but reduced the proportion of patients with prolonged post-herpetic neuralgia.2 Many other reports have dealt with various treatments, but these all need evaluating by controlled studies. Clinical experience suggests that systemic analgesics, either non-steroidal anti-inflammatory drugs or an opioid drug, may moderate the acute pain in herpes zoster, but evaluations of their efficacy have not been reported. Conventional analgesics are ineffective in managing the neurogenic pain, and the most efficacious treatment is probably prolonged treatment with anticonvulsants and tricyclic agents.15 22-25 Baclofen may have a therapeutic role in controlling the pain,26 27 and an epidural injection of steroids is efficacious to the extent that it may be the best treatment for chronic intractable neuralgia when steroids are not contraindicated.28 Acupuncture29 and transcutaneous stimulation30 31 are other possibly effective adjuvant treatments. Finally, preliminary results suggest that topical capsaicin, a neuropeptide active agent, may have an important therapeutic role in post-herpetic neuralgia.32 33

JACQUELINE V JOLLEYS

Principal in General Practice, Belton, Nr Loughborough, Leicestershire LE12 9UJ


Radioactive patients

Patients given radioactive substances represent a small risk to others

Patients given radioactive substances become sources of radioactivity and present a hazard to those with whom they come into contact. Most patients given radioactive substances present only a small risk to others, but guidance needs to be followed carefully to ensure that the risk is kept to a minimum — particularly in the case of those given radiolabelled iodine (iodine-131) therapeutically.

Radiotherapy with sealed radioactive sources is carried out in designated centres on inpatients under controlled conditions of isolation, and the patients are not discharged until the treatment has been completed and the sources have been removed. On the other hand, unsealed radioactive sources are used more commonly in more hospitals, for diagnosis as well as for treatment, and in outpatients as well as inpatients. These procedures result in patients becoming mobile sources of radiation, presenting two distinct types of hazard. Firstly, radiation is emitted from the patient, so that members of the public, porters, nurses, and other hospital staff are exposed. Secondly, specimens of the patient’s tissue and body fluids such as blood and urine may become radioactive, presenting a risk to family members and other people at home and to people in hospital wards, operating theatres, and the mortuary.

The doses of radioactivity used in diagnostic tests are specified by the Administration of Radioactive Substances Advisory Committee, which issues the licence necessary for consultants to give radioactive substances to patients.1 Doses exceeding the specified values are given only in special circumstances. Most unsealed radioactivity used diagnostically is in the form of a radiopharmaceutical containing 99mTc, and its physical half life of six hours means that the radiation hazard is short lived. Special precautions may be necessary only when prolonged close contact with another subject is unavoidable or when there is a risk of contamination. The risk of close contact is greatest when a patient is caring for an infant at home. It may be identified by questioning the patient and minimised by issuing explicit instructions. The risk of contamination will be greatly reduced by following simple principles of hygiene and by adopting the protective measures routinely used for handling biological material.

By far the greatest hazard comes from giving iodine-131 therapeutically. Radioiodide is secreted in body fluids such as sweat and saliva, and more stringent precautions must be taken against both contamination and close contact. Convention al doses for treating thyrotoxicosis are normally given to outpatients, but the higher doses given for thyroid carcinoma mean that patients have to remain in hospital nursed under conditions of isolation similar to those that apply to treatments from sealed sources. If radioactivity retained by the patient when leaving hospital exceeds certain defined amounts then the method of transport will be restricted and an instruction card must be issued to minimise the dose to others, particularly young children.

Statutory control is provided by the Ionising Radiations Regulations 1985,2 and their interpretation for clinical appli-
Enteropathy induced by non-steroidal anti-inflammatory drugs

Often subclinical but may mimic Crohn’s disease

Non-steroidal anti-inflammatory drugs of all groups have long been known to induce ulceration of the stomach and duodenum. Recently they have been recognised—particularly when given long term—to increase the permeability of the small intestine and cause occult blood loss. In a few patients the drugs may cause ulceration and stricturing of the small bowel and a clinical syndrome indistinguishable from Crohn’s disease.

The earliest reports of ulceration and stricture in the small bowel associated with non-steroidal anti-inflammatory drugs were in patients who were commonly taking many drugs. Many cases were classified as idiopathic ulceration of the small intestine. Then Langman and others showed that some patients admitted to hospital with perforation of the small and large intestine were receiving non-steroidal anti-inflammatory drugs, whereas among 44 premature infants receiving indomethacin for closure of a patent ductus arteriosus, four had perforation of the small intestine. Non-steroidal anti-inflammatory drugs also emerged as an important factor in the relapse of ulcerative colitis, and Bjarnason and others showed that patients with rheumatoid arthritis receiving long term non-steroidal anti-inflammatory drugs have increased small intestinal permeability.

Enteropathy affects patients of any age and either sex who have taken non-steroidal anti-inflammatory drugs for six months or longer. More than two thirds of patients taking the drugs long term have subclinical intestinal inflammation and occult blood loss. Most of them remain symptom free, but up to a fifth may have bile acid malabsorption and thus may develop diarrhoeal illnesses. The blood loss is usually subclinical, and iron deficiency anaemia and hypalbuminaemia will develop only if there is additional disease or dietary problems. Probably fewer than 1% of patients develop strictures and ulceration; early ulceration of the mucosa may progress through transmural inflammation to a stricture dominated by submucosal fibrosis. Granulomas, fissuring ulceration, or arthritis does not occur.

Patients with enteropathy caused by non-steroidal anti-inflammatory drugs may present with loss of appetite and weight, an iron deficiency anaemia of uncertain aetiology, or, occasionally, obstruction or perforation of the small intestine, but most patients have no symptoms or physical signs. The differential diagnosis of a stricture includes ischaemia or tuberculosis of the small intestine, lymphoma, post-irradiation enteritis, and inflammatory bowel disease, but only inflammatory bowel disease should pose any diagnostic problem. Sigmoidoscopy, colonoscopy, and examination by barium enema may help in the diagnosis. These show no abnormalities...