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## GUIDELINES

# Management of lower urinary tract dysfunction in neurological disease: summary of NICE guidance

Sharon Swain,<sup>1</sup> Ralph Hughes,<sup>1</sup> Mark Perry,<sup>1</sup> Simon Harrison,<sup>2</sup> on behalf of the Guideline Development Group

<sup>1</sup>National Clinical Guideline Centre, Royal College of Physicians of London, London NW1 4LE, UK

<sup>2</sup>Mid-Yorkshire Hospitals NHS Trust, Department of Urology, Pinderfields Hospital, Wakefield WF1 4DG, UK

Correspondence to: S Swain  
Sharon.Swain@rcplondon.co.uk

Cite this as: *BMJ* 2012;345:e5074  
doi: 10.1136/bmj.e5074

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.

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Previous articles in this series

- ▶ Diagnosis and management of lower limb peripheral arterial disease: summary of NICE guidance (*BMJ* 2012;345:e4947)
- ▶ Risk assessment of fragility fractures: summary of NICE guidance (*BMJ* 2012;345:e3698)
- ▶ Risk identification and interventions to prevent type 2 diabetes in adults at high risk: summary of NICE guidance (*BMJ* 2012;345:e4624)
- ▶ Management of an acute painful sickle cell episode in hospital: summary of NICE guidance (*BMJ* 2012;344:e4063)
- ▶ Management of venous thromboembolic diseases and the role of thrombophilia testing (*BMJ* 2012;344:e3979)

A wide range of neurological conditions can affect the function of the lower urinary tract, potentially causing distressing symptoms and even renal damage. It is important to ask patients with neurological disease about urinary symptoms, as identifying these should lead to appropriate assessment and treatment, improvement in quality of life, and a reduction in long term morbidity. Clinicians can easily overlook urinary tract problems as they focus on other important clinical matters, but a better understanding of how to deal with lower urinary tract problems may increase the confidence of healthcare professionals in this area. This article summarises the most recent recommendations from the National Institute for Health and Clinical Excellence (NICE) on the management of lower urinary tract dysfunction in neurological disease.<sup>1</sup>

### Recommendations

NICE recommendations are based on systematic reviews of the 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 given in *italic* in square brackets.

### Initial assessment

Patients needing assessment include those with newly diagnosed neurological disease; those with known neurological disease and new or changing symptoms suggesting urinary tract dysfunction; and those requiring periodic reassessment of their urinary tract management. The interval between routine assessments will depend on the person (for example, their age or diagnosis) but should not exceed three years.

- Take a clinical history, including information about urinary tract symptoms; neurological symptoms and diagnosis (if known); clinical course of the neurological disease; bowel symptoms; sexual function; comorbidities; use of prescription and other medication and treatments.
- Assess the impact of the underlying neurological disease on factors that will affect how lower urinary tract dysfunction can be managed, such as mobility, hand function, cognitive function, social support, and lifestyle.
- Do a urine dipstick test using an appropriately collected sample to test for the presence of blood, glucose, protein, leukocytes, and nitrites. Appropriate urine samples include clean-catch midstream samples and samples taken from a freshly inserted intermittent sterile catheter or from a catheter port. Do not take samples from leg bags.

- If the result of the dipstick test and the person's symptoms suggest an infection, arrange a urine bacterial culture and antibiotic sensitivity test before starting antibiotic treatment. Treatment need not be delayed but may be adapted when results are available.
- Be aware that bacterial colonisation will be present in people using a catheter and so urine dipstick testing and bacterial culture may be unreliable for diagnosing active infection.

*[All the above points are based on the experience and opinion of the Guideline Development Group (GDG)]*

### Referral for further care

Urodynamic studies (investigations of lower urinary tract function) are needed in some cases to identify causes of incontinence and look for risk factors for renal deterioration. Ultrasound scanning and cystoscopy may sometimes be indicated to look for complicating factors such as urinary tract stones, hydronephrosis, or bladder cancer.

- Refer people for urgent investigation (such as ultrasonography, urodynamic investigations, renal scintigraphy) if they have any of the following "red flag" signs and symptoms:
  - Haematuria
  - Recurrent urinary tract infections (for example, three or more infections in six months)
  - Loin pain
  - Recurrent catheter blockages (for example, catheters blocking within six weeks of being changed)
  - Hydronephrosis or kidney stones on imaging
  - Biochemical evidence of renal deterioration.
- Be aware that urinary tract disease can cause changes in neurological symptoms (for example, confusion or worsening spasticity), and consider further urinary tract investigation and treatment if this is suspected.
- Assess the impact of lower urinary tract symptoms on the person's family members and carers and consider ways of reducing any adverse impact, such as stress, that may harm the person. If abuse is suspected, follow local safeguarding procedures.

*[All the above points are based on the experience and opinion of the GDG]*

### Urodynamic investigations

Although a urodynamic based understanding of the effects of neurological disease on urinary tract function has underpinned major advances in patient management, effective clinical management doesn't always require invasive urodynamic investigations.

### FURTHER INFORMATION ON THE GUIDANCE

The full guideline provides further guidance on improving bladder storage and stress incontinence, including surgical interventions. It also expands on the provision of information and support for patients and carers and on how to improve access to services (see recommendations below).

Providing information for patients and carers is challenging because of, for example, the need to present complex information in a digestible form and to provide information that is accurate, given the many gaps in our knowledge in this field.

- Tailor information and training to the individual's physical condition and cognitive function to promote their active participation in care and self management. *[Based on very low quality evidence from one randomised controlled trial and three observational studies]*
- When managing the transition of a person from paediatric services to adult services for ongoing care of neurogenic lower urinary tract dysfunction:
  - Formulate a clear structured care pathway at an early stage and involve the person and/or their parents and carers
  - Involve the person's parents and carers when preparing transfer documentation with the person's consent
  - Provide a full summary (for the person and the receiving clinician) of the person's clinical history, investigation results, and details of treatments
  - Integrate information from the multidisciplinary health team into the transfer documentation
  - Identify and plan the urological services that will need to be continued after the transition of care
  - Formally transfer care to a named individual(s).

*[Based on high to low quality evidence from qualitative studies]*

#### Methods

The guideline was developed according to NICE guideline methods ([www.nice.org.uk/aboutnice/howwework/developingniceclinicalguidelines/developing\\_nice\\_clinical\\_guidelines.jsp](http://www.nice.org.uk/aboutnice/howwework/developingniceclinicalguidelines/developing_nice_clinical_guidelines.jsp)). The Guideline Development Group (GDG) comprised specialist nurses, a general practitioner, specialists in urology and rehabilitation medicine, a geriatrician, urological surgeons, and patients and carer representatives. This involved systematic searching and critically appraising and summarising the clinical and cost effectiveness evidence. The scope and full guideline was posted on the NICE website as part of a stakeholder consultation. The GDG also conducted new cost effectiveness analysis, for botulinum toxin type A.

NICE has produced four different versions of the guideline: a full version; a quick reference guide; a version known as the "NICE guideline" that summarises the recommendations; and a version for patients and the public. All these versions are available from the NICE website (<http://guidance.nice.org.uk/CG148>). Updates of the guideline will be published according to the NICE guideline development programme.

#### Future research

The GDG highlighted some important questions that need to be answered:

- How do different antimuscarinic drugs compare in this patient population and what are their risks, particularly in relation to central nervous system side effects?
- Do repeated intradetrusor injections of botulinum toxin type A have long term efficacy, and can they protect the kidneys from high bladder pressures?
- How can the burden of urinary tract infection be reduced and by which strategies?
- How do different urinary tract management strategies (such as intermittent self catheterisation, the use of indwelling catheters) differ in terms of complications and quality of life outcomes?

- Do not routinely offer urodynamic investigations (such as filling cystometry and pressure flow studies) to people at low risk of renal complications (for example, most people with multiple sclerosis).
- Offer video-urodynamic investigations to people at high risk of renal complications (for example, people with spina bifida, spinal cord injury, or anorectal abnormalities).

*[Both points are based on very low quality evidence from observational studies]*

#### Treatment

Assessment of the patient allows the patient, carers, and clinical team to formulate options for managing the patient's neurogenic lower urinary tract dysfunction. In

some cases, this will be a relatively simple process requiring treatment of a single symptom, such as urinary urgency in a patient with multiple sclerosis who empties their bladder well. In contrast, a person with spina bifida might have to consider options that include the use of intermittent self catheterisation after a surgical lower urinary tract reconstruction or the containment of urinary incontinence with a penile sheath system or pads. The guideline includes recommendations about the treatment of the various abnormalities that might be present.

#### Impaired bladder storage

Impaired bladder storage is frequently caused by the presence of involuntary contractions (detrusor overactivity) and will typically cause symptoms of urinary frequency, urgency, and incontinence. Possible treatments include the use of various behavioural treatments (such as the timed voiding), the prescription of antimuscarinic drugs, the administration of botulinum toxin type A injections into the bladder wall, and surgical enlargement of the bladder by augmentation cystoplasty. Botulinum toxin type A injections have emerged in recent years as a treatment option in neurogenic incontinence and the guideline includes recommendations about their use in different neurological conditions and in adults and children.

#### Stress incontinence

Stress incontinence is caused by weakness of the urethral sphincter mechanism and arises from damage to the sphincter's nerve supply or through urethral trauma from indwelling urethral catheters. The guideline covers the use of pelvic floor muscle training and surgical procedures such as the use of autologous fascial slings and the artificial urinary sphincter.

#### Impaired bladder emptying

Impaired bladder emptying will often require the use of intermittent self catheterisation or an indwelling (usually suprapubic) catheter. For people using an indwelling catheter, the guideline supports the use of a catheter valve (a tap-like device that can be switched on or off to drain urine from the bladder or to stop drainage) as an alternative to continuous bladder drainage into a bag. The guideline recommends not using an adrenergic antagonists.

#### Antibiotic prophylaxis

Urinary tract infection is common in people with neurological disease, and the challenge is to balance the reduction of the burden of infections on the individual patient with the need to contain the development of antibiotic resistance.

- Do not routinely use antibiotic prophylaxis for urinary tract infections in people with neurogenic lower urinary tract dysfunction.
- Consider antibiotic prophylaxis for people who have a recent history of frequent or severe urinary tract infections.
- Before prescribing antibiotic prophylaxis for urinary tract infection:
  - Investigate the urinary tract for an underlying treatable cause (such as urinary tract stones or incomplete bladder emptying)

- Take into account and discuss with the person the risks and benefits of prophylaxis
- Refer to local protocols approved by a microbiologist or discuss suitable regimens with a microbiologist.

[All the above points are based on moderate to very low quality evidence from randomised controlled trials]

#### Information and support

- Offer specific information and training to people with neurogenic urinary tract dysfunction, their family members, and carers. Ensure that people who are starting to use, or are using, a bladder management system that involves the use of catheters, appliances, or pads:
  - Receive training, support, and review from healthcare professionals who are trained to provide support in the relevant bladder management systems and are knowledgeable about the range of products available
  - Have access to a range of products that meet their needs
  - Have their products reviewed—at the least, every two years.

[Based on very low quality evidence from a randomised controlled trial and observational studies]

#### Potential complications

- Discuss with the person and/or their family members and carers the increased risk of renal complications (such as kidney stones, hydronephrosis, and scarring) in people with neurogenic urinary tract dysfunction (in particular, those with spina bifida or spinal cord injury). Tell them about the symptoms to look out for (such as loin pain, haematuria) and when to see a healthcare professional.

[Based on very low quality evidence from a randomised controlled trial and observational studies]

#### Monitoring and surveillance protocols

- Offer lifelong ultrasound surveillance of the kidneys to people at high risk of renal complications (for

example, at annual or twice yearly intervals). These include people with spinal cord injury, spina bifida, or adverse features on urodynamic investigations such as impaired bladder compliance, detrusor-sphincter dyssynergia, or vesicoureteric reflux.

[Based on very low quality evidence from observational studies and economic evidence with partial applicability and potentially serious limitations]

#### Overcoming barriers

Healthcare professionals from different disciplines should help patients to access relevant services. This requires the cooperation of commissioners and providers—for example, through better “signposting” of referral pathways. Not all centres currently offer botulinum toxin type A injections, and implementing the recommendations will require service development. However, the health economic model showed that botulinum toxin is more cost effective than standard care when augmentation cystoplasty is not appropriate.

The members of the Guideline Development Group were Christine Anderson, Alison Bardsley, Noreen Barker, Amelia Denny, Tamara Diaz, Clare Fowler, Laura Graham, Simon Harrison (chair), Ralph Hughes (health economist), Judith Jesky, Philipp Laramée, Doreen McClurg, Keith McDermott, Susan Orme, Mark Perry (systematic reviewer), Gill Ritchie (guideline lead), Sharon Swain (systematic reviewer), Paul Tophill, Julie Vickerman, Richard Whittome, Alun Williams, Sue Woodward.

**Contributors:** All authors drafted the article. All authors revised it critically for important intellectual content and approved the final version to be published. All authors are guarantors of this article.

**Competing interests:** All authors have completed the ICMJE uniform disclosure form at [http://www.icmje.org/coi\\_disclosure.pdf](http://www.icmje.org/coi_disclosure.pdf) (available on request from the corresponding author) and declare: no financial support for the submitted work from anyone other than NICE; 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.

**Provenance and peer review:** Commissioned; not externally peer reviewed.

- 1 National Institute for Health and Clinical Excellence. Management of urinary incontinence in neurological disease: management of lower urinary tract dysfunction in neurological disease (Clinical guideline CG148.) 2012. <http://guidance.nice.org.uk/CG148>.

## EASILY MISSED?

### Pre-eclampsia

David Williams,<sup>1</sup> Naomi Craft<sup>2</sup>

A 36 year old primigravida woman attended for antenatal care at 10 weeks' gestation with a blood pressure 120/80 mm Hg and no proteinuria. At 28 weeks, she presented to her general practitioner with urinary frequency and mild dysuria. Urine analysis showed 3+ proteinuria, and her blood pressure was 144/90 mm Hg. The fundal height measured 3 cm less than expected for this gestation. A midstream urine sample was sent for culture and a review arranged for a week later. At 29 weeks, her blood pressure was 175/115 mm Hg, proteinuria was 3+, and no urinary infection had been isolated. She was urgently admitted to hospital, but on arrival no fetal heartbeat could be detected. Labour was induced and a growth restricted, stillborn infant was delivered. Maternal hypertension persisted postpartum.

#### What is pre-eclampsia?

Pre-eclampsia is defined by the gestational onset of hypertension and proteinuria.<sup>1</sup> It is, however, a multi-system disorder that can affect all maternal organs.<sup>1 2</sup> Delivery of the fetus and placenta remains the only cure, but preterm delivery may adversely affect neonatal outcome, with complications resulting from prematurity and low birth weight.<sup>3</sup> Pre-eclampsia evolves into eclampsia when maternal seizures develop. Eclampsia is rare in well resourced countries—just 1% of all women with pre-eclampsia develop eclampsia.<sup>4</sup> A severe form of pre-eclampsia characterised by microangiopathic haemolytic anaemia is often termed the HELLP (Haemolysis, Elevated Liver enzymes, and Low Platelets) syndrome.<sup>2</sup>

<sup>1</sup>University College London Hospital, London WC1E 6AU, UK

<sup>2</sup>53 New Cavendish Street, London W1G 9TQ

Correspondence to: D Williams  
d.j.williams@ucl.ac.uk

Cite this as: *BMJ* 2012;345:e4437  
doi: 10.1136/bmj.e4437

## KEY POINTS

For pregnant women with new onset hypertension ( $>140/90$  mm Hg) and  $\geq 1+$  proteinuria, or other features of the multisystem disorder that might suggest pre-eclampsia in the second half of pregnancy, referral to their hospital maternity unit for immediate assessment is needed

Pre-eclampsia can be life threatening to the mother (with complications such as cerebral haemorrhage resulting from uncontrolled hypertension) and to the fetus (with complications of prematurity and low birth weight)

Pre-eclampsia is unpredictable and usually asymptomatic until the condition is advanced. It can evolve rapidly, requiring urgent delivery within hours of diagnosis, or progress slowly over weeks with conservative management

Women who have had pre-eclampsia are at increased risk of chronic hypertension and cardiovascular disease in later life

This is one of a series of occasional articles highlighting conditions that may be more common than many doctors realise or may be missed at first presentation. The series advisers are Anthony Harnden, university lecturer in general practice, Department of Primary Health Care, University of Oxford, and Richard Lehman, general practitioner, Banbury. To suggest a topic for this series, please email us at [easilymissed@bmj.com](mailto:easilymissed@bmj.com).

**How common is pre-eclampsia?**

Pre-eclampsia predominantly affects women in their first pregnancy (2-8% of first pregnancies)<sup>5</sup> and has a variable incidence across nations, being most common in Latin America and the Caribbean.<sup>6</sup> In the United Kingdom, about one in 200 pregnancies is affected by severe pre-eclampsia (about 3500 cases a year).<sup>7</sup>

**Why is pre-eclampsia missed?**

Pre-eclampsia is usually asymptomatic until it is in an advanced state,<sup>2 8-10</sup> and so it can evolve unchecked until the maternal condition has deteriorated to the point of severe organ failure and/or in utero death of the fetus.

In our case study, a urinary tract infection was suspected at 28 weeks, but a urinary tract infection rarely causes  $>1+$  proteinuria. The significance of new onset proteinuria, hypertension, and reduced fetal growth was not understood. This woman should have been referred to hospital at 28 weeks' gestation for further assessment of suspected pre-eclampsia and fetal wellbeing.<sup>9 10</sup>

**Why does it matter?**

The last triennial audit of maternal deaths in the UK reported 22 deaths from pre-eclampsia, of which 20 were associated with substandard care and 14 were thought to be avoidable.<sup>11</sup> The most common cause of maternal death was cerebral haemorrhage secondary to uncontrolled systolic hypertension. Four maternal deaths were attributed to general practitioners' errors, including inappropriate urological referral for proteinuria, outpatient treatment of hypertension alone, and referral to a midwife for follow-up of jaundice that evolved into the HELLP syndrome.<sup>11</sup>

Life threatening maternal complications include uncontrolled hypertension and cerebrovascular accident; eclampsia; placental abruption; hepatic infarction and rupture; disseminated intravascular coagulation; pulmonary oedema; and renal failure.<sup>7</sup>

**How is pre-eclampsia diagnosed?****Clinical**

Guideline bodies advise a diagnosis of pre-eclampsia when blood pressure is  $>140/90$  mm Hg in the second half of pregnancy, with  $\geq 1+$  proteinuria on reagent stick testing, which is confirmed by a protein:creatinine ratio of  $>30$  mg/mmol.<sup>1 9</sup> New onset hypertension without proteinuria but

with other maternal organ dysfunction, such as thrombocytopenia or raised liver enzyme values, may also indicate pre-eclampsia.<sup>1</sup> Some women have an isolated rise in blood pressure without proteinuria or other evidence of multisystem disorder of pre-eclampsia, and this is known as gestational hypertension. About 20% of women with gestational hypertension will go on to develop pre-eclampsia, especially if hypertension develops before 34 weeks.<sup>12</sup>

In the second half of pregnancy, the following symptoms should alert the clinician to check for hypertension and proteinuria: severe headache, with or without visual aura; epigastric pain, with or without nausea and vomiting; and sudden facial, hand, and feet oedema.<sup>8-10</sup>

Women with pre-existing cardiovascular risk factors such as chronic hypertension, diabetes mellitus, obesity (body mass index ( $\text{kg}/\text{m}^2$ )  $>35$  at presentation), renal impairment, older mothers ( $>40$  years old), and those who had pre-eclampsia in a previous pregnancy or who have a family history of pre-eclampsia (mother or sister) are at high risk of developing pre-eclampsia themselves.<sup>2 9 10</sup> Underlying chronic hypertension can be masked during the first half of pregnancy by gestational vasodilatation.

**Investigations**

For pregnant women with new onset hypertension ( $>140/90$  mm Hg) and new onset proteinuria ( $\geq 1+$  proteinuria on reagent stick testing) after 20 weeks' gestation, conduct the following investigations<sup>1 9 10</sup>:

**Full blood count**—To look for platelet consumption (platelets  $<100 \times 10^9/\text{L}$ ) and haemolysis (anaemia with fragmented red cells). In pre-eclampsia the haemoglobin concentration is generally mildly raised ( $>120$  g/L) owing to haemoconcentration

**Urea and electrolytes**—To look for renal dysfunction (raised serum creatinine concentration  $>90$   $\mu\text{mol}/\text{L}$ )

**Liver enzymes**—To look for transaminitis (alanine aminotransferase  $>32$  IU/L; aspartate aminotransferase  $>30$  IU/L)

**Urine sample or 24 h urine collection**—To quantify clinically significant proteinuria (ratio of protein to creatinine  $>30$  mg/mmol) or 24 hour urine collection  $>300$  mg)

**Assessment of fetus**—Ultrasound assessment of fetal growth and the volume of amniotic fluid; and Doppler velocimetry of umbilical arteries.

The results of these blood and urine tests are needed within hours. As it can take several days for blood test results to be received in primary care, general practitioners should send patients to their local maternity hospital for these investigations, as well as for fetal assessment.

Pre-eclampsia can be difficult to diagnose in women with pre-existing hypertension, especially if there is pre-existing renal disease with proteinuria. Under these circumstances, pre-eclampsia can evolve in the second half of pregnancy with a surge in blood pressure or proteinuria, but more usually other elements of this multiorgan syndrome are apparent, such as thrombocytopenia, raised level of liver transaminases, and reduced fetal growth. The uric acid level is often raised in women with pre-eclampsia, but in isolation it is of poor predictive and diagnostic value and should not be tested.<sup>9 10</sup>

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- ▶ Irreversible renal damage from accidental mushroom poisoning (*BMJ* 2012;345:e5262)
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- ▶ Lyme neuroborreliosis (*BMJ* 2012;344:e3250)
- ▶ Mood disorder in the perinatal period (*BMJ* 2011;344:e1209)

## How is pre-eclampsia managed?

Pre-eclampsia may progress unpredictably, within hours or over weeks. NICE guidelines therefore recommend immediate hospital referral for assessment of mother and fetus, with conservative management in a hospital that has facilities for emergency delivery and resuscitation of pre-term infants.<sup>9</sup>

Delivery of the placenta remains the only cure for pre-eclampsia. Childbirth should be considered if pre-eclampsia is identified after 37 weeks' gestation.<sup>13</sup> At 34-37 weeks' gestation, the decision to deliver is a clinical judgment that must weigh the risks to the mother of prolonging the pregnancy against the benefits for the preterm fetus.

Before 34 weeks, clinicians should try to prolong the pregnancy for the benefit of fetal maturity. This involves antihypertensive treatment with nifedipine slow release, labetalol, or methyldopa to keep the blood pressure between 130/80 mm Hg and 150/100 mm Hg.<sup>9</sup> There is little evidence to support choosing any one of these antihypertensive agents over another.<sup>9-10</sup> Magnesium sulphate will reduce the risk of eclamptic seizures,<sup>4</sup> and monitoring the fetal condition will guide the decision for timing of delivery.<sup>9</sup> Antenatal administration of corticosteroids will improve fetal lung maturity in anticipation of preterm delivery.<sup>14</sup>

Maternal hypertension usually recovers within two to three weeks of delivery but can take up to three months.<sup>15</sup> Pre-eclampsia will recur in about 15% of women who had pre-eclampsia in their first pregnancy, although this risk may be as high as 25% if the pre-eclampsia led to birth before 34 weeks and as high as 50% if birth was before 28 weeks.<sup>16</sup> Daily low dose aspirin (75-100 mg) from before 16 weeks' gestation in future pregnancies reduces the risk of recurrent, severe pre-eclampsia.<sup>17</sup>

Some women will continue to have hypertension three months after childbirth. This is presumed to be the result of previously unidentified chronic hypertension or secondary causes of hypertension.<sup>18</sup> Even those who have made a full recovery from pre-eclampsia are nevertheless at risk of hypertension and heart disease in later life.<sup>19</sup> Although the optimal follow-up regimen to minimise the risk of future cardiovascular disease is currently unclear,<sup>9-10</sup> pragmatic steps in primary care include encouraging optimal weight range through diet and exercise, and regular screening for hypertension, hyperlipidaemia, and diabetes.

A proportion of DW's salary is received by University College London Hospital from the Department of Health's National Institute for Health Research.

**Contributors:** Both authors contributed to the planning, drafting, revising, and final approval of the article. DW is the guarantor.

**Competing interests:** Both authors have completed the ICMJE uniform disclosure form at [www.icmje.org/coi\\_disclosure.pdf](http://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.

**Provenance and peer review:** Commissioned; externally peer reviewed.

**Patient consent not required (patient anonymised, dead, or hypothetical).**

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