GUIDELINES

# Diagnosis, assessment, and management of harmful drinking and alcohol dependence: summary of NICE guidance

Stephen Pilling,[1 2] Amina Yesufu-Udechuku,[2] Clare Taylor,[3] Colin Drummond,[4] on behalf of the Guideline Development Group

See also EDITORIAL by Coltart and Gilmore

[1]Centre for Outcomes Research and Effectiveness, University College London, London WC1E 7HB, UK
[2]National Collaborating Centre for Mental Health, University College London
[3]National Collaborating Centre for Mental Health, Royal College of Psychiatrists, London E1 8AA, UK
[4]National Addiction Centre, Institute of Psychiatry, King's College London, and South London and Maudsley Foundation NHS Trust, London SE5 8AF
Correspondence to: S Pilling
s.pilling@ucl.ac.uk

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This is one of a series of *BMJ* summaries of new guidelines based on the best available evidence; they highlight important recommendations for clinical practice, especially where uncertainty or controversy exists. The supporting evidence statements and further information about the guidance are in the full version on bmj.com.

Alcohol dependence affects 4% of people aged between 16 and 65 years in England (6% of men and 2% of women),[1] and over 26% of all adults (38% of men and 16% of women) consume alcohol in a way that is potentially or actually harmful to their health or wellbeing. Yet currently only 6% of people who are alcohol dependent receive treatment.[1] Alcohol dependence is characterised by withdrawal, craving, impaired control, and tolerance of alcohol and is associated with a higher rate of mental and physical illness and a wide range of social problems. Harmful drinking is a pattern of alcohol consumption that can lead to psychological problems such as depression, accidents, injuries, and physical health problems such as pancreatitis. Alcohol misuse is also an increasing problem in children and young people, with over 24 000 treated in the NHS for alcohol related problems in 2008 and 2009.[2] Hospital admissions related to alcohol consumption increased by 81% between 2003 and 2009.[3] Harmful drinking and alcohol dependence therefore represent a considerable burden to individuals, their families, and wider society.

This article summarises the most recent recommendations from the National Institute for Health and Clinical Excellence (NICE) on the diagnosis, assessment, and management of harmful drinking and alcohol dependence.[4]

## Recommendations

NICE recommendations are based on systematic reviews of best available evidence and explicit consideration of cost effectiveness. When minimal evidence is available, recommendations are based on the Guideline Development Group's experience and opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on bmj.com.

### Identification and initial assessment

- Staff working in services provided and funded by the NHS should be competent to identify harmful drinking and alcohol dependence and to initially assess the need for an intervention; if they are not competent, they should refer people who misuse alcohol to a service that can provide such an assessment. Validated tools such as the alcohol use disorders identification test (AUDIT) (figure) are effective in identifying harmful drinking and alcohol dependence in non-specialist settings such as primary care and acute hospitals.
- Consider a comprehensive assessment for all adults referred to specialist alcohol services who score more than 15 on the identification test (figure)[5] This should assess multiple areas of need, be structured in a clinical interview, and cover:
  - -Alcohol use, including consumption and patterns of drinking; severity of dependence (using the severity of alcohol dependence questionnaire (SADQ)[7] or Leeds dependence questionnaire (LDQ)[8]); and alcohol related problems (using the alcohol problems questionnaire (APQ)[9])
  - -Misuse of other drugs, including over the counter medication
  - -Physical health problems
  - -Psychological and social problems
  - -Cognitive function (using, for example, the mini-mental state examination[10])
  - -Readiness and belief in ability to change.

### All interventions for harmful drinking and alcohol dependence

- All interventions for harmful drinking and alcohol dependence should be delivered by appropriately trained and competent staff. Drug interventions should be administered by specialist and competent staff. Base psychological interventions on a relevant evidence based treatment manual, which should guide the structure and duration of the intervention.
- Carry out a motivational intervention as part of the initial assessment to help engage the person in treatment from first contact. The intervention should include helping people to recognise problems related to drinking and resolve ambivalence; encouraging positive change; and adopting a persuasive and supportive rather than argumentative and confrontational position.
- For all interventions, staff should:
  - -Receive regular supervision from individuals competent in both the intervention and supervision
  - -Routinely use outcome measurements to ensure that the person who misuses alcohol is involved in reviewing the effectiveness of their treatment
  - -Monitor and evaluate the person's adherence to the treatment and their own practice competence—

| Questions | Score | | | | | Scoring column |
|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | |
| 1 How often do you have a drink containing alcohol? | Never | Monthly or less | 2-4 times a month | 2-3 times a week | 4 or more times a week | |
| 2 How many drinks containing alcohol do you have on a typical day when you are drinking? | 1 or 2 | 3 or 4 | 5 or 6 | 7 to 9 | 10 or more | |
| 3 How often do you have six or more drinks on one occasion? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 4 How often during the last year have you found that you were not able to stop drinking once you had started? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 5 How often during the last year have you failed to do what was normally expected from you because of drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 6 How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 7 How often during the last year have you had a feeling of guilt or remorse after drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 8 How often during the last year have you been unable to remember what happened the night before because you had been drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 9 Have you or someone else been injured as a result of your drinking? | No | | Yes, but not in the last year | | Yes, during the same year | |
| 10 Has a relative or friend, or a doctor or other health worker been concerned about your drinking or suggested you cut down? | No | | Yes, but not in the last year | | Yes, during the same year | |
| | | | | | Total score | |

**Interpretation of AUDIT scores**
- A total score of more than 8 indicates hazardous drinking[5]
- A total score of 16 to 19 indicates harmful drinking or mild or moderate dependence; the current NICE guideline recommends people with a score of more than 15 should be considered for comprehensive assessment[4]
- A total score of 20 or more indicates severe dependence; the current NICE guideline recommends that people with a score of 20 or more should be considered for assessment for assisted alcohol withdrawal[6]

**Alcohol use disorders identification test (AUDIT), self report version,[5] with interpretation guidance based on the current NICE guideline on diagnosing, assessing, and managing harmful drinking and alcohol dependence[4] and on NICE's public health guideline on preventing the development of hazardous and harmful drinking.[6] An interview version of the AUDIT is also available**

for example, by using videotapes and audiotapes and external audit.

### Psychological interventions for harmful drinking and mild alcohol dependence

- For harmful drinkers and people with mild alcohol dependence, offer a psychological intervention (such as cognitive behavioural therapies, behavioural therapies, or social network and environment based therapies) focused specifically on cognitions, behaviour, problems, and social networks that are related to alcohol.

### Assessment for assisted alcohol withdrawal

- For those who typically drink over 15 units of alcohol a day and/or score 20 or more on the identification test,[5] consider offering:
  - -Assessment for and delivery of a community based assisted withdrawal, or
  - -Assessment and management in inpatient care if you have safety concerns (see below) about a community based assisted withdrawal.
- Consider inpatient or residential assisted withdrawal if the person meets one or more of the following criteria:
  - -Drinks over 30 units of alcohol a day
  - -Has a score of more than 30 on the severity of alcohol dependence questionnaire[7]
  - -Has a history of epilepsy or of withdrawal related seizures or delirium tremens during previous assisted withdrawal programmes
  - -Needs concurrent withdrawal from alcohol and benzodiazepines
  - -Regularly drinks 15-20 units of alcohol a day and has psychiatric or physical comorbidities (for example, chronic severe depression, psychosis, malnutrition, congestive cardiac failure, unstable angina, chronic liver disease) or a learning disability or cognitive impairment.

### Interventions for moderate and severe alcohol dependence

- After a successful withdrawal for people with moderate and severe alcohol dependence, consider offering acamprosate or oral naltrexone in combination with an individual psychological intervention (cognitive behavioural therapies, behavioural therapies, or social network and environment based therapies) that focuses specifically on alcohol misuse. At the time of publication (mid-February 2011), oral naltrexone did not have UK marketing authorisation for this indication. Obtain and document informed consent before prescribing.
- Consider offering interventions to promote abstinence and prevent relapse as part of an intensive and structured community based intervention for people with moderate and severe alcohol dependence who have:
  - -Very limited social support (for example, they are living alone or have very little contact with family or friends)
  - -Complex physical or psychiatric comorbidities
  - -Not responded to initial community based interventions to promote abstinence or moderate drinking.

### Interventions for children and young people aged 10-17 years who misuse alcohol

- For those with limited comorbidities and good social support, offer individual cognitive behavioural therapy.
- For those with significant comorbidities and/or limited social support, offer multicomponent programmes (such as multidimensional family therapy, brief strategic family therapy, functional family therapy, or multisystemic therapy).

**bmj.com** Previous articles in this series

- Management of generalised anxiety disorder in adults (*BMJ* 2011;342:c7460)
- Sedation for diagnostic and therapeutic procedures in children and young people (*BMJ* 2010;341:c6819)
- Management of bedwetting in children and young people (*BMJ* 2010;341:c5399)
- Transient loss of consciousness—initial assessment, diagnosis, and specialist referral (*BMJ* 2010;341:c4457)
- Management of hypertensive disorders during pregnancy (*BMJ* 2010;341: c2207)

**Interventions for depression or anxiety disorders in alcohol misuse**

- Treat the alcohol misuse first as this may lead to improvement in the depression or anxiety. If depression or anxiety continues after three to four weeks of abstinence from alcohol, assess the depression or anxiety and consider referral and treatment in line with the relevant NICE guideline for the particular disorder.[11-14]

## Overcoming barriers

Poor recognition of alcohol misuse is a major barrier to effective treatment[1] and requires a service-wide approach to improve case identification. Current service delivery is also fragmented, with access pathways to services unclear to both patients and professionals. To clarify care pathways and properly implement this and other NICE guidance that relates to alcohol use[5 15] NICE is currently developing an integrated care pathway for the three pieces of guidance.

Limited availability of specialist alcohol services also hinders effective guideline implementation—for example, there is a lack of skilled staff to deliver evidence based psychological interventions and support intensive community based assisted withdrawal, and limited prescribing of cost effective medication such as acamprosate and oral naltrexone to prevent relapse in moderate to severely dependent drinkers. Guideline recommendations on these interventions will need to be supported by effective commissioning.

In addition, safe and effective assisted alcohol withdrawal may require prescription outside the limits of the *British National Formulary*, and the guideline offers clear advice on dose regimens to support this.

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# A disorder of eating or of values?

JM and DS were struggling to find a shared perspective on the management of patients with eating disorders. JM, a middle aged consultant, was educated in the West, in all-female schools and colleges, where dieting and body image disparagement are norms. She has worked for decades with patients who lose weight obsessively and who defend their behaviour as a lifestyle choice rather than an illness. She has seen full recoveries take many years to achieve and has also seen patients die or live spoiled lives as a result of their disorders or of iatrogenic damage.

DS, a male medical student planning a career as a surgeon, still feels scarred by memories of 10 days of severe, involuntary starvation in the tropical jungle. He recalls life in extremis, in a state of desperation and prayer, trying to eat inedible plants. He is horrified by the many months patients spend in the ward resisting renutrition.

A patient's spontaneous account of her difficulties shed some light for both on the paradox. RML told us that each time she has relapsed into the depths of anorexia, she has been aware that losing weight takes over as the most important thing in her life—even more important than the people she loves most as a devoted mother and daughter. The awareness that this dreadful perversion of her values has occurred only adds to her anguish and self hatred.

Later, JM wondered whether DS, even at his most desperate, would have been able to eat again if he had known that, by doing so, he would sacrifice the thing that was of the greatest value to him—the life of a loved one, for instance. Understanding then flooded into the student's face. "Ah then," he said, "I would surely have starved to death."

**Jane Morris** consultant psychiatrist, Eden Unit, Royal Cornhill Hospital, Aberdeen **ejanemorris@talk21.com**
**Daniel Seng** medical student, University of Aberdeen
**RML** recovering patient, Eden Unit

Patient consent obtained.

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A PATIENT'S JOURNEY

# Alcoholism

Anonymous,[1] Adrian M Raby[2]

See also EDITORIAL by Coltart and Gilmore

[1]UK
[2]Abingdon Medical Practice, London W8 6EG, UK
Correspondence to: A Raby
a.raby@imperial.ac.uk

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

This man's drinking habits nearly destroyed his marriage. He recounts how, with help from his wife, family, general practitioner, and other healthcare professionals, he got his life back on track

My wife was shattered and our marriage was nearly shattered, not by my drinking but by the deceit in which I had wrapped it. I am 78. All my adult life I have drunk alcohol, heavily, increasingly. Some years after retirement in 1995, to conceal my drinking I started on vodka. My day became triangulated around alcohol: are the pubs open, does this shop sell half-bottles, dare I ask for another Scotch?

Too often, my wife came home to find me incapable. Once, not understanding, and fearing that I had had a stroke, she took me to the emergency department, and once she called an ambulance. She felt humiliated by the pity the staff showed her and their contempt for me. A crisis erupted around Christmas 2009 when she noticed how frequently I was making large cash withdrawals. I had to tell the truth.

Addiction is selfishness. Having been afraid of the effect on me if my wife found out about my drinking, I had never imagined the devastating effects on her. Suddenly, I seemed to her a fraud. She shrank from family and friends. She developed shingles. She is diabetic: her blood sugar went haywire. Her unhappiness and confusion were palpable. She stated that she could not bear another drunken spell but would leave, and she told our children (each of us had been widowed previously).

At our wedding, her eldest son had expressed gladness that she had found me to look after her. I had not done so. I had devastated her. Nevertheless, he and his siblings were far more supportive than I deserved. My sons were distressed but loving. One of them asked simply, "Which is more important to you, to have another drink or to keep your marriage?"

We have two old friends, a couple, who have been suffering grave health problems. My wife said, "She has coped better than I have." They could each count on the other, whereas my wife could not count on me—I was the problem.

Our general practitioner, Dr Raby, had introduced me to the classic alcohol regime—"maximum four units a day, two dry days a week." I promised to follow this, but a unit became a glass, the glass a tumbler . . . five seems little more than four, six than five . . . today without became tomorrow without . . . and back to where we started. I went to see him again with my wife. She poured out the whole story and he promised to arrange the help I needed.

The NHS was excellent, the staff thoughtful, and the approach well structured. Dr Raby introduced me to a friendly and positive specialist nurse. Her advice was categorical. I must give up alcohol completely for at least six months. Addicts give up often but, at least for my type, there comes a truly decisive moment. A concatenation of old reasons for giving up comes together with a powerful new element. Suddenly, deep inside, the option of not giving up closes.

Years earlier, when I gave up smoking, this new element was observing my father die of emphysema. Now, it was my desperate wish to repair the damage to my wife and to save our marriage.

I saw my specialist nurse every couple of weeks. She encouraged me to foresee occasions when I would want a drink and to work out how to handle them, perhaps by circumventing the situation or finding distraction. Above all, she taught me to prepare mentally so that temptation never took hold.

My wife cut her finger on a broken mug and had to be rushed to the emergency department with septicaemia. They operated on her twice and kept her in for a week. She was on antibiotics for a fortnight after she came out. Her resilience had been badly affected by the shock of discovering my addiction, and for months she remained prone to bouts of exhaustion. The only good thing was that the advice I had received from my specialist nurse paid off. I was worried, and most evenings tired and alone, just right for a quick drink. In fact, I barely thought about alcohol.

My track record gave my wife good reason to doubt me. However, she rejected breathalysers and warily took me on trust. My expenditure, blood pressure, and weight fell sharply. I guess my comportment gradually improved from when I had been drinking.

At social events I would state that I was taking a holiday from booze. Some probably muttered, "Aha, I thought he was an alcoholic," but most, no doubt, had their own concerns and barely noticed. Our children felt that nobody should drink in my presence. They took some convincing that I did not want them penalised by my misbehaviour.

My specialist nurse referred me to a psychiatrist. He carried out a cognitive intelligence test, although I did not realise that's what it was. Presumably he also evaluated whether I needed chemical help or had yet done myself serious physical or mental damage. Undeservedly, I seemed not to have. He urged me to see a psychologist. I did not want anybody fossicking about in my psyche (there is nothing to find), but I did want to convince my wife that I was tackling things seriously, so I accepted.

The psychologist was easy to talk to and always constructive. She said to me, "Tell your wife that you are the same chap but that the alcohol takes over." The structure of my eight sessions with her was largely based on research. She wondered why I drank—for example, was it

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- Rheumatoid arthritis (*BMJ* 2010;341:c7095)
- Heart transplant (*BMJ* 2010;341:c4918)
- Duchenne muscular dystrophy (*BMJ* 2010;341:c4364)
- Vitiligo (*BMJ* 2010;341:c3780)
- At sixes and sevens: prostate cancer (*BMJ* 2010;341:c3834)

to overcome insecurity or improve my chances sexually? The answer was always "No."

We discussed the meaning of the word "craving." To me, craving is sharp, physical yearning, hard to resist. I still feel a craving for tobacco, decades after giving up, if somebody lights up nearby. I have never felt it for alcohol. To the psychologist, craving is merely a wish for something.

She remarked, "There is still a piece of the jigsaw missing." I do not feel any craving for alcohol, so why did I become alcoholic? I regard myself as intelligent and honest. Nevertheless, for decades I drank to an extent that was downright stupid, and that led to downright dishonesty.

Although every drunkard has something in common with every other drunkard, not all drunkards are the same. We are not all "fighting our demons." I have no demons. Although uncomfortable and ashamed when hung over, I am otherwise quite at ease with myself. I suffered no secret childhood harm. I loved and respected my parents and cherish their memory. Admittedly, I went to boarding school, but in war time this seemed normal.

I associate alcohol with freedom and manliness, perhaps thanks to the fictional heroes of my adolescence—Bulldog Drummond's pints, James Bond's martinis—but mostly I drank because I enjoy the taste and effect. However, alcohol rots your judgment—"one more won't hurt." The first "one more" may not. Later comes the one that does—and you are gone.

Once you acknowledge and then understand the problem, you can control it. I left it late. Happily, my wife had the generosity to trust me once more and the persistence to stay with me.

My six months' abstinence would have ended in mid-July 2010. We were in France, out of range of Dr Raby's support system, and my wife feared moderation would be impossible. I extended the abstinence period until our return home in September and then for a further few weeks because of another trip abroad.

In October 2010, after nine months' abstinence, I started drinking occasionally. For me the rule "maximum four units a day, two dry days a week" feels wrong—a maximum easily becomes a norm. Rather than be a steady drinker who takes a regular break, I prefer to be a steady non-drinker who takes the odd glass. It seems to be working.

### PATTERNS OF PROBLEM ALCOHOL USE

*Hazardous alcohol use*—Drinking above the Department of Health's recommended level with no current evidence of physical, psychological, or social harm (estimated 18% of the population in England)

*Harmful alcohol use*—Drinking at a level that is already causing harm (7% of the population in England)

*Alcohol dependence*—A cluster of symptoms including a subjective compulsion to drink, physical withdrawal symptoms, and continued drinking despite evidence of harm

### USEFUL RESOURCES

Screening and Intervention Programme for Sensible Drinking, SIPS (www.sips.iop.kcl.ac.uk)—Supports the National Alcohol Harm Reduction Strategy for England; screening and intervention tools are available on the website

Drinkaware (www.drinkaware.co.uk)—Aims to "increase awareness and understanding of the role of alcohol in society" through "campaigning, educational, and partnership work." The website has advice for drinkers

NHS Choices: Drinking and Alcohol (www.nhs.uk/Livewell/alcohol)—NHS website giving advice for drinkers

### A DOCTOR'S PERSPECTIVE

#### Alcohol

Health problems caused by alcohol use are becoming increasingly common as a result of changing patterns of alcohol consumption. One recent report by the National Audit Office estimates that more than 10 million people in the United Kingdom drink consistently more than the amount recommended by the Department of Health.[1] This correlates with a doubling in the number of deaths related to alcohol use during the 15 years up to 2006.[1] Practitioners in all fields of medicine therefore need to be able to detect problematic patterns of drinking and intervene to prevent the harms that may result from them.

#### Detecting the problem

As a profession the evidence suggests that generally we are not effective at detecting or managing patients with alcohol problems. Data on practice populations from the NHS Information Centre for 2009 combined with estimates of drinking prevalence from the National Audit Office 2008[1] suggest that a general practitioner with an average list size of 2000 in England can expect to have around 230 registered men and 160 women who drink excessively. However, a survey of general practitioners in England found that two thirds reported managing only one to six such patients a year.[2] Common reasons for not screening for alcohol may include feelings of being inadequately trained, fear of upsetting patients, and the belief that interventions are unlikely to be effective.

Several screening tools are available, such as the AUDIT (PC) (www.alcohollearningcentre.org.uk/_library/AUDIT-PC.doc), which takes about two minutes to complete. It requires little training and can be carried out by a nurse or a healthcare assistant. Screening can be conducted at the initial contact with a patient such as at registration at a general practice. Clinicians should also screen opportunistically when a patient presents with a problem to which alcohol could be a contributory factor (such as discovery of abnormal liver function, or hypertension). In the case of the patient in this article, his presentation with falls was directly related to his use of alcohol and offered an opportunity to inquire about alcohol.

#### Interventions

The evidence suggests that even brief interventions (taking as little as 10 minutes to complete) may be effective in reducing the overall level of alcohol use, changing drinking patterns, preventing future drinking problems, improving health, and reducing healthcare costs. Several tools are readily available, such as through the screening and intervention programme for sensible drinking (SIPS) (see resources box). Such tools help clinicians to structure feedback about alcohol use and to develop treatment goals with the patient. Critical to the success in helping patients is the ability to call on the expertise of other health professionals, in this case an experienced specialist nurse and an extended team.

One of the most important lessons that I have learnt from this and other cases is the need to adopt a non-judgmental approach where the plan is tailored to the needs and goals of the individual patient. There is no "one size fits all" approach. Some people may just need simple advice about how to keep drinking within sensible limits; for others, abstinence may be the only option. In the case of my patient a period of abstinence with a return to controlled drinking has proved effective. As with all patients, however, his journey continues with follow-up and support.

Adrian M Raby, general practitioner

**Competing interests:** A Raby has completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years, no other relationships or activities that could appear to have influenced the submitted work.

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RATIONAL IMAGING

# Role of brain imaging in early parkinsonism

David P Breen,[1] James B Rowe,[2] Roger A Barker[1]

[1]Cambridge Centre for Brain Repair, University of Cambridge, Cambridge CB2 0PY
[2]Department of Clinical Neurosciences, University of Cambridge

Correspondence to: D P Breen
davebreen@excite.co.uk

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This series provides an update on the best use of different imaging methods for common or important clinical presentations. The series advisers are Fergus Gleeson, consultant radiologist, Churchill Hospital, Oxford, and Kamini Patel, consultant radiologist, Homerton University Hospital, London. To suggest a topic for this series, please email us at practice@bmj.com.

**bmj.com** Previous articles in this series

- Imaging transient ischaemic attack with diffusion weighted magnetic resonance imaging (*BMJ* 2010;340:c2215)
- Investigating suspected bone infection in the diabetic foot (*BMJ* 2009;339:b4690)
- Acute lower gastrointestinal haemorrhage (*BMJ* 2009;339:b4156)
- Incidental thyroid nodule (*BMJ* 2009;338:b611)
- Definitive characterisation of adrenal lesions (*BMJ* 2009;338: a3092)

Imaging can be helpful in the differential diagnosis of Parkinson's disease, providing it is used in appropriately selected patients and the limitations of the techniques are fully understood

## The patient

A 67 year old man presents with an eight month history of tremor and "lagging behind" when walking with friends. He had labyrinthine symptoms in the past and has taken prochlorperazine for the past four years. Neurological examination confirms a rest and postural tremor affecting the left hand, as well as slight bradykinesia on repetitive fine finger and hand movements, worse on the left. No rigidity, gait disturbance, or postural instability are seen.

## What is the differential diagnosis?

Parkinson's disease is a clinical diagnosis, but even specialists are only 90% accurate.[1] The first step is to decide whether the patient does in fact have parkinsonism. This relies on looking for four cardinal features: bradykinesia, rest tremor, rigidity, and postural instability. The diagnosis of parkinsonism requires the presence of at least two of these motor features. Our patient has evidence of bradykinesia and tremor, together with a degree of asymmetry, and therefore fulfils the criteria.

Prochlorperazine is one of several drugs that can induce parkinsonism; others are neuroleptics, metoclopramine, calcium channel blockers, methyldopa, sodium valproate, lithium, and certain antidepressants. Parkinsonism usually presents soon after the offending drug is started, with bilateral signs and no tremor, so our patient is atypical in this regard. In some patients the drug can be stopped and the response observed, but this is not always straightforward—for example, in those with severe mental health disorder who rely on neuroleptics—and the effect of the drug may take months to wear off.

Our patient has no clinical features pointing towards specific neurodegenerative syndromes that in their early stages can resemble Parkinson's disease. These include multiple system atrophy, progressive supranuclear palsy, and corticobasal degeneration. Although sometimes called "Parkinson plus" syndromes, they are rather different in pathology, clinical features, prognosis, and response to drugs. Classic signs of progressive supranuclear palsy and multiple system atrophy include bilateral disease at onset and less rest tremor. Patients with progressive supranuclear palsy also develop a supranuclear gaze palsy, recurrent falls, apathy, and a frontal dementia. In contrast, patients with multiple system atrophy have early autonomic dysfunction or cerebellar signs, or both. Corticobasal degeneration is extremely variable, but common presentations include asymmetric rigidity and dystonia with apraxia (a "useless hand"), cognitive deficits, and a poor response to treatment with levodopa.

### LEARNING POINTS

- Routine brain imaging is unnecessary in patients with typical Parkinson's disease
- Dopamine transporter (DAT) imaging can help to differentiate patients with Parkinson's disease from healthy individuals and patients with essential tremor or drug induced parkinsonism
- Structural MRI may be performed to rule out alternative diagnoses (including other neurodegenerative syndromes and structural or vascular lesions)

The diagnosis of Parkinson's disease should be based primarily on clinical features. The UK Parkinson's Disease Society Brain Bank Clinical Diagnostic Criteria are commonly used in routine practice and research trials.[2] These criteria do not incorporate brain imaging, which is unnecessary in patients with typical Parkinson's disease. This approach is supported by the National Institute for Health and Clinical Excellence[3] and Scottish Intercollegiate Guidelines Network.[4] In this case, however, imaging is likely to be helpful because it remains unclear whether the patient's symptoms are due to Parkinson's disease or drug induced parkinsonism.

## Clinical imaging modalities

### Single photon emission computed tomography

The dopamine transporter (DAT) is an 80 kDa protein located on the plasma membrane of axonal nerve terminals, where it regulates dopamine concentration in the synaptic cleft. Single photon emission computed tomography (SPECT) uses the density of a ligand radiolabelled with DAT as a marker of dopamine terminal innervation, thus helping to differentiate Parkinson's disease from alternative diagnoses (in this case, drug induced parkinsonism).

In Parkinson's disease, radiotracer uptake is markedly reduced in the putamen and to a lesser extent the caudate (often asymmetrically). Uptake is normal in controls and patients with essential tremor and drug induced parkinsonism (fig 1). Striatal DAT imaging with SPECT differentiates between clinically probable Parkinson's disease and essential tremor with a sensitivity of 79-100% and specificity of 80-100%.[5]

The diagnostic accuracy of DAT imaging depends on the patient population being tested—DAT imaging is more likely to be abnormal in patients with Parkinson's disease with an akinetic-rigid presentation than in patients with tremor dominant disease.[6] Reproducibility of scans is also contentious; one small study of $^{123}$I-β-SPECT showed that radiotracer uptake varied by up to 40% from one day to the next.[7] A different tracer produced better reproducibility,[8] and better measurement of radioligand binding may further reduce variability. Some drugs can affect the DAT scan; these include stimulants and some selective serotonin reuptake inhibitors but not levodopa (for a comprehensive list see

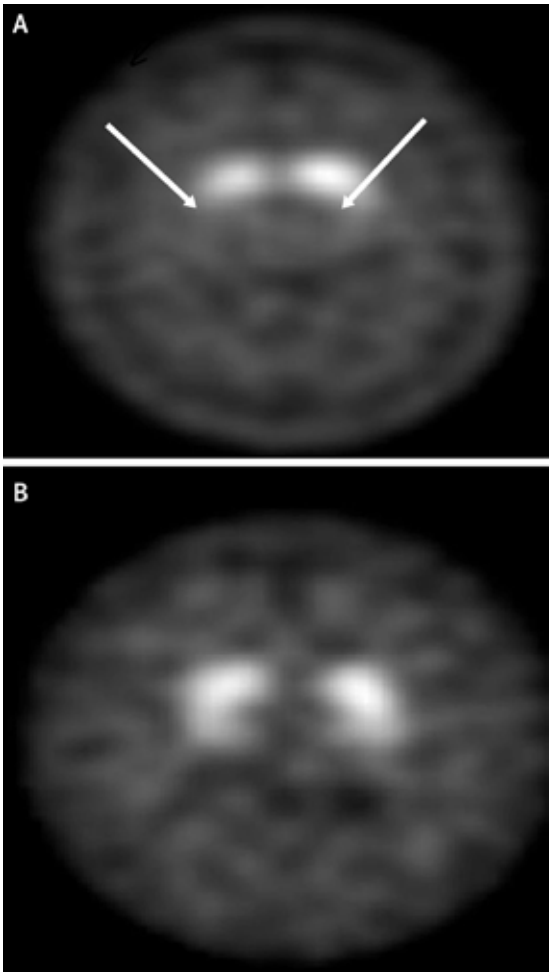


**Fig 1** | DAT scans in patients with drug induced parkinsonism (top) and Parkinson's disease (bottom). Radiotracer uptake is reduced bilaterally in the patient with Parkinson's disease (worse on the right side)

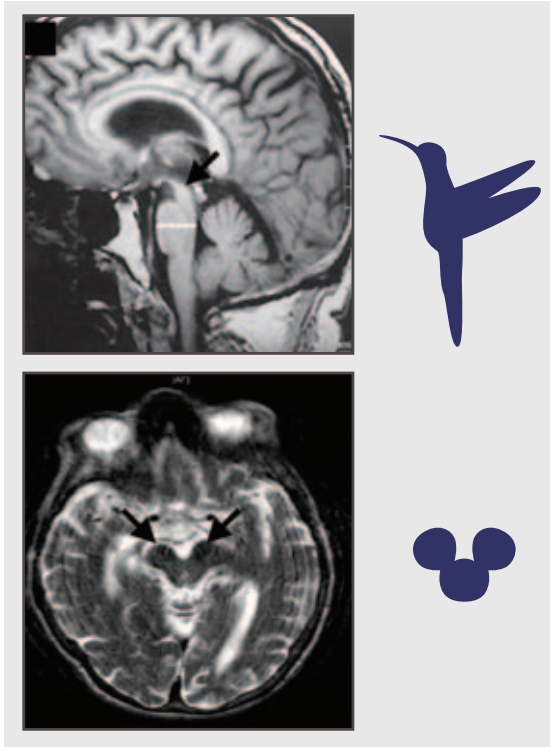

**Fig 2** | Magnetic resonance brain scans in patient with progressive supranuclear palsy, showing characteristic "hummingbird sign" and "Mickey Mouse" midbrain

Kagi et al[9]). DAT imaging cannot effectively differentiate between Parkinson's disease, progressive supranuclear palsy, multiple system atrophy, and corticobasal degeneration, so it should never be a substitute for careful clinical assessment. Given the exposure to radiation that is required and problems with interpretation, it should be requested only by a specialist.

### Magnetic resonance imaging

When clinical features are not typical for Parkinson's disease (young patients with acute or stepwise progression of symptoms, for example), structural brain imaging should be considered to rule out other conditions. Magnetic resonance imaging (MRI) is preferable to computed tomography because of superior resolution and diagnostic sensitivity (especially in the posterior fossa), unless there are contraindications such as severe claustrophobia or metal in the brain or eye. Our case study patient has no atypical parkinsonian features, so MRI is unnecessary.

Structural MRI is generally unremarkable in patients with Parkinson's disease. In vascular parkinsonism (which typically presents in the lower body without tremor), MRI shows ischaemic lesions in the white matter. In elderly patients, however, it can be difficult to know if these lesions are sufficient to account for their parkinsonism. Space occupying lesions, normal pressure hydrocephalus, and lesions of the basal ganglia can also cause parkinsonism with characteristic MRI appearances.

MRI can be helpful in identifying other specific neurodegenerative syndromes. Although not pathognomonic, atrophy of the midbrain tegmentum is seen in virtually all patients with progressive supranuclear palsy (the "hummingbird sign" on sagittal MRI or "Mickey Mouse" midbrain on axial slices; fig 2).[10] Putaminal abnormalities are more common in multiple system atrophy and progressive supranuclear palsy than in Parkinson's disease,[11] but they may be detected only by an experienced neuroradiologist (or not at all) and rarely change clinical management. In corticobasal degeneration, MRI shows asymmetric cortical atrophy in clinically affected areas, especially frontal and parietal association cortex.

## Outcome

The patient reported no improvement in symptoms after stopping prochlorperazine for three months. He was seen by a different neurologist in the follow-up clinic, and a DAT scan showed reduced uptake bilaterally (worse on the right side). He was diagnosed with idiopathic Parkinson's disease and started taking ropinirole, and his motor symptoms improved considerably.

## Research imaging modalities

Research into neuroimaging in Parkinson's disease may lead to facilitation of early accurate diagnosis, prediction of complications such as dementia, a better understanding of the pathophysiology of the condition, and analysis of the mechanisms of cognitive and motor phenotypes in the disease.

### Functional MRI

Patients with early Parkinson's disease typically have difficulty in planning, organising, and regulating goal directed

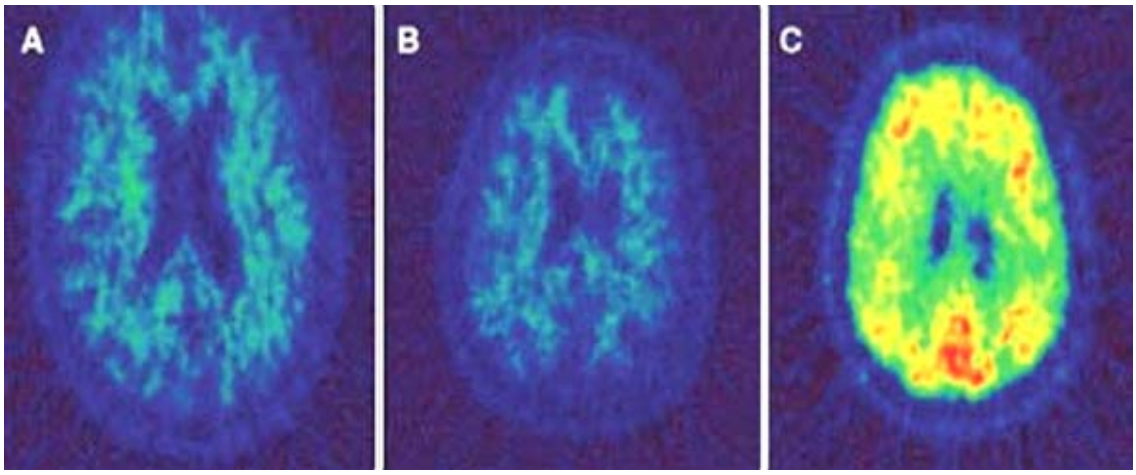


**Fig 3** | PET imaging with $^{11}$C-Pittsburgh Compound B, a thioflavin based marker of amyloid β plaque load: (A) elderly patient without Parkinson's disease; (B) patient with Parkinson's disease dementia with no significant plaque deposition in the brain; (C) patient with Alzheimer's disease in whom amyloid β is extensive[20]

behaviours—so called frontal executive dysfunction. Functional MRI (fMRI) measures the blood oxygen level dependent (BOLD) signal, a function of the changes in cerebral blood flow and oxygenation after neural activity.

Previous fMRI studies have found abnormal frontostriatal activity in early Parkinson's disease.[12] Studies have also suggested that there is a U shaped relation between dopamine concentrations in the prefrontal cortex and neural function, with different optimal dopaminergic states for motor and cognitive functions.[13]

One of the limitations of this imaging technique (and others) is that we do not fully understand the pathological basis of Parkinson's disease. Therefore, fMRI remains an indirect measure of pathology in Parkinson's disease.

### Positron emission tomography

Up to 80% of patients with Parkinson's disease may develop a dementia.[14 15] PET studies of resting brain function in such dementia show an Alzheimer-like pattern of reduced glucose utilisation; posterior parietal and temporal association areas are most affected.[16] Up to a third of non-demented, but cognitively impaired, patients with Parkinson's disease also show reduced metabolism in the parietal and temporal lobes[17]; longitudinal follow-up is needed to assess whether they too develop dementia. In an effort to elucidate the pathophysiology of Parkinson's disease and its evolving dementia, PET ligands have also been used to study amyloid β plaque load ($^{11}$C-PIB-PET; fig 3)[18] and binding to peripheral benzodiazepine receptors on activated microglial cells as a marker of cerebral inflammation ($^{11}$C-PK11195; fig 4).[19]

Moreover, PET can differentiate between normal and abnormal nigrostriatal innervation. In a study of 167 patients with parkinsonism of unknown cause followed up for a mean of 2.6 years, FDG-PET was able to differentiate between Parkinson's disease, multiple system atrophy, and progressive supranuclear palsy (positive predictive value >90% for each condition).[21]

Despite this, the future role of PET outside of research trials remains uncertain, given that this type of imaging is expensive, is not widely available, requires low dose exposure to radiation, and relies on specialist interpretation.

Prospective, longitudinal imaging studies are needed to identify patients with early Parkinson's disease, who are at increased risk of cognitive impairment and dementia. The ICICLE-PD study (Incidence of Cognitive Impairment in Cohorts with Longitudinal Evaluation—Parkinson's Disease; http://public.ukcrn.org.uk/search/StudyDetail.aspx?StudyID=5643) is addressing this important research question using MRI (structural and functional) and PET alongside clinical markers.

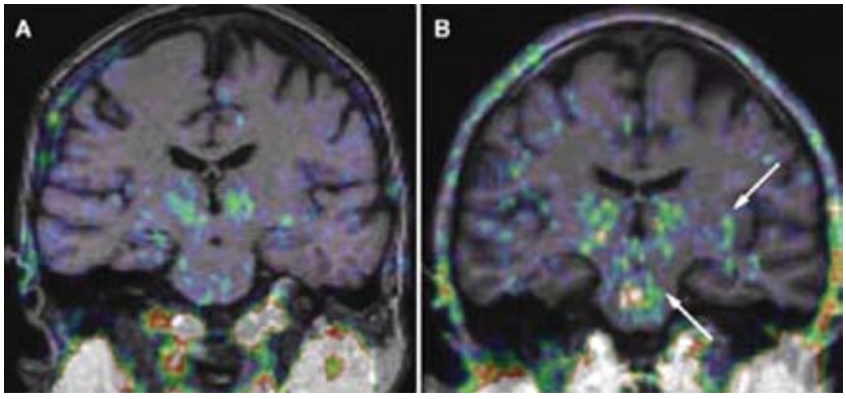


**Fig 4** |Assessment of microglial activation by use of $^{11}$C-PK11195 PET: (A) only mild microglial activation seen in the thalamus of healthy control; (B) raised activation in the midbrain and striata (arrows) of patient with Parkinson's disease, with normal levels of thalamic activation[20]

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## CORRECTIONS AND CLARIFICATIONS

### Obituary: James Mourilyan Tanner

In the obituary for James Mourilyan Tanner (*BMJ* 2010;341:c5374, print publication 2 October 2010, p732) the eponymous scale is formally known as the Tanner scale of pubertal development and is a qualitative assessment of the development of breasts, genitals, and pubic hair in adolescence. Tanner's study at Harpenden was a new study, not a continuation of a wartime study of malnutrition. *A History of the Study of Human Growth* was published in 1981 not 1951. Tanner published more than 300 scientific papers and monographs between 1944 and 2001. Treatment with genetically engineered human growth hormone resumed in 1985, not the 1990s. An error in our production processes meant that the author, Caroline Richmond, was not credited in print or online.

### Mentally disordered or lacking capacity? Lessons for management of serious deliberate self harm

In the second paragraph of this analysis article by Anthony S David and colleagues (*BMJ* 2010;341:c4489, print publication 18 September 2010, pp 587-9), under the heading "Synopsis based on coroner's report" we gave the wrong date for Kerrie Wooltorton's admission to hospital under section 3 of the 1983 Mental Health Act. She was admitted in March 2007 (not March 2009).

### Avoidance of high concentration oxygen in chronic obstructive pulmonary disease

In this editorial by B Ronan O'Driscoll and Richard Beasley an error occurred in the second paragraph (*BMJ* 2010;341:c5549, print publication 30 October 2010, pp 898-9). We wrongly referred to alveolar carbon dioxide tension; we should have said a mean difference in arterial carbon dioxide tension. However, alveolar carbon dioxide tension is also mentioned later, in the fourth paragraph, where it is correct.

### Don't ignore home grown medicine

In this feature by Shahram Aarabi and colleagues (*BMJ* 2010;340:c3187, print publication 19 June 2010, p 1335) we introduced an error into the first author's affiliation address. We should have said University of Washington, Seattle, Washington State (not University of Washington, Washington, DC).

### Communicating with deaf people: risk of ill health is increased

During the editing of this letter by Jeetesh V Patel (*BMJ* 2010;341:c5986, print publication 30 October 2010, p 905) we converted the author's use of "Deaf" (capital D) to "deaf" (lower case d), thus losing some of his intended meaning. The author had used Deaf to indicate he was talking about profoundly deaf people who use sign language. The *BMJ* article that had prompted his letter to the *BMJ* (*BMJ* 2010;341:c4672, doi:10.1136/bmj.c4672) contains a clarification (in box 1) of the distinction between Deaf and deaf: "People who call themselves Deaf (with an upper case 'D') usually use sign language as their first language and consider themselves 'culturally' deaf (that is, they regard deafness as a difference in human experience rather than a disability). They usually have profound deafness, which may be congenital. They may use some lipreading but often prefer to communicate directly in sign language; they may gain little benefit from written material."

### Promotion of cycling and health

In this editorial by Nanette Mutrie and Fiona Crawford (*BMJ* 2010;341:c5405, print publication 23 October 2010, pp 842-3), the authors have alerted us to an error in the second sentence of the fifth paragraph. It should have read: "Of the 25 studies included in their quantitative synthesis, only seven [not 'six'] met at least three of the five validity criteria and only one of these [not 'and none of these'] was based in the United Kingdom, despite the fact that UK studies represented over half of those scrutinised."

### Endgames: Statistical question

In this question about z scores, the numbers of patients given in the example study were incorrect (*BMJ* 2010;341:c6746, print publication 4 December, p 1225). In the first paragraph, the second sentence should start: "A total of 644 children [not "511 children"] aged between 7 and 11 years . . ." Also, not all of these children were followed up at three years; the z scores for body mass index were calculated only for the 434 children who were.