

Miscellaneous Tips

These are like, lots of different tips that, like don't fit perfectly into one of the other categories, like. Starting with like drugs like.

DRUG MANAGEMENT

DRUG MANAGEMENT TIP 1: IT TAKES THREE YEARS TO TREAT POLYMYALGIA RHEUMATICA

Actually three years and ten weeks.

At least.

Doctors make two major mistakes in the management of PMR. The first is to start prednisolone at too high a dose. If prednisolone 15 mg daily makes the disease disappear *as if by magic*, why would anyone start at 30 mg? Particularly since giving 30 mg loses the diagnostic confirmation that the resolution of symptoms *as if by magic* gives you (lots of different diseases will get a lot better on 30 mg, but only PMR will get better *as if by magic* on 15 mg). It's almost as if the connection of PMR with temporal arteritis – where you jump in with 30–40 mg prednisolone just in case the patient's about to wake up next morning blind in one eye – befuddles them into the reckless use of stupidly high doses against the desperate worry that otherwise the patient may wake up next morning . . . still with stiff shoulders.

The second mistake is arguably precipitated by the first. Nobody wants to be on 30 mg prednisolone for very long, so the dose is brought down quite

sharpish every few days to 25 mg, 20 mg, 15 mg . . . and this sequence is continued . . . 10, 7.5, 5 . . . at which point *all the pains come back again*. And unfortunately in my experience once you lose control, very often the whole thing never quite works properly.

So what you should do is start lowish, and you reduce the dose ridiculously slowly. It goes like this:

15 mg	10 weeks
12.5 mg	6 weeks
10 mg	6 weeks
9 mg	6 weeks
8 mg	6 weeks
7.5 mg	6 weeks
7 mg	6 weeks
6.5 mg	6 weeks

. . . and so on, with half-mg decrements every six weeks.

Now, the mathematically astute, and smart-alec guys among you (girls won't have done this¹³⁴) will have spotted that that's 24×6 weeks plus 10 weeks = 154 weeks = slightly *less* than three years. But nobody's really gonna remember to change every six weeks. Much better to change every 1.5 months, so every clinic visit I write down a 'template' for them to follow . . . *1st Jan – 10 mg, 15th Feb – 9 mg, 1st April – 8 mg, etc.* So it's 24×1.5 months plus ten weeks.

By following this slow reduction, patients maximise their chances of getting through to the end without a hitch. They'll be tempted to go faster, but persuade them not to. Tell them 7.5 mg prednisolone is pretty much what their own body normally makes steroid-wise . . . and side effects are very limited once they get down to that level . . . and they will get there quite quickly. I always tell them this, and it might even be true.

If aches come back a few days after a dose reduction, that's OK *as long as they all settle down before the next dose reduction is due*. Then you can go down the next step. If they don't settle, you go up one or (a maximum) two steps, get it back under control, then reduce even more slowly. You never (oops), ever (if you follow the plan) have to jump back up to 15 mg.

In answer to your questions.

- 1 Yes, weekly from the start in any older female. In males, I'd check a densitometry scan first to see if appropriate.

¹³⁴ Again, because it's a waste of time, *not* because they ain't mathematically astute.

- 2 No. When they get OA pains or frozen shoulder pains you don't pretend it's the PMR coming back, and you don't up their steroid dosage.
- 3 What about it? You should only use it to help you with (2). My own theory is that steroids bring it down *per se*, so as the steroids are stopped it will naturally go up towards the normal for a person of that age.¹³⁵

DRUG MANAGEMENT TIP 2: ODs AREN'T BD OR TDS

Theoretically, people who take overdoses are trying to kill themselves. Admittedly, in the real world it's more often a 'cry for help' – though clearly destined to fall on deaf ears since this particular real world is populated by hard-bitten doctors and harder-bitten nurses whose sole priority is to ensure you stay alive overnight (or at least until their shift finishes) rather than sort out all your personal and social problems of which they've got plenty enough of their own, thank you very much . . .

But whether it's a genuine cry for help or a cry for help, it's maybe too much to expect anyone going through either trauma to remember *exactly* 'when did you take the tablets?' But of course that's what we always ask them. Most of the time it's not particularly important, but there is that thing with the paracetamol.

I've always rated paracetamol overdose as a top diagnosis to look after since you get to use the word 'antidote' in all seriousness. People don't really do 'antidote' any more, but it was my favourite medical word from childhood days (certainly well ahead of 'kaolin poultice'). And paracetamol has actually got an antidote! So I take great pleasure in rhyming this off to all prospective antidotees – since all we need to tell us whether or not to use the antidote (see how seductive it is?) is the blood concentration of paracetamol, the time of the blood concentration and *exactly when did you take the tablets?*

But if a patient 'can't remember' in a mumbling voice because they were 'too drunk' (oooh . . . more chance they will need the antidote, as the level requiring antidote treatment is decreased) that puts the kybosh on the entire plan. Maybe.

Back in the old days, there wasn't much you could do other than take a guess whether or not to treat¹³⁶ since somehow, somebody, somewhere got it into their head that the antidote only worked properly in the first eight hours, patchily in the next four hours, after which it actually made things worse. The

¹³⁵ All right then – it's 'what about the ESR?'

¹³⁶ Seems a no-brainer. Treat, just in case. But what if the patient who does get the anaphylactic shock from the antidote does turn out to have taken exactly three paracetamol tablets?

weird thing was that we all swallowed this tosh since the proffered explanation *vis-à-vis* toxic and non-toxic metabolites and preferential pathways and stuff seemed to make sense and charmed the frontal cortexes¹³⁷ of young doctors in much the same way as long-term endowment policies. So there wasn't really time to perform the salutary manoeuvre of checking the blood paracetamol levels twice, and then draw the line between them, showing how fast the levels are dropping – and surmise the likely timing by fitting it onto the standard when-to-treat-paracetamol-poisoning graph which essentially outlines the normal decay of paracetamol levels – and Bob's your uncle.

The fitting-the-line part is a bit of a faff, but there is a quick way to get a yes/no answer on whether to treat. You take the standard graph, thus (see Figure 3).

You then get the first result (with no 'time-since' concept) and slap it on the treatment line ('1'). Then you mark the next blood result at the correct time *relative to the first result* ('2'). If this dot is above the line, treat.

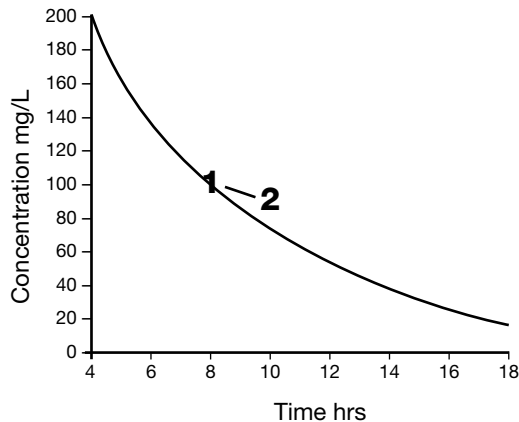


FIGURE 3 Paracetamol overdose

Predicting antidote requirements from two results of uncertain timing. In this case a result of 100 at unknown timing, with a result of 90 two hours later.

137 I know. But everybody seems to get befuddled when I mention 'Drug Kardices' so . . .

(How long should you wait before doing the second level? Not known. Maybe because this is a personal, anecdotal-type tip. It's like . . . long enough to be able to draw a sensible line, within the limits of the reliability of your assay. Maybe two hours? As I say, it depends on your assay. My own theory is that the shortest time possible to get the junior to do a blood test and then do another one should be time enough . . . And I should mention that I invoke this tip only when the decision has been made to NOT treat – to give an extra chance to get it right. I don't delay treatment where otherwise it would have been given.)

DRUG MANAGEMENT TIP 2a: TO BE USED IN CONJUNCTION WITH DRUG MANAGEMENT TIP 2

The antidote is N-acetyl cysteine.

DRUG MANAGEMENT TIP 3: ALTER PHENYTOIN DOSAGE CAREFULLY

There's a story about the best way to get drunk at a party – as told by clinical pharmacologists (not actually told *at* parties since, as outlined elsewhere, clinical pharmacologists do not get invited to such things).

The start of the plan is to drink the alcoholic beverage of your choice reasonably rapidly¹³⁸ until just nicely tipsy. Thereafter you drink at a steady one-unit-alcohol-per-hour as, the theory has it, most people's metabolism when fully up-and-running will break down one unit of alcohol per hour – so you can stay at that nicely-tipsy state. Any faster, and the extra alcohol will accumulate and you'll get hammered.

You might think from this that if you drink $\frac{3}{4}$ of a unit per hour from the start, then you'll pretty much never have any alcohol in your system. But that unit-per-hour is the *peak* rate. If you are drinking less and your blood levels are less, your enzymes don't work so hard, and you'll still get a bit tiddy.

So what's this all got to do with phenytoin? Well, the hourly alcohol story is a bit like the daily phenytoin story. Whilst your system gradually increases its efforts to metabolise phenytoin as the dosage increases, there quite quickly comes a point where its efforts reach a plateau – and the concentration at this point is approximately where phenytoin actually starts to work. The

138 . . . as opposed to 'as rapidly as possible before leaving the house to avoid having to actually buy the stuff yourself instead of taking dad's . . .'

pharmacology chaps like to talk about a changeover from First-Order to Zero-Order kinetics ('saturation kinetics') and stuff like that, but the bottom line is that even small increases in dosage above this level will cause disproportionately large increases in blood phenytoin levels. You can get the idea from a graph of the 'steady-state' blood phenytoin vs. daily dosage for a Mr John Doe (see Figure 4).

So Dr Doh, who doesn't know this concept, might look at a blood level of 10 mg/L when Mr Doe is taking 300 mg of phenytoin per day and think 'we'd like that level to be 15 mg/L,¹³⁹ so I'll give him a 50% rise in dose to 450 mg per day.' A quick look at the chart shows where that would put the patient (in hospital, probably) once the drug reaches its 'steady-state' (classically in five half-lives' time – around 5–7 days in the case of phenytoin, which does at least give Dr Doh a chance to read a medical book, check with a colleague, or leave the country).

Once phenytoin levels get up anywhere near the 'target range', you should increase only in 25 mg increments. The same goes for reducing the dose when

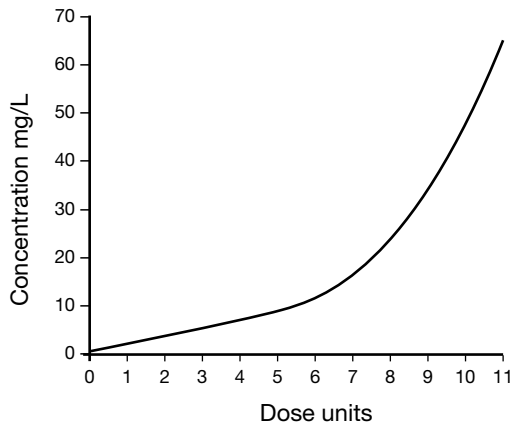


FIGURE 4 Phenytoin concentration vs. dosage

¹³⁹ Middle of 'target range'. But only if the epilepsy is not already controlled. Blood levels are not as important as clinical state.

you think the patient's levels are a bit high. Halving the dose will almost certainly bring levels down to negligible (unless the previous physician was a total tube) so after a missing out a dose or two, a reduction by 25 mg or 50 mg is almost always adequate.

DRUG MANAGEMENT TIP 4: PATIENTS DON'T REALLY NEED FERROUS SULPHATE 200 mg TDS

No clinical evidence for this tip.

But the maths is enough for me.

Men need to absorb 1 mg of iron per day. Women (because of the menses) need 2 mg per day. We absorb approximately 10% of the iron we ingest (as it happens, the % increases if we become iron deficient) so we need 10–20 mg iron per day in our diet.

If you become iron deficient for any of the usual reasons (blood loss, poor diet, etc.¹⁴⁰) there is no conceivable reason why you would want to throw 600 mg per day at the problem – a manoeuvre which can often be counter-productive since a large number of people will get either constipation or diarrhoea (spookily enough) on full-dose iron and will stop taking the tablets. My suggestion would be one tablet per day – 200 mg is ample – and if you have to take them for longer, so be it.

DRUG MANAGEMENT TIP 5: TREAT THE PATIENT'S THYROID, NOT THE POPULATION'S

Thyroid Stimulating Hormone (TSH) is produced somewhere in the brain (I think it's the anterior pituitary) when somewhere else in the brain (I think it's the hypothalamus) decides the body needs more thyroid hormone (thyroxine) floating about. TSH tells somewhere in the neck (I'm pretty sure that one's the thyroid gland) to make more thyroxine. That's why, as somebody drifts towards primary hypothyroidism (i.e. the thyroid itself failing) their TSH keeps getting higher and higher, 'imploring the thyroid to exert itself' as they don't quite say in *Peter Rabbit*. The over-flogged thyroid just manages to keep thyroxine in the 'low-normal range' until finally it packs in completely and we get the low-thyroxine/high-TSH pattern of primary hypothyroidism.

¹⁴⁰ Very occasionally poor absorption might be the primary problem – but that's a totally different and rare ball game (let's call it . . . *pelota*). If you know about it, they may need parenteral iron. If you don't, low-dose iron might help you spot the rarity.

So.

If you treat this with thyroxine, and the patient's thyroxine comes up to 'normal', but the TSH is still high, that means the patient's body wants more thyroxine made. *This 'normal' isn't their normal* (which makes sense since it's just a range in which 95% tend to have their thyroid levels). So in a treated hypothyroid patient where the thyroxine level is normal, but TSH is high, you should give them just a bit more thyroxine.

And you can take it a bit further. If the TSH is slap-dab in the middle of the 'normal' range, but they still have hypothyroid symptoms, even if thyroxine is at a highish level . . . then you can still try a small increase in thyroxine dosage. This is a population normal, not necessarily theirs: bring *their* TSH down into the low normal – maybe that's where it usually sits. Maybe it's actually *up* to persuade their thyroid to make more hormone, which it can't do, so you have to give it in tablet form. Worth a try.

Note, this is for patients on thyroxine already. Not those not on thyroxine who insist they must have hypothyroidism despite the absolutely normal tests and lack of clinical signs because it says in *Red Mariepolitan* magazine that . . .

DRUG MANAGEMENT TIP 6: WARFARIN TAKES A COUPLE OF DAYS TO KICK IN

Let's say it's Monday.

A patient comes in with an unconnected (or indeed connected) illness and the resident/JHO/PRHO/SPV/FY1¹⁴¹ notices they're on warfarin 3 mg, and checks the INR. It's 1.5.¹⁴² The SHO/Reg/ST2/SDF knows it should be around 2.5, so the warfarin is increased to 4 mg.

Tuesday, the INR is 1.6. Clearly the increase wasn't enough so the warfarin that evening goes to 5 mg.

Wednesday's INR is 1.9 so the warfarin is increased to 6 mg.

On Thursday, the INR is 2.5. Everybody's happy, the patient goes home and all's right with the world – except that the INR of 2.5 reflects the increase to 4 mg from two nights earlier beginning to kick in, and she's toddling off home on 6 mg.

Everybody knows it takes a couple of days for the new dose of warfarin to have its effect, but conveniently forgets this since otherwise everything

¹⁴¹ Might have got carried away there. I think one of those is Captain Scarlet's car.

¹⁴² Sometimes I gratuitously ask the junior what the units are, just to watch them sweat – 'cos there aren't any 'cos it's a ratio (vs. normal). Doesn't mean I'm a bad person.

just seems to take too long. It's also a bit counter-intuitive. So if you do have trouble with the mental phase-shift, try this:

Pretend that today's result is the result from two days ago, and see if what you just did with the dosage makes any sense at all.

STATISTICS TIPS

We should all have some basic idea of statistics – if only because it's fun picking holes in people's strongly held convictions so you can win arguments, even when they're right.

STATISTICS TIP 1: UNDERSTAND THE POWER GAME

The Power Game is played in many echelons of the NHS. Particularly in the 'drug procurement' areas of NICE, SMC, major Drugs and Therapeutics Committees, etc. Surprisingly often you'll hear people in these upper echelons, when faced with a surprise positive finding in a drug trial, come out with the 'was the trial powered for that?' canard.

It really doesn't matter.

If you get a significant finding in a trial, it doesn't matter whether it was 'powered' to find that out. It's still a positive finding. Power only matters if you *fail* to show any difference, e.g. between two treatments – then it is important to see if your trial was well-enough designed, had enough patients, clear enough targets . . . , i.e. was *powered* to find the difference if it was there.

As an example, you invent a new antibiotic and you design a trial using 20 patients with pneumonia – 10 get the drug, 10 don't – and you take note of exactly how many die in each group (always good to keep outcomes simple). Arguably the most underpowered trial since Adam 'proved' that you should never do what a woman tells you. No way enough patients. Chances are nobody (or almost nobody . . .) will die in either group, leaving you back where you started. But let's say you happen to do this just at the start of a mini-epidemic of the most virulent pneumonia ever to hit the planet. All 10 patients in your placebo group die. All 10 in your drug group survive. A hugely statistically significant difference. This drug works! You may have been 'lucky'¹⁴³ to prove it – but it is still proven (as much as stats ever prove

143 Lucky enough to end up in jail if you indeed used an inactive placebo.

anything). Small trials often overestimate the extent of a drug's value (the serendipity helping them get the p-value), but the finding that an effect exists remains genuine.

Alternatively, let's say two patients in the placebo group die, but none in the drug group. Since two such events would occur in the same group by chance half of the time, this is no way significant. Even if there were three deaths to none – still not significant. But this would NOT prove that the drug *doesn't* work. Indeed the results are suspicious that it might. This time we do look at the power as we have failed to prove a difference and indeed we see that the trial is hugely underpowered to show a difference that might be there. So this needs a bigger (better) trial.

We'll not go through the actual formula for calculation of power, as I'd have to look it up and that would go against the ethos of this book as well as taking me a few minutes. However, it helps to know it includes such things as:

- 1 Number of subjects in study. More subjects, more power (easier to prove that men are taller than women with 1000 in each group than with five).
- 2 Standard deviation of the thing you are measuring. Less variation, more power (easier to prove which of two classes of schoolchildren is the older, than two disparate groups of random humans . . . say, two railway carriages of people. If we took six kids from each class, it'd be obvious; six people from each carriage, however . . .).
- 3 Expected level of difference between the two groups. Bigger difference, more power (easier to prove 25-year-old men are taller than 3-year-olds than than¹⁴⁴ 15-year-olds).
- 4 How 'significant' you require results to be. Less significance required, more powerful (easier to not-really prove something). Not the *best* way to increase the power.

So the power might be 'an 80% chance of proving at the $p < 0.05$ level a difference in height between the groups (when it's expected to be about 5 cm)'. Such calculations are usually made before a study – at the design stage. The last three are factored in to give an answer as to how many subjects (Factor 1) would be required to give decent power.

So Adam's right then.

144 So why the squiggly red lines?

STATISTICS TIP 2: PICK A CARD . . . ANY CARD . . . TRY AGAIN! . . .

What the person at the top of the previous tip really wanted to know was: *is this 'surprise' a genuine finding or just an incidental when they were looking at so many things and doing so many 'tests' that one of them just happened to be significant?*

We all know the concept. The basic assumption in statistics to us lay-person types is that a p-value of <0.05 is 'significant'. This result would happen by chance less than 0.05 of the time (i.e. less than 0.05 out of 1, i.e. less than 5% of the time, i.e. less than one time in twenty¹⁴⁵). We are happy to accept that level as reasonably unlikely so there must be some significant factor in play. More properly, the assumption is that if you predict something will happen (*see* Statistics Tip 3) and do a study where that something does indeed happen, and you show that the possibility of this happening entirely by chance is less than 5% – then you can take the reasoning behind your prediction as 'proven'.

(Adam should really have asked a whole bunch of women whether he should eat the apple. Not necessarily 20 other women. It's like tossing a coin. Heads/Tails. Yes/No. The first heads is a one-in-two chance. Two heads = $1/4$. Three heads $1/8$. Four heads $1/16$. Five heads $1/32$. So five heads is a less than one-in-twenty chance – if you suspect a double-headed coin, five heads 'proves' it¹⁴⁶ and you shouldn't do what the wife tells you.)

But if you do lots of tests, you will randomly throw up the occasional one-in-twenty chance. We often think of this as 'do twenty tests and one of them will be a one-in-twenty chance.' It isn't as simple as that. If you pick a card from the pack, there's a one-in-four chance it'll be a heart. That doesn't mean if you pick four cards you'll always get one heart. You might get none, you might get two . . . three . . . four. This is one reason why the Bonferroni correction – where you multiply your p-value by the number of tests you've done (e.g. if you do four tests, a $p=0.025$ would become $p=0.1$ and 'lose' its 'significance') is an over-glib attempt to solve this problem, making the criteria too stringent.

There's no simple mathematical way to be precise about the effect of multiple tests. It is perhaps enough to be aware of the problem and view any moderately 'significant' findings picked up in such a way (either by yourself or in others' work) as something requiring further study, but not necessarily proving anything.

145 Not 20–1, but 19–1. Not vital here, but more important down at the lower figures and down at the bookies.

146 Assuming you knew/suspected it was double-headed. *See* Statistics Tip 3.

STATISTICS TIP 3: IGNORE INDEX CASES

So I'm at this conference and this ginger-haired guy's presentation shows: 20% of patients with Disease A will previously have had Disease B – so they must be linked.

And I ask why they looked for this bizarre connection, and it's because they happened to notice three of their patients with rareish Disease A had previously had rareish Disease B so they looked at their whole population.

And I ask if they included their first three 'index' cases . . . and he says . . . 'Yes. Someone told us that we shouldn't do that, but I don't see what the problem is . . .'

Interesting.

It went something like this. They'd got their first three patients – then out of the next 17 cases of Disease A, one had previously had Disease B. Put them together and you get 4 out of 20. 20%! As long as you ignore the good advice: ignore the index cases.

This isn't really a tip. It's a rule.

In the world-sized world of medicine, lots of things will happen by chance. If you notice one of these things and think it may not be by chance, you quite rightly should study this – by starting from scratch. Look at lots of new cases and see if you can prove your theory. In the above example, the best estimate for the Diseases A and B association is 1 in 17, not 4 in 20. 6% rather than 20%.

We can go back to our double-headed coin in the last tip. If we specifically suspect a double-headed coin, then five consecutive heads 'prove' a bogus fourpenny piece at the $p < 0.05$ level. If, however, we suspect a bogus coin – either double-headed or double-tailed – then the first throw doesn't count except to give us an index case. For example, we throw a 'tail'. We now suspect this is a two-tailed coin¹⁴⁷ and the next five throws might 'prove' this. Five heads would not now prove a double-headed coin. To use a longer-odds starter – if someone told you a die¹⁴⁸ was fixed to come up six every time and your first two throws were a six – that would be very suspicious (1:36). If they just said it was fixed in some way and your first two throws were both threes – that's less so (1:6).

Alternatively let's use the Bonferroni too-many-tests concept – using 100 coins to represent 'the world'. We toss them all. Fifty heads (unlikely

147 Not a reference to 'two-tailed significance tests' which concern the tails of a Gaussian distribution, yet spookily having *exactly the same effect* on the stats.

148 If anyone ever complains you're being pedantic using this singular, ask them if they'd ever say 'the dice is cast'.

– but let's not get into that). Toss those fifty . . . 25 heads. Toss those 25 . . . 12 heads. Toss those 12 . . . 6 heads. We now have six coins that have come up heads four times in a row. Toss them again – is anything that turns up heads a bogus coin? Obviously not. But say we hadn't made it so obvious by tossing only the heads coins again. Say we toss all 100 coins the four times. And our ginger-haired guy¹⁴⁹ has been watching closely and is standing near one of our four-headers. He picks it up, and tosses it once more . . .

FROM THE 'OTHER SIDE' TIPS

My writing of this book was interrupted by an excuse for me to spend some time as a patient (well, it's better than doing rheumatology clinics). This prompted me to include a few tips about seeing things from the patient's point of view.

OTHER-SIDE TIP 1: SOMEHOW . . . FIND OUT WHAT IT'S LIKE

Seems a bit bizarre. Like I'm suggesting you go get sick or something. But somehow, somehow, you should try to get a taste of it. I'd always felt I had some insight into the 'other side' – having had a couple of proper childhood illnesses with hospital stays – but recent experience reminded me that things are different as an adult (not sure I'd now tolerate a grown man swinging a leather belt ['tawse'] as hard as he could at my unprotected hand¹⁵⁰) as well as memory being fickle. Patients *do* have a miserable time in hospital, including:

- 1 Food – much worse than I'd ever thought. My concept was of just-a-bit-worse-than-canteen-food, but that totally failed to pick up on its complete awfulness. It is simply inedible. Often cooked miles away and hours earlier (on Friday, I knew it was fried fish for lunch after I smelled its arrival in the building at 9.30 am), the sort of food we throw up at patients (cut out the middleman?) must surely slow down recovery. So if one of them is having food brought in from outside, it doesn't mean they're posh or picky or awkward. Just human. So don't give 'em a bad time.

149 Nothing against ginger hair. They're not a bunch of people who throw coins all the time (whatever that would be). Just an adapted cinematic device so you immediately know who I mean. (Cf. glasses on wife in *Strangers on a Train*, the girl's red coat in *Schindler's List*, Hitler's moustache . . .)

150 On re-reading, I should clarify this is a reference to schooldays. Glasgow paediatricians used a stick.

- 2 Drug rounds – I’ve always filled in timings for drugs based on my simple grasp of maths and my even simpler grasp of pharmacokinetics. What it means to the patient doesn’t occur to me – until it’s eleven o’clock at night and I’m desperately wanting to sleep and I’ve only now just been given my night-time medicines which include diclofenac (yes it *does* give me heartburn/reflux, as it happens . . . and I *am* on anticoagulants at the moment) and lactulose (which gives *everybody* heartburn and was described perfectly by a nursing colleague as like being given ‘someone else’s saliva to drink’) which between them will burn my chest to a frazzle the second my angle-of-recumbency goes below 50° (Tip 1 for in-patients is *learn how to sleep upright*). So, just for fun, try a bit of thought-process next time you’re writing up the times for drug dosing.
- 3 Ward rounds – maybe it’s just me, but I felt the day was not ‘my own’ until the doctors¹⁵¹ had been to see me. So I delayed washing, shaving, phoning my bookmaker, etc. till later in case I missed them. So doing a ward round when expected, and no later than necessary, suddenly makes sense to me.
- 4 Dissociation – one unnerving aspect of being a patient is the realisation that the people coming in to see you . . . doctors, nurses, relatives . . . have all got ‘proper’ lives going on, whilst you are stuck there. If this is emphasised by a group of, e.g. doctors sharing a laugh over last night’s curry, Sunday’s football or other liveliness which no way could involve your battered body, it can become irritating. Of course I like a bit of banter on the ward round – same as everywhere else – but you should make sure that the patient feels he/she is part of the fun (certainly rather than any butt). I’ll often repeat for the benefit of the patient what one of the juniors has said at the case trolley (since only if it’s confidential medical info will they have already shouted it out to the entire ward) if the rest of us have responded with laughter – if it was really funny, I’ll pretend it was me. Amongst other things, it does reassure the patient that it wasn’t at their expense. And lets them know *whose side we’re on*.
- (Before you get confused about which side we *are* on: when we’re diagnosing ‘tricky’ patients it’s *us against them*, when we’re getting the properly sick ones better it’s *us-and-them against the world*.)
- 5 Platitudes – if a patient has a problem or worry, don’t just say ‘it’ll be fine’. Tell them what you think is causing it, whether you need to do anything specific about it, and *why* it’ll (probably) be fine.

151 I say ‘doctors’, but as a consultant I was studiously avoided by anyone themselves below the level of prime minister – leaving my exposure to medical staff quirkily limited to one five-minute contact per day.

So go get sick or something. Or at least be aware of what it's like, and allow for your patient's apparent idiosyncrasies accordingly.

OTHER-SIDE TIP 2: IF YOU ARE ON THE OTHER SIDE, THERE'S NO FOOLPROOF APPROACH

We all know there is a doctor-as-patient problem – quoted by the anti-doctor brigade (that's everyone who isn't himself a doctor) as due to 'not being in control any more' and by the pro-doctor brigade (doctors) as due to 'knowing too much'. Not surprisingly, I fall in the latter camp. When you know what side effects drugs can cause, and what things can go wrong with various procedures, it is nigh impossible to face the slightest manoeuvre with equanimity.

Before my recent admission, the advice from my Clinical Nurse Specialist was to 'do what I was told' (though less abrupt) and I agreed. Let the guys who know what they're doing, do what they do best. Don't try to second-guess. But next day the other CNS said 'don't be afraid to speak up'.

So who's right?

Who's always right?

Both of them. Neither. Nobody.

Just like everything else, the doctor-as-patient scenario can only be dealt with on a case-by-case basis. Indeed an event-by-event basis. Sometimes you need to go with the flow, other times you have to speak up and get things changed. Simple. The only problem is . . . *you will always get these round the wrong way.*

Ergo:

- If you think your epidural's a bit *high*, you'll say nothing, and get lots of low pain.
- If you hear nurses playing around with aforementioned epidural, you'll say something ('how about wearing some sterile mittens!¹⁵²) and they'll point out they're nowhere near the spinal site, and you'll feel a right toffee.
- If your IV drug dosage sounds quadruple what you'd use, you'll say nothing and *skaboossh!*
- If your IV drug dosage sounds a quarter what you'd use, you'll say something ('that won't be enough'), they'll listen to you and *skaboossh!*
- If they're just anaesthetising you and you think you hear somebody saying it's your *right* kidney they're going for and . . . you're . . . sure . . .
. . . tha . . .

152 'Cos you don't want your spinal fluid smelling of chips.

OTHER-SIDE TIP 3: PICK AN IDIOSYNCRATIC FORMAT (YOU LIKE!) FOR YOUR TEA/COFFEE

The first time I was asked in the ward how I liked my tea, I told them a tiny bit of milk and *one-third-of-a-teaspoonful* of sugar.

This was deservedly slagged off as being a bit control-freakish.

The second time I was asked, I told them the same.

This was also deservedly slagged off.

The third time, they didn't need to ask, as they all knew and remembered this slag-offable choice.

OK. I get a totally undeserved reputation as a pernicky blighter, but all the subsequent times I get tea – without their having to ask or getting it all wrong or me having to trouble them – it's how I quite like it (though it would have been better with four-elevenths of a spoonful).

PS Cardiology guru thinks this tip isn't important enough, but what's more critical to recovery than a good cup of tea?

SURVIVAL/CAREER TIPS

SURVIVAL/CAREER TIP 1: MAKE *NEVER SURE TAKE YOU A HAVE LUNCH BIG BREAK LUNCH AT BREAKS ALL*

SURVIVAL	CAREER
1 Taking time over eating is good for your digestion.	<i>You'll get more work done.</i>
2 Taking a proper break from the morning stresses of medicine will 'recharge your batteries' (sorry) and help you cope with the afternoon stresses.	<i>People will think you seriously want to be a manager.</i>
3 Discussing patients with colleagues in an informal scenario gives a chance to 'brainstorm' in a way perhaps inappropriate with formal referrals.	
4 Discussing football, golf, art, TV, films, books, philosophy with your colleagues can foster better relationships.	
5 All the things in 4 also help achieve the target in 2.	
6 Problems with rotas, cover, juniors, seniors, can be teased out in a friendly manner.	
7 You'll live longer.	...mmmm...tricky...

PUBLISHING TIPS

TIP 101: DON'T CALL YOUR BOOK *100 TOP TIPS*

One hundred and one sounds a lot cooler, and it makes people think they're getting some sort of bargain.