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The management of the NHS in England

Loss of experienced managers creates a risk that performance will suffer

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The shape of the reformed NHS has become much clearer since the recent publication of papers on the role of the NHS Commissioning Board and the sources of support available to clinical commissioning groups.[1 2] These papers sound the death knell for strategic health authorities and primary care trusts, which are destined to be abolished in April 2013. Most of the functions of the strategic health authorities and primary care trusts will be taken on by the Commissioning Board and clinical commissioning groups, with the board being organised into four sectors with a total of 50 local offices.

On the surface the proposed arrangements may look similar to the four strategic health authority and 50 primary care trust clusters that exist currently, but in reality the new organisation will be very different. Each sector office and each local office will employ only 50 staff compared with the many more currently employed, so thousands of jobs will be lost. Redundancies among managers will contribute to a 50% reduction in management costs in the new organisation as the government seeks to fulfil its promise to put more resources into front line clinical care.

These changes build on work already done to cut back on the number of managers working in the NHS in anticipation of the controversial Health and Social Care Bill becoming law. Staff working in strategic health authorities and primary care trusts will endure a period of continuing uncertainty and anxiety—not knowing for weeks if not months if they will have a job under the new arrangements. There is a major risk that experienced managers will be lost—through a combination of retirement, redundancy, appointment to other roles in the NHS, and exit to employment outside the NHS—at a crucial point in the transition from the old system to the new. This will make it difficult to provide continuity of business and to ensure that NHS performance does not suffer.

Strategic commissioning is needed to bring about major changes in how specialist services are provided, and this need is particularly acute in London, Manchester, and the other major conurbations. It is widely recognised that the current pattern of hospital provision is unsustainable, and that leadership across a system of care is required to bring about change.[3] A study of hospital reconfiguration undertaken in south east London in 2011 showed that market forces will not be sufficient to make change happen.[4] There is a serious risk that the new system as it is currently designed will lack capacity to undertake strategic commissioning.

Clinical commissioning groups are unlikely to have the appetite or capabilities to lead complex hospital reconfigurations, and the slimmed down Commissioning Board will struggle to fill this vacuum. The board's four sectors will be involved in large scale reconfigurations but, with each sector employing only 50 staff and responsible for a range of other functions, it will be impossible for them to make a substantial contribution. The consequence will be failure to tackle changes in the role of hospitals that are long overdue and much needed to improve quality and safety.

An important weakness in the continuing programme of healthcare reform in England is the government's failure to value the role of managers in the NHS and to recognise the vital contribution they make alongside clinicians in ensuring the provision of high quality care. Although the NHS may be over-administered as a result of the emphasis placed by successive governments on regulation and implementation of national standards and targets, the proportion of the budget spent on management is not excessive compared with other organisations. If anything, the NHS is under-managed and needs to recruit and retain leaders from a variety of backgrounds—clinical and non-clinical—if it is to meet the financial and other challenges that lie ahead, as argued in a recent report from the King's Fund commission on leadership and management in the NHS.[5]

The Commissioning Board's paper on the sources of support available to clinical commissioning groups makes it clear that groups will be expected to draw on expertise from the private sector, third sector, and local authorities, as well as from staff they employ directly. The groups will take on some of the functions of primary care trusts, and they need to attract experienced managers at risk of displacement so that they can assume full responsibility for commissioning as quickly as possible. NHS managers who are not hired by commissioning groups will be housed on a temporary basis by the Commissioning Board, but they will be expected to establish independent organisations by 2016 at the latest, in what the government hopes will become a competitive market in commissioning support.

The vacuum in strategic commissioning could be met by commissioning groups agreeing to collaborate to create the capability to do this work themselves. However, the Department of Health's management cost allowance of only £25 (€30; $40) per head of population means that commissioning groups will face major challenges in acquiring people with the required expertise. This creates a risk that commissioning groups will be unfairly constrained compared with NHS providers who decide for themselves how much of their budgets to spend on management support. Commissioning has been the weak link in successive NHS reforms over the past 20 years, and the limits placed on spending by the Commissioning Board and commissioning groups will do nothing to avoid history repeating itself.

Add to funding constraints the loss of experienced commissioning managers and the risks are plain to see. To say that the NHS is being asset stripped at a point during the implementation of the reforms when effective leadership is needed more than ever is hardly an exaggeration. To take forward the most complex and radical organisational changes in the history of the NHS, and to find the £20bn in savings required under the "Nicholson challenge," are unprecedented challenges. If these changes are not managed well, financial control will probably be lost and quality of care and patient safety will be affected, at least during the transition.

Impassioned discussion about competition and new providers of care may make debate about managers unfashionable, but managers are essential in a well run health service. Some benefits may come from putting doctors in charge of budgets, but without the right support they are doomed to fail. More importantly, in the absence of effective management, the performance of the NHS will suffer to the detriment of patients and the public.

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bmj.com/podcasts
DFID deputy chief scientific adviser Tim Wheeler describes how the department deals with lack of evidence

Agriculture and health

Agricultural research needs to be better integrated with nutrition and health outcomes

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We live in a "triple F" crisis. Global and European financial problems dominate the press and the concerns of privileged people in privileged societies. But for the poor it is fuel and, for most of the poorest, food that present the greatest direct threats to their daily lives. Our ability to sustainably produce and ensure access to a diet of sufficient quantity and quality (in terms of vitamin and mineral content) is central to guaranteeing both food and nutrition security, and it is a major emphasis of current global development policy and planning.[1] In the linked systematic review, Masset and colleagues assess evidence from low income countries on the effects of interventions that aim to improve children's nutritional status by enhancing households' agricultural production and improving diets.[2]

Great strides have been made in ensuring global food security in the past 50 years. Global food production has largely kept pace with population growth, allowing not only more people to be fed but also greater consumption of a wider diversity of vegetable—and particularly animal—source foods, especially by the rich. However, success must be tempered by recognition of serious system failures—recent data suggest that almost 30% of children under 5 years of age are stunted (a marker of chronic undernutrition) and nearly 9% are wasted (a marker of acute undernutrition). Hundreds of millions of people across the globe are deficient in key nutrients that can result in long term poor health and functional losses, such as vitamin A, iron, and iodine.[3] Continuing high and volatile food prices are likely to lead to further deterioration of global nutrition.[4]

The links between agriculture and health are complex and bidirectional: agriculture provides a source of food and income at the household and national level, and healthy populations are more economically productive. Agriculture is important for ensuring food security and thereby reducing undernutrition, but also, by defining the foods available for consumption, agriculture has an increasingly crucial role in the global patterns of non-communicable diseases.[5 6]

Two links between agriculture and health are the "own production" and "market" pathways. The own production pathway (where households eat food they produce themselves) links households' food production directly to their access to food and its consumption, and thereby potentially with changes in markers of nutritional status and health. The wider market pathway (where households eat food bought in markets) links food production with greater and cheaper food availability in markets, higher household incomes, and increased purchases of goods and services (such as food, health, and education). This pathway is an important element within agriculture's powerful role as a driver of wider economic development and increased economic and social prosperity.[7 8]

In this context, critical examination of whether agricultural interventions directly affect nutritional status via the own production pathway is important. Masset and colleagues found no good evidence to show whether or not agricultural interventions that promote improved nutrition in producer households affect the nutritional status of children in these households.[2] The review highlights substantial weaknesses in the evidence base. Of the 23 included reports, 22 were cross sectional or longitudinal comparisons of people or households that were adopting (or had adopted) a range of agricultural interventions, and only some of these studies matched the intervention with control groups. Furthermore, the small size of many of the studies resulted in insufficient statistical power to detect potentially important effects. The review does not investigate the evidence base for the impact of the market pathway on the nutrition and health of children.


An own-production market

There is a pressing need to bridge the research divide between the agriculture and health sectors and to integrate nutrition and health outcomes more fully with agricultural research. Randomised controlled trials that investigate the nutritional and health effects of agricultural interventions via the own production pathway are possible and feasible (effects via the market pathway are more difficult to investigate in this way), and more controlled trials should be conducted in agricultural research. Indeed, Masset and colleagues cite a systematic review that analysed nine trials designed to identify the effect of biofortified maize on child growth in producer households.[9] Innovation is also needed to find ways of measuring outcomes for interventions that cross traditional sectoral thresholds.

The current evidence base that links agricultural interventions directly with child nutrition and health outcomes is limited, and Masset and colleagues show that it is currently not possible to say whether or not agricultural interventions benefit child health via the own production pathway. However the evidence from econometric analyses is that growth in agriculture benefits poor people more than growth in other sectors,[7 8] and that (outside of India) it has a greater impact than non-agricultural growth on child undernutrition and energy supply (although not on dietary diversity).[10] Investment in agriculture for the sustainable production of, and access to, sufficient food of adequate nutritional quality must therefore remain a key development focus. Cross sectoral learning and integration between agriculture and health should also move up the agenda to provide robust evidence for making sound policy in this important area.

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Hypertension and hyperuricaemia commonly coexist. Antihypertensive drugs can increase or decrease the development of incident gout in patients with hypertension

Antihypertensives in people with gout or asymptomatic hyperuricaemia

Losartan and calcium channel blockers are most effective owing to their uricosuric properties

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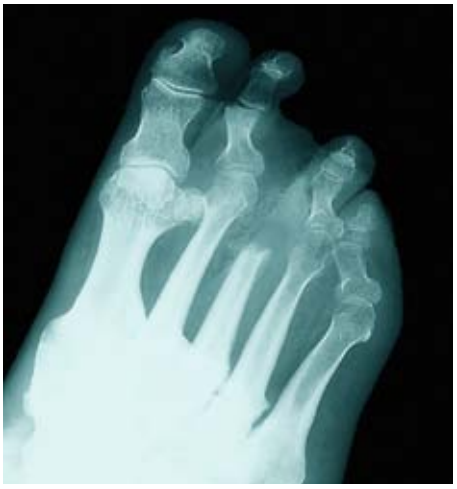
In the linked case-control study Choi and colleagues assess the association between antihypertensive drugs and the development of incident gout, stratified by the presence of hypertension.[1]

The link between uric acid and arterial hypertension was first noted in the 1960s, when prospective studies reported that 26% of untreated hypertensive patients with normal renal function had raised serum uric acid concentrations. This figure rose to 58% for those receiving antihypertensive drugs, and it was particularly high in those taking diuretics (70%).[2] Since then, raised uric acid concentrations in people with normotensive, borderline, and established hypertension have been shown to be associated with decreased renal blood flow, without affecting glomerular filtration rate, and with increased renal and peripheral resistances. This suggests that unexplained hyperuricaemia in patients with essential hypertension probably reflects early renal vascular involvement, specifically, nephrosclerosis.[3] On the other hand, hypertension is one of the most common comorbidities of gout and hypertension is independently associated with incident gout.[4]

Interest in the role of uric acid in cardiorenal disease has recently been reignited. Epidemiological studies consistently find that uric acid concentrations predict the development of chronic kidney disease,[5] and a recent meta-analysis reported that uric acid predicts the development of hypertension,[6] diabetes,[7] and stroke.[8] The association between coronary artery disease and uric acid remains controversial.

Choi and colleagues conducted a case-control study nested within a UK general practice database by identifying all incident cases of gout (n=24 768) and randomly sampling 50 000 controls who were 20-79 years old between 2000 and 2007.[1] They found that the use of calcium channel blockers and losartan in patients with hypertension was associated with a significantly reduced risk of incident gout (relative risk 0.87, 95% confidence interval 0.82 to 0.93; 0.81, 0.70 to 0.94, respectively); this is compatible with their effect of reducing urate concentrations through increased uricosuria. In contrast, diuretics, β blockers, angiotensin converting enzyme inhibitors, and non-losartan angiotensin receptor blockers were associated with a significantly increased risk of gout. Interestingly, similar results were found in people with normal blood pressure.



ZEPHYR/SPL

Radiograph showing gouty changes in foot joints

The authors suggest that urate lowering antihypertensive drugs could help to reduce the high comorbidity burden of gout and hypertension in patients at high risk of developing gout.[4] This potential effect is supported by the LIFE (Losartan Intervention For Endpoint reduction in hypertension) trial, which found that a losartan based regimen reduced cardiovascular morbidity and mortality more effectively than an atenolol based one.[9] Subsequent analysis of the trial showed that the greater reduction in serum uric acid concentrations obtained with losartan than with atenolol explained 29% of the treatment effect on the primary composite end point of fatal and non-fatal myocardial infarction and stroke.[10] Similarly, the decrease in uric acid seen with losartan in the RENAAL study correlated with the extent of the long term reduction in risk of renal damage compared with placebo, which partly explains the renoprotective effects of losartan.[11] Furthermore, a recent study found that allopurinol may have a protective renal and cardiovascular effect in people with hyperuricaemia.[12] Further studies are needed on the protective effects of allopurinol and on the effects of a reduction in uric acid on cardiovascular and renal damage.

In summary, hypertension and hyperuricaemia commonly coexist. Antihypertensive drugs can increase or decrease the development of incident gout in patients with hypertension, with losartan and calcium channel blockers having the greatest lowering effect on blood pressure because of their uricosuric properties. As well as reducing incident gout, a decrease in the concentration of serum uric acid could also improve the cardiovascular and renal prognosis of patients with hypertension.

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1 Choi HK, Soriano LC, Zhang Y, Rodríguez LAG. Calcium channel blockers, losartan, and the risk of incident gout among patients with hypertension: population based case-control study. *BMJ* 2012;344:d8190.
2 Cannon PJ, Stason WB, Demartini FE, Sommers SC, Laragh JH. Hyperuricemia in primary and renal hypertension. *N Engl J Med* 1966;275:457-64.
3 Messerli FH, Frohlich ED, Dreslinski GR, Suarez DH, Aristimuno GG. Serum uric acid in essential hipertensión an indicator of renal vascular involvement. *Ann Intern Med* 1980;93:817-21.
4 Richette P, Bardin T. Gout. *Lancet* 2010;375:318-28.
5 Weiner DE, Tighiouart H, Elsayed EF, Griffith JL, Salem DN, Levey AS. Uric acid and incident kidney disease in the community. *J Am Soc Nephrol* 2008;19:2004-11.
6 Grayson PC, Kim SY, Lavalley M, Choi HK. Hyperuricemia and incident hypertension: a systematic review and meta-analysis. *Arthritis Care Res (Hoboken)* 2011;63:102-10.
7 Kodama S, Saito K, Yachi Y, Asumi M, Sugawara A, Totsuka K, et al. Association between serum uric acid and development of type 2 diabetes. *Diabetes Care* 2009;32:1737-42.
8 Kim SY, Guevara JP, Kim KM, Choi HK, Heitjan DF, Albert DA. Hyperuricemia and risk of stroke: review and meta-analysis. *Arthritis Rheum* 2009;61:885-92.
9 Lindholm LH, Ibsen H, Dahlof B, Devereux RB, Beevers G, de Faire U, et al. Cardiovascular morbidity and mortality in patients with diabetes in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. *Lancet* 2002;359:1004-10.
10 Holeggen A, Alderman MH, Kjeldsen SE, Julius S, Devereux RB, De Faire U, et al. The impact of serum uric acid on cardiovascular outcomes in LIFE study. *Kidney Int* 2004;65:1041-9.
11 Miao Y, Ottenbros SA, Laverman GD, Brenner BM, Cooper ME, Parving HH, et al. Effect of a reduction in uric acid on renal outcomes during losartan treatment: a post-hoc analysis of the reduction of end-points in non-insulin-dependent diabetes mellitus with the angiotensin II antagonist losartan trial. *Hypertension* 2011;58:2-7.
12 Goicoechea M, de Vinuesa SG, Verdalles U, Ruiz-Caro C, Ampuero J, Rincon A, et al. Effect of allopurinol in chronic kidney disease progression and cardiovascular risk. *Clin J Am Soc Nephrol* 2010;5:1388-93.

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- Research: Variation in antibiotic prescribing and its impact on recovery in patients with acute cough in primary care (*BMJ* 2009;338:b2242)
- Research: Effect of antibiotic prescribing in primary care on antimicrobial resistance in individual patients (*BMJ* 2010;340:c2096)

A prescription for improving antibiotic prescribing in primary care

Education programmes can reduce antibiotic prescriptions, but impact on clinical outcomes is unclear

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Over the past 70 years, antibiotics have influenced and improved the treatment of many symptomatic infections. Unfortunately, antibiotics produce side effects and will ultimately lead to a change in the sensitivity of organisms, which can sometimes lead to a reduction in clinical effectiveness.

Many attempts have been made to implement programmes that are designed to improve the use of antibiotics, particularly in primary care. The linked randomised controlled trial by Butler and colleagues describes the most recent of these attempts.[1] The authors used social learning theories to develop an extensive and comprehensive educational programme (Stemming the Tide of Antibiotic Resistance; STAR) aimed at reducing antibiotic use in primary care clinics in Wales. Their multifaceted intervention incorporated many of the approaches other reviews have identified as helpful, such as education, feedback, and patient involvement.[2] Practices randomised to receive the STAR programme dispensed significantly fewer oral antibiotics (26.1 items/1000 registered patients/year)—a total reduction of 4.2% (95% confidence interval 0.6% to 7.7%). The intervention cost about £3000 (€3500; $4713) per practice. The results are similar to (although at the lower end of) reductions seen with other such programmes.[3]

Is a 4% reduction in use of antibiotics clinically important? The authors found no significant differences in hospital admissions or reconsultations for a respiratory tract infection within seven days of an index consultation. Although it was essential to examine these outcomes, the study sample size and the effect on prescribing were too small to ascertain if the decrease in antibiotic use improved or worsened patient outcomes.

The authors did not assess whether resistance patterns changed. In a country-wide programme in Finland, reducing the use of erythromycin by 50% reduced the resistance of group A streptococcal isolates from 17% to 9%.[4] Another study found that a decrease of 50 amoxicillin items per 1000 patients per year reduced resistance by 1%.[5] Others have found that a 20% reduction in the prescription of ampicillin and amoxicillin resulted in 1% fewer resistant isolates.[6] So, although reducing the use of antibiotics can affect resistance, the small reduction seen in the STAR study is unlikely to lead to a clinically important change in resistance patterns.

Most people agree that antibiotic prescribing in primary care needs to be improved. Understanding why antibiotics are prescribed is an essential first step. The ethos of antibiotic prescribing is multifactorial and somewhat unique. Fear on the part of the patient and clinician that the infection may turn into something serious plays a major role in decision making.[7]

Antibiotic prescribing can also arise from a clinician's desire to do something that might help or the perception that the patient wants an antibiotic. This is despite research showing that clinicians accurately distinguish only about half of the patients who want or don't want antibiotics.[8] Patients' satisfaction depends more on improved understanding of their illness, however, than on receiving a prescription.[9]

Most (80-90%) oral antibiotic prescriptions in primary care are for respiratory tract infections, urinary tract infections, or skin and soft tissue infections. In theory, diagnostic certainty should help improve the use of antibiotics. Reliable diagnostic criteria are available for sore throats but not for sinusitis or other upper respiratory tract infections. Decision support tools may help clinicians reduce antibiotic prescribing for upper and lower respiratory tract infections and urinary tract infections. Some tests may help to distinguish bacterial infections from viral ones. For example, the use of procalcitonin as an indicator of bacterial infection reduced antibiotic use from 97% to 25% in primary care patients with both upper and lower respiratory tract infections.[10]

When seeing a patient with a possible community acquired infection, clinicians may find it helpful to outline to their patients some of the potential benefits and harms of treatment. Rational use of antibiotics does not involve quibbling over starting antibiotics in very sick patients, but for non-serious illnesses that may or may not be bacterial a reasonable option to reduce antibiotic prescribing is to use delayed antibiotic prescriptions. This makes clinicians feel they are doing something and gives control to the patient. Delayed prescriptions can reduce the proportion of people who receive antibiotics for upper respiratory tract infections from 93% to 32%,[11] a reduction similar to that seen with the use of procalcitonin. Patients who are not given a prescription initially will still ultimately get an antibiotic 14% of the time. However, delaying antibiotics may worsen outcomes—such as fever at day three—and reduce patient satisfaction, but it may also reduce adverse events such as diarrhoea. The 61% (93% minus 32%) absolute difference in antibiotic use from choosing a delayed prescription may be a worthwhile compromise in areas of uncertainty because a strict "no prescription" approach will only "buy" another 18% (32% minus 14%) absolute difference in antibiotic use.

Most community acquired infections still respond to antibiotics that have been used for decades and many guidelines still support their use. Amoxicillin for respiratory tract infections and cloxacillin for soft tissue infections (unless community acquired meticillin resistant *Staphylococcus aureus* is suspected) are still solid treatment choices, with doxycycline a reasonable alternative for adult patients who are allergic to or intolerant of these antibiotics. For uncomplicated urinary tract infections, nitrofurantoin, co-trimoxazole, or trimethoprim alone are still good choices, especially for patients who are not seriously ill.

Data on the development of resistance suggest that treatment with high dose shorter duration antibiotics may reduce the emergence of resistance.[12] Although several studies show that shorter courses of antibiotics for relatively self limiting infections in primary care are as effective as longer ones, it is never known how an individual patient will respond. Given that, a reasonable approach for most primary care infections would be to tell the patient to stop the antibiotic when they have been asymptomatic or afebrile for 72 hours. Patients also need to be advised what to do if no improvement is seen within 24-48 hours. Patients need to know that the often used warning to finish the whole antibiotic course is not evidence based. Use of the prescription label "Finish all this medication unless otherwise directed by prescriber" should be discouraged.

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- News: Articles disputing link between HRT and breast cancer are "ridiculous" (*BMJ* 2012;344:e513)
- Editorial: The rise and fall of breast cancer rates (*BMJ* 2012;344:d8003)

Hormone therapy for menopausal symptoms

Recent evaluations of the methods of key studies should not change how we advise women

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A recently published and much publicised paper by Shapiro and colleagues, the last in a series of four, evaluated the effects of hormone therapy on the risk of breast cancer.[1] The authors of the four review articles applied epidemiological principles to the findings of two randomised placebo controlled studies from the Women's Health Initiative (WHI; 27 347 women) and two observational studies—the Collaborative Reanalysis (53 865 women) and the Million Women Study (MWS). Shapiro and colleagues concluded in their fourth paper that the MWS had design defects, that it contained multiple biases, and that its findings were thus not robust enough to show that hormone therapy increased the risk of breast cancer.

All observational studies are inherently biased because subjects are not randomly assigned to treatment or control. Adjustment for confounders and careful design of observational studies help to reduce bias. However, because there is no independent variable, such studies can tell us only about association not causation.

The MWS was published in the *Lancet* in August 2003,[2] and a flurry of letters was published in a print issue later that year, many of which raised the same concerns about bias recently highlighted by Shapiro and colleagues. The authors of the MWS replied,[3] and the sequence continued over the years: concerns about the believability[4] and worry about uncritical acceptance of the MWS data,[5] followed by more responses from the authors of the MWS.[6]

Healthy women have a low absolute risk of adverse events, whether they use hormone treatment during early menopause or not

Although observational studies add to our knowledge, they cannot replace randomised trials. Analysis of data from the WHI found a decreased risk of early stage breast cancer and ductal carcinoma in women randomised to receive oestrogen only.[7] Subgroup analysis showed that the reduction in breast cancer was statistically significant only for women who complied with treatment, had not used hormones before study entry, and had started oestrogen more than five years after the menopause. No benefit was seen for women who started oestrogen treatment at the time of the menopause.[8] This "gap time concept" of a reduced risk of breast cancer only if oestrogen is started late contrasts with the gap time hypothesis of a potential decrease in cardiovascular disease if oestrogen is started early.[9] Shapiro and colleagues' evaluation of the WHI findings concluded that treatment with oestrogen only does not increase the risk of breast cancer and may even reduce it, although the last possibility is based on statistically borderline evidence.[10] Fewer breast cancers were diagnosed in the first four years of follow-up in women in the WHI who were randomised to receive combined oestrogen and progestin. This is thought to be the result of hormonally induced increased density of breast tissue, which leads to delayed mammographic diagnosis of cancer. After four years, breast cancer rates were higher in women on combined hormone therapy, and diagnosed cancers were larger and more advanced.[11] Women who had used hormone treatment before joining the study were at higher risk of breast cancer than those who were treatment naive, but a significant increasing trend in risk of breast cancer over time was seen for this last group.[11]

Pretreatment clinical or laboratory characteristics may be discovered that will help identify women who, because of genetic predisposition, are at increased risk of adverse events with hormone treatment. The role of progestogens in the development of breast cancer needs to be clarified. A recent review suggests that women who use progesterone or dydrogesterone instead of progestogens have a lower risk of breast cancer.[12] Because treatment with oestrogen alone seems to be associated with lower risk, local delivery of progestogen to the endometrium is a potential option. However, a recent case-control study found increased odds of breast cancer in women with a levonorgestrel intrauterine system.[13] Other strategies to deliver combination treatment are being investigated; oestrogen combined with selective oestrogen receptor modulators has the potential to improve symptoms without affecting the breast and has positive effects on lipids and bone.[14]

SHEILA TERRY/SPL

How should we advise women while we wait for better treatment solutions? The second article in Shapiro and colleagues' series concluded that potential biases in the combined hormone treatment arm of WHI reduced the robustness of an association between treatment and breast cancer.[15] However, they acknowledged that the use of oestrogen plus a progestogen could possibly increase the risk of breast cancer. This is how most clinicians would frame the issue when discussing the risks of using combined hormone treatment. The increased risk of breast cancer associated with use of combined oestrogen-progestogen (hazard ratio 1.24) is similar to risks conferred by delayed menopause or moderate use of alcohol. Although an increase in risk of this size may be important for public health, individual women may not consider it enough to change their minds about using hormone treatment.

Women should continue with regular breast screening, and those with dense breast tissue may need more frequent screening. The primary aim of the WHI was to see if the use of hormone treatment decreased heart disease, as observational studies had found. The study was not designed to determine the risks of hormone use for symptoms in early menopause. It was not powered for subgroup analysis in the 50-59 year age group, and numbers of adverse events were small. Healthy women have a low absolute risk of adverse events, whether they use hormone treatment during early menopause or not.

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The insertion of an artificial urinary sphincter is an established operation with high patient satisfaction

Urinary incontinence after treatment for prostate cancer

Long term morbidity can be minimised by early referral to specialist centres

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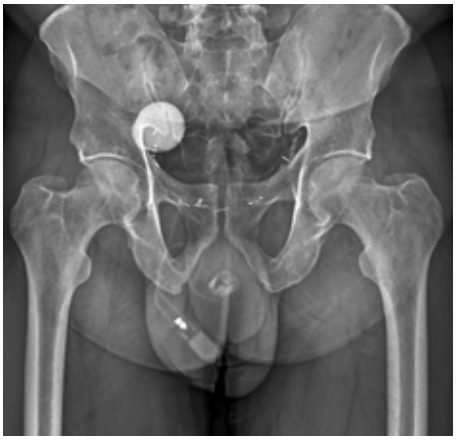
Prostate cancer is the most common cancer in men, making up 25% of all new diagnoses of cancer in men.[1] Most cases are confined to the prostate at diagnosis and therefore amenable to radical surgery.[2] The number of radical prostatectomies being performed in England has increased exponentially from 972 in 1998-9 to 3092 in 2004-5, so the incidence of post-prostatectomy urinary incontinence has probably increased. This problem can be expected only to get worse if routine prostate specific antigen screening is undertaken nationally.[3]

An estimated 14-20% of patients who have undergone radical prostatectomy will need to use absorbent pads as a long term means of managing their urinary incontinence.[4] The morbidity of stress urinary incontinence in men who are relatively young and fit can be devastating and can lead to a loss of confidence, avoidance of social interaction, and even depression.[5] Patients are often poorly advised about urinary containment options such as pads and external collecting devices.

Urinary incontinence after prostatectomy can be the result of bladder overactivity, poor bladder compliance, or failure of the sphincter mechanism. The precise mechanism is likely to be multifactorial, highlighting the need for good quality urodynamic testing (bladder pressure studies). Urinary sphincter weakness is probably related to the surgery, with shortening in the functional length or denervation injury (or both).[6] Studies have suggested that urethral hypermobility may also be a cause.[7]

In most urology centres that perform large volume radical prostatectomies, patients are taught about preoperative pelvic floor exercises and postoperative complications. Early referral for formal pelvic floor muscle training, with or without machine assisted biofeedback programmes, is likely to hasten the return to continence.[8] However, such training is unlikely to affect long term continence status.[8] A recent randomised controlled trial showed that formal one to one pelvic floor muscle training with a therapist does not improve outcome in terms of efficacy or cost effectiveness when compared with information on pelvic floor muscle training alone.[9]



SOVEREIGN ISM/SPL

Radiograph showing an artificial bladder sphincter

The insertion of an artificial urinary sphincter is an established operation with high patient satisfaction. It involves placement of an inflatable cuff around the urethra with a pump (usually situated in the scrotum) regulated by a pressure balloon to allow deflation of the cuff on voiding. The use of this delicate technique is limited by cost, the risk of infection, urethral erosion, and the possible need for revision surgery.[10 11] Patients must also possess the manual and mental dexterity to operate the device. However, because of its high efficacy and durability this technique is recommended by the National Institute for Health and Clinical Excellence (NICE) as the best option for severe urinary incontinence.[1 10]

The use of injectables such as collagen to create a bulking effect on the urethral sphincter has had mixed results. Some studies have suggested good short term results in patients with mild incontinence, although repeated injections are necessary.[10]

Most patients with post-prostatectomy urinary incontinence have mild to moderate symptoms and wear two to three pads a day.[12] Those patients may be best served by a comparatively less invasive operation, such as the insertion of a suburethral sling. Several types of sling exist, but all have a common objective—to augment residual sphincter function and allow spontaneous voiding. The concept of the "male sling" was derived from the popular and successful synthetic midurethral vaginal slings used in the treatment of stress urinary incontinence in women. Nevertheless, the synthetic male sling is relatively new and evidence on its long term results is limited,[11] highlighting the need for more stringent rules on the introduction and evaluation of new surgical devices. Urologists are yet to agree criteria about which patient is most suitable for a sling and who should be offered an artificial urinary sphincter. Randomised controlled trials are needed to tackle this question, and until then, patients should be referred to centres capable of carrying out both procedures. This is reflected in the NICE guidelines on the management of prostate cancer, which suggest that patients with serious incontinence should be referred to specialist centres for further evaluation and treatment.[1]

Although the case for a national screening programme for prostate cancer and the oncological benefits of radical prostatectomy are still debated, our responsibility to those patients remains paramount.

More studies are needed that specifically focus on the functional outcome of treatment for prostate cancer and put more emphasis on the patient journey, quality of pretreatment counselling, functional expectation, and post-treatment support. Adjusting the current resources to cover these areas will be a challenge. In the meantime, patients with urinary incontinence after radical prostatectomy should be identified and supported. If surgery is needed, long term morbidity can be minimised by early referral to specialist centres.

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