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America's uninsured and underinsured

A special issue of JAMA looks at helping America's 40 million uninsured

What is to be done about the system of American health care? Inequality is a feature of all health care systems; in the United States it is endemic. The world's richest nation is well on the way to spending about 15% of its gross national product on the care and treatment of its citizens, yet it fails to provide more than a Third World safety net for 40 million of them. Alain Enthoven, for instance, argues that the American health care financing and delivery system is becoming increasingly unsatisfactory and cannot be sustained; comprehensive reform is urgently required.[1]

There is no shortage of ideas. The recent history of the health policy debate in the United States has been peppered with reform proposals, and the current issue of *JAMA* reviews 13 of these. An associated compendium documents the appalling human consequences of failing to provide for the health care needs of "one in every four Americans on any given day."[2] The editors of *JAMA* are to be congratulated on pulling together so much material and helping to put the problem of the uninsured higher up the political agenda.

The nine monthly specialty journals of the American Medical Association have joined forces to provide "a snapshot of where we are (and how far we have to go) in addressing the question of the uninsured and the underinsured"[3] through the publication of 50 articles, which represent the views of an impressive cross section of American medicine. To a British reviewer too many of the contributions seem parochial, trivial, and unscholarly despite undoubtedly reflecting a sincere and informed expression of genuine concern. Nevertheless, there can be no disguising either the human misery directly related to the lack of adequate health care or the "long-standing, systematic, institutionalised racial discrimination"[4] that is one of the root causes. It is difficult to comprehend, for example, why a poor pregnant woman in Chicago should need to wait 125 days for a consultation with a doctor at a public clinic.

What is to be done? One of the most valuable features of the special issue of *JAMA* is that not only does it provide space for the elaboration of competing reform proposals but it also provides a very useful summary and categorisation of the many, varied, and complex options. Part of the problem in pluralist America, it seems, is its capacity for inventing an almost infinite diversity of ways of getting from A to B, which results in a capacity to go nowhere in particular. And yet, even if the frequent allusions to landing on the moon and giving Saddam a kick in the pants are not entirely convincing, there does seem to be an impressive consensus that something must be done.

Opinion poll data suggest that at least two thirds of all of the most important American constituencies would support universal health care coverage even if—watch my lips—it meant increases in taxation. But when asked to express a choice between competing proposals "leaders of key groups and the public were unable to reach agreement on any single approach to reform."[5] It remains to be seen, therefore, whether the energy created by the genuine concern of health care professionals can be channelled in any purposeful way.

Although the agony looks set to continue for the present, it is important to put the crisis in the American health care system in perspective. It may have the worst safety net in the Western world, but other countries have holes in their nets as well. There is little room for complacency in other parts of the developed world. Inequality of access to health care may not be as starkly exposed in Britain and the rest of Europe as it is in the United States, but it continues to exist. Ginsberg and Ostow remind us that, even if universal health coverage is in place, a wide range of "cultural, demographic, geographic, and institutional factors . . . will continue to impede access to effective care for a significant segment of the population."[6]

One of the objectives of the British reforms of the NHS is to retain and safeguard the principle of universal access, largely free at the point of delivery, to health care. But this ought to mean more than eligibility to a place in the queue, or an entry ticket to the vagaries of the rationing process. The NHS equity principle implies that whatever resources are available should be distributed on the basis of a comprehensive assessment of the health needs of local communities.

This is far from being the case at present. The inverse care law is alive and well; British citizens have differential access to health care, which is unrelated to their clinical needs. For example, the formula of the Resource Allocation Working Party has failed to equalise access to hospital care for Londoners and non-Londoners, the Medical Practices Committee has not ensured that general practitioners are distributed in relation to any properly weighted measure of the need for their services, and no real attempt has been made to link patterns of prescribing with spending on other services. The allocation of purchasing power is badly in need of a fundamental reappraisal.

The NHS may not be as indictable as the American health care system, but inequality of access to health care remains too

much a part of the daily experience of large numbers of British citizens for us to scoff at the inability of our American cousins to tackle their problems. Much remains to be done on both sides of the Atlantic.

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Haemopoietic growth factors

Stimulation of white cells and platelets may transform cancer chemotherapy

The development of effective chemotherapy for cancer has been limited by drug resistance and the toxic effects of treatment on normal tissues, predominantly the bone marrow. The emergence of drug resistance may be prevented by the early use of drug combinations in full "conventional" doses delivered on schedule, and in some tumours resistance may be overcome by escalating the dose.[1] The problem of the bone marrow toxicity of current chemotherapeutic regimens may now also be solvable by the use of haemopoietic growth factors, and these may also allow the safer administration of more intensive treatments. These growth factors regulate the production of the mature cellular constituents of the blood. They include specific granulocyte, macrophage, and granulocyte-macrophage colony stimulating factors; interleukin 3; and erythropoietin.[2] Although the precise part these factors play in maintaining steady state numbers of blood cells is uncertain (except in the case of erythropoietin), the clinical administration of recombinant haemopoietic growth factors does lead to large increases in circulating cell counts.

Very high doses of cytotoxic treatment that would normally cause permanent bone marrow aplasia may be used with the technique of haematological "rescue" with previously stored cryopreserved marrow or haemopoietic progenitors harvested from the peripheral blood.[3] Such procedures, however, cause substantial morbidity, though the mortality is relatively low. The adverse effects are largely due to the period of profound cytopenia while the reinfused progenitors divide and differentiate into mature cells. Both granulocyte-macrophage and granulocyte colony stimulating factors have been shown in several uncontrolled studies to accelerate this process and shorten the period of neutropenia, but these factors have no consistent effects on the recovery of platelets or red cells.[4-6] Prospective randomised placebo controlled trials are in progress to determine whether this enhancement of the recovery of neutrophils has objective benefits such as lower infection rates and a reduced time in hospital. Treatment with haemopoietic growth factors is unlikely to reduce mortality since death from infection often occurs in the period of most severe neutropenia and this is unaffected by such treatment. Death is also commonly caused by toxicity to other organs, predominantly the lungs.[7]

Treatment with haemopoietic growth factors has been well tolerated: the granulocyte macrophage factor may cause low grade fever, bone pain, and a capillary leak syndrome (the last only at very high doses),[5] and the granulocyte factor may cause bone pain.[8] These symptoms are usually controllable by simple measures. Both factors may be given by subcutaneous injection, which provides effective plasma concentrations for 24 hours.[9] Antibodies to granulocyte-macrophage colony stimulating factor have been reported to develop in a few patients, but their clinical importance is as yet unclear.[10] Receptors for growth factors, mainly granulocyte-macrophage colony stimulating factor, are present on several malignant cell lines, including small cell lung cancer cells,[11] but any stimulation of growth of primary cells from biopsy specimens of solid tumours is rare.[12] There has been no suggestion so far of any acceleration of growth or increase in relapse rate of tumours in patients receiving haemopoietic growth factors, but prospective studies are needed to confirm this.

Almost all patients with chemosensitive malignant disease given cytotoxic drugs receive them in standard doses, and several trials have shown that treatment with growth factors reduces the neutropenic morbidity associated with such conventional chemotherapy.[13,14] This, however, seems an expensive way of making already well tolerated treatment regimens more tolerable. What treatment with haemopoietic growth factors also offers is the chance to address the question: does early exposure of a tumour to higher cytotoxic doses or to standard regimens without dose reductions or delays enforced by bone marrow toxicity improve response rates and, ultimately, survival? Eventually, with the drugs currently available other side effects—for example, the cardiotoxicity of anthracyclines—may become limiting.

If higher doses of cytotoxic drugs are given thrombocytopenia becomes more likely and may need supportive treatment.[15] Interleukin 3 and interleukin 6 produce megakaryocyte colonies in vitro, and both are now being evaluated for their ability to increase platelet numbers in vivo.[16] In the future, combination treatment with haemopoietic growth factors—for example, granulocyte-macrophage colony stimulating factor and interleukin 3—is likely to prove more successful than treatment with single factors. Trials are also in progress to assess whether treatment with erythropoietin can improve the anaemia of malignancy and reduce requirements for transfusion of red cells. Early results suggest that it may do so.[17]

In addition to their actions on progenitor cells, growth factors enhance many of the functions of mature granulocytes and monocyte-macrophages, including microbial phagocytosis and killing and antitumour cytotoxicity.[2] These effects have been shown both in vitro and in cells taken from patients receiving growth factors.[18,19] This raises the possibility of using growth factors as adjuncts to standard antimicrobial treatment—for example, in the treatment of systemic fungal infection in immunocompromised patients—and as antitumour agents, perhaps in combination with tumour specific monoclonal antibodies. Not all the effects of haemopoietic growth factors on phagocyte function, however, are necessarily beneficial—granulocyte-macrophage colony stimulating factor has been shown to impair the ability of neutrophils to migrate into an inflammatory site,[20] and the widespread activation of neutrophils has the potential to cause damage to vascular endothelium.