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the assurance that medical schools are producing doctors with the correct competencies at an appropriate standard.

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Is the private finance initiative dead?

It may have failed in the United Kingdom, but that won't stop it being exported

For health professionals working in hospitals that had been starved of investment for decades, often in buildings that were crumbling around them, the United Kingdom's private finance initiative (PFI) must have seemed like a dream come true. Rather than finding the money up front to rebuild hospitals, managers could enter into a contract with a private company or a consortium to finance the building and guarantee that the facilities would be maintained long into the future. In turn the hospital trust would pay an annual fee to cover the costs of financing the project (adjusted for the risk assumed by the private company) and of maintenance. Suddenly it became possible to escape the constraints imposed by buildings that, in many cases, had been designed to meet the needs of the population a century earlier.

The initial enthusiasm by many hospital boards was easy to understand, as the Department of Health made it clear that this was the only way to fund big capital developments. Yet from the inception of this initiative in the NHS there have been voices of caution, most notably Professor Allyson Pollock who, as a consequence, has suffered personalised attacks by those advocating PFI.¹ One of the potential drawbacks to which Professor Pollock drew attention was that the new facilities funded through PFI almost invariably provided less capacity than those they were intended to replace.² Another was that the contracts were extremely expensive (or seemed to be, given that they were shrouded in commercial secrecy) and were not supported by convincing economic arguments.³

Recent developments suggest that the experimental use of PFI in funding health care in the United Kingdom may be coming to an end. The first is the abandonment of the flagship west London development, which sought to create an important new teaching hospital complex by merging several specialist hospitals. There are many reasons for the failure of this project. But, overall, it was simply too complicated for a health system in which—in the name of local responsiveness—those responsible for purchasing care

(along with other interested parties) have become hopelessly fragmented. This has many implications. Perhaps the greatest is the question of how the NHS, meant to be led by primary care in future, can hope to develop a substantial programme of capital investment.

The second sign that political enthusiasm for PFI may be waning is the recognition of the initiative's true costs to the NHS and to contractors. The House of Commons Public Accounts Committee has drawn attention to the large profits made by the private contractor which built the Norfolk and Norwich hospital.⁴ The high transaction costs of bidding for contracts are, however, driving many potential contractors out of the market.⁵

The fundamental flaw at the heart of the PFI scheme is its lack of flexibility. As the pace of change in the delivery of health care becomes ever faster, managers should think twice about signing long term contracts that would levy heavy penalties for even minor changes in building projects. Indeed, one senior NHS manager has argued that new hospitals should have an anticipated lifetime of only 5-10 years.⁶ Such short term contracts are unlikely to interest private investors, unless they are accompanied by very high premiums.

The most serious threat, however, may be one that few people had anticipated. The UK government has set itself two fiscal rules. The first is that over the economic cycle it should not borrow to cover current spending. The second is that net debt should not exceed 40% of the gross domestic product. The first, known as (Chancellor of the Exchequer) Gordon Brown's "golden rule," differentiates between current spending and capital spending. The Treasury recognises that the government has a duty to maintain the level of investment required to meet the economy's needs and to ensure that the public capital stock is kept in good condition to maintain competitiveness and sustain public services.⁷ The second fiscal rule makes no distinction between borrowing for current spending and borrowing to finance investment. Net debt is now around 35% of gross domestic product, projected in

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the budget in March 2005 to rise to 37% by 2008, assuming that projected economic growth will be achieved.

The *Financial Times* published an article this spring suggesting that the Office for National Statistics (ONS) was about to reclassify PFI projects.⁸ Although ONS issued a rebuttal, stating that it “has not taken any decision to change the treatment of Private Finance Initiative schemes in the public finances” as “the element of PFI debts that should be recorded within Public Sector Debt, is an extremely complex and difficult matter,” it acknowledged that “ONS has recognised for some time that estimates need to be made and we have been continuously expanding our ability to cover PFI activities and explore possible sources of information.”⁹

This is important because funds for PFI are treated as “off balance sheet” financing and appear as “additional expenditure” to public sector expenditure and are not currently included in the government balance sheet calculations of net debt. Should the Office for National Statistics change the rules, a major component of capital spending under these contracts could be reclassified as debt. This could easily lead to a breach of the second rule, removing the main justification for the PFI model.

If the private finance initiative dies in the United Kingdom it may still have a life beyond these shores. Rather like general practice fundholding, it has created a cadre of experts who can now offer their services to the rest of the world. The United Kingdom may, once

again, be at least as successful in exporting its failures as its successes.

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Staphylococcus aureus, Panton-Valentine leukocidin, and necrotising pneumonia

A rare but often lethal cocktail that can complicate flu

Panton-Valentine leukocidin (PVL) is one of many toxins produced by *Staphylococcus aureus*. Structurally similar to γ haemolysin, this leukocidin comprises two subunits (F and S) that together are leukocidal and dermonecrotic.¹ Intermixing of γ haemolysin and the subunits of PVL produces toxin molecules with varying cellular affinities and destructive capability, even when the staphylococci may be otherwise sensitive to antibiotics such as methicillin. The death of a fit young soldier in the United Kingdom earlier this year from toxicity to PVL illustrated the extent of that capability.²

Infection with PVL producing staphylococci is rare. Fewer than 2% of clinical isolates of *S aureus* examined in the United Kingdom in 2002-3 had the genes to produce the leukocidin, although it was found in 4.6% of samples from infections of skin and soft tissue.³ Furthermore, “pure” disease caused by those *S aureus* bacteria that produce PVL is rarely life threatening. It presents as recurrent furunculosis or abscesses, it may be either sensitive or resistant to methicillin, and it can be difficult to eradicate among carriers. Three new and more virulent staphylococcal syndromes associated with the leukocidin—purpura fulminans, skin sepsis,

and necrotising pneumonia—have been recognised recently, however.

Purpura fulminans due to PVL producing methicillin sensitive *S aureus* (MSSA) has a mortality of 60% despite such sensitivity.⁴ Skin sepsis due to community acquired methicillin resistant *S aureus* (MRSA) occurs in patients without recent contact with healthcare facilities or known risk factors for such infection. Transmission during close physical contact causes outbreaks in prisoners, military personnel, schoolchildren, and athletes.⁵ Although these bacterial strains are resistant to methicillin, they are, at least, usually sensitive to more antibiotics than hospital strains.

The third manifestation of more serious disease caused by PVL is necrotising pneumonia, which is often lethal. It has been reported in America, Australia, Europe, and the Far East. The pneumonia often arises from bloodborne spread of organisms from infected tissue and can follow viral respiratory infections, especially influenza.

Strains of *S aureus* that produce PVL have a particular affinity for basement membrane exposed by desquamated ciliated epithelium, and they rapidly