cause of apparent resistance to treatment. True resistance cannot be accepted without evidence that the patient was ingesting and absorbing a pharmaceutically satisfactory preparation of vitamin D. Such evidence has not been given for any of the numerous published cases of "resistance" to vitamin D therapy, but adequate assurance on all three counts would be provided by showing that the plasma concentration of 25-hydroxycalciferol had been raised to a level consistent with the dose.[4] I suggest that no further cases of supposed resistance to vitamin D therapy should be reported without this crucial piece of information.

A M PARFITT

Bone and Mineral Research Laboratory,
Henry Ford Hospital,
Detroit, Michigan

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Insurance companies' attitude to psychiatric illness

SIR,—Dr J T Hutchinson (19 March, p 775), says that insurance companies have made it difficult for patients to take out life insurance because they have a history of psychiatric treatment.

The insurance companies have a point in those cases in which the psychiatric condition is likely significantly to shorten life. Take, however, two patients, each of whom has suffered from some form of nervous anxiety. One of them has had psychotherapy as an outpatient and has thereby improved but the other has not and his condition has made less gainful progress. The insurance company in question will penalise the former but not the latter because they do not understand the situation. A third patient may have gone to his family doctor with similar symptoms and have been treated with tranquillisers. He will regard this as "medical" treatment and so will escape the net even if psychotherapy would have done him more good.

My personal view is that where the treatment has been confined to outpatient attendances for psychotherapy and perhaps for mild drug medication this should not be regarded as "psychiatric" treatment. I have on occasion said to a patient that he will have my backing in disregarding such circumstances while filling application forms. Alternatively I would write a certificate to this effect.

D T MACLAY

Dudley Area Health Authority,
Community Health Service,
Dudley, W Midlands

Heparin and pulmonary embolism

SIR,—I should like to report the case of a fit 57-year-old man admitted for routine cholecystectomy three months after an episode of acute biliary colic. As prophylaxis against deep vein thrombosis he was given 5000 U of heparin with his premedication of pethidine 100 mg, promethazine 25 mg, and atropine 0·4 mg, then 5000 U thrice daily postoperatively. The operation was cholecystectomy with exploration of the common bile duct and was uneventful. On the fourth postoperative day he developed massive bilateral pulmonary emboli, which produced a 60-70% loss on lung scan and arteriography and a pulmonary artery pressure of 85 mm Hg. Leg venograms and an inferior venocavagram showed no evidence of thrombosis. Despite immediate heparinisation by constant infusion of 10 000 U four times daily and later 15 000 U four times daily, keeping the thrombin time prolonged by a factor of 3-4, his condition remained critical for several days, necessitating oxygen therapy and transfer to the intensive care unit for close monitoring of his blood gases. He eventually made a good recovery after treatment with streptokinase.

I wonder whether the use of prophylactic heparin transformed what would have been a deep vein thrombosis into early pulmonary embolisation and whether embolism which does occur in the presence of heparin prophylaxis tends to be early rather than late. Conversely, did the prophylactic heparin prevent an otherwise fatal episode?

MARIAN RODEN

Academic Department of Surgery,
Royal Free Hospital,
London NW3

Misdiagnosis of urinary tract infection

SIR,—Professor A W Asscher (21 May, p 1332) is right to stress the importance of making an accurate initial diagnosis when a patient presents with symptoms suggesting a urinary tract infection. But I think he should have mentioned a very important cause for the persistence or reappearance of symptoms after treatment. This is that the symptoms may not be due to a pyogenic infection at all (and organisms cultured from a so-called midstream specimen of urine may be simply contaminants).

I frequently receive referral letters stating that a patient "has suffered from repeated urinary tract infections [*sic*] despite several courses of antibiotics, though the midstream specimens of urine are usually sterile." Each year I see on average two patients with advanced bladder tumour, one with genitourinary tuberculosis, and one with bladder stones whose referral has been delayed by many months *only* because they were subjected to inappropriate and repeated courses of antibiotic therapy and the possibility of the true diagnosis never considered.

ROGER HOLE

North Ormesby Hospital,
Middlesbrough, Cleveland

Rise in antibodies to human papova virus BK and clinical disease

SIR,—Since the isolation of the human papova virus BK seroepidemiological studies have shown that infections with this virus are very common and often occur in children.[1] An accompanying clinical disease has not yet been established, although a mild respiratory illness of children has been recorded at the time of appearance of antibodies against BK virus.[2]

We have investigated retrospectively serial serum samples from 77 patients which had been sent to our department for clinical virological examination. Three cases of primary BK virus infection, one in a child and two in adults, were found as indicated by the appearance of haemagglutination-inhibiting antibodies. The clinical findings and serological data on different days of the illness are shown in the table. Acute upper respiratory symptoms accompanied by neurological involvement occurred in all three patients.

Our findings suggest that primary infection by BK virus in older patients may be followed by acute inflammatory polyradiculoneuropathy (Guillain-Barré syndrome). It remains to be determined whether the BK virus is the aetiological factor. Seroconversions against other viruses (influenza A and B, adenovirus, respiratory syncytial virus, herpes simplex virus, echovirus 9, and measles virus) could not be detected. In one patient (the third in the table) a slight rise (<8 to 32) of complement-fixing antibodies against psittacosis was found.

A causal relationship between upper respiratory tract infections and inflammatory polyradiculoneuropathy has been extensively documented[3] for a number of viruses and our findings suggest that BK virus may also be implicated.

J V D NOORDAA
P WERTHEIM-VAN DILLEN

Virus Laboratory,
University of Amsterdam

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Clinical features and rise in BK virus antibody titre in three patients

Patient No	Clinical features	Age (years)	Rise in antibody titre
1	Upper respiratory tract infection, convulsions	3	<16 (day 3)→64 (day 13)
2	Respiratory infection, Guillain-Barré syndrome	45	<16 (day 9)→128 (day 24)
3	Respiratory infection, Guillain-Barré syndrome	33	<16 (day 13)→128 (day 28)

Further assessment of the normal cholecystogram

SIR,—Mr M H Gough (9 April, p 960) does not mention ultrasound in his excellent review of cholecystography. The value of ultrasound B scanning to assess the presence of calculi in the non-functioning gall bladder has been established.[1 2]

It has become our practice to scan patients with a normal cholecystogram when the clinical symptoms are strongly suggestive of gall stones. Out of a large group of patients we have found gall stones in three when the cholecystogram appeared normal. At laparotomy two of these patients had gall stones but the third has not been explored. We have also detected calculi in the common bile duct in a patient with transient jaundice when a follow-up intravenous cholangiogram appeared normal. This patient had calculi in the duct when operative cholangiography was performed. The presence of pancreatitis in association with cholecystitis may also be demonstrated.

The majority of calculi are rapidly detected by conventional techniques and we do not