in Britain, many of the difficult decisions would not have been necessary.

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1 Wise J. Storage period ends for 4000 embryos. *BMJ* 1996;**313**:189. (27 July.)
2 Dickey RP, Taylor SN, Curole DN, Rye PH, Olar TT. The passage of Florida's statute on assisted reproductive technology. *Obstet Gynecol* 1995;**85**:480-1.

Counselling will be needed when embryos are donated as well as when they are stored

EDITOR,—We agree with Alan Trounson and Karen Dawson that the ability to cryopreserve embryos has presented both opportunities and dilemmas.[1] The authors suggest that consent be obtained at the time of storage, and this may have organisational advantages. Nevertheless, we have misgivings about this suggestion because many units may not have screened couples for HIV infection, cystic fibrosis, and hepatitis B. It is also possible that, in future, additional screening may be deemed necessary. We suggest that couples who consent to the storage of embryos for donation should be told that at the time the embryos are donated they will need to be further counselled and screened and will need to give their consent again. Our present experience is that couples do not wish to return to the unit for further screening, possibly because we did not counsel them about this at the time that their initial consent was obtained.

If a couple who wish to donate frozen embryos return for screening and are found to be positive for HIV or hepatitis B virus then another potential problem arises: there is currently some concern that cross contamination is possible between tissues held in liquid nitrogen storage.[2]

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1 Trounson A, Dawson K. Storage and disposal of embryos. *BMJ* 1996;**313**:1-2. (6 July.)
2 Tedder RS, Zuckerman MA, Gladstone AH, Hawkins AE, Fielding A, Briggs EM, *et al.* Hepatitis B transmission from contaminated cryopreservation tank. *Lancet* 1995;**346**: 137-40.

Postmenopausal cystitis

Bladder cancer may be a cause, but interstitial cystitis is an unusual differential diagnosis

EDITOR,—Linda Cardozo is right to draw attention to the problem of postmenopausal cystitis and the use of topical oestrogens in its treatment.[1] Topical oestrogens are almost certainly underused in this condition. From the urological perspective, however, certain important points should be emphasised.

Firstly, ultrasonography is probably preferable to intravenous urography for imaging. Ultrasonography will quantify residual urine and detects renal and bladder calculi better than plain radiography. Ureteric stones (which ultrasonography will probably miss) are virtually never the isolated cause of recurrent infections. Ultrasonography spares the patient irradiation and the finite risk of a reaction to contrast medium.

Secondly, bladder cancer is an important consideration in postmenopausal women who present with irritative voiding symptoms. There can be few urologists who have not seen patients referred late with bladder tumours after inappropriate management for other presumed causes of lower urinary tract symptoms. In all patients whose urine is not free of cells after treatment of a confirmed urinary tract infection, repeat cytological examination of urine and flexible cystoscopy should be carried out. Cytological examination will detect those patients with carcinoma in situ whose bladder looks normal cystoscopically.

Thirdly, interstitial cystitis is an unusual differential diagnosis in this group of patients. We accept that it creates considerable controversy both in its diagnosis and in its treatment. The putative role of mast cells in this condition is debated; some studies have reported greater numbers of these cells in the detrusor muscle of patients with interstitial cystitis than in normal detrusor muscle, though the value of counting mast cells for diagnostic purposes is still doubted.[2] Indeed, overlapping values are found when the control group includes patients with other types of chronic cystitis[3]: differences are at the statistical, rather than the individual, level. The issue is further confused by the fact that mast cell counts that are related to the surface area of the detrusor muscle will be affected by the state of contraction or distension of the bladder wall and the procedures for tissue fixation and processing. For these reasons, many uropathologists would not routinely count mast cells when presented with a biopsy specimen in this clinical condition[4]: the main value of the biopsy is to exclude neoplasia.

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1 Cardozo L. Postmenopausal cystitis. *BMJ* 1996;**313**:129. (20 July.)
2 Lynes WL, Flynn SD, Shortliffe LD, Stamey TA. The histology of interstitial cystitis. *Am J Surg Pathol* 1990;**14**:969-76.
3 Kastrup J, Hald T, Larsen S, Nielsen VG. Histamine content and mast cell count of detrusor muscle in patients with interstitial cystitis and other types of chronic cystitis. *Br J Urol* 1983;**55**:495-500.
4 Epstein JI. Interstitial cystitis versus denuding cystitis. In: *Differential diagnosis in pathology: urologic disorders*. New York: Igaku-Shoin, 1992:32-3.

Should be managed in urological units

EDITOR,—We take issue with several points in Linda Cardozo's editorial on postmenopausal cystitis.[1] The comment that "for those women in whom oestrogen therapy is ineffective or inappropriate, it is important to exclude underlying pathology" is misleading as it implies that oestrogen should be given before adequate assessment and investigation. The author should have mentioned that urothelial carcinoma should be excluded by microscopical examination of urine for red cells, followed if indicated by cytological examination of urine and flexible cystourethroscopy, before oestrogen is started after antibiotic treatment for bacterial cystitis.[2]

The coexistence of vaginal and urethral stenosis with atrophic vaginitis is well known and is best treated with a combination of urethral dilatation and intravaginal oestrogen for long term success.[3]

Bacterial cystitis can be difficult to distinguish from bladder carcinoma, especially carcinoma in situ. Interstitial cystitis has clearly defined diagnostic criteria,[4] including a particular symptom complex, a systemic response to endoscopic cystodistension, and specific histological appearances on biopsy. The suggested treatment options for interstitial cystitis are incomplete. The therapeutic effects of cystodistension and cystodiathermy are not mentioned; nor is the role of intravesical instillation of silver nitrate, dimethyl sulphoxide, or newer agents such as hyaluronic acid.

This editorial emphasises that postmenopausal cystitis is best investigated and managed in urological units.

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1 Cardozo LD. Postmenopausal cystitis. *BMJ* 1996;**313**:129. (20 July.)
2 Golin AL, Howard RS. Asymptomatic microscopic haematuria. *J Urol* 1980;**124**:389-91.
3 Parsons CL. Urinary tract infections in the female patient. *Urol Clin North Am* 1985;**12**:355-60.
4 Hanno P. Interstitial cystitis. In: Fitzpatrick JM, Krane RJ, eds. *The bladder*. Edinburgh: Churchill-Livingstone, 1995: 175-81.

Sensitivity testing for *Helicobacter pylori* should be more widely available

EDITOR,—Greg Rubin and Richard Stevens are right to ask for the wider availability of non-invasive diagnostic tests for managing *Helicobacter pylori* infection in primary care.[1] We believe that the implementation of management guidelines for peptic ulcer disease also requires the wider availability of tests to determine the sensitivity of *H pylori*, because increased prescribing of antimicrobial drugs to treat the infection may be associated with increased drug resistance. Furthermore, our data indicate that if the global eradication of *H pylori* is deemed desirable then simpler and more effective regimens need to be developed for infected patients.

Table 1 (see overleaf) gives details of seven patients with gastric or duodenal ulcer who received at least three different courses of eradication treatment, including the most commonly used drugs, in an attempt to eradicate *H pylori*. In all cases we used either histological examination (Giemsa staining) plus the rapid urease test or breath testing to evaluate the patients' *H pylori* status at least six weeks after the completion of the treatments. All the patients were fully informed and complied with the treatments.

Failure of treatment may be due to the different antibiotic susceptibilities of the various strains of *H pylori*; phenotypically and genotypically different strains of *H pylori* can be present in the stomach at the same time.[2] We do not yet know whether, in an individual patient, drug resistance occurs through the selection of resistant strains within a mixed population (that is, primary resistance) or through previously sensitive strains becoming resistant (that is, secondary resistance). Because we found that a patient might have to be treated three or more times for *H pylori* to be eradicated, both mechanisms may act in the same patient. In some cases, after the selection of organisms that are primarily resistant to certain antibiotics, secondary resistance to other drugs might develop.

Before treating patients with *H pylori* infection it is thus always important to consider the results of culture and sensitivity tests.[3] The problem is that at present the tests are unavailable in most centres. In addition, the routine isolation of