An attractive hypothesis for the pathogenesis of pouchitis is that the pelvic reservoir undergoes a process of colonic metaplasia, possibly as a result of changes in bacterial flora, and that this metaplasia recreates a colon-like environment that favours the recurrence of ulcerative colitis in the ileal reservoir. More worrying perhaps are the long term implications of colonic metaplasia in the reservoir mucosa. The mucosal epithelium of the reservoir shows increased proliferative activity in patients with and without pouchitis.⁶ The coexistence of colonic metaplasia and hyperproliferation may increase the neoplastic potential of the reservoir's epithelium. In both ulcerative colitis and familial adenomatous polyposis the colonic mucosa shows hyperproliferation,^{24 25} and, although to different degrees, both conditions are associated with epithelial dysplasia and malignancy.

The clinical, pathological, and experimental evidence should be put into context before any conclusions are drawn about the long term outlook for patients with pelvic ileal reservoirs. Current evidence suggests that the neoplastic potential of the pelvic reservoir is low. No convincing cases of epithelial dysplasia have been described in reservoirs constructed for ulcerative colitis. Very few reservoirs have been in place for over 10 years, however, and nearly all cases of epithelial dysplasia in the colon and rectum associated with ulcerative colitis arise after this time. Little evidence exists for increased neoplastic risk in continent abdominal ileostomies, some of which have been in place for 20 years.¹⁷ A recent report describes a carcinoma arising in a pelvic reservoir of a patient with colitis; in the authors' view the carcinoma arose not from metaplastic ileal mucosa but from remaining rectal mucosa within the pouch.²⁶ In patients with familial adenomatous polyposis adenomas have been observed in both pelvic reservoirs and continent abdominal ileostomies,^{4 27 28} but such adenomas could be considered to be part of the normal range of disease in familial adenomatous polyposis.

The optimist's view of the pelvic reservoir is that there is no increased neoplastic potential and that pouchitis is merely a hiccup in the establishment of the operation as the panacea for all diffuse colonic mucosal disease. The pessimist would suggest that all patients with an original diagnosis of ulcerative colitis will eventually develop pouchitis, that some will develop dysplasia and carcinoma, and that many patients with familial adenomatous polyposis will develop adenomatous dysplasia in the reservoir mucosa with high rates of malignancy. The truth, of course, lies somewhere in between. Until we have more objective evidence of the long term consequences of ileal reservoir construction surveillance by endoscopy and mucosal biopsy is warranted.

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National Cervical Screening Programme

Enforcement and coordination are required: guidance and interpretation are not enough

In the mid-1980s defects in the programme of screening for cervical cancer in Britain were highlighted in a series of research reports and in press, television, and radio programmes. Deaths were occurring that should have been prevented; women had to wait for months for the results of their tests, others were not recalled when they should have been, and many women were still not being screened at all.^{1 2}

These deficiencies led to a reappraisal of the NHS programme, and in 1988 the Department of Health issued a guidance circular intended to improve standards.³ Shortly afterwards the National Association of Health Authorities looked at the results being achieved and concluded that "there is every reason to believe that the NHS has risen to the challenge of translating policy into reality."⁴ The department should be congratulated for now (perhaps bravely) commissioning an independent audit (p 915) of the performance of district health authorities.⁵

Some of the findings certainly suggest that all is well. All districts now have computerised call-recall programmes, and 93% have fail safe mechanisms to follow up abnormal smears. An average smear reporting time of less than four weeks in three quarters of districts is a result likely to receive favourable publicity—although the new general practitioner contract, aiming at achieving 80% target coverage, has recently substantially increased this time.⁵ Three quarters of the districts were "fairly satisfied" with their programme; but, interestingly enough, this was not assessed by the prime performance indicator of effectiveness (not evaluated by Elkind *et al*) but simply reflected the hard work and commitment of those concerned.

The report should, however, cause some disquiet as well as some satisfaction. Substantial variability was found among districts. Some findings, such as restricted choice of smear taker and non-notification of results, are important causes of distress to women. Others warrant urgent explanation. Why,

for example, are 30% of district health authorities not expecting to meet the target date of 1993 to complete their initial call? Why are 46% of laboratories not undertaking the full recommended quality control,^{7,8} and, most critically, why have four laboratories with no quality control been permitted to continue screening at all?

Both the National Association of Health Authorities survey⁴ and Elkind *et al*⁵ have shown that the workload being put on laboratories is becoming a serious problem—reflecting both inadequate funding and a lack of trained staff. It seems that NHS screeners are being attracted to the private sector by its higher salaries—and private practice is then being fuelled by NHS inadequacies. In a timely study Raffle *et al* (p 907) (who have experienced staff shortages) have audited workload rates and practices in Avon.⁹ A recall interval of three years has substantial medical support,¹⁰ and the audit by Elkind *et al* showed that it was operating in full or partially in half of all NHS districts.

The Avon study, however, shows that districts still struggling with a five year interval are invariably handicapped by numerous inappropriate opportunistic smears. Avon's successful policy of returning such smears unprocessed to the taker has proved successful and deserves widespread introduction. Their findings with respect to follow up smears, however, signal danger. Once they have implemented national follow up recommendations,^{11,12} the authors' projections suggest that by 1993 half their present capacity will be required for this purpose. The message is clear: priority should be given to the investigation and treatment of severe rather than minor cytological abnormalities. Such a policy would improve cost effectiveness by conserving scarce laboratory and colposcopy resources and improve screening specificity. To date, there has been a preoccupation with programme sensitivity because of the emotive response to false negative results. It is now essential to redress the balance and give more consideration to the adverse effects of over-diagnosis and overtreatment.

The 1988 circular should by now have led to the NHS having a successful cervical screening programme. Sadly, however, after a promising start, many district health authorities have sought economic refuge in the "guideline" nature of the circular. The word guideline has been seen as permission to interpret widely and thereby avoid expenditure on all but the most sensitive issues.

The remedy is plain. The circular requires departmental enforcement to ensure appropriate funding—especially in the present climate of white paper flexibility.

At the moment each district cervical screening programme works in isolation, and this will not be changed by the implementation of the circular—nor has it been helped by the abandonment of the departmental committee on gynaecological cytology. Current problems in general practice are further evidence of inadequate central coordination and advice. It is wrong, for example, that some work conforming to the circular does not qualify for payment under the new contract. The NHS cervical screening programme requires leadership, integration, and standardisation in all its aspects, including management, clinical issues, information systems, quality assurance, development, education, training, and research. The Faculty of Community Medicine must be congratulated for initiating the national coordinating network with these objectives.¹³ As with the circular, however, the national coordinating network could be ineffective if not given departmental support and "bite" to guarantee adequate resources.

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Diagnosis and prevention of congenital and perinatal infections

TORCH screening should be discouraged

The acronym TORCH was coined in 1971 to draw attention to the need to diagnose congenital infection caused by *Toxoplasma gondii*, rubella virus, cytomegalovirus, and herpes simplex virus.¹ Although paediatricians and obstetricians often send serum samples from infants and pregnant women with a request for a TORCH screen, virologists have recently been questioning the value of such requests, which often lead to unnecessary and expensive serological tests.² Moreover, a TORCH screen does not include all congenital and perinatal infections. It does not, for example, include congenital syphilis, although four or five cases occur each year in Britain; parvovirus B19 infection (which causes hydrops fetalis or fetal death in the second trimester in about 9% of cases when infection occurs in early pregnancy³); HIV and varicella zoster virus (which may cause both congenital and perinatal infections^{4,5}); or hepatitis B virus, human T cell leukaemia/lymphoma virus type I, enteroviruses, *Chlamydia trachomatis*, and several bacteria that can all cause perinatal infections.²

A TORCH screen also gives the false impression that all congenital infections can be diagnosed with a single serum sample. Although this may be enough to diagnose congenital rubella and maternal toxoplasmosis and B19 infections, other samples are usually required to diagnose the other infections. Because maternal antibody will complicate the interpretation of specific IgG results and IgM specific for *Toxoplasma gondii* and cytomegalovirus may be detected in only about half of all congenitally infected neonates, serological results must be interpreted with caution. Kits for TORCH testing using serological methods should therefore be used with care. Congenital cytomegalovirus is best diagnosed in the first three weeks of life by detecting virus in the urine by culture methods, immunofluorescence, or electron microscopy as babies with congenital cytomegalovirus excrete high titres of virus but may have no detectable IgM specific for cytomegalovirus. Neonatal herpes may be diagnosed similarly, but the features of neonatal herpes may not develop for 10-14 days after birth and about a fifth of infected babies have no skin lesions.⁵ Lesion swabs, cerebrospinal fluid, urine, and throat and eye swabs should all be inoculated into cell cultures. Electron microscopic examination of scrapes from lesions will