1) Ketamine is in the news again. There is a Turkish study that said that midazolam blunts the well known emergence reaction. (Ann Emerg Med 57(2)109). The methods admittedly were not bad, but see the related editorial from Ketamine guru Steve Green and yes-EMU reader Baruch Krauss. (ibid 115) They point out that not everyone has bad emergence reaction – there is a continuum- some are slightly bad and some are very bad; some even have a pleasant reaction. So to do a study as "yes/no" emergence reaction can be deceiving. My comments are as follows. Firstly, I do not know Steve Green but I have to thank Baruch for putting ketamine on our map. They didn’t discover this drug- it has been around for fifty years; but they put it to the forefront. My problem is that in kids midazolam did not work in previous articles (ibid 36(6)579). Perhaps you’ll say that is because they do not get emergence reactions but there was only a six percent difference in the incidence of emergence reactions in kids and adults, so why should midazolam work only in adults? In addition, reader Dr. Al Sachetti recently pointed out in EMRAP that he uses ketamine often without any midazolam and has never seen this reaction. To be quite honest I have used ketamine very often especially years ago when propofol was less popular and indeed I never saw the reaction either. And it’s popularity as a street drug seems to also agree with what Dr. Krauss said above. I think in conclusion, use ketamine but do not give everyone midazolam as a matter of routine. I will just add my peer reviewer’s comment: agree – never seen a bad emergency reaction (I’m a spring chicken however!) Pre-ketamine coaching is crucial to avoiding an unpleasant subjective experience – I like Reuben Strayer’s approach: http://emupdates.com/2011/01/27/taming-the-ketamine-tiger By the way, you could use alfentanil also for sedation (ibid 57(2)117) but 40% needed some kind of mild airway intervention similar to deep sedation of propofol. I use this drug only for sedation in intubated patients- see Sept EMU roundtable. One last note on sedation – patients with sleep apnea can be given conscious sedation (SMJ 104(3)185). It seems that this is
correct; but this was for endoscopy and I am not sure what agent they used. Be careful since many of these patients are fat and may be hard intubations. Did I say that was the last point on sedation? Well, I lied. We discussed ketofol in the past. Many folks do not understand the need for two sedation agents, but remember-ketamine is a pain reliever in sub dissociative doses, such as 0.15mg/kg-0.3mg/kg. It is good to add a pain reliever when ever doing a painful procedure when a pure relaxant is on board (such as midazolam or propofol). My peer reviewer adds: This ties in with anesthetic literature suggesting that ketamine may reduce the incidence of chronic postsurgical pain and attenuate opioid-induced tolerance and hyperalgesia. It has been shown that this relieves pain that may be present after the procedure in for example a fracture. See AEM 18(3)237.

2) There is always the possibility you will see this injury, even outside of the ED. The patient is usually a wrestler or a weight lifter and they feel a pop with pain in the upper arm. There is occasionally ecchymosis on the inner aspect of the arm. The chest film will be normal. If you do not note asymmetry in the chest when you have the patient abduct and externally rotate the arm, you will miss a pectoralis major rupture. These patients do poorly when managed conservatively so do not miss this injury. (JEM 40(2) 208). Now while we are on diagnostic challenges try this one on for size "A patient with atopic dermatitis presents with vesicles growing on an area of atopy on the face, there was some fever, and some crusting and Cephalexin did not help. (JEM ibid p167) This is a hard one, so let's give you some help here is a picture.
Let us give you some choices as well as we are all bad on diagnosis in Derm:
- Eczema herpeticum
- Transient acantholytic dermatosis
- Papular urticaria
- Pustular psoriasis

3) Many years ago Clairol used to advertise "Is it real or isn’t it? Only your hairdresser knows for sure". Same could be said about this study. They did a nice randomized placebo controlled study and saw that paracetamol (acetaminophen) does increase INR. (Eur Clin J Pharm 67(3)309). Perhaps, but this study was tiny- only 45 patients in each arm and they took 2 or 3 grams of the drug every day for ten days which does not mirror what occurs in real life- at least I do not think so. So think about this- but consult with your hairdresser in any case.

4) If you listen to Risk Management Monthly—Greg Henry and Rick Bukata's excellent CD series (that's a plug guys- you owe me one) you will know how much in the last few months they have been
cautioning the practice of ordering tests in the ED that you can't follow up on- stuff like PSA etc. (ibid p225) Now in my ED- our director has eliminated the taking of urine cultures and blood cultures on patients that are not admitted- for the same reason. I definitely support this idea, but the subject of x rays is especially important to EPs in Israel – and perhaps FPs as well. If you order them- you must follow up on the results- because you may have noted there was no infiltrate but you may have missed the coin lesion that was the size of a shekel (or a dime). In many high volume EDs- no one goes over chest films- and you are liable. Chris 's comment is important- we let him speak again: Reminds of the storm Billy Mallon recently kicked up on EM blog: http://www.epmonthly.com/columns/in-my-opinion/the-life-cycle-of-a-parasitic-specialist/ If this bothers you, try speaking to your radiologist chair- probably will accomplish as much as speaking to your hairdresser. Are they one and the same?

5) Absolutely loved this study and if you are not a trauma doctor- read on any way. In Greece at least- patients who went to centers with ATLS trained physicians had worse outcomes than those treated by physicians without such training (Rescus 82(2)180). Now of course there can be many reasons for this, being that there are no trauma centers in Greece but these badge courses do not really certify anything. I think it is self evident that some one who deals with cardiac arrests daily in and day out should be better than one who takes an ACLS course. But that is just me perhaps. Great idea for a paper though. Again, B Paul and Yoram my trauma guys and Mike D- what do you have to say? Ken?

6) Internal jugular vein DVT is not as rare as you think and think of it if there is neck swelling. It is usually caused by cancer, CVP lines and patients who are receiving infertility therapy – the ovarian hyperstimulation syndrome. They commonly progress to PE and to post thrombotic syndrome, so be on the lookout (QJM 104(3)209). Actually this should be no surprise to you as an older article highlighted this syndrome as well as a cause of jugular
DVT ([JEM 37(1)29](#)). The lead author was a guy named Leibman. He's a hairdresser.

7) Kobi M this is for you. There is a member of the trauma team that will never give you grief. Never will scream, never will call you an idiot. (Gee can I marry a computer?) They studied computer use in traumas and found that when used in real time ([Arch Surg 146(2)218](#)) it reduced errors. Now don’t go crazy with using this idea- the computer – probably like a wayward GPS did call our errors – but these were to adherence to guidelines and protocols- and we all know what they are worth. Morbidity did go down from aspiration pneumonia, "errors" were less, and there was less morbidity from shock management, but the p values are not impressive at all, and only the use of blood products –which is rightly guideline driven –was lessened. I think there is a place for computers in the management of patients – but there still needs to be human input. Kobi M- do you agree? Even if you don't computers are invading another area of medicine- computer assisted personalized sedation. Gastro guys still are not licensed (?) able?) To give propofol for their wonderful procedures so they have a computer that measures vital signs, capnography and blood pressure and provides propofol in accordance to these. It lowers the rate of infusion if the signs take a turn for the negative. ([Gastroent Endo 73(3)423](#)). Betcha some singers and their doctors wished they had this machine.

8) Oh what is the best dressing for burns? We have discussed this many times- Silver Sulfadiazine is not to be used on the face and it does soothe but it is very expensive. Does it have any advantage? In my opinion no, but don’t take my word for it. In this Pakistani study, done on humans- the healing rate was faster by about three days when patients received honey dressings. ([Int Wound J 7(5)413](#)) Honey is an effective antiseptic. Aloe Vera also has antiseptic properties and while it may cause a local allergic dermatitis- it worked in this Iranian study with healing taking the same amount of days honey. ([Surg Today 39(7) 587](#)) True these
studies appeared in questionable journals with questionable methods but at least they were done on humans and these results have been reproduced elsewhere. Biafine- very popular in Europe-works as well as Silver but it shows no clear advantage and while this study was good, and in a good journal it is a pig study (AEM 11(4)339) (In Israel no less- where are those pigs coming from?) I like regular Vaseline to keep the place moist.

9) DO NOT SKIP THIS PARAGRAPH. It is not about critical care. Two months ago we brought an article from CCM that praised Hydroxyethyl starch known as Hetastarch). It stated it had an excellent safety profile; use resulted in less catecholamine use and less overall fluid requirements. (CCM 38(11) 2260) But see Analg Anest 112(3)507. Apparently a doctor by the name of Joachim Boldt printed a positive paper on hetastarch that only had 50 patients- not a real impressive endeavor, but when reviewing the article for publication it was found that the data was found to be falsified. Fair enough, the journal printed a retraction and a little study like this is not likely to turn too many heads. However, checking the ISI database, it was discovered that 85 of Dr. Boldt's articles dealt with volume therapy and 30 of these studies and 2 of his reviews have been incorporated into recent meta analyses and reviews. All the studies included made up data. Now it is true that most guidelines, including Cochrane still urge caution with this drug, we see from the article in Critical Care Medicine that this may have influenced them. In their defense they claimed that the newer heta starch was safer, but a closer look shows that the new product had been studied in 56 RCTs and one third of these were by Boldt and the remaining had serious methods problems.

10) I can't say I learned a lot from this review of the workup and treatment of diarrhea (CMAJ 183(3)339) but I will just present what might be new information. Testing is rarely necessarily in the outpatient setting or the ED in patients without bleeding, or high fever. WHO oral rehydration fluid is great and BRAT less so, but do not recommend sports replacement drinks because they have too few electrolytes for replacement (same could be same for mineral
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waters), and they have too many carbohydrates. Pediatricians still recommend not using sugary drinks (Ilene Claudius Oct EM RAP).

Careful with antibiotics. They may be problematic in E. Coli O:157 and while they did shorten non bloody diarrhea by two days, they also had more side effects, and more problems of resistance in the community. Cryptosporidium can spread by swimming pools for two weeks after symptoms resolve. Could speak about this subject some more, but I gotta run.

11) A really great little article with absolutely no evidence. I would have used it for an essay but last month the essay wasn’t clinical, so I can not do it two months in a row. The article dealt with treating VIPs—anywhere from royalty to the hospital administrator’s son’s little league coach. They start by emphasizing the principles of treatment—aptly abbreviated as VIP- Vow to value your skills and judgments—especially as they will want to bring in all sorts of experts, I is for intention to command the medical aspects of the situation – stay in control and leave the press and other paparazzi for some one else to deal with and P is for practice medicine the same way for all patients. Now the principles that he presents. Don’t bend the rules. They are usually designed for the best operations of the system to provide for good medical care. They bring the example of Eleanor Roosevelt who was tragically misdiagnosed because they wanted to spare her the pain of a bone marrow aspirate. Principle number two is to work as a team even when all sorts of consultants are convinced they know better. This leads to the third principle—communication. Not just with consultants but with security, press secretaries and people not related to the patient. Resist Chairperson’s Syndrome where a senior physician or medical director may wish to take over—this should only be allowed if indeed he is the most experienced clinician for the type of illness being dealt with. Be careful about where care should be delivered if it must be in the ICU and there is no privacy—do not consider making their present room into a mini ICU. Accepting gifts may be problematic—during the acute phase it
may be seen as a bribe of sorts, but declining may be a problem depending on the culture of the VIP. (Cleve Cli 78(2)90)

12) OK, enough playing around- what was the diagnosis above in number 2? Eczema Herpeticum. Now the more important question- who cares? The answer is- like with herpetic whitlow and this- if you do not start acyclovir the patient won't get better. If you start steroids, they will do worse. I am not a dermatologist so I cannot tell you why this wasn’t the other choices, but the point is with vesicles- be on the lookout for herpes. (JEM 40(2)167) In case you are wondering here is herpetic whitlow

13) Really nothing new here -- and that is the key. They compared men and women undergoing PCI in Italy and indeed, female patients were older on average by seven years. Nothing new here. They did about the same after PCI- also nothing unexpected here. But in this study they also less likely to get drug eluting stents even though they had more risks that would dictate them getting these types of stents. That is also not new and that is the problem after all these years, women get inferior care. (AJC 107(5)651). Well,
this may be true. The question is can we identify why they did not get the drug eluting stents? In the past they did not put drug eluting stents in patients with acute MI and thrombus, but the science isn't great for this and it isn't clear in this study who had ACS and who had an MI. And then, also one must ask if drug eluting stents are really better, although it makes sense. If it is true though, we are still making the same mistakes. By the way, family docs- in QI studies females are less likely to get the proper therapies as outpatients as well

14) I have deliberately ignored the issue of CT for coronary artery disease in the ED because I did not think it had proven itself yet. This article seems to imply that this is a test with good sensitivity but there is always the problem of calcifications that may confuse the picture. Here they took a verified clinical suspicion scale and applied CT to it. It worked best in intermediate risk but caused unnecessary PCI in low and high risk patients. (Arch CV Dise 104(1)29). I liked the idea of using clinical suspicion but one thing I don't understand. It could be that if there is a low suspicion and the CT was falsely positive that it led to unnecessary PCI. But how did it lead to unnecessary PCI in high risk patients if positive- or even negative? Chris says: Is the study saying that some high risk patients don’t actually have significant coronary artery disease, but these patients tend to have positive CTs? This would lead to ‘unnecessary PCIs’. The other cause for ‘strategy failure’ would be a major adverse cardiac event in a high risk patient for whom the CT was negative. But I would say with regards to the first answer: that that can not be called unnecessary PCI. But I agree on the second point

15) I know non clinical studies are also important part of our practice but many find these studies somewhat nebulous so let's combine two here. They followed medical students 10 years through residency to practice. Their goal was to determine what psychological factors led to success in the medical field. Academic advancement was related to competence (no, wise guy, it meant they were more competent not less so) but not to satisfaction in
life. Coherence was related (not sure what that was) but the influence of stress was variable. (Med Teach 33(3)e163) The big problem here is that this was done in Poland- in other systems it may be different. But I liked the idea for the study and think it should be repeated in different countries. Another quickie- this author feels that patients would be better served if we taught our residents safety by going over closed claims cases. Rick B- and Greg- run with this! (Acad Med 86(3)282)

16) Peds gastroenteritis very often needs no special ED treatment, so why not just educate the parents and that way they will know how to deal with this at home? It sounds like a great idea, but after one and six months they caregivers had no idea what they were taught and indeed – ED visits from gastroenteritis in the education group increased. (AJEM 29(3)271) Now probably the problem was that the education program was way over their heads, and probably just made them worry more, but that isn't to say that a proper program might work. And that is what you should take from this study- aim low and you will have more success. Maybe. Let us recall what Einstein said- there are only two things in the world that are infinite- one is the universe and the other is human stupidity and I 'm not certain about the former

17) This isn't the first article on the subject, but it is so futuristic I thought I had to mention it. TPA is still controversial in strokes (you can see my problems with TPA from my article in IJEM Feb 2005), but now they are doing catheterization of the brain vessels and ultrasound can help here. Ultrasound waves can cause negative pressure waves which enhance the fibrinolytic activity of TPA. It also cause more bleeding, but this is controlled by using micobubbles (Exp Rev Nruother 11(2)265). Sometime in the future someone may mention this and you'll say I saw it in EMU, and they will think your nuts but that is OK, I know you aren't.

18) The subject and the hypothesis are interesting and the second author Marianne Gausche Hill is one of the big names in Peds EM, but I have some reservations about the study. The hypothesis was that pediatric epididymitis doesn’t require antibiotics. Adult
epididymitis is usually sexually transmitted or from UTIs (reflux) - and so they checked for STDs and for UTIs and so they concluded that these were not bacterial in origin if the urine culture was normal. (PEC 27(3) 174). However the study was retrospective going by ICD codes which could have missed quite a few. Furthermore very few patients underwent epididymitis aspirate cultures, and a few did not have ultrasounds so there could be some doubt as to the diagnosis and in addition, it isn’t clear to me that this covered all possible bacterial causes. After all, an obstruction can cause a secondary infection that may not appear on a urine or STD. We have a few USC readers – what do they have to say? At least the question has been asked – perhaps it is correct, but I would be cautious about not using antibiotics in this sensitive area.

19) If you are a very old physician or nurse you for sure know about hypodermoclysis- when they used to inject fluid subcutaneously instead of giving IV. This went out of vogue but it is coming back for kids without easily found access. Alternatives that we have today are the intraosseous route, the nasogastric route, the rectal route, the intraperitoneal route, and the subcutaneous route. They searched for articles in databases and found the following: Of course it goes without saying that the oral route is the best. IO is equal to IV for rehydration purposes.Rectal rehydration was not studied at all and all the others have very poor data (Peds 127(3)e748) There is some disagreement on this, Ilene Claudius (who is from USC) (and is married to an Israeli) was recorded on EM RAP and quoting a study that NG rehydration was effective at 50 ml/kg per three hours and was well tolerated- although the well tolerated part seems hard to me. In her facility they do give fluids subcutaneously. She places a 24 gauge IV catheter in the subcutaneous tissue and instills 150 units of hyaluronidase and then gives IV fluids at a maximum rate of 20cc/kg/hr. The hyaluronidase aids in absorption of fluids from the subcutaneous tissue. If this is true, it could be pretty helpful although the 24 gauge IV seems pretty scary. If you do get in an IV remember the
typical rehydration rate is 20 ml/kg/hr up to 60ml/kg if you need to give it faster. Here is Chris again- like his comments: At Princess Margaret Hospital in Perth the standard protocol for rapid rehydration in children not tolerating orals is 50mL/kg over 4 hours – this is well tolerated in my experience.

20) This just scratches the surface of a critically important subject. Medical errors are inherent in our practice. Why do they happen? Is it due to uninformed patients, or doctors who are too overworked or saddled with unnecessary paperwork to provide enough patient care? What is interesting is that medical residents on average work more than 28 hours a week more in the USA than in the EU. (Lancet 377(9774)1289) I believe this is a system problem however the legal and licensing systems have found it more convenient to punish physicians instead of realizing this point. If you have never erred in your practice than you are truly great. And a liar as well.

21) In the USA there is now a fellowship for palliative medicine. So I want to spend a minute on the subject. And indeed while this was in their journal, it was from the EM program at Mount Sinai. Patients with end stage illness present to the ED when they have lost control of their symptoms. Despite this, they want to die among friends and family and express strong faith in G-d. They do not want to be burdens to their family but still want to die at home (J Pall Medicine 14(3)293). We really need to take these in to consideration and be sympathetic to these concerns. True there were only 13 patients and mostly Spanish, but we should keep an eye on this subject and I will do my best to bring more information as it comes out in journals.

22) Before we turn to this month’s letters, one last article from CMAJ 183(3)e195 on the subject of non invasive ventilation To summarize their findings- and the evidence was graded-COPD: use it for severe exacerbations- but just BiPAP; CPAP has never been proven. Heliox has no role. In asthma surprisingly, no evidence for BiPAP or CPAP being beneficial. Pulmonary edema: both work well. No info in acute lung injury, or chest trauma. Immunosuppressed
patients with acute respiratory failure – BiPAP yes CPAP no info. BiPAP will help get patients off of mechanical ventilation and keep them off. No word on use in pulmonary embolism. There is no mention in this paper on BiPAP efficacy in carbon dioxide narcosis which has been proven. Now if you have never used these modalities – here is a short tutorial. Pressure does improve ventilation but it can lower blood pressure- a perfect tandem in pulmonary edema for example. Use 7.5 CPAP as a starting point for most patients; I rarely need to go over 12.5 .BiPAP – usually start at 8/3 (the first digit is IPAP the second EPAP) and keep IPAP 5 above EPAP. If the patient has hypoxemia EPAP and IPAP should go up in 2 cm H2O increments. Hypercarbia- increase IPAP in 2 cm increments. Most of all – get a user friendly machine- you do not need all the bells and whistles.

23) A letter to the editor from Nurse Sherri who had some good points about nursing ratios- which of course can not take in to account acuity. There is another interesting way of dealing with this problem in a similar journal which was beyond me, but may interest those who want to analyze this subject more in depth See BMJ Qual Safety 20(1)15.

24) Alex S. writes again on the subject of superficial head trauma in those taking Coumadin and states that in Tel Hashomer- a large teaching hospital in Ramat Gan- the admit all head injured patients taking Coumadin since bleeds are typically slow – just like we said last month. I can not say I am in favor of this idea- it is exposing many elderly patients to infections when the need for admission is questionable- I think if the family is responsible- send them home. Alex also took me to task on the new dronedarone. But more on that later. Thanks for writing.

25) And a last letter from Chris, who is a EP in Perth Australia, who had some other ideas about my clinical challenge last month- Wallenberg’s Syndrome Also, I’ve just re-read the Nov issue neuro case. I don’t think it is a classic description of Wallenberg syndrome. Classically, there should be contra-lateral loss of pain and temperature sensation in the limbs and ipsilateral loss of pain...
and temperature sensation in the face (as well as ipsilateral Horner's and limb ataxia). Facial weakness suggests facial nerve involvement - CN7 is situated more medially, just lateral to the CN6 nucleus. Involvement of CN7 is more suggestive of a hemi-brainstem lesion, but it does follow a loopy course and "lateromedially" situated. However, I know some purists reserve Wallenberg's for dorsolateral medullary infarction and don't include CN7/ facial weakness as part it.

BTW the best (simple)description I've seen for localising brainstem lesions is Gate's rules of four - I have a few blogposts on it:  
http://lifeinthefastlane.com/2011/06/the-rule-of-4-of-the-brainstem/  
http://lifeinthefastlane.com/2009/05/helpful-brainstem-figures/  
http://lifeinthefastlane.com/2009/05/using-the-brainstem-1/  

Anyway, loving the EMU  
I stand corrected.

26) And just a message on EMU getting around. We have been featured on Rick Bukata's EM-Blog- now defunct but EMU can now also be found on the EM Central and Life in the Fast Lane sites. On the latter, Chris tells us:  
We have a project on Life in the Fast Lane that could use your reader’s input.  

It's called R&R in the FASTLANE and involves a team of expert emergency medicine and critical docs from around the world recommending papers to read (to read, not just know about!). Contributors are allowed to suggest up to 3 papers a week. It is kind of eminence meets evidence for the ADHD doctor... There is a distinct absence of contributors from your neck of the woods.  

Also, intermittent/ occasional contributions are welcome (ideally just
want papers that people should actually be reading - they read EMU to find out about all the latest cutting edge stuff)

This is how the latest R&R looks (the first edition got 1500 page views in the first week): http://lifeinthefastlane.com/2011/11/rr-in-the-fastlane-002/

These are the contributors so far http://lifeinthefastlane.com/education/rr-in-the-fastlane/
This is a worthy project-I would encourage you all to participate. EMU is also working on a website of its own.

**EMU LOOKS AT: Migraine and Axillary Rain**

We have many times spoken about migraines, and we present extracts this month from three articles on the subject-what makes this time different is we will look at complementary therapies.

**MIGRAINE**

1) We will not discuss the clinical presentation of migraine nor the pathophysiology, although they claim to be pretty close to defining the pathophysiology (some egghead thing with neurotransmitters) and as such some really far out experimental drugs are now in trials and if it really interests you (and you qualify as an egghead) see [Curr Treat Options Neuro 13(1)1](#)

2) Treatment –see [ibid 13(1)15](#). The principles are the following- start early- it is easier to abort a migraine early on. Medication overuse can lead to refractory headaches so migrainers have to be careful about overuse. Have them be selective as to which headaches they will treat – allowing minor ones to subside on their own.

3) NSAIDS and Tritpans are proven to work. NSAIDS work on the inflammatory cascade so are very effective here. Often our patients have tried them already before coming to us. Naproxen has a slight
advantage in having a longer half life than ibuprofen- the former 12-15 hours the latter 2 hours.

4) Triptans work great, but should be used early. There are few differences between the many drugs in this class and we will go over them below. These by and large are safe drugs but there may be some minor side effects like chest pressure, myalgias, fatigue, flushing, or asthenia. Contraindications include cardiac, cerebral or peripheral vascular disease. They do increase blood pressure. They should not be given in basilar or hemiplegic migraine. Serotonin syndrome is a concern when used with SSRIs and MAO inhibitors, but there is little evidence at this point.

5) Sumatriptan has the advantages of be able to be given intranasally, by injection by pill form and with naproxen in one pill. Naratriptan has no MAO or P450 interactions but can not be given in renal impairment. Frovatriptan has the longest half life of any triptan- 26 hours. Almotriptan has also no MAO interaction. That is about all there is in differences between these meds.

6) Want a great therapy? Try aspirin 1 gram. One study showed good results- about as good as NSAIDS- which makes sense.

7) Butalbital (a barbiturate) was popular in the past for migraines- this med has been banned in most parts of the world because of ease of developing dependence. Opiates are discouraged but are still the most widely prescribed drugs for acute attacks. The theory is that they are vasodilators, pro inflammatory and maybe pro nociceptive. Caffeine is not discussed although it is included in many medications, I am not sure why- the last thing I want when I have a migraine is to be awake.

8) Other options were also not included. The article seems to feel ergotamines are no longer indicated. Steroids are often used- I have seen articles in CMAJ that were very enthusiastic. One article in Headache showed that propofol helped. High flow oxygen is used by many. Droperidol is supposed to be the best therapy, but it is black boxed in the USA and not available in many parts of the world. Haloperidol seems to be effective if you do not have this- see Headache 2006. An Israeli study used Valproic acid.
IV with remarkable results, but it was open labeled and took a full 50 minutes to work. They needed 900 - 1200 mg. See Acta Neuro Scand 123(4)257

9) Here is Chris’ take on steroids:
10) Role for steroids is primarily meant to be in reducing recurrence, as backed by meta-analyses like http://www.ncbi.nlm.nih.gov/pubmed/18541610 but only the 1999 was actually statistically significant. A more recent DB-RCT found no effect: http://www.ncbi.nlm.nih.gov/pubmed/18272089

11) Now let’s speak about non pharmacologic treatments, from this article (Curr Treat Options "Neuro 13:28) which has headache guru Rich Lipton as one of the authors. Foods are considered a trigger to migraines but the evidence is actually sparse. Those often implicated are MSG, aspartame, nitrates, red wine and caffeine (more than 200 mg a day). Chocolate and tyramine have even less evidence to support their role in causing headaches. Lifestyle causes include stress irregular meals and lack of sleep. Exercise tends to be protective. Menstruation can also be a trigger. Most migraners have identified their triggers.

12) Relaxation therapy shows that about 50% of people have a 50% or more reduction in head ache frequency

13) Biofeedback is “the digital capture of physiological processes which are converted back into a medium that is fed back to the patient” I have no idea what that means. Whatever it means it is done by EMG feedback, hand warming feedback and blood pressure feedback where they learn to contract blood flow to the temporal artery. They are enthusiastic about this therapy as it works well against depression and anxiety- results are similar to relaxation therapy.

14) Cognitive behavioral therapy which is done with a therapist or psychologist works as well as the above. They identify what stressors cause the headaches and how to manage these stressors.

15) Pharmacological therapy is better than behavioral therapy over the short term, but once patients learn how to use behavioral
therapy- they equalize in efficacy. One study however showed amazing results using combination drug- behavioral therapy.

16) Complementary therapies have been plagued by remarkably poor studies. It escapes me why practitioners are unwilling to do RCTs like other accepted therapies. Acupuncture is probably the best hope, but there is publication bias- only positive studies are published. So we cannot really know.

17) Spinal manipulation: Case studies and small studies are the rule here- so there is no strong science that this works at all. PT/OT has similar evidence problems,

18) Massage does lower anxiety, hear t rate and cortisol levels- but again evidence is lacking. This may depend on who is doing it.

19) Yoga actually has one controlled trial that showed benefit. On the other hand homeopathy has failed in many trials to show benefit Reflexology which is related to reflex zones in the foot showed possible benefit in one study but it was uncontrolled. Seems most CAM is not first line.

20) Natural medicines actually have somewhat stronger evidence but still not RCTs that would give us a clear answer. Butterbur rhizome is somewhat effective in doses of 100- 150 mg a day in divided dosages.

21) Vitamin B2 at 200 mg twice a day and coenzyme Q10 have some benefit as well. Q10 has one RCT. The article states that most therapies are safe although I do not know how we know this with the paucity of data

22) Not mentioned in the article is a therapy we mentioned in the past- greater occipital nerve block (Curr Pain Headache Reports 2007). Some neurologist I spoke to say it helps. But like all the above - it isn’t clear how these work and if they work and there may be a significant placebo effect.

Hidradenitis Suppurativa

1) I can’t really understand how this sentinel article was published only in Curr Opin Infect Dis 24:118. For those with this problem it isn’t funny, and it would be helpful to give these patients hope.
The disease presentation is pretty typical—painful nodules, abscesses, draining sinuses, draining sinus typically in skin fold regions. Differential diagnosis included infected epidermal cysts (what we called sebaceous cyst), Bartholin’s gland abscess, nodular acne, and cutaneous Crohn’s disease. This is located in the area of apocrine glands. However they are probably not involved. It seems this probably starts with follicular inflammation and keratin plugging. Bacterial infection is secondary and not the cause. And indeed often the cultures are sterile and antibiotics have variable response rates.

2) High BMI and smoking seem to be involved— and these factors make it worse. Pregnancy seems to improve the situation and premenstruation and oral contraceptives seem to make things worse. Anti androgen therapy seems to work proving a hormonally association as well.

3) Whatever the cause, it is icky and so treatment is desired, even though the disease is usually harmless. Antibiotic creams such as clindamycin, fusidic acid and the like have an effect in mild disease. If there a few lesions, the result of injected the lesions with 2-5mg of triamcinolone is not that painful and does help the pain rather quickly.

4) By mouth, tetracyclines are often used but results have been disappointing. Clinda and rifampin by mouth had impressive results- but only in some patients.

5) Females seem to benefit from estrogen therapy, or finastride (helped their BPH too!) or spironolactone. Isotretinoin is ineffective. Dapsone and cotico steroids seem to be effective. TNF blocking agents have worked in a single case report.

6) Surgical options: incision and drainage usually is not successful as nodules do not drain and often there is scarring and recurrence. Wide excision surgery has the best results but the science is lacking. Laser treatment may be less invasive with similar success.

7) I must say this article makes this clearer to me but there are still too many question marks as to what works. What doesn’t work though- is now clearer to me. How about you?
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17) That is it, friends- 13 years publishing monthly. I hope you have enjoyed it as much as I have.
Now our bonus essay from Dec 2006

EMU LOOKS AT: *Diabetes Type II*

What could I possibly tell you that you do not know already? Firstly, not everyone is an internist. Secondly many new drugs have come out since many of you have trained. The purpose of this essay is not to treat diabetes but rather to understand what patients are taking when they come to the ED and also to start or correct therapy when these patients are poorly controlled. The source for this article is the ADA Consensus Statement in Diabetes Care, Aug 06, and the NEJM, 2 Nov 06. The focus is on the medications, not the theory.

1) Fasting glucose is a good guide, but nowadays, Hemoglobin a-1-c is the standard. Remember the number <7% - this is where your HgB1-a-c should be and perhaps even <6%. Fasting glucose measured a
few times during the space of a week can correlate well with A1C. For following glucose, fasting and preprandial are the best measures
2) We still recommend lifestyle adjustments to lower A1C. Surprisingly, according to this guideline, they can lower A1C as well as insulin. In those who have had surgery for weight reduction, for example, many have seen their diabetes disappear completely. Many people see benefit even with a loss of as little as 4 kg. However, these measures often fail within the first year.
3) Step one with medications is Metformin (Glucophage). This drug is cheap, easy to take, decreases hepatic glucose output and lowers fasting glycemia. Rarely causes hypoglycemia or weight gain. Lactic acid in folks with kidney problems is the scary side effect, but it is rare.

3) Step two is sulfonylureas. We are well beyond the days of Diabenase and Tolinase (in Israel Orsinon) but the second generations are dependable and cheap. They cause hypoglycemia, though, and weight gain which results in perhaps worse cardiovascular mortality. Overdose is treacherous, and of course even in non overdose cases, hypoglycemic events need admission due to the long half life of these drugs. These drugs work by enhancing insulin release In 2011 I rarely use these

5) Step two could also include TZDs. These drugs are insulin sensitizers. These drugs work more modestly in bringing down A1C, somewhere between .5 and 1.4%. They cause fluid retention, pedal edema, and weight gain. They do however, improve lipid profiles. The prototype for this group is rosiglitazone, also known as Avandia- (now in 2011: off the market).

5) After step two, consider other drugs, although you must give thought to going straight to insulin. Glinides work like the sulfonylureas, but bind to different sites. They have a much shorter half life. Seems they cause less hypoglycemia. Repaglinide is the most effective in the group (Novonorm in Israel), but it is very expensive.
6) Alpha Glucosidase inhibitors reduce digestion of polysaccharides in the small intestine, they reduce A1C about .5-.8%. They do not cause malabsorption, but they will cause a lot of gas and discomfort, as more carbohydrate is delivered to the colon. Almost 50% of the patients taking this do not continue therapy. The prototype is Prandase.

7) Glucagon like peptide agonists (exenatide) stimulate insulin release, and are a naturally occurring hormone. They lower postprandial spikes, prevent glucagon release, and lower A1C modestly. Vomiting and diarrhea are very common. It is unavailable in the USA. Pramlintide is a Amylin agonist works similar to and has the same side effects as exanatide. I could not find this drug in the Israeli list of medications (MEDIC).

8) Insulin. This is very effective of course, but you need larger doses in type II to overcome insulin resistance. HDL will go up, triglyceride will go down, but there will be weight gain. New non peaking insulins make hypoglycemia unlikely, and inhaled insulin is the newest rage, but whether it can lower A1C to less than 7% remains to be seen. Recall if your patient is taking Lantus (a long acting non peaking insulin) you need to admit for observation also if they do have hypoglycemia. If is insulin does not work completely, you may increase it or add metformin or a TZD. In sick hospitalized patients, avoid sliding scales- give a small basal rate and add a regular insulin before meals.

9) I never understood why people send patients to the ED with high sugars who have no ketones on urine tests and are not in a hyperosmolar state, and this guideline does not understand why these patients are hospitalized.