ROYAL COLLEGE of PHYSICIANS of EDINBURGH

60th St Andrew's Day Festival Symposium

Tuesday 24 November 2020 – Friday 27 November 2020

Tuesday 24 November 2020

How to manage acute severe and life threatening asthma – Dr Donald Noble

Q. When would you choose 50mg Prednisolone vs 40mg/30mg?
A. There is no clear evidence to support a higher dose than 40mg. However in cases where there is persisting blood eosinophilia or a high BMI, I would consider a higher dose up to 60 mg/d.

Q. Do you modify your assessment for the clinically obese chronically hypercapnic asthmatic?
A. Yes, although in this type of situation you need to consider whether asthma is the main condition or obesity hypoventilation. The latter is more likely to respond to treatment with non-invasive ventilation in cases of decompensated respiratory failure

Q. When to start magnesium and aminophylline?
A. Intravenous magnesium is indicated for cases of acute severe asthma they do not respond rapidly to treatment with nebulised bronchodilators. Intravenous aminophylline is likely to provide little additional bronchodilation and there is no strong recommendation for use in acute asthma. Aminophylline is still used occasionally for resistant acute severe asthma, although this should only be considered following discussion with the critical care team.

Q. Is there definitely no role for IV salbutamol? - I believe there is a Cochrane review that states there is no difference in effectiveness vs IV aminophylline but reduced AE with Salbutamol?
A. The only role for iv Salbutamol is in cases where the oral route is not available

Q. How much and how frequent salbutamol nebuliser one can we give in acute severe asthma?
A. If the initial response to nebulised Salbutamol limited, can give repeat doses at 15–30 minute intervals or give continuous nebulisation of salbutamol at 5–10 mg/hour.

Q. How frequently can you give IV magnesium in acute severe asthma, or is it just a once off?
A. Single dose. There is a risk of hypermagnesaemia with repeat dosing

Q. Can I ask if I understood correctly that b2 agonists can increase serum lactate?
A. Yes, high doses of B2-agonists can often cause an elevated serum lactate level. High dose B2-agonist treatment leads to an increase in pyruvate in muscles which is metabolised to lactate. It is difficult to know if this causes harm, but if you see this and the patient is otherwise responding well, B2 agonist treatment should be tapered back.

Q. Can asthma in childhood lead to lung restriction in adulthood?
A. Yes, chronic uncontrolled asthma in developing lungs can lead to lung function abnormalities in adulthood

Q. When I did Ecg for 2 patients with acute asthma I found 2 times inverted t waves which become normal after a while. Any relation with hypokalemia?
A. This is possible if the patients were indeed hypokalaemic. However, a more common situation in the context of acute asthma is the presence of hyperventilation which can this type of ECG abnormality.

Severe hypertension: managing hypertensive urgencies and emergencies – Professor David Webb

Q. Same day referral with BP >180/120...does it have to be the systolic AND diastolic above these values, or only 1 of the 2?
A. NO – it can be either the systolic ≥180mmHg OR diastolic BP ≥120mmHg. In any case, it is important not to get too fixated on exact cut-offs because some people (especially those with pre-eclampsia or renal failure) can sometimes present with BPs a little lower. The key thing is whether there is evidence of organ damage/stage 3 or 4 retinopathy, which indicates hypertensive emergency and the need for urgent referral to hospital.

Q. How good are we at picking up relevant changes on fundoscopy in those who do get it done?
A. Mostly, as one is looking for fairly florid changes of bilateral haemorrhages or exudates, with or without papilloedema, this should not be too difficult for a reasonably experienced clinician. If unsure, or there is a reason that the back of the eyes cannot be seen (e.g. cataracts) then one might wish to err on the side of caution and admit the patient.

Q. How do you know if the BP in these hypertensive emergencies is the cause or effect?
A. I am not sure what is meant here. A hypertensive emergency has a raised BP AND evidence of organ damage, so the organ damage is likely to be related to the BP. However, it may be that an ischaemic or haemorrhagic stroke, for instance, causes an increase in BP (i.e. secondary to the stroke), and that is perhaps why the evidence that BP should be rapidly lowered in this situation is less clear.

Q. Stage 3 HTN without end organ damage - need admission?
A. This group of people meet the definition of hypertensive 'urgency', NOT emergency. They do not, generally, require admission. They need assessment to exclude organ damage, repeated BP measurement and, if appropriate (sustained hypertension, no suggestion of poor adherence to treatment, no relevant drug use, etc.), oral treatment can be started with a calcium channel blocker (like amlodipine) or ACE inhibitor (like lisinopril), as an outpatient, and the patient reviewed within a week (preferably with home BPs in the meantime).

Q. Why would you choose GTN infusion for hypertension in those with ACS/pulmonary oedema but not for other indications? Is there evidence of benefit from using labetalol instead?
A. GTN infusion causes a reduction in venous tone (reducing preload), large artery compliance (reducing arterial stiffness) and arteriolar tone (reducing cardiac afterload and BP). In addition, GTN may reverse coronary spasm. This combination of effects is particularly helpful in left ventricular failure and myocardial ischaemia/coronary insufficiency.

Q. What's the common BP criteria for PRES?
A. PRES is a rare condition. The BP criteria for PRES in hypertension are as for all of the hypertensive emergencies - systolic ≥180mmHg OR diastolic BP ≥120mmHg. However, importantly, this is a clinical syndrome within the broader diagnosis of accelerated hypertension, so the key clinical features are as follows: Seizure, altered mental state, headache, hemiplegia, speech problems, visual disturbance (commonly hemianopsia). The diagnosis can be confirmed by classical features on MRI. The findings most characteristic for PRES are symmetrical hyperintensities on T2-weighted imaging in the parietal and occipital lobes; this pattern is present in more than half of all cases. FLAIR sequences can be better at showing these abnormalities

Q. What about patients with a BP >240/120 mmHg without emergency features?
A. People with a systolic ≥180mmHg have a hypertensive urgency or emergency. The key thing is whether there is evidence of organ damage/stage 3 or 4 retinopathy, which indicates hypertensive emergency and the need for urgent referral to hospital.

Q. 240/120 mmHg implying auto-regulatory failure?
A. No specific level of BP causes autoregulatory failure. If BP rises slowly, a sustained BP of 240/120 may well not cause autoregulatory failure. It is a rapid change of BP, upwards or downwards that cause autoregulatory failure by either hyperperfusion on hypoperfusion, respectively.

Q. What features of the history would make you think of phaeochromocytoma seriously enough to avoid labetolol?
A. Phaeochromocytoma is rare, but easily diagnosed with a 24-hr urine test or a blood test for catecholamines/metanephrines. Alternative treatments in this situation would include IV nicardipine, IV sodium nitroprusside or iv GTN. Once diagnosed, phentolamine (irreversible alpha-blockade) needs to be established before labetalol (beta-blocker). Key symptoms that would suggest phaechromocytoma include: headache (not very specific), tachycardia, sweating/diaphoresis, usually paroxysmal in nature. Other fairly common features include panic attacks, anxiety, pallor and orthostatic hypotension.

Q. Patients with home monitors get lower readings than the one you get in clinic, what would be the best way to clarify whether they are really hypertensive?
A. Home BP monitoring is more reliable than using clinic BPs (which are not terribly reliable), but clinic BPs are a good screening tool (if they are normal). The UK 'gold standard' is ambulatory BP measurement, which is recommended by NICE in the UK and most international guidelines.

Q. Difficult to see in older patients who are intolerant to light and those ones with cataract - Any advice for them?
A. Yes - These are likely to be difficult. If they had no evidence of organ damage, it would be a clinical judgement whether to treat them as an outpatient with oral agents and follow up within a week or admit them for hospital review. Each such case (not so common in my experience) would need to be judged on its merits.

Q. What about the use of oral nifedipine as a short acting calcium channel blocker?
A. There are no grounds for using any short-acting calcium channel blocker in patients with severe hypertension. Patients with a hypertensive emergency should be established on iv therapy to reach their relevant target BP over the relevant time (see talk and papers cited). Those without, should be given an oral calcium channel blocker (like amlodipine) or ACE inhibitor (like lisinopril).
See these references for further information:
NICE Guideline on Hypertension in Adults (NG136; updated Aug 2019)
Bert-Jan et al. *Eur Heart J (CV Pharmacother)* 2019;5:37-46
Astarita *et al*. *J Hypertens* 2020;38:1203-10

Targeting treatments for Covid-19 – Sir Michael Jacobs

Q. When it says no oxygen required, does that mean the patients maintaining saturations between 90-94% (COVID acceptable saturation range?)
A. Yes

Q. Any evidence that monoclonal antibody treatment will reduce infection rates?
A. None yet

Q. Can someone be infective for months after COVID and what is the current data about reinfection with covid?
A. Maybe in rare immunocompromised states, but in general no. PCR positivity can persist for a few weeks but this does not equate to infectious virus. There have been a handful of convincing cases of reinfection, one of whom at least developed severe disease, but it does not seem common yet. It may become more so as immunity wanes – it simply isn't known.
Q. Is there a role for the use of ARBs in the prevention of deterioration due to the pivotal role of tissue RAS?
A. There is no evidence to suggest so. Registry data looking at patients who were taking ARBs and became infected do not suggest better outcomes.

Q. Given increasing rates on re-infection, is vaccination really an option?
A. The early vaccine data suggest impressive short term protection, but it will take time to understand the durability of protective responses both from vaccination and after natural infection

Recent onset polyarthritis and red hot joint – Dr Euan McRorie

Q. Have you seen reactive arthritis after covid in patients? I haven't but have seen fibromyalgia symptoms not uncommonly.
A. I have not personally seen reactive arthritis post-COVID though such case reports are beginning to appear e.g. Waller at al Rheumatol Adv Pract. 2020 Oct; 4(Suppl 1): rkaa052. Published online 2020 Nov 3. doi: 10.1093/rap/rkaa052

Q. Sometimes there is diagnostic uncertainty about a red joint and high CRP. Is Procalcitonin helpful at differentiating between septic joint vs gout / inflammatory arthropathy?
A. This is not a routine part of our routine assessment when faced with an acute hot joint. I undertook a PubMed search and came across this recent paper which suggests that procalcitonin is not useful in discriminating between gout

and septic arthritis: Chouk et al, *Clin Rheumatol* 2019 Aug; 38(8):2265-2273. doi: 10.1007/s10067-019-04542-0. Epub 2019 Apr 16.

Q. Would you treat as for RA if your patient's presentation was an extra-articular presentation eg a pleural effusion. or would you require assoc joint symptoms first?
A. It would be difficult to diagnose RA if the only clinical manifestation was a pleural effusion, but I would have a low threshold for undertaking imaging of joints if the patient were strongly sero +ve for RF or ACPA.

Q. Which treatment would you use in patients with renal dysfunction e.g. stage 3 CKD and a gout flare? Is colchicine risky in this group?
A. I would use colchcine and allopurinol at low dose in the context of CKD III eg starting dose colchicine 500mcg od and allopurinol 50mg daily, keep a close eye on renal function and cautiously escalate allopurinol titrated against serum urate and renal function.

Q. Procalcitonin - can it differentiate gout from septic arthritis?
A. See answer to Q2 above.

Q. In pseudogout, is ultrasound useful for diagnosis as in gout?
A. I put this question to a Rheum/Radiol colleague; their view is that US does not have a clear role in the diagnosis of pseudogout.

The red leg: assessment in the acute medical unit – Dr Girish Gupta

Q. How did the authors make the diagnosis of cellulitis when developing the clinical prediction models? What was considered gold standard?
A. Most studies were retrospective or if prospective, lacked large numbers. Evidence level was generally poor and there were no RCTs. Gold standard was clinical diagnosis of cellulitis made by the clinician in the majority of studies. Clearly further work is required in this area with better designed trials and scoring systems also need to be validated.

Q. Interestingly there is an increased number of referrals with a query of bilateral cellulitis? how common is this? (or is this a thing?)
A. Not sure how commonly these pts are referred from primary care with "bilateral cellulitis" as these referrals do not come to us but bilateral cellulitis is extremely rare. Can happen however but I have not seen this.

Q. Should we even accept referrals into hospital with 'bilateral cellulitis'? Is there any pre-referral advice we could give our GP colleagues to avoid the numerous hospital referrals? The vast majority don't really need hospital attendance at all in my experience.
A. Difficult not to accept without examining the skin. May be worth auditing figures in Lothian to see what the yield is. Agree that most will not be cellulitis and wonder if the algorithm on the last slide would help primary care/clinicians in MAU.

Skin cancers to recognise and refer – Dr Colin Fleming

Q. Is there a way that can hint to systemic metastasis of melanoma clinically?
A. History of previous melanoma, rapid onset, rapid growth, association with systemic features e.g. lymphadenopathy.

Q. If a cancer was missed by AI, where would the responsibility lie?
A. This will need to be worked out with society, along with other applications of AI e.g. autonomous vehicles.

Q. How effective is immunotherapy in melanoma in terms if prognosis of metastatic disease?
A. Works in approximately 50% of cases

ROYAL COLLEGE of PHYSICIANS of EDINBURGH

Wednesday 25 November

The ECG in the poisoned patient – Professor Michael Eddleston

Q. Is the sort of ST elevation in aVR in the ECG you showed characteristic of amitriptyline overdose?
A. The shape of the AVR lead in an ECG for amitriptyline toxicity often takes this shape as the QRS duration prolongs out to about 160 msec.

The behaviourally disturbed patient – Dr Catriona Howes

Q. Do you recommend any specific medications or any dose changes for lorazepam/haloperidol in patients with renal or liver impairment?
A. Yes, in liver failure would suggest halve the dose of haloperidol. Lorazepam at low dose is acceptable in mild-mod liver impairment, if severe avoid altogether or use very short acting benzo (oxazepam)
In renal failure, can use both but start at low doses and monitor closely.

Q. If needing to use IM sedation on frequent occasions is this an indication for detention under the mental health act or is an AWI enough?
A. No, IM in an informal patient is only acceptable if significant risk to them or another person under common law. In Scotland, if likely to need as more than a one off, patient should be reviewed for detention under STDC. Mental welfare commission should also be informed of use of IM medication without consent in an informal patient by completing a T4 form.

Q. Challenging behaviour often may be due to a number of causes including substances. Any thoughts on how their management may be different?
A. I would use benzodiazepines and at higher doses in patients a) withdrawing from substances or b) in acute toxidrome. Antipsychotics will reduce seizure threshold in those withdrawing from alcohol and should be avoided.

Q. As a Geriatrician I am interested at the recommendation of lorazepam first line in elderly rather than haloperidol. Our local policy is the other way around with advice that benzos typically prolong delirium.
A. I would recommend benzodiazepines as first line in the acute setting, where cause of agitation is undetermined as they are generally safe. However, if likely to be caused by delirium I agree that low dose haloperidol is a better choice- benzodiazepines will prolong delirium.

Diabetes – Dr Nicola Zammitt

Q. Recent case with DKA, despite resolution of ketones and hyperglycemia patient remained mildly acidotic with a persistently raised lactate (4-5) (not septic, BP normal, good U/O). Was told this was normal and would resolve in due course. Is this true?
A. Yes, would generally expect lactate to resolve with ongoing fluids provided other causes of lactate have been treated. I see that he patient was not septic. worth making sure patient is not on metformin (less common in type 1, but is used in some overweight patients) and that there is no tissue ischaemia e.g. rhabdomyolysis after a long lie, ischaemic foot in person with diabetes and peripheral vascular disease.

Q. The DKA protocol is the busiest one we use in our trust. It can be overwhelming for people to follow particularly in face of other patient demands. Do you have any tips? Re CP lactic acidosis likely from hyperchloremic acidosis from excess N Saline.
A. Tips: read through the protocol at a time when you are quiet so as to familiarise yourself with it, rather than trying to work it out on a busy take. 0.9% saline can cause a hyperchloraemic metabolic acidosis, but would not push the lactate up as such.

Q. Recently assessed 2 patients started on Empaglofizin with angioedema apparently known SE of unknown frequency. Have you encountered this in your experience? Any wisdom on this.
A. I have not encountered this side effect myself

Q. Should patients in the community with COVID be asked to stop SGLT2 inhibitors?
A. We have stopped these in all patients with type 1 diabetes (dapagliflozin is licensed in type 1) but not for type 2. however, via community pharmacy, all patients on SGLT2 inhibitors have been given "sick day rules" information asking them to stop the SGLT2 inhibitor if they develop any Covid symptoms. We reinforce this at our clinic appointments.

Q. If euglycaemic DKA should we diverge from DKA protocols and start insulin at lower rate?
A. You could start insulin at half the usual rate e.g. 3 units per hour. however, it is the combination of insulin and glucose which will switch of ketone body production, so we still need to give insulin but with adequate 10% dextrose alongside it.

Q. How soon would you restart SGLT2 antagonists post covid-19 if one was previously on it?
A. You can restart within 4 or 5 days, but if they were in DKA we are generally discontinuing it long term

Q. The evidence from NHS England seems to be at odds with the more recent evidence published from Belgium in relation to the risk of death with type 1 diabetes and Covid-19. Do you think we have enough evidence yet?
A. Evidence still evolving.

The abnormal potassium – Dr Matt King

Q. Do the labs not account for the haemolysis before issuing a result to clinicians? If the sample is grossly haemolysed they would usually just release a 'haemolysed' result.
A. Yes to some extent but there is no correction for haemolysis. Also this is not a clear cut yes there is haemolysis or there isn't haemolysis. I have had a sample too severely haemolysed for no magnesium result yet a potassium result was released. So a result will be given even where there is mild haemolysis. Also on ABG and Li samples you can't recognise haemolysis so haemolysis is still an important clinical consideration.

Q. Would you give calcium chloride/gluconate even if there are no ECG changes and k+ less than 6?
A. As mentioned later in the talk, no. Even at potassium levels upto 7 mmol/l in my opinion calcium is not required if no ECG changes.

Q. Chronic hyperkalaemia (6-6.5) with no ECG changes and no symptoms, is this acceptable?
A. Yes in a well patient. I would suggest 6.1 or above needs an ECG but if less than 6.5, ECG ok and little risk of it getting higher then no treatment and discharge. However not ideal long term at 6-6.5 so at or above 6.1 I would suggest dietary advice and maybe stopping relevant drugs, such as ACE-I.

Q. Would 10% or 20% dextrose be a better option than 50%?
This has been debated for some time. 50% dextrose is like treacle and can damage veins. Nephrologists in particular don't like the volume associated with using the equivalent dose of 10% or 20%. So in a non-dialysis patient or non anuric patient using a lower concentration may be better. Down side is that it keeps it simple if one rule is used for all!

Q. How quickly do you administer the sodium bicarbonate?
A. Depends on the situation and the concentration of sodium bicarbonate. I would treat a bag of 1.26% just like a bag of Hartmanns/plasmalyte/0.9% saline. It cannot cause a lot of harm unless your patient is already in pulmonary oedema. If patient has 'space' and no known heart failure then quick bags can be given. Hypertonic is more tricky and is more an ICU treatment with much more risk of causing pulmonary oedema.

Q. When the med reg gets called at midnight with a K+ reading, is there a value at which you think admission is mandated? Especially as historical K+ results are unfortunately not always available.
A. If you do not know the patient this can be tricky. Assuming the patient is a well outpatient then I would say assessment could probably wait until the next morning unless above 7. Even in the high 6's it is likely the true level will be lower and safe because of the spurious effects on community bloods. If patient is unwell or has Aki then risk is more so I would say urgent assessment if above 6, with a minimum of an ECG.

Q. For a patient who's still passing urine, how many rounds of insulin dextrose would you try before calling the friendly nephrologist for ?dialysis.
A. I think this depends on the situation. Remember that treatments do not in general actually help remove potassium so unless something else changes like potassium dietary intake, drugs or renal function you might not be any better off after just a round of insulin and dextrose. Also, potassium is mainly intracellular so even when renal function improves it can take some time for potassium to be excreted from the body. It is therefore quite common for hyperkalaemia to develop from an acute problem to a subacute ongoing one. This is why I think anion exchange resins and diet do have a role (others would not agree!). Given that hyperkalaemia is usually associated with either advanced CKD or AKI I would think nephrology discussion should be relatively early unless clearly for supportive treatment only or maybe delay a little longer in cases where a rapid improvement is expected, e.g. catheter for acute obstruction.

Q. What is the safest alternative thromboprophylactic agent where heparin is felt to be the cause of hyperkalaemia?
A. I have not come across a situation where I think heparin is the main cause of hyperkalaemia. The average effect of heparin is in the order of 0.2- 0.3 mmol/l increase. There are some case reports of high increases but there are likely other factors responsible. In particular, diabetes may increase the risk of heparin-associated hyperkalaemia but maybe this is because diabetes tend to have very volatile potassium levels anyway. There seems to be risk with LMWH's but other agents nay not produce the effect. I would suggest that the risks of alternative anticoagulants or the risk 0f stopping prophylactic usually outweigh any small benefit.

Q. If ECG no changes when should we consider giving calcium gluconate?
A. No evidence to answer this but I would probably say above 7mmol/l. (Maybe above 6.5 if risk of going higher quickly). You could argue that calcium may not be needed at all unless ECG changes but in most medical units there isn't someone looking at the monitor every 5 minutes so safer to give for very high potassium levels even if no ECG changes. It's possible some of the arrests associated with 'no ECG changes' may have had rapidly rising potassium levels with a changing situation.

Q. Our guideline for hyperkalaemia is 10ml of 10% calcium gluconate. Should we be giving 30ml as you mentioned in your talk?
A. The dose suggested is artificial. So sometimes ECG may not change even after 30mls. However 10mls of gluconate is a low dose so yes I would suggest to give more.

Q. We have had massive resistance from pharmacists about replacing potassium peripherally at a rate of 10mmol/L without cardiac monitoring. I find it somewhat unreasonable to have people on cardiac monitors getting 40mmol KCL in 500 ml of fluid. Any thoughts?
A. Do not have a complete answer for this one but I share and recognise your frustration as I also get this from pharmacists. Calcium is dangerous to the skin if given peripherally in high concentration. This is of much more concern than cardiac risk but rapid Iv calcium has been associated with bradyarryhmia. I can't see any logic in monitoring requirements needing to be different between peripheral or central, rate and quantity are the important factors. There is risk of cardiac arrest in cases of hypocalcaemia and hypomagnasaemia associated with long QT. So, to me the risk is always more to do with the low level rather than the replacement. I think this might be where the concern however comes from. In a similar way the risk of arryhmia is almost certainly much higher from the hyperkalaemia itself rather than the infusion of calcium. Pragmatically don't delay giving the calcium waiting for monitoring in real cases where Qrs is prolonged. If calcium is given slower then this maybe means it's more likely for a tissued venflon to be missed and more risk of skin necrosis. Unfortunately, I cannot quote you an evidence based safe speed for calcium administration.

Hypercalcaemia – Dr Rachel Williamson

Q. Why is surgery still first line treatment for primary? Risks much be greater than cinacalcet?
A. Whereas there is evidence for both in terms of calcium-lowering, there is not yet evidence for the effect of cinacalcet on other outcomes such as osteoporosis/fractures/renal stones. Risks of cinacalcet are felt to be low, but there is a proportion of patients who are not able to tolerate it due to nausea.

Q. Can over the counter Vitamin D 2000units lead to hypercalcemia?
A. Hypercalcaemia would not be expected on this dose of 25-hydroxvitamin D, and if it occurred then I would recommend looking for other causes. One group of patients in whom even modest doses of Vitamin D can cause

hypercalcaemia is those with granulomatous diseases, such as sarcoidosis or lymphoma, in which there is extra-renal activation of Vitamin D – Vitamin D supplements should be avoided in these people.

Q. We quite commonly try some steroid when struggling to manage malignancy caused hypercalcaemia, what is your advice on this?
A. Guidelines suggest use of steroids for hypercalcaemia only in malignancies (or other conditions) associated with over-production of 1,25-dihydroxyvitamin D, and I am not aware of evidence for benefit out with these individuals.

Thursday 26 November 2020

Chest pain – Dr Anne Scott

Q. No option for "Angina Episode" - do we call that ACS?
A. Angina is indeed distinct from ACS. It wasn't included in the poll simply because it wasn't the correct answer in any of those 5 cases.

Q. In retrospect was there anything on the CXR for the patient with dissection?
A. Unfortunately, not really. It was AP and within normal limits for such a study.

Q. What about MINOCA - myocardial infarction with normal coronary artery? do you ever diagnose this?
A. MINOCA can be a useful working diagnosis where you have fulfilled the definition of myocardial infarction but seen normal coronaries. However, MINOCA encompasses a heterogenous group of patients with differing aetiologies and who require different treatments. It is therefore not helpful to stop with this as a final diagnosis and further exploration of aetiology is necessary for effective treatment.

Q. Is there a role for coronary calcium Index in diagnosing ACS?
A. The short answer is no. Coronary calcium scores help risk stratify on a population basis for the presence or absence of coronary disease but we have all seen patients with no coronary calcium but critical disease due to soft plaque or fibromuscular dysplasia so it is not useful for ACS.

Q. Would you agree that serial ECG's are essential? Too often I see juniors only do 1 ECG & 2 tropnins & I've seen dynamic change on 2nd ECG but normal troponin.
A. Absolutely, and thank you for raising. The ECG is hugely important and we shouldn't be over reliant on troponin. The dynamic nature of an ECG is very helpful and I agree patients should have a minimum of 2 ECG's when being investigated for chest pain that may be cardiac.

Q. Are we over-using Type 2 MI diagnosis? Haemodynamically patients with no oxygen supply:demand mismatch, no overt 'Sepsis' with ischaemic ECG, rising troponin and classic chest pain seem to get labelled this to avoid need for specialist input.
A. Interesting perspective. Recall the positive predictive value for the algorithm I showed was only 70-75% so there will be many patients who have a cause other than type 1 MI for their chest pain and troponin rise. Most commonly a type 2 MI will indeed be due to some supply:demand mismatch but the term also encompasses things like coronary spasm and dissection. Type 1 MI is defined by its atherosclerotic aetiology. My own perspective is that type 1 MI is over diagnosed and I spend much of my time unpicking the diagnosis. Of course we can all misjudge diagnoses; specialist or generalist.

Q. Why are some patients without chest pain get troponin done in some ED? how would you interpret at that point?
A. Excellent question, and one I frequently ask. Unless a patient has a history consistent with a cardiac event (usually chest pain, though in the context of a type 1 diabetic perhaps an acute episode of autonomic arousal) then there is no reason to check the troponin; it is most likely to lead to excess or inappropriate treatment or investigation. 'After the fact' it is usually possible to figure out an explanation for a raised troponin but it is better never to find yourself in that situation.

ROYAL COLLEGE of PHYSICIANS of EDINBURGH

Syncope – Dr John Davison

Q. Should every hospital have a syncope follow up clinic? This would change the focus from "falls clinic". It can be difficult to discharge a patient who you feel does not need to stay in if it is only you who is going to check their follow up investigations!

A. Yes - in my opinion there should be an outpatient clinic with access to prolonged ambulatory ECG / 24hr BP / beat-to-beat BP equipment / tilt bed (both for prolonged head up tilt test, and also for erect CSM) to assess for neurally mediated causes of syncope and for rhythm monitoring.
C. O'Dwyer, R.A. Kenny, Syncope clinics and the older adult, European Geriatric Medicine,
Volume 1, Issue 1,2010 gives an outline of what is involved. There is evidence that access to this expertise as an outpatient reduces inpatient LOS. The best clinician to run a syncope clinic depends on your local expertise and interest - cardiologist or geriatrician with links to neurology

Q. There is often friction in terms of where to admit these patients. Often cardiac monitored beds are limited in hospital. Any thoughts?

A. Unless there are ECG or clinical pointers to cardiac disease (from history, past history, vascular risk factors or examination abnormalities), a cardiac monitored bed is rarely needed. The ESC guidelines point to only admitting high risk patients - e.g. those where there is an abnormal ECG, the history is of palpitations or chest pain with syncope, syncope with exertion and nothing to support a neurally mediated cause. With lower risk groups they rarely need admission unless there is evidence of an acute volume loss / dehydration which would require correction in association with orthostatic hypotension / vasovagal syncope. Another risk stratification tool to consider is the Canadian Syncope Risk Score, which again flags those to admit in high-risk group linked to the above factors.

Q. Should we be doing CSM only with cardiac monitoring?

A. *Yes. Rarely CSM can precipitate arrhythmia, so cardiac monitoring essential for this. If CSM is +ve it is important to be able to document the length of ventricular standstill for referral on for PPM treatment. As one third of patients with carotid sinus syndrome have vasodepression only without bradycardia, it is also important to have access to beat-to-beat BP monitoring during CSM.*

Q. Previously liberally stopped Tamsulosin, but some bad experiences - AKI with 'asymptomatic' retention etc. Now more cautious. Any advice about planned follow up for stopping Tamsulosin e.g. bladder scans, starting alternative agents etc.?

A. *I usually take a history for lower urinary tract symptoms and check if there has been previous urinary retention. If stopping Tamsulosin, I usually start finasteride. I don't routinely undertake post void bladder scans. Agree there is a risk of asymptomatic urinary retention and recommend that when stopping tamsulosin flagging symptoms to be aware of - e.g. increased urinary frequency with low volume might indicate incomplete bladder emptying which would trigger bladder scans.*

Q. Could you clarify the driving restrictions for patients presenting with what appears to be a single episode of vaso-vagal syncope? I would have let her drive after the first episode. Is that wrong?

A. *DVLA guidance for a single vasovagal episode where the 3 'P's are met - upright posture, prodrome (duration sufficient to safely bring a vehicle to a standstill) and precipitant (a trigger which is unlikely to occur when driving) is that the patient can continue to drive and does not need to inform DVLA.*

*If LOC with vasovagal syncope occurs when **seated** and there is an avoidable trigger which is unlikely to occur when driving, they can continue to drive. However, **if recurrent** VVS when seated and the trigger may be unavoidable, e.g. abdominal pain as the trigger, DVLA states they must stop driving for 4 weeks, inform DVLA and are judged safe to resume when the annual risk recurrence is assessed as less than 20%. This latter requirement is a challenge for clinicians as there is no evidenced risk stratification score to formalise this, other than frequency of events and symptoms.*

The rationale in the case presented for stopping driving is that the patient developed recurrent loss of awareness without prodrome. For dissociative attacks, DVLA requires that these are 'controlled' before driving eligibility is returned.

Q. You referred to her ECG as abnormal - but isn't it reasonable to consider partial RBBB in a young patient a variant of normal?

A. *Agree. Partial RBBB in a young person is a variant of normal. The reasons to investigate further are 1) when they now present with syncope, and 2) when there is a family history of sudden cardiac death or cardiac disease.*

Acute medical problems in pregnancy – Miss Joanna Girling

Q. What are some of the possible reasons for such a big discrepancy in Maternal Mortality state among different ethnic groups?

A. A paper is about to be published in BJOG about this. Multifactorial, including complex and multiple disease processes, socioeconomic deprivation, microaggressions, institutional racism

Q. PE in Pregnant Women. What is your view regarding CTPA vs V/Q scan. Our Local radiologist suggest to go for CTPA after explaining the risk of breast cancer as V/Q scan often turn back as inconclusive and patient ends up with CTPA....

A. Departments need to have access to both and decide on clinical basis – for an intrinsically well young woman, starting a treatment dose of LMWH and awaiting a VQ scan minimises her risk of breast cancer. For a sick woman or those in whom other conditions are suspected – such as COVID – a CTPA is usually better. The RCOG Greentop guideline 37b gtg-37b.pdf (rcog.org.uk) summarises the options

Q. Do you think AMUs are the best services to be addressing or identifying the impact that any pre-existing medical conditions could have on future pregnancy?

A. Everyone has a role to play in identifying women who will benefit from prepregnancy planning, including staf in AMUs. We need to change the mindset for all health care professionals and women, so that every interaction is an opportunity to consider contraception / preplanning direction. Often it will be an opportunity to signpost a woman to the correct services, rather than actually providing the guidance

Q. Why such a significant biochemical hyperthyroidism in hyperemesis gravidarum? Would you do any other tests?

A. HCG has identical alpha subunits to TSH, so HCG binds to TSH receptors on the thyroid; and some HCGs are more thyrotropic than others. In hyperemesis gravidarum other tests might be required depending on the full clinical history and examination, including renal and liver function, and tests for other causes of vomiting if there is any doubt about the diagnosis of HG

Q. How do you differentiate hyperemesis causing abnormal TFT from true hyperthyroidism apart from doing TRAB ?

A. Usually by taking a thorough history and performing a full examination. With hyperemesis, the symptoms start clearly after the pregnancy started. There will not be any eye signs, onycholysis or skin changes (which are TSH Rec Ab driven); if there is goitre it will be small and symmetrical. Typically women with biochemical thyrotoxicosis are lethargic and dull, not buzzing and lively.

If in doubt, measure TSAH rec Abs, which will be negative in gestational thyrotoxicosis; but to do so will be unusual

Q. This is why it can be difficult in hospitals where pregnant women under 30 weeks are seen in general medicine. We are not just so comfortable in knowing what is normal for a pregnant woman & I mean observations and blood tests. On that subject had d dimer got any use? I know it is - generally high.

A. … and this is why education and team working are so key! No current place for D Dimer or non-pregnant scoring systems in diagnosis of VTE in pregnant or recently delivered women

Q. MEWS score in AMU at all stages of pregnancy?

A. I would say YES. Physiology changes early in pregnancy; and women tend not to arrive – especially when acutely unwell or collapsed – with a clear gestation already determined. I would argue that once the pregnancy test is positive, we move monitoring for sick pregnant women ot MEWS, not NEWS

Q. But CT(PA) increase risk of breast cancer and hence advice to avoid CT if not strongly indicated?
A. I agree

Q. What about CTPA in breast-feeding? Can you give advice on best resource to explain breast cancer risk? Radiology often refuse on this basis but my understanding is risk low if baseline risk of mum is low.
A. https://www.acr.org/-/media/ACR/Files/Clinical-Resources/Contrast_Media.pdf page 101; this was updated in 2020 Drugs Factsheets - The Breastfeeding Network - https://www.breastfeedingnetwork.org.uk/drugs-factsheets/

Q. As an acute physician, we get asked to see a lot of "suspected PE's" in pregnancy with somewhat dubious chest pain, some SOB which can easily be attributed to the later stages of pregnancy, and an equally useless D dimer result. What is your experience in these scenarios? Do you scan all patients?
A. I agree, it is a hard diagnosis to make clinically, an important one not to miss given the 5-10fold increased likelihood in pregnancy / puerperium and the 'high' mortality, and a diagnosis not to make inappropriately given the lifelong implications of the diagnosis.
And no, I don't scan everyone; but I do scan 'lots'. The clinical assessment, including history, examination, ECG / CXR / leg examination can help. If the clinical suspicion is reasonably low (but not low enough to dismiss) I would do a VQ scan whilst she takes a treatment dose of LMWH

Oncology emergencies: new drugs, new problems – Professor Ruth Plummer

Q. What differentiated the small number of long-term stage IV melanoma survivors (pre checkpoint inhibitor)?
A. We do not know, but it is assumed that they were able to mount some host immune response giving the long term control, and also melanoma and renal cancers are the two tumour types where we see rare spontaneous remissions, so possibly this also shows the most immunogenic. The previous chemotherapy drugs used, being DNA damaging may have helped trigger the immune response

Q. Immune suppression to treatment autoimmune disease can cause cancer, so how do you treat autoimmune disease caused by immunotherapy for cancer treatment and will that affect cancer therapy?
A. We treat conventionally, and within the trials there did not seem to be a reduction in efficacy

Q. How have you managed screening for such side effects during the remoteness imposed by Covid-19?
A. Good CNS support and patient information leaflets.

Q. For colitis post immunotherapy, do you wait to rule out infection before giving IV steroids etc.?
A. Most admitting hospitals do, what we try to avoid is this significantly delaying starting steroids as if the colitis becomes "established" it seems harder to settle down, and there is the perforation risk if severe

Acute liver failure: my local – your global – Professor Debbie Shawcross

Q. Role of urgent psychiatric assessment in patients with ALI being referred to the transplant centre?
A. As patients with acute liver failure are often in grade 3/4 encephalopathy and ventilated, acute psychiatric assessment is often impossible to undertake. We often therefore rely on liaising with the GP/psychiatric team regarding their pre-morbid psychiatric state prior to making decisions on transplantation. The main concern is whether they will be compliant with immunosuppression post-transplant.

Q. Is DVT more common in ALF? Prophylactic LMWH?
A. A procoagulant state is more commonly seen in the context of chronic liver disease rather than acute liver failure. Generally thrombosis risk is independent of INR/platelet count. DIC increases risk of thrombosis in the context of acute liver failure. We do not routinely give LMWH to patients with acute liver failure.

Q. ALF: I might have missed this, but how do we diagnose the condition? Is it by LFTs? Clinical etc.?
A. It's a clinical triad of coagulopathy, encephalopathy and peripheral vasodilatation/cardiovascular collapse. It is primarily a clinical diagnosis.

Q. Do you have a cut off level for ammonia where you would want to start RRT?
A. Not specifically but Will Bernal has shown that the risk of brain oedema and intracranial hypertension significantly increases once the ammonia level rises above 150 umol/L.

Q. I recognise alcohol consumption is a contraindication for liver transplant, in POD how do we assess the line between alcohol dependence and social alcohol use?
A. Many patients take paracetamol overdoses with alcohol and patients are more vulnerable to paracetamol toxicity when they have a history of alcohol excess (steatotic liver). However, unless there is a clear history of alcohol excess/addiction a moderate history of alcohol excess is rarely a contraindication to proceeding with transplantation.

Q. What is the reasons to maintain high sodium level in ALF?
A. Maintaining a high serum sodium >145 mmol/L helps to control brain oedema. A low serum sodium exacerbates astrocyte swelling/brain oedema. We commonly give boluses of hypertonic saline.

Reducing admissions and changing behaviours – Dr James Marple

Q. Patients who are frail elderly and acutely unwell often have very little input when they are scooped into ED in the middle of the night. Much better to anticipate these occasions and ask them / family early. How can we improve our professional communication so that we improve their care?
A. I think that we need to start having these sorts of conversations earlier & in a more routine fashion. Perhaps it could be done like screening is done at specific ages by sending out information & questionnaires. It could be done in Primary Care using the Electronic Frailty Index. Could be done in Memory Clinics & other OPD settings for anyone with advanced organ failure. Could be linked to getting a Power Of Attorney organised. I suppose we need to try & make it the default option for everyone rather than the exception. Like making a will. We could really do with a public campaign.

Could it be functional? A positive approach to negative tests – Dr Elizabeth Visser

Q. How often do functional problems co-exist with chronic pathological ones? And any tips to deal with them?
A. In the handbook of Clinical Neurology it is described that comorbid neurologic disease occurs in around 10% of cases with functional neurological disorders. It probably varies depending on the functional symptom. In order to address this explaining to the patient which symptoms are FND and which are not is important. Video EEG for instance, can help educate patients and carers as to events that are dissociative seizures rather than epilepsy in patients who experience both. It is not always easy to distinguish. We would still look for positive functional signs on examination and to also illustrate that to the patient can help. Management is as discussed in my talk, acknowledging the symptoms, addressing possible “triggers” and directed symptomatic management. To use dissociative seizures as an example, patients will not respond to higher doses or multiple antiepileptic treatments but a therapeutic explanation and input from a psychologist is more appropriate.

Friday 27 November 2020

Pain in advanced disease: assessment and management in the Acute Medical Unit – Dr Fiona Finlay

Q. End of life is defined in Primary Care as in the last year of life for palliative care. Is it right to prescribe anticipatory medication for these patients who may NOT be immediately dying?
A. I'd suggest considering the patient in front of you is the best way to decide about anticipatory drugs.
Some people benefit from having oral/sublingual medications on an 'as required' basis, if they are not imminently dying (thinking of opioid and benzodiazepine) - these can be helpful for symptom control, and taking control/having autonomy over symptoms, if they are available at home for a patient to use on an as needed basis. I wouldn't necessarily advocate for someone who has an intact oral route, and the ability to use medicines as required, to have a supply of subcutaneous anticipatory drugs in this context.
As I am a hospital palliative medicine doctor, I tend to prioritise ensuring anticipatory medications (both po and sc) go home with a patient where I think prognosis is in the region of maybe a small number of weeks, and by NOT supplying these drugs, I might risk delay in them being available when the patient deteriorates, especially if I think this is likely. It's also very helpful that in the last several months, we have been able to complete a Community Kardex for any patient going home to somewhere in the Glasgow city area, so that all sc prn meds are already supplied/legally prescribed on a prescription on discharge to prevent additional work for the GP/potential delay for patients receiving a sc medication.

Cancer of unknown primary – Dr Sally Clive

Q. Was poor continuity part of the perceived SDM (?). Would it have been significantly different if 'pre-Covid'?
A. In this particular case, one of the difficulties was doctors working in different 'zones' – on call so working in red zone but then patient moving to green zone and, as per advice at that time, that doctor not working between 'red' and 'green' and therefore relying on more junior staff to look after the patient once transferred. Junior staff followed instructions but didn't change plan according to changing clinical situation and patient wasn't officially handed over to another consultant colleague, as that consultant was still on site working and the named consultant. So – yes – Covid probably impacted the continuity in managing this case. But the same thing could easily happen pre or post Covid for numerous other reasons.

Sexual health in the acute medical unit: the Great pretender and the magic curtain? – Dr Sarah Allstaff

Q. Why do you think the incidence of syphilis is increasing?
A. The rise in syphilis we have observed exceeds any rise in STI testing. It is largely observed in men who have sex with men although there have been a few outbreaks amongst heterosexual groups in Scotland. There are likely multiple behavioural factors feeding into this including increasing numbers of sexual partners and group sex both of which are more easily facilitated by geosocial networking apps. The introduction of good HIV prevention (regular testing, universal treatment and HIV pre-exposure prophylaxis) coupled with the emergency of "chemsex" (the sexualised use of certain drugs such as crystal meth, mephedrone and GHB) have contributed to reduced condom use. Syphilis (and gonorrhoea) are also very infectious by oral sex and sexual repertoires have expanded to include more oral sex as it is low risk for HIV transmission. As secondary syphilis may present with non-specific findings it often goes undiagnosed and as such it is important that we are aware of its epidemiology and be sure to test people who present with symptoms, signs and unusual presentations.

Decision making for the intensive care unit – Dr Nazir Lone

Q. Guidance for frail elderly patients if undergoing emergency surgery is for HDU level support post-op?
A. The key decision making should happen before going to theatre: this should include ICU/anaesthesia/surgery senior decision makers along with patient/family. Imaging may narrow the likely differential diagnosis. As always, an individualised approach of weighing benefits vs burdens of surgical intervention is needed, including discussion of likely outcomes (including death). If the decision is to proceed to surgery, a period of post-operative monitoring in critical care is usually necessary, although a time limited treatment trial/ceilings of therapy may need to be discussed in advance with family/patient. For emergency laparotomy, be aware of national quality improvement activities/audit and risk stratification (NELA).

Q. Has COVID changed your approach when deciding to offer level 3 and if so how?
A. COVID-19 has not changed my approach to offering level 3 care. As we have developed familiarity with the clinical course of the disease in critical care, this has allowed better informed discussions with patients and families. The biggest difference is that many patients and families have heard of the treatments offered in ICU such as 'being on a ventilator', and a number of patients have already ruled this out as a potential treatment option before being assessed by critical care teams.