Wow 2012 already- how time flies? - Which means it is time to quote Groucho Marx. "Time flies like an arrow. Fruit flies like a banana." Didn't like that one? How about" Marry me and I'll never look at another horse again". (that was Groucho too) All seriousness aside, however, this article that I am about to present was designed for testing residents but it the tables are a good basis for a practice that is not common in many countries. Triage levels are important for us to organize our work. This allows us to also sort the patients out to determine who needs a higher level of care that may need an EM specialist instead of the intern seeing them. If your ED is already doing this, fine- but there are many ways how to do this and I would welcome to hear the methods you use in your ED. (AEM 18(3)E8)

Small study and it studied kids between the ages of 11 to 17 years – which is a big range in age, but the subject is very big now. Many of kids who play contact sports can get knocked out and we are often asked who can be allowed to return to the game The best tests include verbal memory, processing speed and reaction time which when done at the time of injury most correlates with the improvement scores that will be seen two weeks down the line (AEM 18(3)256). What is the current teaching now is not to let them go back to the game so fast, so in your clinic or in the ED- do not give recommendations for when the athlete can return to sports unless you will be following up with him and can do these tests.
EMERGENCY MEDICINE UPDATE

3) The classic teaching is that subsegmental pulmonary embolism is less likely to cause trouble. Actually, someone has actually looked at this in Korea. They found that these were generally safe with no mortality in their study, and with much less oxygenation and hemodynamic instability issues. (Respir 80(6)500). There are problems here. Many of the patients that had subsegmental embolisms received anticoagulant therapy as treatment and perhaps that is why they did well. There were only 334 patients, and they used three types of imaging- pulmonary angio (anyone still doing that?), CT and indirect CT venography which I am not sure what that is. In any case we know that CT misses many subsegmental PEs, so we can't really be sure about the benign nature of these PEs since we do not know who had them and went home since the CT was normal. The big question- is this a risk for a bigger PE down the line or are subsegmental PEs just a normal everyday occurrence that we all may experience and has no clinical relevance.

4) This is perhaps one of the worst journals I screen but it is hard to screw up when you are meta analyzing a well known concern. Since however, may be some new readers (EMU averages new ones almost every week) I will mention it. The use of acetaminophen (paracetomol) seems to increase the risk of childhood asthma. (Clin Exper All 41(4)482). However, there were only six studies and their quality was not evaluated. Furthermore the risk ration is only 1.21
which is only a slightly increased risk. Since we believe that this is the most acceptable drug in pregnancy- exercise some caution.

5) Here is a bone for my EMS readers (I hope to dedicate a EMU roundtable to you guys- will welcome your questions). But please read the whole paragraph. They did trans cranial dopplers on 18 head injured patients to help stratify the patients (Acta Anaes Scand 55(4)422) Not clear if this improved care or mortality, but in a long transit time this could help. Now this was a French study and their system is much different – they have specialized hospitals- ones for heart, ones for lungs etc, and docs often are in the ambulances, specifically anesthesiologists. However, in Israel, and some other countries physicians do ride the ambulances but they are basically unable to perform procedures that the paramedics are unable to. Furthermore they are usually poorly trained. Should we go to the USA system where physician accompaniment is rare?

6) Resuscitation news- They compared two self teaching shorter versions of BLS with the traditional course and found that the students did just as well if they took the self teaching course. (Resuc 82(3)319). Now these were medical students and the way they established equality of skills seemed to be asinine, but if you need to have badge courses (and I am against them for emergency physicians) this way of teaching would expose more people to this critical skill. Indeed youtube could be a great source as well (ibid 332) but the quality of these videos were highly variable.
7) Hi, ICU guys- you are probably are making fun of me for something you have known for quite a while, but statins have anti inflammatory effects (I knew that), anti oxidant effects (can't see why that would help) immunomodulatory effects (depends what those are) and anti apoptotic effects (no idea what that is) and they think it will work well for septic shock. (Eur J Int Med 22(2)125) The evidence though in this review is kind of sketchy. They are now looking into this also for pretreatment before PTCA. (Circ 123(15)1622) This was also a meta analysis but I do not know how much was confounded by the fact that so many patients undergoing PTCA are taking statins anyhow.

8) Trauma guys laugh it me a lot as well, but please reconsider pancraniography – in kids at least and in everyone as well. While a standard chest film is only .05 milli Sieverts; kids in this study got on average 3 scans per patient and received 17.43 milli Sieverts. That is about 350 chest films (J Trauma 70(3)724). Considering that little kids get more radiation distributed over smaller surface areas, this could be devastating. They found these dosages to be in the range for increased chances for solid cancers, thyroid cancer and leukemia.

9) I always welcome good evidence, and looking at manipulation therapy for treatment of radiculopathy they found that there is moderate evidence it works for lumbar radiculopathy, but only if it is acute. For chronic lumbar and cervical radiculopathy there is no evidence. (Phys Med Rehab Clin No Amer 22(1)105)
10) Ken I.- sit down before you read this. They polled academic physicians and found they on average see one death a month in the ED. The overwhelming majority received little or no training on how to cope with this. Debriefing occurred almost never and many reported insomnia and fatigue as well as sadness and disappointment after witnessing deaths. Common coping mechanisms included talking with colleagues and – you guessed it- continuing to work. (AEM 18(3)255)
It seems that powerful experiences with death – like an unexpected one- teach physicians a lot on coping (Acad Med 80(7)648). I for one wish I was given more training in this- I still- after 26 years in the business do not know how to do this well. Ken I, Mike D- would you have some pointers for our readers?

11) An interesting perspective on ultrasound written by a residency director. Often we feel befuddled when we see our residents whizzing by with the machine and we do not even know where the on off button is. There are many relevant questions that could be asked on this technology which has truly changed EM practice. What about places that do not have access to this technology in the ED? What about studies we do but are not ready to take full responsibility for (like Doppler of the lower extremity?) And what to do when the technology is unavailable- we can put in a CVP blindly but can our residents who have never done it without ultrasound? (AEM 18(3)309)
I think we need to embrace the technology and make sure everyone is well trained in this—even if it is by learning from radiologists or our own
residents. We need to assure competency in the method as well. We can no longer stand against this and we cannot ignore it either. So be honest- how many of you are as good with the probe as you are with the ET tube? (and are over the age of 45)

You do not have a lot of time with the acute scrotum and the six hours often quoted is ischemic time on dog testicles- not overly relevant to humans. There are a lot of scrotums in China (I bet you didn't know that- see- it is good you get EMU) and when they studied this they found that there was a lot of overlap in the signs of an acute scrotum meaning that physical exam is not reliable and ultrasound was also not reliable. They concluded that one must have a low threshold for exploration in these cases. (PEC 27(4)270). They looked at scrotums in the UK (where by the way the have a lot less scrotums) and found that torsion was the most common finding, but only barely –that is 51%. They therefore recommend exploration for everyone with an acute scrotum but they ignore that in 49% of the cases the surgery was unnecessary (and painful I assume) ( BJU Int 107(6)990). I think the solution must be that someone must invent a laparascope for the scrotum. Two other points: Firstly both studies took over 18 and ten years respectively-but it took that long to get enough patients. The UK study by the way found that age didn't help either to rule out torsion. Hot Flash: (pun intended) I just spoke to one of our senior urologists who corrected my misconceptions- a scrotal exploration involves an
incision of 1 cm and is a short procedure that is painless. So if in
doubt get that urologist in and let him take a look.

13) This has got to help someone somewhere. None of these drugs really
work, but there may be some family guys that swear by them so let's
go. Transplant patients can not take all OTC cough and cold preps.
Diphenhydramine has anticholinergic properties that can directly
affect even a denervated heart. It also interacts with cyclosporine.
Dextromethorphan is OK, except in liver transplant patients. Guaifensin
is OK in transplants patients except those with kidney or lung
transplants or renal impairment. Codiene is OK for all transplant
patients except those with renal impairment. Now I know you all saw
this article already- for sure Alex S saw it in Up to Date, but there may
one person out there that does not get this journal so I brought it. (Progress Transplant 21(1) 6)

14) The talk of the town- everyone is very interested in Ian Stiell's newest
project- the rapid discharge of patients with AF who –per his protocol-
get a one hour drip of procainamide and then electrical shock if it
doesn't work. Some of the positives from this article that I really like-
procainamide is a good drug and I am glad that amiodarone is not the
Messiah any more- it really doesn't work that well. Furthermore, he
advances that rate control is not necessary and in an interview he said
that it may even make conversion harder- though I have no evidence
for this supposition. (CJEM 12(3)181). Now the bad part. I spoke with
Dr. Odaya who reviewed this article for a presentation in our journal
club, and I would like to present her comments verbatim. "The study was a cohort study with no placebo group, which is not a bad thing in this case, but a comparison group with electrical cardioversion alone or versus amiodarone for example would have been nice. Many of these patients presented more than once, so actually more patients presented more than once than those who presented only once. So it could be that the patients that it helped re-presented and if there sample size was greater they would have found more who did not respond. Looking carefully on the charts in the study, 120 patients received anti arrhythmics before coming (including 8 who were taking procainamide already) and great amounts were taking rate controlling meds- which could have influenced the results.) Other questions I asked the charts in the article showed that patients received 25 -2000 mg of procainamide despite the protocol being for 1 gm- could it be on the low side these patients converted spontaneously? And why were A flutter patients in the ED for an average of 6 hours? I heard Ian speak in an interview on EM RAP and he admitted that electrical conversion is also just fine, and I find it helps us discharge our patients must faster. Ian did admit no American journal would accept this article- which I find odd. By the way, not to dis Dr. Stiell but Dr. Odaya is our intern. Bright kid, no?

15) This article describes on going research which will interest me only when the article is finally published, but the statistics were important. Intracranial aneurysms occur in 0.4-0.6% of the general population;
the total risk of rupture may be about 2% for all aneurysms, but indeed if they are less than 7 mm in diameter, the risk is only 0.1%. This is important for us to know because often we do CT's for headaches, and find aneurysms that are not leaking—and it appears we do not need to do much for them. Surgical treatment for aneurysms cares a 1 in six morbidity rate and a mortality rate of 2.6% to 15.7%—probably a wide range due to location of the aneurysms. Coiling is much safer but has an incomplete occlusion rate in 40% and aneurysms recur in 34%. Risks for rupture include: diameter greater than 7 mm, posterior circulation location, small parent artery, smoking, and hypertension. (Can J Neuro Sci 38:191)

16) Cardiologists can be cantankerous group and they do not often agree with each other (or as Jay Leno says—9 out of ten doctors agree the other one is an idiot). And indeed the Canadians, the Americans and the Europeans all came out with their own guidelines for the treatment of Atrial fibrillation. All of these guidelines agreed to more lenient rate control (if you want this)—now 110 beats per minute are acceptable, but the Europeans and the Americans agree to this only with provisos whereas the Canadians are OK with this throughout. Rhythm control—the Canadians are the only ones that do not restrict the use of Sotalol and class IC(propafenone, flecanide, ibutilide) from those who have LVH. The Canadians also do not believe that the use of dronedarone is reasonable on the basis that it saves admissions for AF, although they agree that it can be used. The Americans strongly recommend the use
of ablation for a fib that fails anti arrhythmic therapy. The Canadians are the only ones recommending dabigatran to prevent strokes as being superior to warfarin, but if you read EMU, you know that this is not true. In any case, it seems from the article that the Canadian approach is the most enlightened but I would have expected that being that this article is from the Can J of Cardio 27(1)7. While we are speaking about subjects close to my heart, EM RAP recently asked Amal Mattu if troponin elevation is pathologic in SVT. Dr. Mattu said no, but presented no evidence for this. Well there is an article that shows that is of no significance see ibid 105. Alas, wouldn't it be great if it was good evidence, but there were only 73 patients of whom only 24 had an increase in troponin and 19 of them underwent stress tests of which 2 were positive and one needed intervention- if you can trust stress tests which you can't. But I still do not measure troponin in SVT and think you shouldn't either. All though I do sometimes wonder why all the SVT patients I send home die. (only kidding- really)

17) Just what exactly are you bringing home with you? If you live in the UK you are probably undergoing mandatory screening to see if they can detect your pet MRSA. And as can be expected there is no or little literature with regards to whether routine screening is useful, what the prevalence of MRSA among health care workers is, how it is transmitted and what to do with the results of this screening. (J Hospital Inf 77(4)285). However this bug in the community so we
EMERGENCY MEDICINE UPDATE

could expand the screening to the clinics too. If we knew it made a difference- which we do not know.

18) I am certainly not a genius in this subject and other than Didi B don't know anyone who is, but if you took your boards you learned that the oximes – use with atropine of course- are the treatment for organophosphate poisoning. If you are uncertain as to what this medication is – in the USA it is Pralidoxime (2 PAM) and in Israel Toxiganon. Cochrane as usual says there is no proof it works (or that it doesn’t damage) but add that the organophosphate re adheres to the receptor after the oxime is done working in massive overdoes and doesn't work when dimethyl organophosphate presents in a late fashion. (Cochrane 5085:2011) Does this help anyone? Well, maybe. If your poisoned patient doesn't get better, so up the atropine and perhaps the Pralidoxime as well- if it is safe to do so- we don't know this either.

19) I have mentioned this before, but what can I say- it takes time for internists to clean their ears out. Also you FPs may see patients taking these drugs if they are pre dialysis patients. Sodium Polystyrene Sulfonate- also known as Kexylate in most of the world is used to lower potassium in the blood. It can given by mouth or by enema. It takes time to work so other therapies (calcium, Ventolin inhalations, insulin and glucose, loop diuretics) are often given first. This article says Kaexylate may take a very long time to work; in fact it may not work at all. Indeed this medication was introduced in
1958 when safety and effectiveness did not have to be proven. There have actually been no studies that have proven it works. Can it hurt? Well when given with sorbitol it causes colonic necrosis. (J Am Soc Nephrol 21(5)733). We do have other options after the acute care including dialysis and loop diuretics but I think many of us would believe from our experience that it does work- which leaves us with- when does it and when does it not? Only your hairdresser may know for sure.

20) Synthetic cannaboids are out there- they are legal in many countries, and they are available over the internet. They like bath salts are legal because they are labeled not for human consumption, but when smoked or ingested give a high. Toxicity data are limited. There are some case reports that this is not innocuous. One report after ingestion is seizures and SVT (Clin Tox 49(8)760). Hallucinations, hypertension and chest pain also occur but all resolve within 2 -4 hours. Are there long term effects? I know some folks in Oregon that would probably say no (Ann Pharm 4593414)

21) NSAIDS bashing again. (Maybe it is time I got a life, no?) The safest NSAID in cardiac disease remains naproxen- which we have mentioned in the past .However, there is still an increase of 30% MIs in those taking these naproxen- meaning other NSAIDS are even worse (Nature Rev Card 8(4)193). So you will say to me- Hey, you can't outsmart me, I read EMU-I will recommend they take aspirin for pain- which is fine if you are in the USA, but in many countries- Israel included – you can't
EMERGENCY MEDICINE UPDATE

get higher dose aspirin, only the 100 mg and you will get strange looks when you recommend that patients take 5 – 10 pills at a shot.

22) OK, so you are not an EP and you do not give propofol in general, but read on, because you may be in for a surprise. Propofol can sting when given IV about 60% of the time. Giving lidocaine IV or ketamine with it will reduce the pain. However the most efficacious way of preventing pain is by giving it in an antecubital vein as opposed to a hand vein. (BMJ Mar 2011) I always believed this anyway- where there is more muscle mass- it will hurt less. Need to take arterial blood gas? (Not sure why you would, but no matter)? Femoral probably hurts less than radial. While we are on the subject of sedation- we have two letters from last month as replies to the use of ketamine-

Lisa Amir from Schneider pediatric megapolis says:
After the update on ketamine use in Annals from Jan 2011 - we've gone exclusively to using ketamine without midazolam in kids. 1.5-2 mg/kg (little ones sometimes need more). Not only do we virtually never see emergence reactions, the duration of action for ketamine without midozolam is about 10 minutes. For most of our procedures this is long enough and we have the kids out the door about 60 minutes after completing the sedation. If the procedure is longer (suturing, e.g. ) we just give additional boluses of ketamine 0.5 mg/kg

Pinny Halpern from Tal Aviv’s Ichalov Hospital writes:
In our department we have using a midazolam-ketamine combination for many years, with thousands of satisfied patients and many very satisfied EPs and orthopods. In fact, this is the sole sedation method allowed orthopods. It is successful approx 95% of the time, so much so that I really don't find the need for propofol (which I love – as a former anesthetist). Emergence phenomena are so rare I can't think of the last time I saw one. Etomidate (+/- fentanyl) is my fall back drug when needed.

I think the etomidate idea is an interesting one- what advantage does it have over propofol? (it doesn’t hit the blood pressure as hard but
EMERGENCY MEDICINE UPDATE

these are usually young patients that can handle that). I have never seen an emergence reaction either. My problems with ketamine and midazolam are that I rarely get these patients out the door that fast. Thanks for your comments.

23) BETS- the EBM series from the EMJ – these folks asked: kids that can fully extend their elbow- does that rule significant injury? They claim that it doesn't but the studies are few and not of great quality, and besides, I would ask- what is considered a significant injury? (EMJ 28(4)334). Even more interesting is their BET number four where they ask how useful bowel sounds are. They claim they have a high specificity for obstruction but low sensitivity meaning hearing normal bowel sounds does not rule out obstruction. (EMJ 28(4)336) This conclusion is based on only one good paper, and in addition, there are many other signs of obstruction that make checking bowel signs less relevant. I personally never check for them, and I think this article brings into question the whole concept of what in the physical exam is really helpful. I am not trashing everything we do, but we do need to question – a little. Isn't that why you read EMU? (Or is it because you can't find a Reader's Digest in the smallest room of the house?)

EMU LOOKS AT: UTI and You're Making me cry

This months essays deal with two very different subjects, one which we return to often but those treating such patients in the trenches will probably thank me for these pointers. The sources for the first essay are Expert Opinion in Pharm. 12(6)865 and Journal of Pain 12(3s supp1) s14.
EMERGENCY MEDICINE UPDATE

1) Both of these articles deal with pain. However they deal with the two problem cases- the elderly and kids. We will not go over pain principles; if you are an EMU reader, you know we have gone over these enough. (By the way, the EMU web site is almost ready and will have a search function)

2) Acetaminophen or paracetomol for our Europeans is still listed in most guidelines as the first line for pain in the elderly. ALT can go up transiently and it is of no significance. inflammatory conditions respond less to this. The article doesn’t say if it is because there is less anti inflammatory effect with this or because it just is not a great pain reliever.

3) NSAIDs –can cause much more GI bleeding when taken with aspirin and most elderly folks are taking both- aside from the fact that NSAIDS also block the beneficial effects of aspirin. COX 2 also increases the GI bleeding rate, despite the advertising. Naproxen causes less Mls- see above in number 21. They like topical NSAIDS and so do I. Overall- avoid them as a class. They do not mention aspirin as a pain reliever in the elderly- why not?

4) Opiods: I do not have to bash propoxyphene any more because it is finally off the marker (Darvon and Darvocet in the USA, Algolysin in Israel). If you somehow find this- it is linked to falls; it has no pain relief damage over aspirin and has significant nervous system and CV effects. It makes a lot of the elderly constipated. Another bad drug which is rarely used anymore is
Pentazocine (Talwin) which causes confusion and agitation. Merperidine (penthidine) is often sacked because of neurotoxicity- but the risk is mostly in doses of greater than 600 mg/day for greater than 48 hours and there are those who do not believe it is clinically relevant. Other opioids seem to be well tolerated, even in long acting formulations- but in this article they did not look at parental formulations.

5) Adjuvant analgesics are recommended by the American Geriatric Association especially for neuropathic pain, but not TCAs, because of their anti cholinergic and cognitive side effects. They do not mention Lyrica. The adjuvants they do like are topical agents like lidocaine patch, capsaicin and NSAIDs, but advise to avoid benzos, muscle relaxants calcitonin for pain, and say the evidence is scant to recommend ketamine, chondrotin, cannaboids, and the like.

6) Now over to kids. The myths are well known and we won't spend much time on them. We do a lousy job in taking care of kid's pain, but then again the studies show we do a lousy job in everyone. Pain pathways are working from birth (and maybe before?) so kids do feel pain at infancy and remember it, so please quit doing those heel sticks, will you?

7) Paracetomol is used a lot in kids, but is not without its controversies. Most studies recommend 10 -15 mg/kg or 20 mg/kg found that these do not reach the levels in the blood to give pain relief (the boxes you buy in the store recommend 10-15 mg/kg).
There are those who have reported better efficacy by 40 mg/kg po and 60 mg/kg by rectum but if you are not so heroic, just know that 20-25mg/kg as a first dose is fine, and if you gave a little more, no big deal. They like IV formulations, but the expense has scared many away. There seems to be some people with a heightened susceptibility to toxicity and you cannot who has this, so you have to be careful- sometimes you just may not get to therapeutic levels. A Nitroactomenophin preparation is in trials and it doesn't cause hepatotoxicity.

8) Now NSAIDS- for kids. Studies have shown that Ibuprofen at 10 mg/kg was better than paracetomol 15 mg/kg and codeine 1 mg/kg- but the latter is rarely given because of side effects and we just discussed that with the former that 15 mg/kg may not be enough. You can combine both paracetomol and NSAIDS for better pain management. Asthma is always a concern with NSAID use in kids, this is still controversial. You can use NSAIDS after tonsillectomy and they do not increase bleeding.

9) Tramadol can be used. But it doesn't work for everyone- 5-10% of Caucasians, and 1% of Asians see no effect. Hard to know how to understand the studies that have shown Tramadol is less effective than NSAIDs and pethidine( meperidine) but better than IV Morphine because of industry sponsorship of these studies. Keep in mind seritonin syndrome with tramadol, but it does have the advantage of no effect on respiration.
10) Opiods: the salient points for opiod use in kids include many important points. By six months infants develop the ability to clear morphine to about 80% of the clearance rate of adults- so be careful with morphine in these younger infants. Their review of the literature found hydromorphone equivalent to fentanyl, and morphine but with much less side effects. Oxycodone was only slightly better than codeine. In any case, there is a nice table on how to dose fentanyl, and morphine in the neonate and infants. Last points on opioids- fentanyl can be nebulized at 3- 4 mcg/kg and works as well as IV, which I do not entirely believe because I know that nebulized morphine does not work however Fentanyl is so potent, it may work better. Remifenatnil is very short acting, but can cause bradycardia and hypotension, and I have little experience with it. Propofol can also cause hypotension but we know it is transient and of limited clinical relevance.

11) Ketamine is one of my favorite meds and we have said many times before that in dosages of 0.1 mg/kg you get analgesic effect without putting the patient out. There have been concerns about ketamine’s effect on developing brains – it does cause neuorapoptosis (what ever that is- I am sure Chris N knows what it is). But nothing has even been proven.

12) Nitrous oxide- known as laughing gas- I have used this and been disappointed. It has very weak analgesic effect- and the sedative effects are hard to achieve without getting to 70% nitrous concentration. They say that there is only 6% vomiting rate, but I
saw a lot more cases of vomiting with kids. Fasting doesn't help prevent this either.

13) Do not forget about sucrose and breast feeding for kids as this is a very effective manner of pain relief for kids.

UTI
UTI is a boring subject but we are looking at kids where workups can be extensive. I am not sure who needs a complete workup and what kind of workup, after this essay hopefully it will be clearer (but don't bet on it). The source of the essay is Ped Surg Int 2011 27:337

1) Why do pediatric UTIs bother us and adults do not? The reason is that there can be reflux of urine to the ureters and this can result in renal scarring. More recent research has shown that renal scarring is likely to be ante natal (meaning before birth) in boys but in girls it is acquired. Another important point- some kids have repeated UTIs and have been found to have reflux and never develop renal damage. This much is known however, - it you surgically correct their reflux they develop less UTIs compared to having these kids on antibiotics all the time. The point of this essay is that the investigations are not benign and you should be somewhat selective in which approach you decide on

2) The numbers game. UTI before age 7 is seen in 8% of girls and 2% of boys- not that common and still more girls than boys but not that much more common in girls. They do not like bagged urines for
diagnosing UTIs because of contamination, but I think with fever and white cells in the bagged urine – you should be able to make the diagnosis. Reflux is found in 30-40% of children with UTI and does increase the rate of pylo although their definition of pyol is fever and an UTI. As kids get older, most reflux resolves. Renal scarring occurs with high grade reflux more than 90% of the time and it occurs in half the kiddies with acute pylo. 17-30% of those with significant renal scarring will develop hypertension as children. This scarring can be from reflux, but it doesn't have to be.

3) So let's start. You have a one year old kid in your ED or clinic with a UTI. If you are from the older generation you would do a VCUG- a voiding cystourethrogram which is done but injecting dye into the bladder via a catheter-not a fun experience for Mom or kid. So there are some things to think about. Since boys usually have developmental abnormalities causing their reflux they are often already diagnosed with the problem when the ultrasound is done in utero. Also you now have other modalities- ultrasounds and DMSA nuclear scan of the kidney. IVP is not used anymore for this and almost anything else.

So it comes down to checking from the bottom up or from the top down. The bottom approach will start with VCUG and ultrasound of the urinary tact. If reflux is found they do a DMSA scan and if there is hydronephrosis they do a MAG-3 Fusid(Lasix) renogram or an MR. The top down approach starts with a DMSA scan and if a pylo is
found they do a VCUG and if there is hydronephrosis they do an ultrasound,
4) The bottom up approach will find reflux and those with more likelihood of repeated UTIs, but it will not say who will develop renal scarring. No sedation for this approach is generally needed. AAP guidelines recommend this approach for UTIs in kids from 2 months to two years of age. This is from the first UTI. These guidelines however are from 1999. Since reflux does have a strong relationship to renal scarring, finding reflux is a paramount concern.
5) The top down approach looks at the kidney- which is what interests you in any case. That sounds appealing but there are some disadvantages including the need for sedation; DMSA scans need to be repeated to show if there is permanent damage and there is more radiation from them than from VCUG. Since reflux is not the cause of all scarring, it is important to identify scarring and not just reflux. Now the tests that we use for the bottom up approach also have limitations –the ultrasounds are normal in 60% of reflux cases and misses 50% of abnormalities found on DMSA scan. VCUGs also miss up a lot of cases.
6) There is another way to do this investigation which takes into the account all the drawbacks- and that is a selective approach which takes into account risk factors, such as atypical UTIs (serious case, poor urine flow, abdominal masses, elevated creatinine, failure to respond within 48 hours. These are the NICE criteria. If they are under six months of age; then they get an ultrasound. If they have a
recurrent UTI- they get a DMSA scan. If they have poor flow or an abnormal ultrasound- they get a VCUG. Sounds great, but a recent study showed they would miss 1/4 of the refluxes and about the same of renal scarring.

7) So if you are not completely confused yet, so let us try to bring it home. DMSA is the best test. It is the most sensitive in identifying scarring and pylo and the moderate and severe cases of reflux will show a positive DMSA scan meaning those with reflux unlikely to cause scarring will be weeded out. So yes I would conclude that DMSA by this article seems to be the test of choice but there are technical considerations and often the kids have to be sedated so I would probably in most cases waive doing the test in the first UTI, and worry about doing it in recurring ones.

8) One last word on treatment- using prophylactic antibiotics is definitely passé and not with out its dangers. Surgery is effective in treating reflux 98% and endoscopic injection – done just once-works in 83% of the time.

9) Sorry, but this is the best I could do – but it should be clear that this is a disease whose management is currently changing so the definite pathway to management is still in flux.

**EMU ROUNDTABLE** returns next month due to technical concerns.

Here is our bonus essay from Jan 2006-which is Mario’s favorite subject- I have no idea if the info is still current.
**EMERGENCY MEDICINE UPDATE**

**YOU LOOKS AT**: Acute Porphyria

Alright, stop moaning- it is not the most glamorous of topics. But it is out there, and an emergency physician needs to know about it- from an *emergency physician* standpoint. This review was extracted from an article in the Annals of Internal Medicine, 15 Mar 05

Porphyria is a disease of heme synthesis that can have acute life threatening attacks. It is an autosomal dominant disease. It comes in four flavors, but the one you have to know about it AIP or acute intermittent porphyria

There are many clinical manifestations- here are the common ones

a) Abdominal pain- usually with a silent abdomen, which gives an impression of conversion disorder. Have vomiting, but no peritoneal signs, no fever, no leukocytosis. They look like they are suffering from psychiatric illness- this is especially difficult to deal with, due to the tendency to a neurological origin. There may be muscle weakness and paresis. This can look like heavy metal poisoning or multiple sclerosis, or again like they are faking it

b) Heart- tachycardia is common

c) Electrolytes- hyponatremia is particularly common

These are common but not always present in cases of acute porphyria. Failure to treat in a timely fashion can result in long term sequelae

What will make you suspect you are dealing with this disease? Dark or red urine, hypertension, proximal muscle weakness, or other strange neuro symptoms, dieting (see below). Since there are many asymptomatic carriers, family history may not help you

Triggers can help you. Medications can set off an acute attack (steroids, hormones, anticonvulsants). Careful- in pregnancy- metoclopramide (Reglan, Pramin) can set off an acute attack, while promethazine (Phenergan) is safe. Other stressors- crash dieting, smoking, stress, alcohol) Many meds can cause an attack- the patient should come to the ED with a list or you can look it up on the internet.
Treatment: Of course stop using the offending drug. Some studies show the use of carbohydrates (in the form of glucose) will help- just be careful because large amounts will result in hyponatremia. Hemin is the treatment of choice and will reverse attacks. It is safe in pregnancy.

Failure to treat can result in permanent neurological sequelae including respiratory paralysis.

As is our custom, we now give homage to those who make EMU what it is - our peer reviewers. Thanks guys

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