RATIONAL TESTING

Investigating polyuria

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This series of occasional articles provides an update on the best use of key diagnostic tests in the initial investigation of common or important clinical presentations. The series advisers are Steve Atkin, professor, head of department of academic endocrinology, diabetes, and metabolism, Hull York Medical School; and Eric Kilpatrick, honorary professor, department of clinical biochemistry, Hull Royal Infirmary, Hull York Medical School. To suggest a topic for this series, please email us at practice@bmj.com

Polyuria represents a common presentation in primary care that can often be a diagnostic challenge requiring careful consideration. This article provides a structured, logical approach for investigating polyuria, highlights the importance and relevance of various tests, and advises on when to refer to a specialist

A 43 year old male teacher presented to his general practitioner with frequent urination and polydipsia for six weeks. He was passing large volumes of urine throughout the day and night, but he denied dysuria, hesitancy, or urgency. His fluid intake was approximately two litres a day and included two cups of tea. He had no relevant medical history; reported no weight loss, visual changes, or bowel disturbance; and took no drugs, including over the counter drugs or herbal remedies. There was no family history of diabetes or renal disease. Clinical examination was unremarkable, and his blood pressure was 138/84 mm Hg.

Box 1 | Glossary of terms

Polyuria—The production of "abnormally" large volumes of urine (>3 L/day in adults)

Urinary frequency—The excessive need to urinate, which is not normal for the patient. The total volume of urine passed is within normal limits

Polydipsia—Excessive thirst as a symptom of disease or psychological disturbance (resulting consumption of >3 L/day)

Nocturia—The need to wake from sleep and pass urine (urinating once a night is presumed to be within normal limits)

Osmolarity—The measurement of a solute concentration affected by water content, temperature, and ambient pressure. It can be calculated, using laboratory derived data, as $[2Na^+]+[2K^+]+[glucose]+[urea]$ (mmol/L)

Osmolality—The measurement of a solute concentration that is independent of temperature and pressure. This laboratory measurement is presented as the number of moles of solute per litre (mmol/kg)

LEARNING POINTS

In patients with increased urination, take a thorough clinical history to determine whether it is urinary frequency or polyuria; a home fluid balance chart may help to make the distinction

Exclude use of drugs (for example, caffeine, alcohol, diuretics, and lithium) and diabetes mellitus in all patients with polyuria

Urine dipstick testing is useful, and specific gravity can direct further investigations/referral

Plasma/urine osmolality may help to distinguish between different diagnoses

What is the next investigation?

Clinical history

A thorough clinical history is essential to distinguish between polyuria and urinary frequency (box 1). Urine volume can be difficult for patients to quantify, and use of fluid charts (measuring fluid input and output) may be helpful. The presence of urinary symptoms should be established, along with the timing of urination (nocturnal, throughout the day, or both). This may help clinicians to distinguish between possible causes by the relation to tubular disorders versus anatomical abnormalities, such as overactive bladder syndrome in which frequency and urgency of urination are present rather than polyuria or prostatic disease.

Polyuria and polydipsia are regularly experienced together and share similar causes. Box 2 lists the population prevalence of diseases that can cause polyuria. Diabetes mellitus affects more than 1 in 20 people in the United Kingdom and is the most common cause of polyuria in both children and adults.[1] Diabetes should be suspected, particularly if the patient has an associated history of weight loss (type 1 diabetes) or a family history of diabetes (type 2 diabetes). Patients with heart failure may experience nocturia owing to fluid accumulating as pedal oedema and returning to the blood stream when the patient lies flat. Older patients with hypertension may also have nocturnal polyuria, as a result of the effects of hypertension on cardiovascular and renal physiology.[2] Moderate to severe chronic kidney disease has a prevalence of 0.2% in the general population and can occasionally give rise to either polyuria or nocturia.[3] Therefore, check for visible or microscopic haematuria and systemic causes of renal disease (arthralgia, rashes, and constitutional symptoms), as anecdotal evidence suggests that patients with vasculitis and systemic lupus erythematosus may occasionally present with polyuria.

A full drug history is essential to rule out a potential pharmacological cause. Ask particularly about substances that increase urine output (diuretics, lithium, alcohol, and caffeine), as well as over the counter drugs. Excess vitamin D supplements may lead to hypercalcaemia, which may in turn cause polyuria and polydipsia.[4] However, primary hyperparathyroidism and malignancy account for 90% of patients with hypercalcaemia.[5] Malignancy is present in 1.1-3.9% of the general population, and hypercalcaemia develops in up to 30% of patients with cancer.[6]

Diabetes insipidus results in the excretion of large amounts of dilute urine and can be subdivided into central or nephrogenic types. It is very rare, with an incidence of 3 in 100 000 in the general population.[7] Central diabetes insipidus is characterised by a lack of antidiuretic hormone (also known as vasopressin) production caused by infiltration of or damage to the pituitary as a result of a tumour, a head injury, neurosurgery, haemochromatosis, or sarcoidosis. Nephrogenic diabetes insipidus occurs when

Box 2 | The population prevalence of diseases causing polyuria

Common (>1 in 10)
Diuretics/caffeine/alcohol*
Diabetes mellitus[1]
Lithium[8]
Heart failure*

Infrequent (1 in 100)
Hypercalcaemia[6]
Hyperthyroidism[9]

Rare (1 in 1000)
Chronic renal failure[3]
Primary polydipsia*
Hypokalaemia*

Very rare (<1 in 10 000)
Diabetes insipidus[7]

*Prevalence unknown but categorised approximately

Box 3 | Summary of investigations

Primary care
Home fluid balance chart
Urine dipstick
Capillary blood glucose
Serum urea and electrolytes, calcium
Random/fasting glucose or glycated haemoglobin (HbA_{1c})
Urine and plasma osmolality
Urine electrolytes

Secondary care (endocrinology)
Water deprivation test*
Desmopressin administration
Measurement of plasma antidiuretic hormone
Anterior pituitary hormones†
Magnetic resonance scan of brain/pituitary

*Urine and plasma osmolalities are measured in response to fluid deprivation and subsequent administration of desmopressin (antidiuretic hormone)
†Thyroid stimulating hormone, prolactin, luteinising hormone, follicle stimulating hormone, growth hormone, adrenocorticotrophic hormone

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Previous articles in this series
- Investigating low thyroid stimulating hormone (TSH) level (*BMJ* 2013;347:f6842)
- Abnormal liver function tests in pregnancy (*BMJ* 2013;347:f6055)
- Investigating hypokalaemia (*BMJ* 2013;347:f5137)
- When to order an antinuclear antibody test (*BMJ* 2013;347:f5060)
- High sensitivity cardiac troponin in patients with chest pain (*BMJ* 2013;347:f4222)

the kidneys become insensitive to the effects of antidiuretic hormone, often because of acquired kidney disorders or therapeutic lithium use. Up to 40% of patients taking lithium develop nephrogenic diabetes insipidus.[8] Hyperthyroidism is a common endocrine disorder found in 1.3% of the general population.[9] Elicit symptoms of thyrotoxicosis (sweating, heat intolerance), as this stimulates thirst and causes an increase in circulating natriuretic peptides, resulting in greater excretion of both sodium and water and hence leading to symptoms of polyuria.[10]

Determine the patient's daily fluid intake to assess whether it is in excess. This can be done by using a simple fluid chart that monitors fluid intake and urine output through the day. Primary polydipsia is the excessive intake of fluid, in the absence of a physiological stimulus to drink, caused by a mental disorder or disordered thirst. It is common among institutionalised psychiatric patients (6-20%)[11]; the prevalence in the general population is unknown, but it is thought to be rare. Polyuria throughout the night is uncommon in this condition. Primary polydipsia is difficult to treat in the community and, owing to the risk of hypotonic hyponatraemia, may require admission to hospital.[11]

Clinical examination

Note the patient's blood pressure, general appearance, and signs of dehydration (skin turgor, capillary refill, and mucous membranes). The presence of weight loss may support causes such as diabetes or thyroid disease, whereas muscle weakness may be a manifestation of metabolic derangements. Clinical examination rarely elicits positive findings; however, it is important to exclude a bitemporal hemianopia, which may be a sign of a tumour causing compression and leading to central diabetes insipidus. If a central cause is suspected, referral to secondary care is indicated for further investigation including magnetic resonance imaging of the brain and measurement of pituitary hormones. Palpate the abdomen for masses, particularly an enlarged kidney, which would warrant an ultrasound scan to rule out hydronephrosis and urinary tract abnormalities. Finally polyuria can occur after paroxysmal tachycardias including atrial flutter and fibrillation.[12]

The history and elicited clinical findings should then direct appropriate investigations and referral. Box 3 lists the basic investigations likely to lead to a diagnosis; these are discussed below.

Urinalysis: urine dipstick

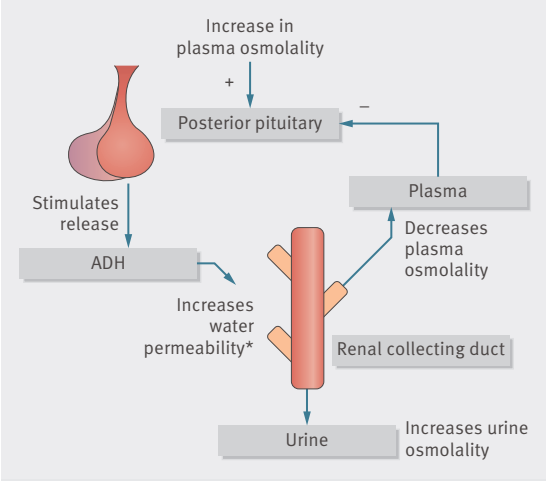
The presence of glycosuria necessitates a fasting or random glucose blood test or HbA_{1c} measurement to rule out diabetes. The renal threshold for glucose may be lower in children and pregnant women, as well as in renal glycosuria when even normal blood glucose concentrations produce a positive dipstick. A venous blood glucose concentration can help to identify these patients and aid further investigation and treatment.

The presence of leucocytes or nitrites in conjunction with urinary symptoms should direct the clinician towards a diagnosis of a urinary tract infection. Specific symptoms such as dysuria and frequency (without vaginal discharge or irritation in women) raise the probability of a urinary tract infection to more than 90%.[13] The combined presence of leucocytes and nitrites on urine dip, together with clinical symptoms, has a sensitivity of 68-88% with a negative predictive value of 84-98%.[14] Most studies have concluded that a negative urine dipstick without specific urinary symptoms is sufficient to rule out a diagnosis of a urinary tract infection.[13 14]

The specific gravity of the urine gives an estimate of its concentration and thus an indication of its osmolality. A high or low specific gravity (normal: 1.010-1.025) should prompt evaluation of osmolality to assist diagnosis. Twenty four hour urine collection, although useful in quantifying a patient's urine output, is not practical in the community and is best reserved for secondary care.

Urine/plasma osmolality with urine electrolytes

Osmolality is monitored by osmoreceptors in the hypothalamus, which detect very small changes and control release of antidiuretic hormone accordingly. Antidiuretic hormone acts on the collecting ducts to increase reabsorption of water (figure). If antidiuretic hormone production is lacking or the kidney has become insensitive to it, such as in



Mechanism of antidiuretic hormone (ADH) and feedback loop. ADH is controlled by hypothalamus in response to changes in osmolality of blood. Hence rise in plasma osmolality leads to increase in ADH, and fall in osmolality leads to decrease in ADH secretion.[15] Low specific gravity (<1.010) should prompt further investigation into osmolality of patient's urine.
***Of collecting ducts, increasing water reabsorption**

diabetes insipidus, the urine osmolality will be low but the plasma osmolality will be high. In primary polydipsia, urine osmolality will be low, with plasma osmolality found to be normal or low.

Blood tests

Capillary blood glucose, plasma glucose, and HbA$_{1c}$—Capillary blood glucose is a quick and easy screening test that can help to determine whether a fasting plasma glucose/HbA$_{1c}$ is needed. A diagnosis of diabetes mellitus can be made if the patient has a typical history (polyuria, polydipsia, weight loss), along with a random glucose or two hour glucose tolerance test above 11.1 mmol/L, or a fasting plasma glucose above 7.0 mmol/L. An HbA$_{1c}$ of 48 mmol/mol or above (or >6.5%) may also be used for diagnosis of diabetes, if no conditions precluding its use (for example, in children, steroid use) are present. Diabetes mellitus can be largely excluded as a cause of polyuria if the fasting plasma glucose is within the normal range; however, this is not true for a normal HbA$_{1c}$.[16]

Serum urea, creatinine, and electrolytes (including calcium)—These simple tests enable assessment of kidney function and may aid diagnosis. Hypernatraemia is a good marker of true water depletion,[17] whereas hypokalaemia, a common electrolyte disturbance, is an uncommon cause of polyuria. Calcium abnormalities may warrant parathyroid hormone, phosphate, and alkaline phosphatase measurements to explore underlying causes.

Thyroid function tests—Thyrotoxicosis can occasionally cause polyuria, as previously described.

Outcome

A urine dipstick was negative with a low specific gravity (<1.005). The absence of urinary symptoms made a urinary tract infection unlikely. The patient's kidney function, calcium, and potassium were normal. Advice from a renal physician recommended checking the urine and plasma osmolality. Plasma osmolality was high (318 mmol/kg; normal 280-295 mmol/kg), and urine osmolality was low (150 mmol/kg; normal 300-900 mmol/kg), indicating a problem with the patient's ability to concentrate urine. He was referred to secondary care. A water deprivation test had no effect on the urine osmolality, which remained low, excluding primary polydipsia. Subsequent administration of desmopressin (an antidiuretic hormone analogue) led to an increase in the urine osmolality. The measured antidiuretic hormone concentration was low, confirming diabetes insipidus. Computed tomography and magnetic resonance scans of the head, pituitary hormone concentrations, and autoantibody screening (arginine vasopressin antibodies) were negative. The patient was diagnosed as having idiopathic central diabetes insipidus, which accounts for about 30% of cases of diabetes insipidus. He was treated with 10 µg of nasal desmopressin daily, which improved his symptoms and normalised his biochemistry. His sodium and plasma osmolality were monitored to ensure that adequate fluid homeostasis was maintained.

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GUIDELINES

Prostate cancer: summary of updated NICE guidance

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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.

Further information about the guidance, a list of members of the guideline development group, and the supporting evidence statements are in the full version on bmj.com.

Prostate cancer poses a major health problem in many countries and is the commonest cancer in men in the UK.[1] Its incidence and mortality rate are higher in men of African-Caribbean origin. Since publication of the original NICE guideline in 2008,[2] there have been several changes in diagnosis and management of prostate cancer. Great improvements in the treatment of hormone relapsed metastatic disease with the introduction of several new treatments (cabazitaxel, abiraterone, enzalutamide, and radium-223) have been assessed by NICE's Technology Appraisal Programme[3 4] and are not covered by this guidance.

This article summarises recently updated recommendations from the National Institute for Health and Care Excellence (NICE) on the diagnosis and care of men with prostate cancer.[5] Many recommendations have not been updated from the previous guidance and can be found in a previous *BMJ* summary.[6]

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 in the full version of this article on bmj.com.

Information and support for patients

- Discuss all relevant management options recommended in the guideline with men with prostate cancer, and their partners or carers, irrespective of whether they are available through local services and use a validated, up to date decision aid.[7]

Initial assessment and diagnosis

- To help men decide whether to have a prostate biopsy, discuss with them their prostate specific antigen (PSA) level, digital rectal examination findings, age, black ethnicity (associated with an increased risk of prostate cancer), and comorbidities (which may affect the decision to offer curative treatment), together with any previous negative prostate biopsy.
  - The serum PSA level alone is a poor predictor of the presence of prostate cancer and should not automatically lead to a prostate biopsy; moreover, many cancers diagnosed on this basis alone will be of low risk, having little or no impact on life expectancy.
- A core member of the urological cancer multidisciplinary team should review the risk factors of all men who have had a negative first prostate biopsy, and inform them that:
  - There is still a risk that prostate cancer is present *and*
  - The risk is slightly higher if any of the following risk factors are present:
    - The biopsy showed high grade prostatic intraepithelial neoplasia (HGPIN)
    - The biopsy showed atypical small acinar proliferation (ASAP)
    - Abnormal digital rectal examination.
  - (New recommendation.)
- Consider multiparametric magnetic resonance imaging (MRI) (using T2 and diffusion weighted imaging) for men with a negative result from transrectal ultrasound guided biopsy (extracting 10-12 cores) to determine whether another biopsy is needed: do not offer repeat biopsy if the MRI is negative unless any of the risk factors in the previous recommendation are present.(New recommendation.)
- Consider multiparametric MRI (or computed tomography if MRI is contraindicated) for men with histologically proven prostate cancer if knowledge of the extent of primary tumour or condition of regional nodes could affect management. (New recommendation.)

As MRI is recommended for staging and after a negative first biopsy, the GDG considered recommending that multiparametric MRI be carried out on all men before prostate biopsy, but the evidence on clinical and cost effectiveness did not support such a recommendation at this time.

- Before treatment, warn men (and, if they wish, their partner) that radical treatment and long term androgen deprivation therapy for prostate cancer will result in an alteration of sexual experience and may result in loss of sexual function including loss of ejaculation and fertility. Offer sperm storage. (Amended recommendation.)

Active surveillance for localised prostate cancer

- Offer active surveillance as an option to men with low risk localised prostate cancer (see table 1 for risk stratification) for whom radical prostatectomy or radical radiotherapy is suitable, and consider using the protocol in table 2. (New recommendation.)

Table 1 | Risk stratification for men with localised prostate cancer

Level of risk	PSA level (ng/mL)		Gleason score*		Clinical stage†
Low risk	<10	*and*	≤6	*and*	T1–T2a
Intermediate risk	10–20	*or*	7	*or*	T2b
High risk‡	>20	*or*	8–10	*or*	≥T2c

PSA=prostate specific antigen.
*Gleason score = The sum of the predominant histological pattern of cancer (graded from 1 to 5) and the next most common pattern. For biopsies (as opposed to radical prostatectomy specimens), it is not possible to allocate a pattern of <3 because of the small quantity of tissue obtained. Therefore, the lowest possible Gleason score on a biopsy is 6 (3+3).
†Clinical stage = The anatomical extent of the cancer, informed by the gross resection specimen (in men having a prostatectomy) or by biopsy and rectal findings, sometimes augmented by magnetic resonance imaging. T1-T2a describe low volume disease confined to <50% of one prostatic lobe. T3 and T4 cancers extend beyond the prostate.
‡High risk localised prostate cancer is also included in the definition of locally advanced prostate cancer.

Table 2 | Protocol for active surveillance of localised prostate cancer

Timing	Tests*
At enrolment in active surveillance	Multiparametric MRI if not previously performed
Year 1 of active surveillance	Every 3–4 months, measure PSA level† (monitor PSA kinetics throughout active surveillance‡)
	Every 6–12 months, perform DRE§
	At 12 months, repeat prostate biopsy
Years 2–4 of active surveillance	Every 3–6 months, measure PSA level† (monitor PSA kinetics throughout active surveillance‡)
	Every 6–12 months, perform DRE§
Year 5 and every subsequent year until active surveillance ends	Every 6 months, measure PSA level† (monitor PSA kinetics throughout active surveillance‡)
	Every 12 months, perform DRE§

MRI=magnetic resonance imaging. PSA=prostate specific antigen. DRE= digital rectal examination.
*If there is concern about clinical changes or changes in PSA level at any time during active surveillance, reassess with multiparametric MRI or repeat biopsy, or both.
†May be carried out in primary care if there are agreed protocols for shared care and recall systems.
‡May include PSA doubling time and velocity.
§Should be performed by a healthcare professional with expertise and confidence in performing DRE.

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Previous articles in this series

- Intravenous fluid therapy for adults in hospital: summary of NICE guidance (*BMJ* 2013;347:f7073)
- Secondary prevention for patients after a myocardial infarction: summary of updated NICE guidance (*BMJ* 2013;347:f6544)
- Management of urinary incontinence in women: summary of updated NICE guidance (*BMJ* 2013;347:f5170)
- Management of autism in children and young people: summary of NICE and SCIE guidance (*BMJ* 2013;347:f4865)
- Acute kidney injury: summary of NICE guidance (*BMJ* 2013;347:f4930)

- Consider active surveillance for men with intermediate risk localised prostate cancer who do not wish to have immediate radical prostatectomy or radical radiotherapy, but do not offer active surveillance to men with high risk localised prostate cancer. (New recommendation.)
- Offer radical treatment to men with localised prostate cancer who have chosen an active surveillance regimen and who have evidence of disease progression. (New recommendation.)

Radical treatment

- Commissioners of urology services should consider providing robotic surgery to treat localised prostate cancer and should ensure that robotic systems for surgical treatment of localised prostate cancer are cost effective by basing them in centres that perform at least 150 robot assisted laparoscopic prostatectomies a year. (New recommendation.)
- Offer men with intermediate and high risk localised prostate cancer a combination of radical radiotherapy and androgen deprivation therapy rather than either therapy alone. (New recommendation.)
- Offer men with intermediate and high risk localised prostate cancer six months of androgen deprivation therapy before, during, or after radical external beam radiotherapy and consider extending this for up to three years for men with high risk disease. (New recommendation.)
- Consider high dose rate brachytherapy in combination with external beam radiotherapy for men with intermediate and high risk localised prostate cancer. (New recommendation.)

Managing adverse effects of radical treatment

- Ensure that men have early and ongoing access to specialist erectile dysfunction services.(Amended recommendation.)
- Ensure that men with signs or symptoms of radiation induced enteropathy are offered care from a team of professionals with expertise in radiation induced enteropathy (who may include oncologists, gastroenterologists, bowel surgeons, dietitians, and specialist nurses). (New recommendation.)
- Carry out full investigations, including flexible sigmoidoscopy, in men who have symptoms of radiation induced enteropathy to exclude inflammatory bowel disease or malignancy of the large bowel and to ascertain the nature of the radiation injury. Use caution when performing anterior wall rectal biopsy after brachytherapy because of the risk of fistulation. (New recommendation.)

Hormone treatment

- Before starting androgen deprivation therapy, tell men and, if they wish, their partner, that long-term androgen deprivation will cause a reduction in libido and possible loss of sexual function.(New recommendation)
- Consider intermittent therapy for men having long term androgen deprivation therapy (but not in the adjuvant setting after radical treatment with radiotherapy or surgery), and include discussion with the patient, and his family or carers if he wishes, about:
  - The rationale for intermittent androgen deprivation therapy
  - The limited evidence for reduction in side effects from intermittent therapy
  - The effect of intermittent therapy on progression of prostate cancer.
  - (New recommendation.)
- For men having intermittent androgen deprivation therapy:
  - Measure PSA level every three months
  - Restart androgen deprivation therapy if PSA level ≥10 ng/mL or if there is symptomatic progression.
  - (New recommendation.)

Managing adverse effects of hormone treatment

- Offer medroxyprogesterone (20 mg/day), initially for 10 weeks, to manage troublesome hot flushes caused by long term androgen suppression and evaluate the effect at the end of the treatment period and consider cyproterone acetate or megestrol acetate (20 mg twice daily for four weeks) if medroxyprogesterone is not effective or not tolerated. (New recommendation.)
- Tell men that there is no good quality evidence for the use of complementary therapies to treat troublesome hot flushes. (New recommendation.)
- Ensure that men starting androgen deprivation therapy have access to specialist erectile dysfunction services and consider referring men who are having long term androgen deprivation therapy, and their partners, for psychosexual counselling. (New recommendation.)
- Offer phosphodiesterase type 5 (PDE5) inhibitors to men having long term androgen deprivation therapy who experience loss of erectile function. If these fail to restore erectile function, offer a choice of intraurethral inserts, penile injections, penile prostheses, and vacuum devices. (New recommendation.)
- Consider assessing fracture risk in men with prostate cancer who are having androgen deprivation therapy, in line with NICE guidance on osteoporosis fragility fracture.[8] (New recommendation.)
- Offer bisphosphonates to men who are having androgen deprivation therapy and have osteoporosis, and consider

denosumab if bisphosphonates are contraindicated or not tolerated.(New recommendation.)
- Tell men who are starting androgen deprivation therapy that fatigue is a recognised side effect of this therapy and not necessarily a result of prostate cancer and offer supervised resistance and aerobic exercise at least twice a week for 12 weeks to reduce fatigue and improve quality of life. (New recommendation.)

Follow-up

- Men with prostate cancer who have chosen a watchful waiting regimen with no curative intent should normally be followed up in primary care in accordance with protocols agreed by the local urological cancer multidisciplinary team and the relevant primary care organisation(s). Their PSA levels should be measured at least once a year.
- Check PSA levels for all men with prostate cancer who are having radical treatment: at six weeks after treatment at the earliest, at least every six months for the first two years, and at least once a year thereafter.
- After at least two years, offer follow-up outside hospital (for example, in primary care) by telephone or secure electronic communications to men with a stable PSA level who have had no significant treatment complications, unless they are taking part in a clinical trial that requires formal, clinic based follow-up. Direct access to the urological cancer multidisciplinary team should be offered and explained.

The recommendations on the management of metastatic prostate cancer have not been updated and can be found in the previous *BMJ* summary.[6] Several new treatments for metastatic prostate cancer has been licensed and these have been subject to NICE technology appraisals.[3 4]

Overcoming barriers

Although the technology to perform multiparametric magnetic resonance imaging (MRI) of the prostate gland is available in most hospitals diagnosing prostate cancer, the expertise to report these scans is not widely available across the UK. The Royal College of Radiologists is aware that this training issue might delay implementation of recommendations relating to MRI.

As there was no universally accepted protocol for active surveillance of men with localised prostate cancer and no comparative evidence, the consensus protocol developed by the Guideline Development Group should become the UK standard.

Recommendations on commissioning of robot assisted laparoscopic radical prostatectomy should ensure wider availability of this treatment.

Resource issues are likely to delay implementation of the recommendations on management of radiation enteropathy, sexual dysfunction, osteoporosis, and exercise programmes to combat fatigue from hormone therapy.

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A lesson from a bike shop

Have you ever wondered how much medical "success" is simply down to patients failing to tell us what we've got wrong? Insight into patient experience comes from surprising sources at times. I recently learnt a lesson in a bike shop.

My second favourite bike needed a service. The front gear wheel wasn't engaging properly, and after three years of punishment I knew every bit that moved was clogged with muck and oil. So I took it to a man I trusted in a place I trusted, a place I'd used for years.

The problem turned out to be a stretched chain—I wasn't expecting that. A few days later the chain was changed, and I set off from the bike shop ready for another run in the mud.

Only now the chain kept slipping when I peddled in high gear.

My man was deeply apologetic—he hadn't tried it in top gear; it was clear the problem was the front gear wheel itself was worn. The new chain I hadn't realised I needed was just too good for the old wheel. Should have spotted it. No worries. A few more days and a new front gear wheel later, and I was back in the saddle again.

The chain kept on slipping.

Another rapid return, more embarrassment, more apologies. He hadn't changed the rear gear wheel cartridge. Basic requirement with the new chain I hadn't realised I needed—stupid to miss it, sorry. He's a lovely man, I've known him ages—no need for a fuss. And he was great: took the cartridge off a brand new bike and turned me around in 10 minutes. And this time the chain was running beautifully.

But the front gear wheel still wouldn't engage.

And that's what I'd gone in about in the first place. I didn't go back again. I couldn't face the bother. I decided to lump it. No bad feelings towards my man. He'd done his best and would do so again in the future. There was even a bit of me worried he might think I was a pest. And at least he'd admitted what he'd got wrong and apologised. And I can still ride my bike ... sort of. And I've got a brand new chain I didn't know I needed.

Anyone else feeling uncomfortable?

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