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Primary care in the inner cities

In 1980 the then Minister of State for Health, Dr Gerard Vaughan, rejected the call for a general inquiry into London's health care problems on the grounds that: "We have had a series of inquiries on London... what we need is some action."¹ In 1986 the government proclaimed in the discussion document on primary health care its firm resolve "to tackle the problems of providing high standards of primary health care in inner cities."²

It would be reasonable—but wrong—to conclude from these ringing declarations that nothing had happened during the intervening years to improve the quality of primary health care in London and other large cities. For one thing, the Acheson report on primary health care in inner London was published in May 1981,³ offering 115 specific recommendations ranging from a planned programme of retirement for principals in general practice to providing car loans for nurses. More than two years later—in October 1983—the government announced a £9m package for the inner cities spread over three years and earmarked mainly for higher improvement grants for premises, increased training for health visitors and district nurses, and the encouragement of innovative ideas.⁴

Taken together the Acheson report and the limited financial response of the government have achieved a measurable improvement in the training of nurses, the use of cost rent schemes and improvement grants, the expansion of primary health care teams, and the appointment of facilitators. Equally importantly, the broad thrust of the report's analysis of the problems of primary care in large cities has become conventional wisdom. Yet the response of central government and health authorities to the Acheson report has been characterised more by piecemeal uncoordinated initiatives than by integrated strategic thinking about inner city deprivation. And presumably the government's awareness of the limited impact of the Acheson report underlay its renewed commitment in the discussion document to "do something" about primary health care in the inner cities.

Why has this happened, and why, more than five years after the Acheson report, has little progress been made? These are the questions addressed by a careful and perceptive new analysis from the Policy Studies Institute,⁵ in which the authors tease out the tangled story of the Acheson report

from its origins in the 1970s to the period just before the publication of the discussion document. Their analysis is an outstandingly good example of a difficult form of social research—a case study of the policy making process. It will repay careful study by anyone interested in why things do not (and sometimes do) happen in the public sector.

From one perspective the answers offered by its authors are unsurprising, although they are salutary reminders to those who believe that the policy process is quick, flexible, or rational. Part of the reason for the desultory response to the Acheson report lay in the nature of the report itself. The study group ignored the request to rank its recommendations in order of priority (thereby making it difficult for the government to respond rationally even if it wanted to), and it presented its findings in a way that had obvious implications for the inner areas of all large cities, not just London.

This in turn compounded the government's difficulty, for while it had hoped to fund any recommendations for London out of savings in the hospital sector, the Acheson report said that new money was needed for an adequate policy for inner city areas throughout Britain. Such a proposition was distinctly unpalatable to a government committed to reducing public expenditure, and its hopes of confining the repercussions of the Acheson report to London were further damaged by the profession's opposition to London being treated as a special case.

There were, therefore, strong political forces militating against a swift and adequate response by government, and they were compounded by the divided structure of the NHS at the local level, where the policies for change would have to be implemented. Where, then, does this leave the government's new intention to improve primary health care in inner cities?

There are, as Jarman has recently pointed out,⁶ several things that could be done quickly and cheaply, including implementing the discussion document's proposal for the compulsory retirement of doctors at 70. Moreover, as the authors of Policy Study Institute study themselves remark, the beneficial impact of the Acheson report is likely to continue for some time. Yet further substantial progress must depend on a clearly articulated strategic policy for inner city areas backed up by the necessary resources. Such a

policy would address itself particularly to the roles of the Department of Health and Social Security, the regional and district health authorities, and the family practitioner committees, and it would regard the inner cities as a special case requiring special solutions. A more flexible approach to virement between the cash limited allocations to the health authorities on the one hand and the open ended expenditures on the family practitioner services on the other would sharpen the debate about respective roles and functions. Whether such a clear strategic lead will be forthcoming is a matter of guesswork. The lessons from the Policy Study Institute study are not encouraging.

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Immunohistochemistry in lymphoma diagnosis

Careful examination of the histological appearances of a pathological lesion is the mainstay of pathological diagnosis and will undoubtedly remain so. Nevertheless, the distinction between lymphoma and carcinoma and between different types of lymphoma can be difficult using conventional histological techniques but is of great clinical importance.^{1 2} Immunohistochemical demonstration of antigenic determinants on cells helps pathologists to distinguish more reliably between most neoplastic and reactive proliferations of lymphocytes and non-lymphoid disorders.^{2 4}

Until recently frozen sections of fresh tissue have been needed as most lymphoid antigens do not survive conventional fixation and processing.^{2 5} Since most specimens arrive at the laboratory in fixative immunohistochemical methods have thus been of limited use in diagnosing lymphoid disease. Fresh tissue also presents practical difficulties: the risk of infection to laboratory staff is increased; the tissue requires careful freezing and storage; and the morphology of frozen sections is inferior to that of paraffin sections. Facilities and staff for frozen section immunohistochemistry are mostly available only in major centres, and, while several methods have been advocated to circumvent the need for frozen section,^{6 9} they are unlikely to be applicable in most general laboratories.

Reagents that can simply and reliably identify lymphoid and related antigens in conventionally processed specimens would thus be of great value, and some such reagents are now available.^{10 13} The most valuable are those antibodies that recognise leucocyte common antigens since these allow the distinction between lymphoid and epithelial malignancies when used together with epithelial markers—for example,

cytokeratins. The lineage of many undifferentiated tumours can now be determined more often and more objectively than was previously the case.

Do lymphoid markers help in the distinction of Hodgkin's disease from non-Hodgkin's lymphoma? Claims for the existence of specific markers of Hodgkin's disease have been made.¹⁴ The monoclonal antibody Leu M1 has been widely regarded as a marker of Hodgkin's disease, but recent evidence shows that the antigen is found in many tissues¹⁵: Leu M1 immunoreactivity is neither a sensitive¹⁶ nor a specific¹⁷ marker of Sternberg Reed cells. Hence the diagnosis of Hodgkin's disease remains essentially a morphological one. The disease may, indeed, be pathologically and immunologically heterogeneous and not a single biological entity.^{18 19}

The value of immunohistochemistry is more clearly established in non-Hodgkin's lymphoma. Showing immunoglobulin light chain restriction has long been advocated as a useful discriminant between reactive and neoplastic B cell proliferations,^{2 4} but some have urged caution in interpreting immunoglobulin staining on paraffin sections²⁰ (personal observations). T cell clonality can be shown on frozen sections using antibodies that recognise the relatively few proteins produced by V region genes for β chain T cell receptors.²¹ Such reagents should enhance our understanding of T cell neoplasia, which is being recognised increasingly often.

New reagents can also identify both B and T lymphocytes and their neoplasms in paraffin sections.¹¹ These antibodies are not ideal, but they greatly improve our ability to study and diagnose lymphoreticular disorders. The use of these reagents has already shown that a series of gastrointestinal lymphomas associated with intense eosinophilia are of T cell origin, which has contributed to defining this disease.²²

New markers for lymphoid and accessory cells active in paraffin sections will soon become available, and immunohistochemistry on paraffin sections is likely to improve the standard and reproducibility of lymphoma diagnosis. But all is not perfect: interpreting results with existing agents is difficult; the reagents are expensive; and the methods are demanding in time and expertise. Further studies will be required to establish the true value of paraffin section immunohistochemistry in diagnostic lymphoma pathology.

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