must be relatively small compared with the considerable expenditure in the National Health Service on a variety of management information systems of unproved value.[24] We hope that outcome studies by the medical profession are developed further to become a regular part of postgraduate medical education and to make a contribution to the assessment of the quality of care.

We thank the consultants and coroners who provided us with the information which made this study possible. We particularly thank Professor P F Jones for invaluable help during the drafting of the paper and Dr W Kirkup for statistical advice.

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(Accepted 25 September 1987)

SHORT REPORTS

Cryptorchidism in Scotland

Using data from the Hospital In-Patient Enquiry for England and Wales for 1962-81, Chilvers *et al* showed that the number of boys discharged from hospitals in England and Wales each year with a diagnosis of cryptorchidism increased by a factor of 2·3.[1] As part of a study into the validity of using the Scottish morbidity record system[2] to monitor trends in the incidence of a disease we studied the trends in hospital discharges and deaths of patients with cryptorchidism in Scotland from 1961 to 1985.

Methods and results

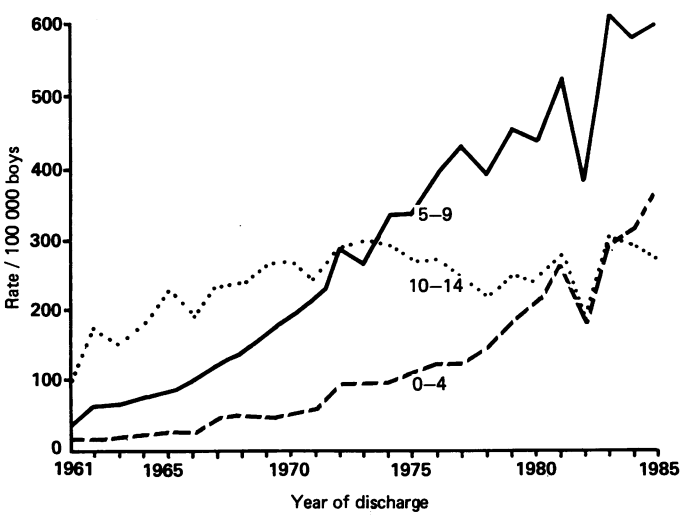
The data were obtained from the Scottish Morbidity Register (hospital inpatients record summary sheet; SMR1) for 1961-85 and the mid-year population estimates for 1961-85 of the Registrar General (Scotland). Annual data on cryptorchidism by year of discharge and date of birth were obtained from the Information and Statistics Division of the Scottish Health Services Common Services Agency.

The annual number of hospital deaths and discharges of boys aged 0-14 years diagnosed as having cryptorchidism increased from 326 in 1961 to 2084 in 1985. The rate per 100 000 boys increased 26-fold in those aged 0-4, with the greatest change in the last 10 years; 16-fold in those aged 5-9, with an almost linear increase over the 25 years of the survey; and threefold in those aged 10-14, the rate remaining relatively stable from 1972 (figure).

Comment

We found substantial increases in discharges of boys with cryptorchidism in different age groups during the 25 years 1961-85. Successive birth cohorts also showed similar increases for orchidopexy (unpublished observations). From the early 1970s the data for all age groups have shown a greater increase in Scotland than in England and Wales, the greatest difference being in the age group 0-4 years. Since 1980 SMR1 has increasingly included day cases, whereas the Hospital In-Patient Enquiry has not; removal of these day cases from the analysis, however, would not eliminate these differences. Possible errors in the information contained in SMR1 both over time and between hospitals are too small to explain changes of this magnitude (unpublished observations). It seems, therefore, that surgeons operate on younger boys in Scotland than in England and Wales.

Between the postneonatal period and puberty the diagnosis of cryptorchidism may be complicated by the cremasteric reflex and partial absorption of the processus vaginalis.[3,4] We studied the prevalence of cryptorchidism in neonates at examination on discharge from hospital and at examinations at school entry and school leaving age (unpublished observations; routinely available Scottish data). The rate of diagnosis in neonates on discharge from hospital remained virtually constant at 14/1000 live male births after reliable recording began in 1976; the rate on examination at school entry and school leaving age declined after peaking in 1973 (47·8/10 000) and 1974 (8·5/10 000), respectively. The cumulative rate of orchidopexy to age 14 in the cohort studied was predicted to be 3·8% compared with the rate of cryptorchidism of 1·4% recorded in the neonatal discharge records. A possible hypothesis to explain this apparent discrepancy is that surgeons may be operating inappropriately on children with retractile testes or that cryptorchidism is acquired after birth.



Discharge rates for cryptorchidism by age group 1961-85.

Surveillance of this trend is required. An increase in the number of orchidopexies without a true increase in the incidence of cryptorchidism should be discouraged. If the incidence of cryptorchidism has truly increased the reasons for this change need to be ascertained. We agree with Jones that babies need a careful postnatal examination and that the finding of a testicle outside the scrotum should lead to a surgical opinion before the first birthday.[5]

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(Accepted 19 August 1987)

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Fatal renal and hepatic toxicity after treatment with diltiazem

The calcium antagonist diltiazem has been recommended as the first choice for patients with ischaemic heart disease.[1] We report on a patient taking diltiazem who developed renal failure and severe liver damage shortly after starting treatment.

Case report

A 72 year old man (an ex-smoker) presented with three days of chest pain after a 17 year history during which both oesophagitis and ischaemic heart disease had been confirmed. Previous treatment had included nifedipine and isosorbide dinitrate and at least two courses of ranitidine. There had been no documented myocardial infarction but he had been taking frusemide 40 mg and amiloride 5 mg per day since an episode of pulmonary oedema one year before. He took minimal alcohol and had no history of allergy.

On examination he was not distressed. His blood pressure was 130/90 mm Hg, his pulse was 80 beats/minute and regular, and there was no evidence of cardiac failure or cardiomegaly. His peripheral pulses were all palpable with left femoral bruit, and a pansystolic murmur was heard at the apex, radiating to the axilla and the base. Respiratory and nervous systems were normal. A smooth, non-tender liver edge was palpable. Rectal examination showed melaena. The results of investigations performed on admission were haemoglobin 100 g/l, mean cell volume 76 fl, potassium 4·0 mmol/l, bilirubin 13 μmol/l (normal <17 μmol/l), albumin 42 g/l; the results for urea and creatinine concentrations and alanine aminotransferase activity are shown in the figure. A chest x ray picture showed cardiomegaly with some upper lobe blood diversion, and results of electrocardiography were the same as in previous investigations.

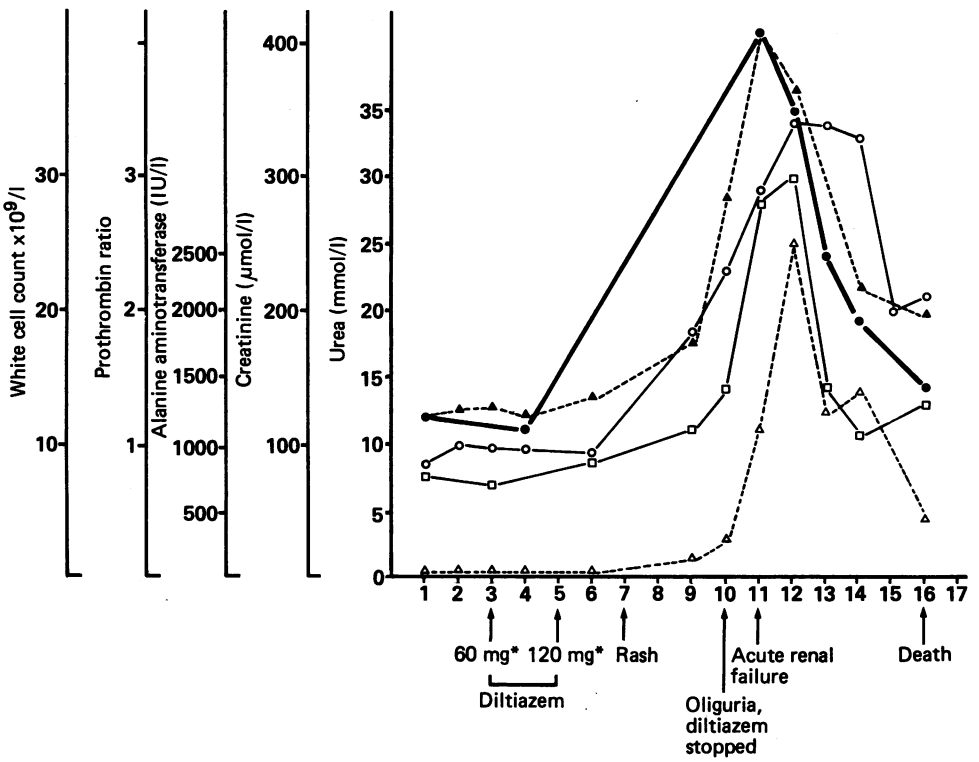
He was treated with bed rest, continued diuretics, isosorbide dinitrate 30 mg/day, nifedipine 15 mg/day, and ranitidine 300 mg/day. The melaena did not recur and was attributed to oesophagitis; he was never hypotensive. After two days of continued pain diltiazem 180 mg/day was substituted for nifedipine and isosorbide and was increased to 360 mg/day after two more days. Three days after starting diltiazem a mild urticarial rash developed on his trunk and arms. After two more days he became oliguric (200 ml/day); diltiazem was stopped and the next day he was in established acute renal failure with acidosis (pH 7·18) and hyperkalaemia (potassium 7·0 mmol/l). He was treated with intravenous calcium, insulin, and dextrose; and ultrasonography showed no postrenal obstruction. Jaundice was noticed with biochemical and coagulation evidence of severe liver damage (figure). During the next five days renal and liver function and urine output improved but he then developed intractable and fatal pulmonary oedema with cardiogenic shock. Necropsy showed pulmonary oedema, coronary calcification, no gross liver abnormality, and oedematous kidneys. No histological examination was performed.

Comment

Renal failure secondary to haemorrhage was unlikely in this patient as he was not hypotensive before its onset. The coincidence of renal and hepatic dysfunction and the urticarial rash makes it much more likely that these conditions were caused by a drug, although he had received isosorbide dinitrate, nifedipine, and ranitidine previously without ill effects.

A search of reports published in English and communication with the Committee on Safety of Medicines and the manufacturers showed no reports of severe liver damage and only one of (non-fatal) acute renal failure associated with diltiazem, in which serum urea and creatinine concentrations became abnormal three days after starting the drug but returned to normal after stopping it.[2] During studies of 193 patients given diltiazem over four years six patients developed a mild rise in transaminase activity, which returned to normal on withdrawal.[3,4] Ranitidine does not interfere with the cytochrome p450 system, which is the main metabolic pathway of diltiazem.

We conclude therefore that this patient developed sudden acute renal failure and severe liver damage seven days after starting treatment with diltiazem as an adverse reaction to the drug. If a patient develops a rash after



Results of biochemical investigations (urea concentration ●——●, creatinine concentration ▲----▲, prothrombin ratio ○——○, white cell count □——□, alanine aminotransferase activity △----△).
*Diltiazem given three times daily.