No single aspect of the history is unconcerning, however features that are less likely ACS bay be "stacked." These include pain that is pleuritic, positional, reproducible with palpation, stabbing (though "stabbing" is present in 3-5% of ACS), localized to a small area.

Finally, Dr Mukherji pressed learners not to rely on the HEART score blindly, impressing upong us that the EKG, troponin and history are the most useful tools we have to guide chest pain management.

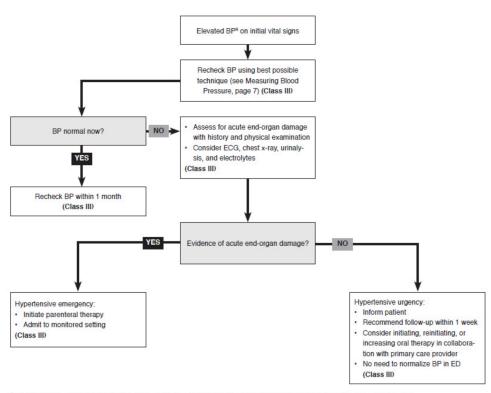
Under Pressure: Blood Pressure Management in the ED - Dr. Trivedi

Hypertension is common in the emergentcy department. When should we be managing BP in the ED? What are common pitfalls in BP management? Dr. Trivedi bravely led an expedition through the tumultuous topic of high blood pressure.

Oscillometric cuff pressures measure the MAP and extrapolate SBP/DBP.
Therefore we should define Hypertensive Emergency as end-organ damage and an acute elevation of MAP to 135-140 in a patient who is normally normotensive.



Clinical Pathway For Management Of Elevated Blood Pressure In The Emergency Department



[®]All patients with elevated blood pressure should have it addressed in the clinical decision-making notes or discharge instructions.

Abbreviations: RP, blood pressure: ECG, electrocardiogram: ED, empressure denartment

Abbreviations: BP, blood pressure; ECG, electrocardiogram; ED, emergency department. Mechanism of Dose Adverse Effects Contraindications Drug Action Sinus bradycardia, Sick sinus Hypotension, 500 to 1000 μg/kg syndrome, Second-Dizziness, Peripheral ischemia Cardioselective ß1or third-degree heart block, Heart failure, i.v. bolus in 1 min or 50–250 µg/kg/min decreased cardiac Infusion site continuous i.v. Cardiogenic shock. output reaction. Pulmonary hypertension, Asthma, COPD Symptomatic Asthma, Heart postural 0.25-0.5 mg/kg i.v. and β-adrenergic failure, Second- o hypotension bolus or 2–4 mg/min i.v. infusion, thereafter 5–20 mg/h blocker resulting in decreased cardiac third-degree heart block, Cardiogenic LABETALOL Flushing, Acute left ventricular failure, output and direct Shock, Severe Bronchospasm, vasodilation bradycardia Bradycardia 1-2 mg/h i.v. Allergies to infusion, increase Block L-type calcium sovbeans, sov every 2 min with 2 mg/h i.v. bolus or channels, which leads to coronary Systemic hypotension, Reflex products, eggs or egg products, 15-30 mg/min and peripheral tachycardia Defective lipid vasodilation metabolism, Sever aortic stenosis 5 mg/h continuous Dizziness, Flushing Block L-type calcium Reflex tahycardia dose by 2.5 mg/h Liver failure NICARDIPINE leads to coronary Nausea, Vomiting, every 15 min to a Increased intracranial pressure mg/h Known history of Headache, Reflex intracranial 5-200 µg/min tachycardia, pressure. Severe continuous i.v. infusion, increase by Vomiting, Flushing, nemia, Right-sided NITROGLYCERINE Methemoglobinemia, 5 µg/min every 5 min Syncope infarction, Venodilator Concurrent use with PDE-5 inhibitors ED workup of hypertension should first confirm the reading with a repeat measurement, then assess for end organ damage suggested by symptoms/history/ exam. This testing may include US (aortic dissection? volume overload? LVH), labs (Upreg, BMP, troponin, UA, Utox), EKG, imaging (CTH, CTA H/N/C/A/P).

If you diagnose Hypertensive Emergency, how do you treat it? BP goal should be to decrease MAP by 20% in 1-2 hours. If you drop your

patient's pressure too fast you may cause ischemia. After the initial drop, slowly lower the MAO to 125 over the next 2-6 hours. initial drop, slowly lower the MAP to 125 over the next 2-6 hours. If managing storke, BP goals are still listed as SBP, with hemorrhagic goal SBP <160, ischemic CVA trated with tPA/endovascular intervition goal <185/110, but should allow permissive hypertensiion for ischemic strokes that do not receive tPA/andovascular intervention up to 220/110.

What meds to use to treat BP in the ED? Dr. Trivedi recommends nicardipine drip (onset 5-15 min, lasts 1 hour, contraindicated in cirrhosis patients) or clevidipine drip (onset 2 min, lasts 10 min, contraindicated in patients with issues 2/2 lipidemia). If you need to push a medication, Dr.

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intubated, s/p ROSC, with STD in V2,V3: According to the COACT and PROCAT I and II trials we do not have evidence to push cards to take patient to cath unless initial rhythm was shockable and patient meets STEMI criteria.

Trivedi recommends labetalol pushes (onset 5-10 min, lasts 3-6 hours, contraindicated in bradycardia, heart block, cardiogenic pulmonary edema, asthma exaccerbation, cocaine/ sympathomimetic intox).

Should we treat asymptomatic uncontrolled hypertension in the ED? According to ACEP guidelines, routine ED medical intervention is no trequired. However if patient is likely to e lost to follow up it is reasonable to start an antihypertensive; Dr. Trivedi recommends amlodipine for its low side-effect profile. Regardless, all patients with a single reading of high blood presure in the ED should receive guidance on the importance of blood pressure management and lifestyle modifications in addition to primary care follow up

Focused Case Review - Dr. Remy

Dr. Grossman introduced Dr. Remy, stating that "the artist formerly known as M&M" is no more. He detailed a recent law suit in which evidence thought to be inadmissible in court was obtained. We will have new opportunites for case review but M&M is over.

Dr. Remy presented a Focused Case Review, which was not recorded by your faithful scribe due to issues of privacy and confidentiality.

