59th RCPE St Andrew's Day Festival Symposium: Updates on Acute Medicine

Thursday 28 November – Friday 29 November 2019

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Shared decision making – Dr Caroline Whitworth

Q. Should we be embracing formal SDM tools?
A. I think they have a role, particularly if they assist in the steps required to explore patient preference and patient context. Some SDM tools however do not effectively enable 'weaning out' of un-realistic options and therefore may detract from clinician and patient focussing on more appropriate options accounting for the patient context.

Q. Do you think there is a role for the use of clinical frailty scales in helping to decide treatment goals and plans with patients?
A. Yes, very much so. There is evidence particularly in my own specialty of assessing clinical frailty - and using that to help decision making but also help identify what other actions are required to help a patient generally.

Q. Having access to personalised data narrowing uncertainty as in the VM case seems essential to be able to help in shared decision-making, yet seems incredibly hard to deliver. Why do you think this has been so challenging and what can we do?
A. I agree 'Big Data' could, in time, help us have more certainty about probability of outcomes for patients following different options. We have to get over the barriers to collecting that data effectively, addressing issues of ensuring individual patient confidentiality. Currently we do struggle to identify appropriate data to help inform our patients. Access to Information technology that enables rapid access to such data, during a consultation is needed. Time is also a significant barrier in the clinic.

Q. How do we guard against further health inequality? The most literate/educated getting the opportunity to make the best decisions?
A. I think it is the clinician's responsibility is to 'put themselves in the patient's situation', so explore their level of understanding, their preferences, their individual concerns, and by doing that help ensure that SDM is realistic. SDM should not rely on patient literacy/education, but will rely on good two-way communication between patient and clinician. However, SDM will not get rid of the inverse care law.

Q. Thanks a lot for your wonderful talk. How do you tackle the "older style" patient who says "you are the doctor" and "I will accept whatever you decide" "I know you will do whatever is best for me"?
A. In that situation, I usually respond with "It is important then for me to understand a bit more about you and what matters to you, so that I can work out with you what might be the best". Talk through the options with the patient, stating that you are trying to put yourself in their shoes to help reach the right decision for them. I will always talk through all of the options and explain why I think one option might be the best, and why some options would not work.

Q. How do we balance patient safety and their autonomy?
A. Different cultures will answer this in different ways. In western medicine, we place autonomy as the most important of the four core ethical principles, and if a patient, who is judged to be competent, makes a decision that in our view, is not in their best interest, we have to respect

it. Other eastern cultures and religions will not necessarily concur, ranking beneficence and non-maleficence as more important, and we should respect that. There is also the question about what 'patient safety' really means. To be entirely 'safe', we might choose to remove a patient's freedom to make their own decisions, live independently etc., and place them in a supervised (supported) environment. Is that necessarily right or wrong?

Understanding the 'human' factor – Dr Vicky Tallentire

Q. In more short lengths of stay and differences in AMU physician management- how much assessment was there on the stress of caregivers receiving more rapid short lengths of stay?
A. I'm not sure this has been studied in much detail, but is certainly a consideration. We are all under pressure to move patients on more quickly and it most certainly impacts on stress levels for all members of the team.

Q. What if your whole organisation is infected with incivility? Only option is to leave?
A. Yes, this is certainly a sad state of affairs when it occurs, and is often a vicious cycle (in families too, not just organisations!) I wouldn't wish to work somewhere that people are rude to each other on a day-to-day basis. We are all products of our environment, at home and at work, so be careful which environments you choose to be most influenced by!

Q. Do consultants who publicly humiliate subordinates not know that they are doing harm by lowering self-esteem of their "victims", any advice?
A. I think that there is often a total lack of insight and/or empathy. Look for good role models, they are out there, it's just that their behaviours might not be quite as memorable. And there is value too, in seeing the sort of person who you don't wish to become – it is powerful and even helpful in its own way.

How do I manage pyrexia of unknown origin – Dr Elham Khatamzas

Q. There is a huge pressure to give antibiotics for "Sepsis" when this may often be a viral illness with pyrexia and some aspects of SIRS. Is there any evidence for observation and delayed antibiotics in fever?
A. Whilst there is as you say significant amount of evidence for timely administration of antibiotics in sepsis, there is no good quality studies that have demonstrated the lack of harm in using clinical judgement to observe patients with fever. For PUO cases that have by definition had a "fever" for more than 3 weeks and are most likely clinically stable, the consensus is to observe and make diagnosis unless high clinical suspicion of endocarditis, miliary TB or patient becomes clinically unstable. In my view clinical judgement taking into account patient history and risk factors should make the basis of any decision about initiating antibiotics and stopping after 72 hours if no improvement.

Q. Can thyrotoxicosis present as PUO?
A. Yes it is rare but is both thyroiditis and thyrotoxicosis have presented as PUO belonging to the miscellaneous causes.

Q. How common is antibiotic associated temperature?
A. In my experience more common than we think but I'm biases as an infectious diseases specialist. It should be considered after careful exclusion of other causes and typically fever ceases quickly on withdrawing the drug. Whilst all agents can induce a fever, some antibiotics such as beta-lactams and also glycopeptides are more commonly implicated, but that may just be a reflection of their frequent use. I'm not aware of any good quality studies about the incidence.

How do I manage the acutely jaundiced patient – Dr Michael Williams

Q. Role of UCDA in cholestatic jaundice?
A. There are theoretical reasons why UDCA may be helpful in a range of cholestatic liver diseases, but the evidence for its use in conditions other than PBC is lacking. Personally, I do use it in PSC and ischaemic cholangiopathy (it improves liver enzymes in many patients), and occasionally in drug-induced cholestasis if very symptomatic but there is no good evidence it affects outcome.

Q. Will you use of iv nac for any form of acute liver failure?
Q. Is there a role for N-acetylcysteine in non-paracetamol liver injury/failure?
A. I do not currently use NAC in non-paracetamol-related acute liver failure. There is some weak evidence to suggest that NAC may be beneficial in this setting. However, this is a very diverse group and we know that different aetiologies have different likelihoods of spontaneous recovery. Larger studies are therefore needed to be confident of a benefit.

Q. Comments on steroids vs pentoxyphylline in alcoholic hepatitis?
A. The evidence from the STOPAH trial is clear that pentoxiphylline has no effect in severe alcoholic hepatitis. The evidence for steroids is borderline and open to differences in interpretation. Optimists point to a slightly improved 1 month survival; pessimists point to no effect on survival at 3 months or 1 year. Work continues to identify a subgroup of patients who may have more benefit (e.g. those with a neutrophil/lymphocyte ratio of 5-8) but this remains speculative.

Q. There's often a lot of reluctance to give lots of fluids to patients with acute liver failure and ascites. Which fluid resus regime would you recommend e.g. crystalloid versus albumin?
Q. Role of iv human albumin and terlipressin in acute liver failure?
A. Patients with acute liver failure can develop a degree of portal hypertension due to architectural distortion of the liver, but large volume ascites is relatively uncommon in this situation (as these patients do not have the same chronic sodium retention that is seen in cirrhotics). I would generally therefore recommend crystalloid but would switch to albumin if ascites was becoming problematic. Similarly, hepatorenal syndrome is more common in the setting of cirrhosis but is described in acute liver failure. Progressive renal failure in this setting is likely to require renal replacement therapy, and will be treated by transplantation. However, terlipressin can be considered if ascites and persistent renal dysfunction despite hydration.

Q. Is it ethical to refuse transplant for patients with psychiatric illness?
A. Selection of patients for transplant does frequently raise ethical issues, as we currently still have a shortage of donor organs relative to the number of potential recipients. Decisions on organ allocation therefore take into account predicted survival with and without a transplant to ensure the most equitable use.
I would not refuse transplant in a patient with treatable psychiatric illness who was felt to have a reasonable likelihood of complying with treatment post-transplant. Our concerns usually focus around those with a pattern of repeated overdoses (usually in the setting of untreatable personality disorders) or ongoing substance misuse problems that might result in non-compliance, as this has a detrimental effect on graft outcome.

Q. How reliable is fibrosan to diagnose cirrhosis?
A. Fibroscans are very helpful in assessing fibrosis but come with several caveats. Firstly, the cut-offs for diagnosing cirrhosis are disease-specific so should be interpreted with care. Secondly, estimates of sensitivity and specificity are limited by the lack of an absolute gold standard. Many studies compare Fibroscan to liver biopsies, but we know that there can be significant variation in fibrosis

staging in two biopsies taken from the same patient on the same day. We can therefore assess for correlation but where discrepancies arise, it is difficult to say with confidence which test is correct.

Q. When should I worry that it might not be Gilbert's? Is History enough?
A. I would be confident diagnosing Gilbert's in a mildly jaundiced patient (bilirubin <80) with normal liver enzymes and normal haemoglobin, especially if history of previous similar episodes. If concern, check conjugated bilirubin level. Genetic testing (TATA box test) possible but usually only needed in complex cases (e.g. Gilbert's and coexistent liver disease).

How do I manage anticoagulation – Professor Henry Watson

Q. Supra-renal function is a contraindication to some DOAC's but not others - but is it likely to be a class-related problem?
A. I suppose it is a phenomenon of any drug that is dependent on renal clearance where the AUC becomes lower at high GFR.

Q. Is there a role for ROTEM/TEG style machines in assessing anticoagulation with DOAC use?
A. No none of the TEG/TEM measurements correlates well with DOAC concentrations as far as I know.

Q. Patients with APS who were historically put on DOAC due to very difficult to control INR who have not had clots since then, would you put them back on warfarin due to recent MHRA guidelines?
A. These cases are difficult as you can argue that in the event of poor INR control you might be better off with a DOAC. However, the rate of events was so high in the rivaroxaban group of the TRAPS study that you could almost argue that the rivaroxaban was detrimental – not just of poor efficacy.

Q. Would drug levels be better than PTr in monitoring anticoagulation in patients with VTE Disease?
A. I am not sure what you are asking. For patients on warfarin the INR should be used, for patients on DOACS there is no need for any monitoring of anticoagulant effect or drug concentration in 99% plus of cases.

Q. What is the role of low dose aspirin in patients with Lab criteria of APL without clinical criteria?
A. None. The clinical trials of aspirin in patients with antiphospholipid antibodies but no clinical event showed no evidence for benefit. This is covered in the current BCSH guideline on APS.

Q. We don't routinely screen for antiphospholipid syndrome in most patients with unprovoked VTE. Should this practice change? Most of these end up on DOACs.
A. Well we have started to do this because part of the differential for unprovoked VTE is APS and that diagnosis would change your management – so unfortunately I think the answer is yes.

Q.Is it important to distinguish mechanical valve anticoagulation (mandates warfarin) and biological valves (generally no anticoagulant but DOAC OK for AF with biological valve prosthesis)?
A. Of course

Q. Any role of anticoagulation in seronegative APLS?
A. I don't really recognise seronegative APS as an entity. If you do then I assume that you test patients with stroke, TIA and VTE and if they are negative – call then seronegative APS. That makes no sense to me.

Q. In intracranial haemorrhage due to DOACS when used for AF would reversing anticoagulation actually significantly increase prothrombotic risk and risk concomitant ischaemia due to the underlying AF?
A. The whole clinical entity of life threatening haemorrhage increases the risk of thrombosis for several reasons. These include decreased O_2 delivery subsequent upon the reduced blood pressure, compensatory tachycardia and ↑SV increase O_2 requirements, platelet activation induced, transfused cells low 2,3,DPG increasing Hb affinity for O_2 – sink effect, transfused cells consune NO and maybe most importantly antithrombotic drugs are discontinued for several days. However, in the context of significant ICH it would be the correct thing to attempt to stop bleeding and then try to deal with the situation subsequently.

Q. How long do we need to wait after last dose of NOAC to performing procedures like diagnostic pleural tap or diagnostic ascitic tap? (assuming no other risks of bleeding and moderate urgency to perform the procedure).
A. In someone with normal renal function 24 hours.

<u>The public understanding of risk – Dr Sander van der Linden</u>

Q. Any tips for the best way of explaining 95% confidence limit to a lay person?
A. This depends on whether it is a Bayesian or Frequentist interval.

1) A Bayesian model interpretation would be quite straightforward: there is a 95% probability that the true value lies within the interval.

2) A standard frequentist 95% interval is more complicated to explain. The ideal explanation is this: if the exact same procedure/surgery were repeated many times over, and a 95% confidence limit would be constructed each time, then 95% of the time, the confidence limit would contain the true population value.

The more we simplify this, the less correct it gets, but a simpler way:

3) If the assumptions of the model are correct, 95% of such intervals would contain the correct value. I think the key issue here is to be careful not to give the impression to the patient that a) the 95% limit is a probability or likelihood (it is not, only if Bayesian) and b) that the true value lies within the specific interval (we don't know, we only know that 95% of intervals contain the true value, not whether any specific interval contains it).

4) Least accurate, but OK if confident that the model is correct: the 95% confidence limit gives a range of plausible values for the true [population] outcome.

This is probably the most patient friendly explanation, but stress that this is only true if we assume that the underlying model is correct and the assumptions hold.

Q. Is realistic medicine going to introduce even more uncertainty into the clinical work (not doing scans on certain patients due to frailty or perceived lack of treatment options)?
A. I am not sure if I am qualified to answer this question but I generally support the approach to empower patients to engage in shared decision-making with a full and transparent overview of treatment harms and benefits to determine what course of action is in the best interest of the patient rather than solely relying on the "doctors know best" principle.

Q. Occasionally I quote a risk to a patient and they reply "well that will happen to me because bad things always happen to me". How should I reply?
A. There's several approaches here. One is to explain optimism and pessimism bias. Optimism bias is a cognitive bias that causes people to think that they themselves [in particular] are less likely to experience bad outcomes ("oh I've smoked my whole life, cancer won't happen to me") vs. pessimism bias where people overestimate the probability that bad things will happen to them.

Here it can be useful to give a reminder that bad things can happen either way: The treatment has risks, and not being treated also has risks and highlighting both how serious as well as how likely the risks are in each case. And likewise the benefits (of treatment and of non-treatment), of course. For people who see themselves as "unlucky" when it comes to very low-probability risks, Calman's Risk language and dialects can be useful for making clear exactly how unlucky you'd have to be. For example, for a 1 in 1000, "This level of risk is analogous to the chance of an individual being selected at random out of a line of people standing one metre apart stretching for one kilometre. In community terms it means that "you could expect to find about one person affected in every population grouping the size of a rural village or an inner city housing estate." So out of all those unlucky people who have this medical issue and are facing a treatment decision similar to this one, you'd have to be the unluckiest one.
In short, help contextualize people's judgement.

Q. How do we identify fake news in medical literature and symposia where there is author bias?
A. Here are some helpful rules of thumb. Do check:

A) The credibility of the journal the research is published in (e.g. is this a predatory publisher? E.g. "The British Journal of Medical and Health Research" – sounds legitimate, but it probably isn't. There is a long list of predatory journals you can check against: https://predatoryjournals.com/journals/

B) The funding source at the end of the paper is probably the most important thing; https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5015869/ gives some idea of how mind-bogglingly serious funding bias can be. Beyond checking funding sources & conflict of interest (COI) statements,

C) it can be worth Googling and searching https://retractionwatch.com/ for author names to see if they are involved in any ongoing controversies (and if so, looking more closely into the nature of the controversy; Retraction Watch occasionally 'names and shames' even for relatively insignificant violations of publishing norms, so it's important to read the post to see what the author is actually accused of).

<u>Pulmonary emboli – Dr Jay Suntharalingam</u>

Q. If D-Dimer is only helpful in patients with a low clinical probability and a negative result - should its routine use in all chest pain and shortness of breath presentations be discouraged as it encourages mis-labelling of VTE as diagnosis or false reassurance in higher risk patients?
A. Yes, exactly. D-Dimer should only be used in patients in whom PE is suspected but score as 'low risk' on a two level Wells score. Indiscriminate use may lead to patients being over investigated or undergoing delayed investigation

Q. Do you use age-adjusted d-diner cut offs?
A. Using age related D-Dimers can certainly reduce the chances of false positive results in more elderly patients. We do not use age-adjusted D-Dimer cut offs in our own institution but there is a good evidence base for doing this in patients with a low probability of PE, to reduce unnecessary CTPA requests

Q. When to check for hereditary thrombosis syndromes leading to hypercoagulability when you have already started the patient on anticoagulation?
A. There is little firm guidance on which patients to consider thrombophilia testing in, so I would recommend being guided by your local haematology department. As a rule the majority of patients do not require thrombophilia testing as it rarely changes management, so I would not recommend empirically testing for this in patients before starting anticoagulants – decisions regarding the duration of anticoagulation are usually influenced by many other factors.

Q. Would you routinely check troponin level when investigating for PE?
A. The latest ESC guidance would suggest doing this in all patients, to help risk assess patients more robustly. Conversely the latest BTS guidance, which focusses purely on potential low risk patients who may be treated at home, suggests that a troponin is only required in clinically low risk patients (ie low sPESI, PESI or Hestia) who have evidence of right heart strain on CT or echo. Both guidelines advocate that all patients with high clinical risk should have their troponin levels checked.

Q. To PERC or not to PERC?
A. There has been some interest recently in developing PERTs (PE Response Teams) to help manage high risk or intermediate high risk patients acutely. The teams typically draw on a number of disciplines (eg Cardiology, Respiratory, Radiology, Haematology, Critical Care) and interact 'live' to help develop the best individualised management plan for patients. Although ideal this solution does depend on having resources available to staff a team. Modern IT solutions, such as web-based teleconferencing, may help facilitate development of these teams in smaller or more remote hospitals in the future.

Q. Has the PESI score ever been used in the non-PE population? - it occurs to me these are risk factors for death in most emergency presentations....
A. I'm not aware that it has been validated in other diseases. High risk PE may exhibit differently to eg high risk ACS though, so I would be surprised if it performed well in all conditions.

Q. Do miller scores inform management?
A. Miller scoring is a radiological tool for quantifying clot burden to a segmental level. Although this would give a little more information, it would be better in the first instance for all hospitals to improve their standard of reporting, to comment on the presence or absence of right strain and a global description of clot burden (ie low or high) – the NCEPOD data suggests this is not happening in >50% of sites.

Q. What is the guidance on thrombolysis for refractory hypoxia? Perhaps using 50mg dose rather than 100mg?
A. Hypoxia is not necessarily an indication for thrombolysis, and usually improves with time. In patients who are haemodynamically stable, and who have evidence of good end organ perfusion, I would avoid thrombolysing purely for hypoxia and simply observe them in a high dependency setting, in case things change. Hypoxia can usually be corrected with nasal high flow as an interim in this patient cohort.

Q. Why can't you put a shocked patient in an ambulance and drive them 30 miles? If there is a high risk of them dying at the base hospital and a potentially lifesaving treatment available at the tertiary centre then transfer could be a sensible choice.

A. This may certainly be the right choice for an individual patient if there are no local options available to them. Conversely, eg a percutaneous approach may be available locally and may be delivered quicker and therefore more effectively. A PERT (see earlier) can help make the best decision, based on the available resources, in this situation.

Q. Assessing for PE, when should we start acting for thrombolysis?
A. Those patients with a suspected PE who are haemodynamically unstable should undergo rapid assessment with a bedside echo or CTPA. If either shows evidence of PE then they should be thrombolysed as soon as practicable.

Q. Is there a role for thrombolysis in the CVS stable but rising lactate population (no other cause)?
A. Patients who have evidence of end organ perfusion should certainly be considered for thrombolysis. It would be a little unusual to see a rising lactate in isolation (e.g. with normal haemodynamics, normal cerebral function, good urine output etc.) so worth excluding any other pathology first.

Q. The data on lack of use of formal risk tools is alarming. Do you have examples of health systems where risk assessments have been routinely incorporated? Is this a technology or leadership problem?
A. I suspect that this a multifactorial problem and is partly due to a lack of awareness – strong leadership at a local level would probably help highlight this and improve things going forward.

Q. If a patient is not suitable for early discharge, for how long should they remain as an inpatient?
A. There is no firm guidance on this but one suggestion has been that once the reversible parameters on a PESI or sPESI have improved to 'low risk' status then this could be considered a safe time to discharge a patient.

Q. How long should be the anticoagulant in unprovoked PE and provoked PE?
A. In a truly provoked PE, 3 months' anticoagulation is usually recommended. In unprovoked PE then the potential risks of recurrence needs to be balanced against the potential bleeding risk if anticoagulation was continued long term. There are scoring systems (e.g. DASH, HERDOO) that can help inform clinician-patient discussions.

Q. What is best medical management of CTEPH if frail / not fit for surgery?
A. Only approximately two thirds of patients have disease that is proximal enough to be amenable to pulmonary endarterectomy (PEA) surgery. Of these some patients are too frail for surgery, although this decision should only ever be made by a PH centre, following discussion at an expert PEA MDT. Other options do exist for these patients, including medical therapy (including PDE5 inhibitors, sGC stimulators and ERAs) and, more recently, pulmonary angioplasty.

Q. When would you use catheter targeted thrombolysis?
A. I would only consider thrombolysis in patients with massive PE ie those who are haemodynamically compromised as a result of acute PE. I would normally use systemic thrombolysis first line and only consider catheter directed thrombolysis if there was a contraindication to systemic thrombolysis.

Q. Incidental small peripheral PE found on CT - treat or not?
A. The answer to this is unclear. The UK is starting a prospective study to answer this question directly so hopefully there will be clearer guidance on this in the future. The most recent ACCP guidance suggests that patient with subsegmental PE and no DVT could be managed with surveillance alone when the risk of VTE recurrence is low.

Q. What would you consider "appropriate follow up"?
A. Follow up should cover assessment of ongoing symptoms, review of compliance, assessment of side effects, a check that limited screening for malignancy has been completed, plans have been made regarding duration of anticoagulation and long term complications such as CTEPH have been excluded.

Q. In stroke we have to thrombolyse within 4.5 hours. In PE can thrombolyse a monitored patient who destabilises but is there a time limit max between first symptoms and lysis?
A. No, there is no set time limit. The majority of patients start to improve shortly after they start anticoagulation. If patients deteriorate on anticoagulation due to PE extension then thrombolysis could be considered, without any necessary arbitrary time limit.

Q. What is discharge criteria for intermediate risk? How long should they be an in-patient?
A. See earlier response to similar question

Q. When is the right time to repeat CTPA following a diagnosis of PE as very often patients re attend in A&E in less than 3months?
A. If you are concerned that the patient has developed CTEPH then I would advocate waiting until the patient has completed at least 3 months' anticoagulation. If you are concerned that the patient is very pro-thrombotic and has developed more acute clot, despite anticoagulation, then you may need to consider a scan before this.

Q. How to differentiate p.htn from other causes rather than from embolic disease?
A. Imaging is the key modality that differentiates CTEPH from other forms of PH. VQ is the most sensitive test, but is not always specific. CTPA can be sensitive but is operator dependent. Current guidelines suggest using VQ as the first screening tool in patients with suspected CTEPH – a normal VQ discounts the diagnosis.

<u>The difference a plan can make – Dr Juliet Spiller</u>

Q. We have lots of screening programs where age and comorbidity trigger routine screening (i.e. "well man" clinics) - Should we not be extending this to Advance care planning in those >70yo / chronic medical problems / recurrent exacerbations / reducing functional ability?
A. Absolutely, and this is what many tools like eFrailty or SPICT can be used to do. However an embedded and consistent approach to advance care planning (anticipatory care planning in Scotland) is still far from routine practice so the ReSPECT process, whilst only addressing the emergency bit of ACP, could be embedded as a consistent, manageable and recognisable place to start with someone who has 'triggered' in primary or secondary care.

Q. Any tips about how to open a conversation with a patient about dying in the next x weeks to months (where the patient isn't expecting it)? "What matters to you" is great but a broad stroke and doesn't particularly convey death or dying...

A. You're right so a general question about the patients understanding is a safe starting point that lets you know where their thinking is at. You can then gently explore what might be ahead for them if they are able to do that. 'Are you up to thinking about some 'what if' situations -like 'what if you were to suddenly become much less well?' Always let them know they have choices and that the focus of anything offered will be achieving what really matters to them. The REDMAP structure below can be useful to frame a conversation.

EC4H Effective Communication for Healthcare — THE UNIVERSITY of EDINBURGH — NHS SCOTLAND — Effective Communication F

Talking about Care Planning: RED–MAP		
R eady	**Can we talk about your health and care?**	When would be a good time to talk? Who should join us? This about making good plans for your treatment and care.
E xpect	**What do you know?** **What do you want to ask?** **What are you expecting…?**	How have you been doing recently? What has changed? How do you see things going in the next days/ weeks/ months….? **Some people** think about what might happen **if…?** Can we talk about what might happen if you get less well?
D iagnosis	**We know…** **We don't know…** **Questions or worries?**	What is happening with your (*health problem*) is… We **hope that**…, but I **am worried about**… **It is possible** that you might not get better because… We **don't know** exactly when…, can we talk about that? Do you have questions or worries you'd like us to talk about?
M atters	**What matters to you?**	What's **important to you** that we should know about? Are there things **you'd like** or **wouldn't want** for you?
A ctions	**What can help…** **This does not work…**	Things we **can do** are…. **Options** we have are… This **does not work** because…./ **will not help** when/if….
P lan	**Let's plan ahead for when/ if….**	Can we make some plans so everyone knows what to do? Talking and planning ahead **'just in case'** helps people get better care.

Agreeing that such conversations with patients and relatives is everyone's responsibility, do you think "40 seconds" Are enough to do that in a ward round or OP consultation? Or does it just refer to time required to win their confidence on you, and THEN the conversation starts ... ?

The '40 seconds' simply refers to the evidence that being compassionate in every clinical interaction does not take much additional time (contrary to what most clinicians believe). Being perceived as compassionate has all of the added benefits including being perceived as trustworthy and competent which are both essential to any conversation around ACP. If you regard ACP conversations as a core part of your job which should be true for every clinician then practicing compassion will make it easier to have effective ACP conversations – a win-win!!

Q. Thoughts on 'palliative' antibiotics?

A. Oral antibiotics can certainly provide palliation in terms of improving symptom control and reducing delirium as can IV antibiotics. However, the added symptom burden of antibiotics can be significant (nausea, diarrhoea, drug interaction, tablet burden/IV cannulation etc). Also a palliative patient who is sick enough to warrant IV antibiotics needs to be aware of the significant risk that they may end up dying in hospital. It is a case of being realistic and honest about the potential benefits and potential burdens and thinking about less harmful and less intrusive alternatives to managing the symptoms the patient is struggling with. Informed shared decision-making and finding out what really matters to the patient is key to helping the patient/family make the right decision for them.

Q. Do you think the resus council's Respect forms are going to help encourage better discussions?
A. ReSPECT is a process not just a form and it is the education to support the culture shift that is key to having better and earlier conversations. However, the form is designed to prompt and support the process of early conversation and easy documentation of realistic and person-centred clinical guidance for an emergency. We know from early adopter sites that using the form as a prompt and focus for the conversation helps clinicians feel more able to do that and patients and families also find the form a helpful visual aid for the conversation. Please don't think that simply dropping the forms into a clinical area will solve the conversation culture issue – embedding the process with education, quality improvement and facilitated engagement is the way to make progress.

Friday 29 November 2019

Wearables in medicine – Dr Ali K Yetisen

Q. Potential harms from wearables? And increase in demand on services (maybe from worried well)?
A. The users should ensure that their medical wearable products have CE or FDA approval and the intended use. Medical wearable devices without regulatory approval should not be used for medical diagnostic applications. However, consumer wearable devices for lifestyle applications are designated as low risk.

Q. Is this technology not likely to meet resistance by patients with chronic health conditions who's insurance companies may require a period of monitoring to derive more refined cost penalties?
A. Medical wearable devices have been used in chronic health conditions. For example, continuous glucose monitoring systems for type I diabetic patients have led to a significant increase in the quality of life of diabetics, as well as preventing long-term disability. These continuous glucose monitors have been endorsed by the NHS (https://www.diabetes.org.uk/get_involved/campaigning/flash-glucose-monitoring).

Q. Is there a possibility in future that we depend on these technologies/devices and lose acuity of our clinical acumen?
A. The penetration of technology in our lives is inevitable; medical wearable devices will allow for monitoring diseases in real time. However, such wearable devices should not be perceived as the decision makers. They have been designed to assist medical professionals based on their clinical acumen.

Q. Any medical school's starting to incorporate this in their curricula?
A. Technology has become an integral part of medical education. Wearables as emerging technologies should be integrated in existing medical education curricula. There are several initiatives, such as the Clinician Engineer Hub, (www.clinicianengineer.com), to bridge the gap between engineering and medical education.

Exercise and Health – Dr Gregor Smith

CMO Guidelines main report
https://www.gov.uk/government/publications/physical-activity-guidelines-uk-chief-medical-officers-report

Technical reports for each age group which accompanied the report
http://www.bristol.ac.uk/sps/research/projects/physical-activity/final-working-group-papers/

Tachyarrhythmias – Dr Paul Broadhurst

Q. How big is the risk of Adenosine in patients with severe asthma?
A. Risk of aggravating asthma with iv adenosine is largely anecdotal or theoretical but it would seem sensible to avoid it in severe asthma and use an alternative e.g. iv verapamil in such cases.

The jugular venous pulse – Dr Andrew Flapan

Q. What happens to JVP in acute VSD post mi?
A. Depending on the state of the R.ventricle it may not change that much but if the R.ventricle dilates there will be a raised venous pressure often with V waves as the patient develops tricuspid regurgitation.