Spring Snippets

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Wenckebach phenomenon is the heart block we all know - or do we? I was puzzled recently by an ECG that looked like Wenckebach (with classic "grouped beating", in triplets) but where the 2nd and 3rd PR interval seemed to be of identical duration prior to the dropped beat, instead of the classical description of each successive PR interval getting longer prior to the dropped beat. It turns out that "classical" Wenckebach (which we are all taught) only accounts for 15-30% of Mobitz Type 1 2nd-degree HB! All the rest - the majority - are "atypical".

A fab open-access article in Clinical Cardiology (Barold, 2018)¹ highlights a "useful but generally ignored" 1978 WHO definition: "intermittent failure of impulse conduction in which the blocked impulse is preceded by prolongation of conduction time relative to the first conducted impulse" and recommends, "...at a minimum, the term "progressive" pertaining to prolongation of the P-R interval in the traditional definition of type I block should be replaced by the word "variable" to describe the changing P-R intervals before a blocked impulse."

Whilst on the topic of ECGs, our usual practice assumes standard lead positions when acquiring a 12-lead ECG. Modified limb-lead positions are used during exercise stress testing (the "Mason-Likar" or "M-L" ECG lead adaptation), but ECGs taken in the M-L lead positions can have clinically significant differences to standard ECGs², most commonly right axis deviation and they can "produce or obscure changes of myocardial infarction". If you need to use an ECG with modified lead positions, interpret with caution and make sure it is clearly written on the ECG that the lead positions were non-standard.

Bronchiectasis patients may grow some weird bugs in their sputum, some sensitive to antimicrobials limited to the IV route. Be aware that most (?all) respiratory consultants will want to know and personally decide if treatment is required, so discuss with them before acting on a sputum culture result.

Welcome to my first "Bog Blog" of 2019 - designed to be printed out and put on the back of the loo door! It's a pot-pourri of things I have learned/re-learned/asked about/thought about recently. The content will mainly be relevant to GP & EM, but hopefully there'll be something relevant to any clinician

In Emergency Medicine, any thoughts about Myasthenia Gravis tend to be about the potential for respiratory failure but don't forget that many commonly-used drugs, including some antibiotics, can be dangerous to patients with Myasthenia Gravis. MG patients are usually well briefed and very aware of the need for caution, but there are emergency situations where one could forsee a trip-up in a resus room situation.

For example, aminoglycosides are a particular hazard in Myasthenia Gravis, so if gentamicin is included in the antibiotics your institution uses for suspected urinary sepsis, be mindful of this. Ciprofloxacin, erythromycin and many other antibiotics are a problem too.

Parenteral magnesium is an issue, and with the recent LOWMAGHI trial results³ supporting a 4.5g dose of magnesium in ED patients with fast AF, it's not too difficult to imagine a situation of an obtunded patient with MG being brought to ED and accidentally given drugs they shouldn't be exposed to. Always check!

13% of oesophageal cancers in the UK are said to be caused by alcohol drinking⁴, with a greatly increased risk in heavy drinkers, so it's sadly not uncommon to have patients with oesophageal varices who also develop an oesophageal SCC. Oesophageal stents, however, reduce the risk of variceal bleeding (the stent tamponades the varices) and so if the meds to control portal hypertension (i.e. spironolactone, propranolol) are causing problems in a patient with an oesophageal stent, you might be OK to stop them.

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- Comments? Questions?
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Refs:

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