imagine in the highly controlled system of government in Britain. Secondly, it would not remove pressure for increased funds and, with greater visibility, may actually increase it. Thirdly, without an increase in overall tax, any increase in health spending would mean a corresponding decrease in other areas such as transport, housing, or the environment—all areas contributing to population health. Fourthly, it would be vulnerable to changes throughout the economic cycle. Finally, other spending departments would soon press for their own hypothecated funding.

The UK faces three quite separate issues. How much money does it need to run a health service that is at least comparable to that in neighbouring countries? What should it spend the money on? And how should it be collected? It is more important to answer the first question before the last.

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Improving cancer outcomes through radiotherapy

Lack of UK radiotherapy resources prejudices cancer outcomes

The British government has recently stated its aim of reducing deaths from cancer by a fifth over the next decade, with contributions from prevention, early diagnosis, and better treatments. British cancer cure rates are poorer than those of continental Europe and North America,[1] and underresourcing of radiotherapy services contributes to this.[2] The government target could be accomplished by providing adequate radiotherapy facilities to deliver proved clinical treatments.

After surgery, radiotherapy is the most effective curative treatment for cancer. Between 30% and 40% of the population will develop cancer, and at least half require radiotherapy at some time in their illness. Of patients having radiotherapy about 60% are treated with curative intent, often in combination with surgery and chemotherapy. Improving the effectiveness of radiotherapy would thus have a substantial impact on cancer cures in the United Kingdom.

Provision of adequate radiotherapy facilities would improve the outcome of cancer treatment through three main mechanisms. The first is reduction of the waiting list to start radiotherapy. Some consider this of negligible importance,[1] but sound evidence exists to the contrary.[3] Delay may allow progression of tumour stage, which is associated with worse survival.[4] In breast cancer a direct link between treatment delay and survival has been shown,[5] and a similar relation between delay and reduced local control is found for other tumours, including head and neck cancer.[6] A two week interval to plan and start curative radiotherapy treatment is considered reasonable by the Joint Council for Clinical Oncology.[7] At many UK centres, however, a six week wait is typical, and this interval is directly related to the level of resource provision.[2]

Six weeks is the approximate volume doubling time of many tumours,[3] and introducing an additional delay of four weeks between planning and starting radiotherapy must prejudice outcomes because more tumour cells are present when treatment starts. When the tumour volume doubling time is six weeks, an extra four weeks' delay allows a 67% increase in the number of tumour cells. In a clinical setting where the tumour control probability is 50%, this increase in the number of tumour cells would be estimated to reduce it to 31%.

The second mechanism is to avoid, or compensate for, gaps that occur during radiotherapy. Worldwide, including the UK, only about a third of patients complete their radiotherapy in the prescribed time, the remainder taking longer because of interruptions.[7] Overwhelming evidence exists that this worsens outcome, with an average calculated loss of tumour control probability of 1.6% per day of treatment prolongation.[8] For breast cancer a loss of local control of 3% has been described for each day of protraction between external beam radiotherapy and brachytherapy boost.[9] An audit at one centre showed that only 15% of patients undergoing breast radiotherapy met strict criteria of overall treatment time.[10] Missed fractions can be compensated for by treating the patient twice a day, but to do this requires additional linear accelerator capacity. An alternative is to continue treatment over a weekend. This does not require more machinery, but it carries extra salary costs.

The third mechanism is to introduce altered fractionation schedules, particularly giving more than

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one fraction per day. Hyperfractionation involves increasing the number of fractions and reducing the dose per fraction, typically with two or three fractions given per day, and randomised controlled trials have shown important improvements in outcome with such schedules. The EORTC hyperfractionation trial in head and neck cancer produced a 49% improvement in five year local control (absolute 19%) and a 33% improvement in survival (absolute 10%).[11] Continuous hyperfractionated accelerated radiotherapy (CHART, which includes reducing the overall treatment time as well as hyperfractionation) for treating lung cancer has delivered a 43% increase in two year survival (absolute 9%). For squamous carcinoma the increase in two year survival is 65% (absolute 13%).[12] Continuous hyperfractionated accelerated radiotherapy was conceived and evaluated in the UK, but it has not been generally implemented because of lack of resources.

Our calculations, based on the laboratory and clinical data available, suggest that an overall relative improvement in cancer cures of around 25% could be achieved simply by providing adequate radiotherapy facilities. The purchase of new linear accelerators for radiotherapy from the New Opportunities Fund announced recently is welcome and will help. However, many of the new machines will simply replace ageing ones; rather fewer will be additional, and there is no additional funding for the extra staff required to deliver the treatment (radiographers, physicists, and oncologists). A strategic review of radiotherapy resources, as the basis for a planned programme of national investment, is needed to address these important issues. Although further gains are to be expected from new radiotherapy technologies, such as conformal radiotherapy, the measures outlined above use existing technology, and all that is required is adequate resourcing.

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The rise in bacterial resistance

Is partly because there have been no new classes of antibiotics since the 1960s

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Almost since the beginning of the antibiotic era bacterial resistance has been seen as the major obstacle to successful treatment. Hardly any group of antibiotics has been introduced into clinical practice to which some bacterium has not developed resistance. Quantifying the impact of this resistance has often proved difficult, and misconceptions have often resulted from incomplete surveillance. Now that our surveillance methods are much better, we know that levels of antibiotic resistance are rising inexorably—as illustrated by this week's paper on trends in England and Wales (p 213).[1] Yet it has taken a long time to realise the extent of the problem, and there is still much that we need to learn about the mechanisms.

Resistance was often minimised as a problem simply because the problem was not known or recognised. At the end of the 1960s the surgeon general of the United States stated that "we could close the book on infectious diseases." Although those words seem naive now, at the time they were said the emergence of resistance did not seem to affect therapeutic options. Certainly, *Staphylococcus aureus* had become resistant to benzylpenicillin and was showing some resistance to methicillin, but it remained sensitive to gentamicin and thus infections could be treated.[2] Most of the bacteria responsible for community infections remained sensitive to the myriad of antibiotics available to treat them, and the surplus of available antibiotics masked the problem of emerging resistance.

At the start of a new century, some 30 years later, things look very different. We are facing a potential treatment crisis for some infections, with an escalating rise in resistance that we have difficulty in controlling.[2] What has changed? At the end of the 1960s we did not realise that we would face the next three decades with much the same antibiotic groups as we had then. Antibiotic discovery and development had been exponential since the 1940s, but no new clinically useful structures were discovered after 1961, and almost all the drugs that have been launched since the 1960s are modifications of antibiotics that we already have. This meant that bacteria that had "learnt" how to resist one member of a chemical drug class did not have to learn much more to overcome its later modifications. If bac-

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