

PRACTICE POINTER

Interpreting research findings to guide treatment in practice

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When applying research findings to individual patients, practitioners can use the PICO approach, which considers characteristics of the patient or population, intervention, comparator or context, and outcome. Patient centred practitioners should however identify the outcomes which are important to individual patients

Trials are important but not sufficient for good clinical decision making. Recommendations derived from trials in groups of patients must be interpreted and adapted by clinicians to the context of each individual patient seen in practice.¹ The spectrum of patients in primary care is often very different from that in secondary care and clinical trials. In general, practitioners have two options: to consider how the treatment's benefits and harms will differ given the severity, risk, and context of the individual patient, or to use a "try it and see" approach (or, more formally, do an "n of 1" trial). There is a range between these options. Take the example of the case of depression described in box 1.

Do depression guidelines help clinicians for this individual case?

Current guidelines in Australia and New Zealand, Canada, the United Kingdom, and the United States all recommend similar treatments for major depression or dysthymia (chronic mild depression) diagnosed according to psychiatric classification systems (DSM-IV or ICD-10).³⁻⁶ The two management strategies they most strongly support—antidepressants or cognitive behavioural therapy (CBT)—have been evaluated mainly among patients with major depression, mostly in secondary or tertiary care settings.

Guidelines from NICE (the UK's National Institute for Health and Clinical Excellence) on antidepressant treatment were based on a review that included 31 outpatient studies, three inpatient studies, and only one study from primary care.⁵ A subsequent systematic review of primary care based trials identified 12 studies, but most were small, commercially funded studies of short duration and low methodological quality.^{7,8} Little

of the evidence about antidepressants is derived from the type of depression seen more commonly in primary care, which is less severe than major depression and less chronic than dysthymia. The most recent systematic review of 35 published and unpublished trials from the US Food and Drug Administration database included only one trial in mild depression.⁹ Most trials have excluded patients with considerable alcohol use or comorbid physical conditions such as epilepsy that could be adversely affected by antidepressants.¹⁰ The treatment recommendations also fail to acknowledge patients' reluctance to take drugs and do not address whether treatment works in the face of the social adversity usually associated with depression in primary care.^{11,12} More primary care based evidence exists to support the use of counselling and cognitive behavioural therapy, but only for patients with depression of a severity comparable to that of patients seen in outpatient settings.^{13,14} For milder depression, the guidelines mention the need to deal with social issues and suggest patient education and self help strategies based on principles of cognitive behavioural therapy,

Box 1 A patient with depression after marriage break up

A 54 year old man presents with low mood after separating from his wife of 30 years. He is usually seen only twice a year for review of his epilepsy drugs, tends to play down his symptoms, and has no history of depression. He has been unable to work for three weeks owing to reduced sleep, energy, appetite, and lack of motivation. He is now living alone in bed and breakfast accommodation and drinking more alcohol, is having thoughts about suicide but has no specific plans, and is not keen on antidepressants as he believes they can be addictive. He scores 14 out of 27 on the patient health questionnaire (PHQ-9), indicating mild major depression.² The GP discusses the pros and cons of antidepressants, explores with the patient what strategies he will use to address his situation, and certifies him unable to work.

A week later, with his wife now taking steps to divorce him, he says he feels more down, and his PHQ-9 score is 18, indicating moderate to severe major depression. However, a friend has given him a room to stay in for a few months; he has stopped drinking and now wants to try antidepressants so he can get back to work.

but the evidence for these interventions in primary care settings is limited.¹⁵

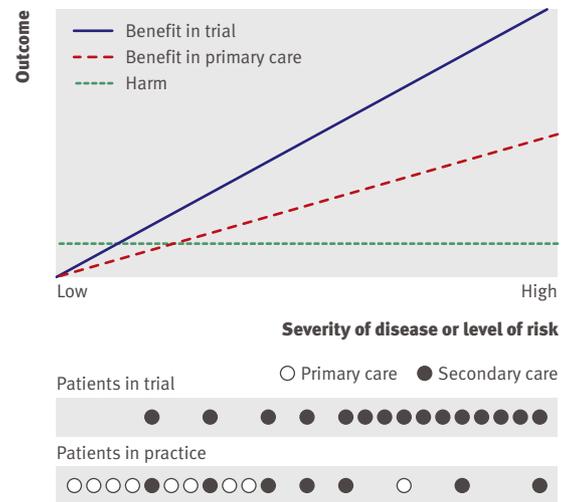
How might benefits and harms in clinical settings differ from the research setting?

The figure provides a model to help think through the issues of transferability and application of research findings.¹ In general, benefits will be higher in patients with higher risk or more severe illnesses.¹⁹ The top line shows the likely increase in benefit if the trial’s results apply across the spectrum of risk or severity of the condition. However, the relative effectiveness in primary care may be less (lower line) for several reasons—for example, because compared with everyday practice, trials may use tighter diagnostic rules, ensure higher levels of adherence to treatment, and fail to follow up patients with a poorer outcome. As trials often include a higher proportion of patients with severe disease or at high risk, the reported net benefit will be higher than in actual practice. This difference will be even greater for primary care, where most patients are likely to be at lower severity or risk. However, a primary care patient who is similar to the patients in the trial (the patient represented by the open circle near the right in figure 1), should receive similar benefit. Every clinician must answer the following question: “For this patient does the severity or risk reach the threshold where benefits outweigh harms such as adverse effects and inconvenience?”

How should we estimate an individual patient’s likely response to treatment?

Let us apply this approach to the depressed man in box 1. Box 2 shows some of the factors to be considered.¹⁶

In the UK the commonest immediate decision is whether to prescribe an antidepressant, as cognitive



Applying evidence from trials. Diagonal lines show thresholds for benefit; horizontal line shows threshold of harm

behavioural therapy is usually not available or has a waiting list of several months. Since 2006, UK general practitioners have been remunerated for using questionnaire measures of the severity of depression at the outset of treatment, and the patient in box 1 scores just above the threshold for major depression, suggesting that antidepressants may be of benefit. However, the doctor is aware that the positive predictive value of the PHQ-9 for major depression is likely to be lower in primary care than in secondary care due to the relatively lower prevalence, and therefore the patient may be a false positive case given his borderline score.

So the doctor does not prescribe antidepressants on the basis of one marginally raised PHQ-9 score alone, especially as the patient is not keen on taking them and may be at risk of taking an overdose while acutely distressed. Also antidepressants may lower the threshold for epileptic convulsions. Instead the doctor discusses with the patient the possible benefits and side effects of antidepressants and arranges a review a week later. The patient clearly needs follow-up, as he tends to play down his symptoms, his functioning is impaired, and—being an older, socially isolated male with increased alcohol use—he is at increased risk of suicide (although the doctor is aware that having all these epidemiological risk factors is still a poor predictor of actual suicide in any individual case).

At review the symptom severity score is higher, indicating moderate to severe major depression. The patient’s preference is now for antidepressants and he is less socially isolated, reducing the suicide risk. He is now seen to be more like the patients included in trials of antidepressants, and the balance of the decision tips towards prescribing antidepressants. Caution is needed however, given the possible adverse effect of precipitating epileptic convulsions, with possible risks to the patient’s driving licence and job, and it would be reasonable to monitor the patient for longer, as his social situation is in flux and changes over the next few

Box 2 Factors to take into account in the individual case: the PICO approach¹⁶

Population—is the individual in front of you sufficiently similar to trial participants to be likely to gain a similar relative benefit from treatment?

Is the patient diagnostically similar to patients in the trials? (Take into account the limitations of secondary care diagnostic tests in the primary care setting)

Are the likely benefits and harms of treatment similar to those in the trials?

Intervention—how similar will the treatment be to that given in the trial?

Is sufficiently similar treatment available and accessible?

Will the patient adhere to it? (Have you explored the patient’s preferences?)

Context or comparator treatments—are there specific contextual issues for the individual that are very different from the trial context and likely to influence the outcome for the patient? What are the alternatives to the specific treatment being considered?

Does the patient have complications or co-morbid conditions which would affect the likely benefits or harms?

Are there other prognostic factors which were not measured in the trials?

Is the patient likely to improve without treatment anyway?

Outcome—are the outcomes assessed in the trials, the same outcomes that are important for this individual?

Have you established what is important to the patient?

weeks may lead to considerable improvement.¹² Prescribing can also be viewed negatively as a way of “disposing” of the patient which medicalises understandable reactions to life’s difficulties, precludes further in depth exploration of the person’s circumstances and own resources, and risks subsequent adverse effects on the doctor-patient relationship.^{12,17} If the doctor does decide to prescribe antidepressants, further discussion of the benefits of treatment, prompted at regular follow-up encounters, is likely to maximise the patient’s adherence to treatment.¹⁸ Encouraging questions, in a collaborative patient centred relationship, is likely to lead to a better outcome.¹⁹ Case management programmes have been shown repeatedly to ensure the best outcomes through such engagement, but at present are not widely available in primary care.²⁰

The decision to apply research findings in practice depends not just on the setting but on the risk, severity, and context of the individual patient; the relationship between doctor and patient, and the availability of other resources to offer the patient as an alternative to medication. The transferability and applicability of secondary care evidence to primary care will vary between disorders. For depression, the spectrum seems quite different, making it difficult in many cases to base treatment decisions on evidence from trials. In many cases, the default position should be not to treat, at least initially, to avoid doing the patient harm, while watching for the development of more severe depression. The same principles could be applied to other common conditions such as migraine, for example. Yet in other conditions, the trial evidence can be applied directly. For example, for the use of statins after myocardial infarction the trial participants may have been recruited in hospitals but are actually the set of patients discharged to primary care for treatment, and hence the guideline recommendations are much more readily applicable.

N of 1 trials

For some conditions we can use the “n of 1 trial” approach to check whether a particular treatment works for a particular individual. For example, to determine whether a non-steroidal anti-inflammatory drug (NSAID) is better than paracetamol for arthritis, we could ask a patient to alternate paracetamol and the NSAID for a few days at a time over some weeks and record pain daily to measure the relative benefit. This would be most rigorous if similar preparations of each medication were used and the patient was unaware which was which—this would reduce the placebo effect, which would tend otherwise to favour the NSAID.¹⁶ Other examples of this “try it and see” approach, which will be familiar to general practitioners, include trying different creams on different parts of an apparently eczematous rash, or different eye drops in each eye for dry eyes.

Unfortunately, the n of 1 approach doesn’t work well in the case of depression, as antidepressants take some weeks to work, and a wash-out period is often needed

between different types. Patients who have tried various treatments in the past can obviously tell us which seemed to work best for them. Otherwise we have to consider how well the evidence from groups of patients might apply in the individual case.

How can the evidence base be improved to be useful in individual cases?

To better inform treatment decisions we need more studies of the course of conditions without treatment, to identify predictors of who needs active treatment. We also need trials that include patients with mild as well as more severe conditions, and with comorbidities that might affect the relative benefits and harms of treatments. Patients’ preferences need to be taken into account in research designs, and outcomes important to patients need to be measured.

Studies will often need to be large, so that they have sufficient power for subgroup analyses to measure the effects of important predictors of response, including age, sex, and ethnic minority differences and variable adherence to treatment. Such studies would be better carried out in primary care, and clinicians should be prepared to take part in studies that will directly inform their practice, facilitating the negotiation of a truly informed decision between clinician and patient. However, some extrapolation from trial populations, whether they are from primary or secondary care, will always be necessary, as research studies can never include every possible type of patient presenting in practice.

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UNCERTAINTIES PAGE

What is the optimal management of partial epilepsy uncontrolled by a first choice anticonvulsant?

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Up to 70% of people who are treated with a single anticonvulsant will enter remission within a short time of being diagnosed with epilepsy.¹ Optimal first choice treatments have been identified in some comparative randomised controlled trials and guidelines from the National Institute for Health and Clinical Excellence.^{2,3} Lamotrigine or carbamazepine are usually the preferred initial treatment of seizures with localised onset in the brain, whereas valproate is preferred for generalised epilepsy syndromes.^{4,5}

However, despite optimal doses of a first line anticonvulsant, many patients with localised onset seizures (10-15/100 000 each year worldwide) do not enter remission and continue to have seizures of varying severity and frequency, which are associated with considerable psychosocial distress.^{6,7} The evidence base to support management of these patients is minimal,² and it is uncertain which of the following available options is optimal:

- Continue with the existing first line treatment as monotherapy.
- Switch to an alternative second line drug as monotherapy.
- Add a second drug to the existing first line treatment.

What is the evidence of uncertainty?

A single small randomised study has compared the options of switching anticonvulsants or adding a second drug.⁸ No differences were detected, but the study was small and lacked sufficient power to detect clinically important differences.

A large number of placebo controlled regulatory studies in patients with refractory localised onset seizures have indicated some short term reduction in

the frequency of seizures after adding a second anticonvulsant to the existing treatment.⁹ This potential benefit is, however, accompanied by an increased incidence of adverse events. The longer term effect of this risk-benefit ratio on patient perceived quality of life is uncertain. Studies in patients with less severe epilepsy (those in seizure remission on drug treatment and those with few or infrequent seizures at diagnosis) show that the benefits from improved seizure control are equally balanced by worse quality of life as a result of taking anticonvulsants.^{10,11}

We also have no evidence on which second line drug is optimal. Over the past two decades, many newer anticonvulsants have been licensed for add-on treatment for localised onset seizures. Consequently, patients with refractory seizures have had their treatment regimen changed frequently, with uncertain benefits.

Is ongoing research likely to provide relevant evidence?

There are some existing and planned industry sponsored studies comparing the addition of different second line drugs. However, these are of short duration and are not suited to looking at important longer term patient based outcomes that are crucial to guide treatment choices in a chronic disorder such as epilepsy.

What should we do in the light of the uncertainty?

In patients with continuing localised onset seizures despite optimal first line therapy, consider adding a second anticonvulsant. A consensus evidence-free view is that when drugs are combined they should have differing primary mechanisms of action and no pharmacokinetic interaction.¹² Most first line

This is a series of occasional articles that highlights areas of practice where management lacks convincing supporting evidence. The series advisers are David Tovey, editorial director, BMJ Knowledge, and Charles Young, editor of *BMJ Clinical Evidence*, and editor in chief, BMJ Point of Care

RECOMMENDATION FOR FURTHER RESEARCH

Population: patients with localised onset seizures without remission after adequate sequential treatment with two or more antiepileptic drugs

Intervention and comparison: add-on treatment with one antiepileptic drug compared with add-on treatment with an alternative antiepileptic drug compared with continued optimised treatment with the existing single drug

Outcome: frequency of seizures

anticonvulsants (carbamazepine, lamotrigine, phenytoin, oxcarbazepine) act on sodium channels. Of the commonly used first line add-on anticonvulsants, our current preferred options include clobazam (which acts at GABA (γ -aminobutyric acid) receptors) or levetiracetam (which binds synaptic vesicle protein 2A).⁹ No evidence is available to support our choice of add-on—topiramate, pregabalin, and zonisamide are all reasonable alternatives.

A randomised controlled trial is urgently needed that compares adding placebo with adding one or two second line agents to existing optimally dosed treatment with carbamazepine, lamotrigine, phenytoin, or oxcarbazepine in patients with continuing localised onset seizures. Such a trial should evaluate longer term clinical, patient based, and health economic outcomes. Input from patients and carers is essential to help understand the balance between the effects of improved seizure control and the additional burden of drug treatment. In the meantime, patients should be offered the addition of a second line anticonvulsant, such as clobazam or levetiracetam, with appropriate counselling about risks and benefits and the opportunity to review outcomes over the short to medium term.

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LESSON OF THE WEEK**Acute phosphate nephropathy after sodium phosphate preparations**

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Predisposed individuals may develop chronic kidney disease after administration of sodium phosphate purgative before colonoscopy

Oral sodium phosphate preparations are used as bowel purgatives before colonoscopy. Subsequent renal impairment is increasingly being reported.¹ We describe a case of acute phosphate nephropathy with persistent renal impairment after administration of sodium phosphate.

Case report

A 76 year old woman was admitted with rectal bleeding. Her past medical history included hypertension—treated with nifedipine—and long standing use of tobacco. Physical examination was unremarkable.

Laboratory results were normal—haemoglobin 106 g/l, white cell count $7.2 \times 10^9/l$, platelets $357 \times 10^9/l$, sodium 132 mmol/l, potassium 4.3 mmol/l, urea 6.2 mmol/l, and creatinine 98 $\mu\text{mol/l}$.

She underwent flexible sigmoidoscopy after being given a sodium phosphate enema (Fleet Ready-to-

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use; De Witt) the night before (day 1). Colonoscopy was performed on day 4 after she took two sachets of oral sodium phosphate solution (Fleet Phosphosoda; De Witt). Histological findings were consistent with chronic active ulcerative colitis. She was discharged and prescribed mesalazine.

On day 6 she presented with acute renal failure (creatinine 541 $\mu\text{mol/l}$). She received intravenous fluids and mesalazine was replaced by prednisolone enemas. A renal consultation was obtained.

Urinalysis was unremarkable. She was normocalcaemic (2.4 mmol/l) but mildly hyperphosphataemic (1.54 mmol/l). Results of a screen for glomerulonephritis and renal tract ultrasonography were normal. Interstitial nephritis secondary to mesalazine was considered and she underwent renal biopsy.

Twenty seven glomeruli were identified; one was globally sclerosed and the others were normal. The striking feature was widespread tubular calcification (figure). Von Kossa stain accentuated the phosphate component of these concretions (figure). The tubules were dilated and the tubular epithelium was

flattened. Lymphocytic infiltration was minimal, and focal fibrosis was seen in less than 10% of the cortical tissue. Deposits of immunoglobulin or complement were not seen on immunofluorescence microscopy.

We made a diagnosis of acute phosphate nephropathy with persistent renal impairment secondary to the administration of a sodium phosphate purgative. The patient was discharged on day 22 with prednisolone enemas and erythropoietin injections. Serum creatinine had stabilised at 271 $\mu\text{mol/l}$ (estimated glomerular filtration rate 15 ml/min/1.73 m²), and serum phosphate was 1.15 mmol/l.

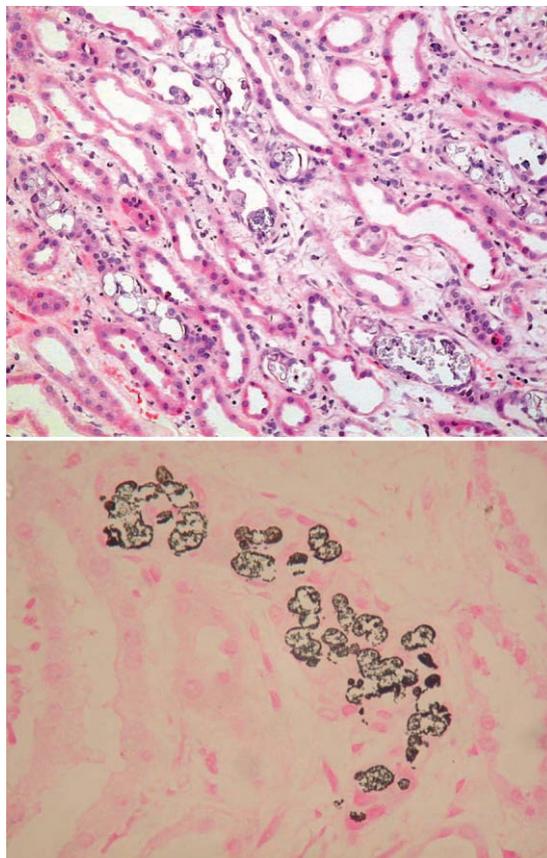
Discussion

Acute phosphate nephropathy refers to renal impairment caused by diffuse tubular damage as a result of deposition of calcium phosphate in the distal tubules and collecting ducts. Interstitial oedema is often present. Tubular atrophy and interstitial fibrosis indicate irreversible tubular injury. Unlike nephrocalcinosis, in which hypercalcaemia promotes an insidious loss of renal function through chronic tubulointerstitial injury secondary to the deposition of calcium crystals in the renal parenchyma,^{1,2} this new pathological entity is typically acute in onset and occurs in the absence of hypercalcaemia.²

Acute phosphate nephropathy is an increasingly reported cause of chronic kidney disease.¹ The rise in serum creatinine needed for a diagnosis of acute phosphate nephropathy has varied in studies to date; a recent study used a $\geq 50\%$ rise in creatinine from baseline over 12 months after colonoscopy.^{1,3} Although the true incidence is therefore unknown, it may occur in up to 1 in 1000 patients who receive sodium phosphate and is probably underdiagnosed.^{1,2}

Oral sodium phosphate preparations promote colonic evacuation by drawing large volumes of water into the colon (1-1.8 litres of water per 45 ml of preparation).⁴ They provoke transient hyperphosphataemia (an increase in serum phosphate of 0.165-0.195 mmol/l),⁵ which is most profound in elderly people.⁶ This is rarely associated with untoward events. Factors that promote hyperphosphataemia predispose patients to acute phosphate nephropathy. Such factors include inappropriate dosing, increased bowel transit time (for example, bowel obstruction), and reduced ability to excrete a phosphate load (such as renal impairment).⁷

Factors that promote tubular precipitation of calcium phosphate also predispose to acute phosphate nephropathy. These include inadequate hydration during phosphate administration, hypertension with arteriosclerosis, and drugs such as renin-angiotensin



Extensive tubular calcification with little chronic damage as shown by haematoxylin and eosin stain (top; original magnification $\times 200$) and von Kossa stain (bottom; original magnification $\times 200$, courtesy of T Toth)

inhibitors, diuretics, and non-steroidal anti-inflammatory drugs.² This first group of drugs limits the kidneys' capacity to compensate for the reduced renal perfusion of volume depletion and accentuates bicarbonaturia by inhibiting angiotensin II, thereby enhancing alkalinisation of the urine and increasing calcium and phosphate precipitation.⁸ Heart failure and cirrhosis are further risk factors,^{9,10} and the condition seems to be more prevalent in women.²

Acute phosphate nephropathy may arise in patients receiving sodium phosphate solutions (Fleet Phosphosoda; De Witt) or tablets (Visicol; Salix Pharmaceuticals).^{2,11,12} Renal insufficiency is sustained in most patients, but this varies according to the increase in creatinine needed for diagnosis.³ In one study, 19% of patients developed end stage renal disease,² whereas in another study only 16% returned to their baseline renal function.³

Absolute contraindications to oral sodium phosphate preparations include renal impairment (phosphate excretion is extremely compromised once the glomerular filtration rate drops to 30 ml/min),⁶ bowel obstruction, dehydration, electrolyte disturbances, and heart failure.⁸

Our patient had active colitis, a relative contraindication to phosphate preparations.⁷ Older age (those aged over 57 are at increased risk of acute phosphate nephropathy) and previous hypertension are further relative contraindications.^{6,13}

Prevention of acute phosphate nephropathy requires adequate volume repletion during phosphate administration.¹⁴ Electrolyte rehydration solutions may help.^{15,16} Admission for intravenous prehydration may be necessary. Drugs that alter electrolyte balance (diuretics) or reduce renal perfusion (renin-angiotensin inhibitors) should be withheld before phosphate administration. The appropriate doses should never be exceeded.

To facilitate evaluation of the risk of acute phosphate nephropathy, we recommend that the patient's age, sex, electrolyte profile, comorbidities (glomerular filtration rate, hypertension, heart failure), and drugs are incorporated into the colonoscopy referral form. Alternative agents that do not provoke such dramatic osmotic shifts, such as polyethylene glycol solution, might then be considered in high risk patients—although sodium phosphate preparations are more effective and better tolerated.⁵

We also recommend that clinicians consider checking serum creatinine about a week after colonoscopy in high risk patients. Because the identification at a later date of non-progressive chronic kidney disease in a typical patient (an elderly person with hypertension and minimal proteinuria) is unlikely to provide a strong indication for renal

biopsy, and because the link between colonoscopy and renal impairment is less likely to be noticed as time elapses, failure to check serum creatinine may lead to cases of acute phosphate nephropathy being missed. Management strategies will not then be instituted and the patient may receive further sodium phosphate preparations.

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Patient consent obtained.

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10-MINUTE CONSULTATION

Sleep disorder (insomnia)

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A 53 year old man comes to you complaining of not having slept well for many years. He always feels tired the next day. He has tried sleeping pills, which sometimes help, but he is not keen on taking them continually and has found that the benefits they give him don't last. He spends about 9-10 hours in bed each night (going to bed about 9 30 pm or 10 pm and getting up at 7 am) and has trouble getting to sleep. His actual hours of sleep are 5.5 to 6 each night. He wakes about three times a night and describes the quality of his sleep as poor.

What issues you should cover

Assessment

Rule out secondary causes. To assess whether he has depression or anxiety, for example, ask screening questions, take a full history of depression and anxiety, or use a scale such as the Hospital Anxiety and Depression Scale, which gives a score for both conditions. Consider sleep apnoea if he snores a lot at night, has periods of apnoea, falls asleep easily during the day (for instance, as a passenger in a car, in lectures, or in waiting rooms), and is overweight. (Sleep laboratory assessment may be needed if this diagnosis is not clear.) As his general practitioner you will know whether he has severe pain or a breathing condition—these may need treatment. Delayed sleep phase is where people prefer to go to sleep and wake up more than two hours later than societal norms. You can ask directly about parasomnias (sleep walking, sleep talking, and restless legs syndromes). If he has none of these he probably has primary insomnia—his insomnia is not due to any other cause and hence is a diagnosis of exclusion.

Documenting insomnia

You could ask him to keep a sleep diary over 1-2 weeks. Or ask him what time he puts out the lights, how many minutes it takes him to get to sleep, how many times he wakes up after first falling asleep and how long he stays awake, and what his final waking time is and the time he gets out of bed. From this information you can calculate how much time he spends in bed and how much time asleep, which can be expressed as the sleep efficiency—the percentage of time spent in bed during which he is asleep. He sleeps for about six hours and is in bed for nine hours, so his sleep efficiency is about 66%. A sleep efficiency of 80% to 85% is considered optimal. More than 90% may indicate sleep deprivation, and below 75% is considered to be a sign of poor quality sleep. Ask how he feels when he wakes up and during the day, and ask him to describe the quality of his sleep.

Causes of sleep disorder

Common causes

Depression
Anxiety
Sleep apnoea
Pain or a serious breathing condition
Daytime naps, especially if late in day
Evening consumption of coffee, nicotine, or alcohol
Vigorous physical activity close to bedtime
Shift work
Primary insomnia (in reality all those cases that don't have a specific cause)

Causes of unknown prevalence in primary care

Delayed sleep phase ("night owls")
Parasomnia (sleep walking, sleep talking, and restless legs)

What you should do

In primary insomnia the behavioural option of sleep restriction (restricting the amount of time spent in bed just to the usual sleeping time) has been shown to work, with or without cognitive behavioural therapy. As he is sleeping for only 5.5 to 6 hours, advise him to go to bed much later than he currently is; thus if he gets up at 6 am he could go to bed at 12 midnight so that he spends less time in bed.

Some patients find this regime quite difficult but within about two weeks find themselves sleeping much better and report a better quality of sleep. If they achieve this in a few weeks they can start increasing the time spent in bed, restricting it again if the quality of sleep deteriorates. Once the patient achieves better quality sleep he can choose to remain on that schedule.

It is common for patients with primary insomnia to get a much higher quality of sleep once they begin sleep restriction. Their usual response to poor sleep is to spend more time in bed, which is in fact counterproductive.

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Useful reading

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This is part of a series of occasional articles on common problems in primary care. The *BMJ* welcomes contributions from GPs