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London's health care again

This time may be different

Nearly every decade this century someone has tried to solve the interlinked problems of London's health care, medical teaching, and research. Through a combination of unclear direction, vested interests, and lack of political will they have all failed: both the process and the problems continue. This autumn the Tomlinson inquiry will make its recommendations on London to the Secretary of State for Health, and this week the King's Fund London Commission has produced the final report of its £500 000 study into acute care in London.[1] Might the problems be solved this time?

The problems are familiar: an acute sector dominated by specialist services and provided from multiple hospital sites; fragmented teaching and research; and underfunded and underdeveloped primary and community care.[2] One reason for London's high costs is medical staffing levels 30% higher than elsewhere, which have not declined in line with resources or beds. Indeed, one perverse outcome of financial cuts over the past decade is that they have fallen disproportionately on general medical and surgical beds serving local populations. The result is that substantial groups of Londoners do not get as good or responsive health care as many outside London.[2]

The London Commission's final report does not name the institutions that should close—though it thinks that many should. Instead it sets out a vision for the year 2010 of responsive health care and internationally excellent teaching and research and suggests a mechanism and a source of funding for getting there.

The vision is one that will be familiar to health strategists: of a service led by primary care practitioners, who not only provide more services but also orchestrate a whole series of secondary and community services much more than now. Hospitals are fewer because much is done outside them and because highly technological care is concentrated in a few—to use resources efficiently but also to ensure that high volumes maintain high levels of skill. While some hospitals will still have a range of acute specialties to back up accident and emergency departments, others will specialise in day care or short stay elective procedures. The teaching hospital will disappear; research will be based round four university centres, which will contract with many provider units to supply the clinical experience needed by both undergraduates and postgraduates.

The specific recommendations (see p 1651) include reducing the numbers of both medical staff and medical students by a third and reducing 41 acute hospitals in London to no more than 30. The capital and revenue thus released (about £250m) should be used to develop primary and community health services, reshape acute services, and consolidate teaching and research. The mechanism for achieving these aims is a task force answerable to the secretaries of state for health and education (and to the Chancellor of the Duchy of Lancaster for research) that would work with "and direct" regions, district and family health services authorities, provider units, and the university on the details of developing primary care and reshaping acute hospitals.

The London Commission has shown that the traditional pattern of services and teaching in London is unsustainable. In such circumstances the detail of any recommendations becomes almost irrelevant: what is important is that the strategy should be agreed—and then implemented. In fact ministers are unlikely to do anything until the Tomlinson inquiry reports—and clearly the commission hopes its own recommendations might influence what Tomlinson has to say. Although the report's refusal to identify specific institutions that should close seems rather coy, there is sense in not doing so. Firstly, it avoids provoking an immediate defensive response. More fundamentally, no one group can have the wisdom to lay down a detailed blueprint for all of London. One of the London Commission's strong messages is that services have to be tailored to different communities and take into account what their publics want.

The barriers to this vision are, of course, immense. Changing habits and challenging institutional cultures are hard, though already there are signs that the explicitness over activities and costs brought about by the internal market is beginning to force change.[3] Bringing general practice in London up to the standards of the best in Britain would itself be an enormous task—but the commission is asking for more. Building the sort of community based health services that the

report envisages—with completely new ways of delivering care—may actually prove harder than reshaping acute services and redirecting medical students. The biggest barrier, however, may be London itself. The report touches on the fact that health services cannot be seen in isolation: poverty, unreliable transport, violence, collapsed social services, rotten housing, and poor education all work against the sort of health service that the commission wants to see.

Ministers have said that the political will exists to change London's health care, but they will need a lot of it to overcome the barriers and promote these radical—and untested—solutions. Unless he adopts completely the London Commission's ideas and analysis Tomlinson is unlikely to be as radical, and ministers might well be tempted by a smaller investment of political will. The danger is that anything less might not be enough.

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New research in tuberous sclerosis

Probably more common than was thought—with more adult complications

Tuberous sclerosis is a serious dominantly inherited condition best known for its presentation during childhood with fits and mental retardation. Articles on the disease have doubled over the past 10 years and have focused largely on improved diagnostic criteria and clinical features, disease prevalence, and genetics. Cranial magnetic resonance imaging is beginning to throw light on the contribution of cerebral tubers to neurological outcome, and complications of the disease in adults are gaining greater importance as both survival and ascertainment improve.

The diagnostic criteria first suggested by Gomez in 1979 have evolved.[1,2] Facial angiofibromas, ungual fibromas, retinal phakomas, multiple cortical tubers, subependymal glial nodules, and bilateral renal angiomyolipomas are considered pathognomonic. The condition can be diagnosed in an individual with an affected first degree relative if any of the following are found: a shagreen patch, a forehead fibrous plaque, multiple cardiac rhabdomyomas (on histological examination or, in a young child, on echocardiography), a giant cell astrocytoma, an isolated retinal phakoma, or an isolated cortical tuber (on histological examination, computed tomography, or magnetic resonance imaging). Without a family history these lesions are highly suggestive but not diagnostic and further investigations are required. This is also true for the following lesions whether or not there is a family history and at least two should be present to make the diagnosis: polycystic kidneys, typical hypomelanic macules, an isolated renal angiomyolipoma or cardiac rhabdomyoma, pulmonary lymphangiomyomatosis, and multiple cortical or subcortical hypomyelinated lesions. Several other features also suggest the possibility of tuberous sclerosis: seizures, gingival fibromas, molluscum fibrosum pendulum in a child or young adult, rectal hamartomatous polyps, hepatic angiomyolipomas, and renal cysts.

The incidence of tuberous sclerosis at birth is unknown. Recent epidemiological studies report a prevalence of 1 in 12 000 to 1 in 15 000 in children aged under 10 years,[3,4] but mildly affected cases are rarely diagnosed in childhood so there is a selection bias towards those with mental retardation. Thus the real frequency of mental retardation may be lower than previously thought; a recent study in an unbiased population of patients with tuberous sclerosis showed a prevalence of 38% (95% confidence interval 19% to 56%).[5] This suggests that the true incidence at birth may be as high as 1 in 6000.

In 1987 Fryer *et al* reported significant linkage with the ABO blood group, suggesting a locus for the tuberous sclerosis gene at chromosome 9q34.[6] This finding was not initially confirmed, and the chance discovery of an infant with an unbalanced 11/22 translocation was followed by linkage in American families at 11q22–q23.[7] A collaborative study combining all available data has confirmed genetic heterogeneity and refined the position of the chromosome 9 locus to within two megabases.[8,9] There is weaker evidence for a second gene on chromosome 11, and since several pedigrees are negative for both loci a third and possibily more loci may exist. Except perhaps in very large informative families, antenatal diagnosis will not be achieved by closely linked markers but must await the discovery of the gene itself.

About two thirds of cases are new mutations, so a common clinical problem is counselling apparently normal parents about the risk of a second affected child. The risk is 2-5%.[4] The most important assessment is clinical examination, including examination of the skin with a Woods lamp in a darkened room and direct funduscopy through dilated pupils.[10] Cranial imaging is unlikely to cause confusion, and as lesions of tuberous sclerosis have been seen in patients who are clinically normal this should be offered. If renal ultrasonography is used it should be remembered that single renal cysts are common in the normal population but do occur in tuberous sclerosis. Angiomyolipomas would always be suggestive. A blind controlled study of echocardiography for genetic counselling found that this was unreliable.[11] Likewise, a skeletal survey is unhelpful.[10]

Cranial magnetic resonance imaging has proved more sensitive than computed tomography at detecting cerebral tubers, although the relation between findings on magnetic resonance imaging and clinical outcome is not clear cut.[12] The simple number of tubers is unlikely to predict outcome: most children with more than 10 tubers will be severely retarded, but a quarter of those with less than five are severely retarded and in a series of patients with normal intellects one child had nine.[13] Tuber size and location may be more important. The number of large tubers seems to correlate with the number of electroencephalographic foci, and lesions in the occipital lobe correlate best with interictal electroencephalographic abnormalities.[14] The combination of posterior (occipital and temporal) tubers with bifrontal parasagittal tubers and marked bilateral synchrony on an electroencephalogram seems to signal a particularly poor prognosis.[15]

For many, however, the outlook is not so poor. Although patients with tuberous sclerosis do not have an average life span, they live longer than was once thought. An analysis of 40 deaths showed 11 from renal disease and 10 from brain