surgeries? Find out number of patients and area covered. (*b*) Other possible queries concern dispensing; deputising services; records (Record envelopes or A4? Problem-oriented?); teaching medical students, vocational trainees, and others; research interests; whether the practice is RCGP-oriented; and whether patients see one doctor or can see any doctor.

(3) Partners. (*a*) Ages. (*b*) Interests: medical; medicopolitical; clinical assistantships; police; industrial; old people's homes; political and religious; and other. (*c*) Attitudes: to private practice; to termination of pregnancy—are they willing to accept change? Do they get on well together?

(4) Facilities. Is there open access to: a pathology laboratory; *x*-ray facilities (including contrast studies); physiotherapy; maternity and medical beds; audiograms; and a dietitian.

(5) Equipment owned by surgery and special clinics: ECG; peak-flow meter; vitalograph; cautery; radiotelephones; and other—for example, audiometer, sigmoidoscope. Do they do suturing and minor operations? Are they equipped to go to road accidents? Is there a baby clinic; well-woman clinic; antenatal clinic; or other clinics?

(6) Staff: number of receptionists; secretary (are letters typed or handwritten?); do they have a practice manager? What is the relation with district nurses, health visitors, and social workers? Is there a "health-team" atmosphere?

(7) Hospital: relation with consultants: How long are waiting-lists for *x*-rays, outpatients, and routine surgery? Is there a postgraduate centre?

(8) Time off: off-duty rotas; holidays; and study leave. Is there provision for sabbaticals?

(9) Money: how is it shared out? What is the starting salary? Find out about income at parity and capital required, and possibly look at accounts (see text).

(10) Area: what is the provision for private and state schools? Check theatre, arts, and sports facilities and shops; parking and traffic problems; and public transport. Find out whether any changes are expected and about the likelihood of finding a job for your wife.

(11) Housing: any chance of accommodation while you find a house?

Today's Treatment

Drug-induced diseases

Drug-induced gastrointestinal disease

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Surveys of hospital inpatients show that 20-40% of all side effects of drugs affect the gastrointestinal tract. Because most drugs are taken by mouth, patients tend to blame medication for their gastrointestinal symptoms; nearly all oral drugs have gastrointestinal side effects. Gastrointestinal symptoms are important because they may reduce patient compliance, and also because they may herald the development of a drug-induced disease. In this article attention will be restricted to specific gastrointestinal diseases attributed to drugs. Usually, these diseases have pathological changes that do not resolve immediately the drug is withdrawn.

Mouth

Aspirin, potassium supplements, corticosteroids, pancreatic enzyme tablets, and sublingual isoprenaline cause ulceration of the buccal mucosa if the tablets are sucked. Patients, especially children, should be advised to swallow tablets with water. Buccal ulceration may also occur as part of a generalised hypersensitivity reaction, the most severe form being the Stevens-Johnson syndrome. When drug-induced, usually after sulphonamides, barbiturates, or penicillin, it responds to withdrawal of the drug.

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Bone marrow suppression induced by antimitotic drugs, carbimazole, or chloramphenicol may present first as buccal ulceration, or as petechial haemorrhage or infection within the mouth. Herpetic ulcers are common during immunosuppressive treatment. Oral candidiasis may complicate treatment with antibiotics, corticosteroids (including inhaled beclamethasone), or immunosuppressive agents.

Oesophagus

Candidiasis may also effect the oesophagus and can cause dysphagia. It may not be suspected clinically if the mouth is not affected, but can be diagnosed endoscopically or radiologically. There are many reports of oesophageal ulceration from slow-release potassium chloride tablets; in some cases a tablet has been found adjacent to the ulcer, providing strong evidence for a causal relationship.

Drugs known to cause mucosal ulceration should not be given in conditions where there may be a delay in oesophageal emptying, as in oesophageal stricture, scleroderma, enlargement of the left atrium, or immediately after cardiac surgery. Symptomatic gastro-oesophageal reflux may occur with anti-Parkinsonian drugs of the anticholinergic group, which are known to decrease lower oesophageal sphincter pressure.

Stomach

Peptic ulcer has been reported in association with many drugs, but as it is common in the general population it can be expected to occur at least as commonly in patients taking drugs. The incidence of peptic ulcer is said to be higher in patients with rheumatoid arthritis, irrespective of treatment, than in the general population; this has given the drugs used to treat

rheumatoid arthritis—salicylates, corticosteroids, and non-steroidal anti-inflammatory analgesics—a notoriety that they may not deserve.

Surveys in Australia and the USA have shown that women with chronic gastric ulcer consume more salicylate than control subjects attending outpatient clinics with non-ulcer dyspepsia; no difference has been found for men or for patients with duodenal ulcers. Other surveys have shown that patients with major gastrointestinal haemorrhage consume more salicylate than patients admitted to hospital for any other reason. This association is strongest with chronic continuous salicylate use. Salicylate-induced haemorrhage occurs more often from acute erosions than from chronic peptic ulcer. Salicylates increase the rate of occult blood loss in patients with peptic ulcer and in healthy controls. The incidence of such occult blood loss does not correlate with the incidence of major ulcer haemorrhage. Dispersed ("soluble") and buffered aspirin cause less occult blood loss than acetylsalicylic acid. Although the exact relation between salicylate and gastric disease is still disputed, it seems sensible not to prescribe salicylates to patients with gastric ulcer or dyspepsia, or to those who have previously had dyspepsia or gastrointestinal bleeding with salicylates.

Corticosteroids have long been reputed to cause peptic ulcer. An analysis of 42 double-blind controlled trials of steroids in the treatment of a variety of diseases including rheumatoid arthritis has shown no difference in the incidence of dyspepsia or gastrointestinal haemorrhage between active and placebo groups. In studies where a history of peptic ulcer was common, and did not exclude patients from the trial, there was no evidence of reactivation in the group taking steroids. These epidemiological data are consistent with experimental evidence that steroids do not increase occult gastrointestinal blood loss or cause gastric erosions.

Phenylbutazone and indomethacin commonly cause dyspepsia in patients with rheumatoid arthritis. Peptic ulcer is no more common in patients treated with phenylbutazone or indomethacin than in patients treated with other drugs.

The newer non-steroidal anti-inflammatory analgesics (ibuprofen and its relatives, mefenamic acid, diclofenac, and naproxen) appear to be better tolerated than salicylates in patients with a previous history of dyspepsia. Gastrointestinal haemorrhage has been reported during treatment with each of these drugs and is said to be particularly common with naproxen. No systematic investigation of the incidence of peptic ulcer and gastrointestinal haemorrhage has been performed, but one study in progress suggests that these drugs may be more commonly associated with haematemesis and melaena than salicylate. The non-steroidal anti-inflammatory analgesics are all more expensive than aspirin, and many are less potent. They should therefore be reserved for patients who develop dyspepsia while taking soluble aspirin. Patients who have been shown to have a peptic ulcer should if possible be treated with an analgesic such as paracetamol, which has no anti-inflammatory action.

Small intestine

Ulceration, perforation, and stricture of the small intestine are now well-recognised complications of enteric-coated potassium chloride tablets. All preparations containing enteric-coated potassium chloride have now been withdrawn, but the introduction of a slow-release preparation has not abolished the problem. Since 1970 there have been several reports of mid-ileal ulcers and strictures in patients taking Slow K. Compound tablets containing a diuretic and slow-release potassium chloride have also been implicated. It seems sensible therefore to use potassium-sparing diuretics when the small intestine is abnormal.

Paralytic ileus may be induced by anticholinergic drugs (including anti-Parkinsonian drugs of this class), tricyclic antidepressants, phenothiazines, and opiates. Tricyclic antidepressants and clonidine can also cause the syndrome of intestinal pseudo-obstruction; recognition of this can save patients from an unnecessary laparotomy. Both conditions resolve on withdrawal of the offending drug.

Infarction of the small intestine due to thrombosis of the superior mesenteric artery has been reported many times in women taking high-dose oestrogen-containing oral contraceptives. This severe and irreversible catastrophe is otherwise extremely rare in young women; its attribution to the oral contraceptive is consistent with oestrogen-induced thrombosis elsewhere. Thrombosis of the coeliac artery, inferior mesenteric artery, hepatic vein, and portal vein have also been reported.

Drugs can cause malabsorption in several ways, and this may lead to failure of concurrent medication. Several drugs inhibit pancreatic lipase, but only mefenamic acid has been reported to cause steatorrhoea. Neomycin and cholestyramine bind bile acids leading to malabsorption of fat and fat-soluble vitamins. Neomycin can also damage the enterocyte and reduce disaccharidase activity; at high doses partial villous atrophy may ensue. Colchicine and antimitotic drugs reduce cell turnover in all rapidly dividing tissues. They can cause partial villous atrophy during treatment, with restoration of normal architecture between courses.

Many drugs interfere with absorption or transport of vitamin B_{12} but only neomycin, cholestyramine, para-aminosalicylic acid, isoniazid, and metformin have been reported to cause anaemia by this mechanism. Anaemia from malabsorption of folic acid is common in long-continued anticonvulsant treatment, particularly with phenytoin. Folic acid malabsorption has been reported with the oral contraceptive but does not cause anaemia unless the diet is inadequate.

Large intestine

Broad-spectrum antibiotics cause diarrhoea in up to 30% of patients. With some antibiotics, such as ampicillin, the diarrhoea is attributed to an alteration in colonic flora. Diarrhoea after treatment with antibiotics usually improves when the drug is withdrawn, as does that due to other drugs such as magnesium containing antacids, iron salts, guanethidine, and methyldopa.

Diarrhoea that persists after withdrawal of antibiotics may signal the development of pseudomembranous colitis. This disease was rare before the introduction of antibiotics. It first became widely recognised as a common and potentially lethal complication of treatment with lincomycin and clindamycin in the early 1970s. Ampicillin, penicillin, sulphonamides, chloramphenicol, and tetracycline also cause this syndrome, although less often than lincomycin and clindamycin. The diagnosis should be considered in a patient who develops persistent diarrhoea after any antibiotic. The disease appears to be particularly common when antibiotics are used after colonic surgery; signs of septicaemia and pelvic sepsis may falsely suggest a breakdown of the anastomosis. Recognition of the disease can save the patient from unnecessary surgery or even death. Pseudomembranous colitis may start during treatment with an antibiotic, or up to two weeks after stopping it. It may present as mild diarrhoea, or as an acute fulminant colitis. Sigmoidoscopy is usually diagnostic and should never be omitted, however ill the patient. The characteristic appearance is of raised plaques with a specific histological appearance. Barium enema may show characteristic changes but is dangerous in fulminant cases. Pseudomembranous colitis is now thought to be caused by overgrowth of *Clostridium difficile* in the colon, with production of a necrotising toxin. Both the organism and the toxin can be detected in the stool and may distinguish pseudomembranous colitis from non-specific post-antibiotic diarrhoea. A recent controlled trial has shown that vancomycin, a non-absorbed antibiotic to which this organism is sensitive, rapidly improves the condition of the patient, reverses the sigmoidoscopic appearances, and eliminates the organism and its toxin from the stool.

Staphylococcal enterocolitis is an extremely rare but severe complication of broad-spectrum antibiotic treatment and may

be complicated by septicaemia, portal pyaemia, and metastatic abscesses.

The oral contraceptive can cause a severe form of ischaemic colitis that is otherwise extremely rare in young women. Unlike the infarction due to arterial thrombosis affecting the small intestine, this complication may resolve on withdrawal of the drug.

Laxative abuse is sometimes not suspected as a cause of chronic diarrhoea, because patients do not realise that their consumption of purgatives is excessive, or because they deliberately conceal it. Diarrhoea may be accompanied by weakness due to hypokalaemia, which if prolonged may lead to renal impairment. The anthraquinone laxatives (senna, cascara), phenolphthalein, and magnesium sulphate are commonly responsible and may be detected by appropriate tests. Barium enema appearances of a dilated featureless colon may suggest ulcerative colitis, but characteristically the changes are most pronounced in the ascending colon and transient pseudo-strictures may be seen on screening. Rectal histology may be normal or may show pigmentation (melanosis coli) with arthraquinone cathartics. In long-standing cases permanent damage to the myenteric plexus may cause persistent diarrhoea, which is best treated by faecal bulking agents.

Pancreas

Many drugs have been reported to cause acute pancreatitis on the basis that recurrent attacks cease when the offending drug is withdrawn. The evidence is strongest for corticosteroids. Focal pancreatitis is more common in necropsies of steroid-treated patients than matched controls. The incidence of acute pancreatitis in children with the nephrotic syndrome is reported to be greater in those treated with steroids. In patients treated with steroids the symptoms and signs of pancreatitis are often suppressed, and the prognosis is poor even if the drug can be withdrawn. Pancreatitis is often identified as the cause of death only at necropsy. Its incidence in steroid-treated patients is unrelated to the dose or duration of treatment.

Acute pancreatitis has been reported in women taking oral contraceptives, and responds to withdrawal of the drug. Thiazides, sulphonamides, clofibrate, methyldopa, and guanethidine are the other main drugs reported to cause acute pancreatitis. A careful drug history should always be taken in pancreatitis, and withdrawal of all drugs should be attempted.

Miscellaneous

Clofibrate and oestrogen-containing oral contraceptives increase the cholesterol saturation of bile. The incidence of gall stones is increased in patients treated with these drugs.

Sclerosing peritonitis is now a well-recognised complication of treatment with practolol. It has not been reported with any other beta-blocker apart from one dubious report with oxprenolol. Methysergide causes retroperitoneal fibrosis that may lead to narrowing of the colon or mesenteric vessels, but it is rarely used nowadays.

MATERIA NON MEDICA

Moroccan travels

Trouble was only to be expected when 13 of us climbed into two Land Rovers and prepared to camp our way round Morocco. Our start was delayed by a minor fault in one of the trucks, so we couldn't reach the proposed night stop. Offered the town rubbish dump as an alternative, the whole party, with an unanimity never again to be achieved, said "non," so we detoured to an oasis. It was spectacular and romantic, but it cost us several hours. A robbery in the "guarded" camp at Ouarzazate meant time wasted there. We arrived therefore too late at Zagora to see the famed sunset from its mountain. Never mind. A 5 o'clock start, a brisk walk, and we would see the sun rise. And, indeed, by 6·30 most of the party were seated on the summit, cameras clicking.

It was near dusk on the fourth day that we suddenly realised that the second vehicle was not behind us. We were now on pistes, deeply rutted dirt roads, so badly eroded by the recent rains that we had to turn back twice. The truck stopped, four of us climbed out, and suddenly it was gone, leaving us to face the inevitable interested crowd. A French-speaking Berber arrived and we were invited to take mint tea. It was a solemn ceremony in a small clean room, our quartet uneasily shifting on blankets while a lad made the tea in an antique silver pot, and then the old man poured it from pot to glass to pot again before handing it round. Conversation through the interpreter was stilted, but I will long remember the calm gravity of our host and the beauty of the women, with their wool embroidered gowns and elaborately made up faces. Just as we had exhausted all the usual topics of children and marriage the Land Rovers returned, one with a broken spring.

So we camped in the desert. There was no hardship: we had food, water, and wine in abundance. The acacia tree became our reading room—"Quiet please." We explored the oasis and found two ancient diesel engines, instructions uncompromisingly printed in English.

Our ascent of Mount Zagora had been prefaced by one of Nature's more imperious summonses. When my husband had rejoined me I had noticed a cylindrical white object encased in a plastic bag shimmering in the false dawn. "You're surely not taking that up the mountain?" I had asked.

"Certainly not," he had replied with dignity "I'm going to hide it." He "hid" it on top of the wall, and, not surprisingly, when we had got back it was gone. Our desert camp was complete, therefore, in all but one particular. Fortunately we never travel without our favourite reading material, and it is at times like these that one realises the full value of the *BMJ*.—ANNE SAVAGE (Tsolo, Transkei).

Jerusalem

Nearly twelve years after reunification under the Israelis, Jerusalem in the spring must be one of the most peaceful cities in the world. The Americans have not yet swamped it, the tourist industries are still rubbing the winter out of their bleary eyes, and the richly polyglot population is strolling the streets in the late afternoon and evening, ripe for the patient observer of multilingual conversation. The army is still much in evidence, and baggage checks and body searches are the order of the day, but the tension has gone, and the city is blossoming with renovation and development. The present walls of the Old City, largely dating from Ottoman rule under Sultan Suleiman in the sixteenth century, can rarely have seen such untroubled times. Under the aegis of the Jerusalem Foundation, they are being surrounded by landscaped parks and gardens—fulfilling the idea of a garden zone around the city first proposed by the British municipal authorities in 1918.

Despite "made in Taiwan" ethnic garments, Coca-Cola (which admittedly looks quite exotic when printed in Hebrew), Hong Kong plastic, fast food counters, and awful mass-produced wood carvings of New Testament scenes, the Old City manages, somehow, to preserve its timelessness. Extensive and idiomatic renovations, begun by the Israelis after the 1967 war, rub comfortable shoulders with the meticulously preserved buildings of the Christian and Armenian quarters, and even the completely rebuilt Jewish quarter, containing *yeshivot* (places of Talmudic study) and high-class apartments, will soon be an exciting area to visit.

But all is not idyllic. Jerusalem is in the middle of a property boom, and the cranes compete with the minarets in those romantic sunset snapshots from the Mount of Olives. Fortunately the Jerusalem town planners are strict, having taken a timely lesson from Tel-Aviv's urban chaos, and if you want to build a skyscraper, you will have to clad it in Jerusalem stone—which of itself does not create good buildings, and it is difficult to disguise a Hilton Hotel as anything but a Hilton Hotel—but if they are bad, they are less blatantly so, and will still obligingly glow with the suffused pink Impressionistic tint that envelops the whole city at dusk. It is little wonder that it has been fought over for 4000 years.—DAVID LEVY (medical student, London).