evidence that there is an increasing demand for this technique, especially in the field of selective breeding.⁸⁻⁹ The uses to which amniocentesis may be put are wider than the medical purposes for which it was originally devised.¹⁰ In this case the decision taken was one where the general practitioner's suggestions carried no more weight than the woman's own request. Yet medically he had a much deeper knowledge of her circumstances and whether or not the pregnancy was desirable or not.

Although women are increasingly applying pressure for termination for social rather than medical reasons, the final decision for abortion is a medical one alone. In the last decade a more liberal¹¹ attitude has been apparent, and perhaps this patient's request was just ahead of its time. Nevertheless, it does raise the issue of abortion on demand with all its ethical and social implications, and the legal rights of all concerned.¹²

SHORT REPORTS

Intestinal pseudo-obstruction in alcohol abuse: report of two cases

Acute intestinal pseudo-obstruction presents with the clinical features of bowel obstruction without any demonstrable lesion within the bowel lumen.¹ It is of unknown aetiology, but we report its occurrence in two patients with proved alcoholic liver disease and a recent history of alcohol abuse.

Case reports

(1) A 45-year-old man presented with 36 hours of abdominal distension, vomiting, and no bowel action for 72 hours. He had alcoholic hepatitis on liver biopsy one month earlier, when he had been dried out. He had subsequently relapsed to a level of one bottle of spirit daily. He had signs of intestinal obstruction with a distended tympanitic abdomen and accentuated bowel sounds. There was no evidence of cardiorespiratory disease or a generalised systemic disease. Radiographs of the abdomen were consistent with colonic obstruction. Haemoglobin was 11.5 g/dl, white cell count $7.0 \times 10^9/l$ (7000/mm³). Plasma electrolytes were chloride 80 mmol(mEq)/l, bicarbonate 13 mmol(mEq)/l, potassium 3.2 mmol(mEq)/l, sodium 123 mmol(mEq)/l, urea 7.0 mmol/l (42.0 mg/100 ml), creatinine 175 μ mol/l (1.92 mg/100 ml), and glucose 7.1 mmol/l (128 mg/100 ml). Liver function tests showed serum glutamate oxaloacetate transaminase 36 IU/l, bilirubin 15 μ mol/l (0.87 μ g/100 ml), alkaline phosphatase 96 IU/l, and gamma-glutamyl transpeptidase 435 IU/l. At laparotomy there was dilatation of the transverse colon but no demonstrable site of obstruction. A right defunctioning colostomy was performed. A barium enema later showed no abnormality. The colostomy was therefore closed, and he has made a good recovery off alcohol.

(2) A 79-year-old man presented with increasing abdominal distension, nausea, and no bowel action for 72 hours. Signs of chronic liver disease were present but the main signs were of acute obstruction confirmed by abdominal radiographs. Haemoglobin was 15.8 g/dl, WCC $9.6 \times 10^9/l$ (9600/mm³). Electrolytes were chloride 92 mmol(mEq)/l, TCO₂ 26 mmol(mEq)/l, potassium 3.9 mmol(mEq)/l, sodium 136 mmol(mEq)/l, urea 10.5 mmol/l (63 mg/100 ml), and creatinine 175 μ mol/l (1.92 mg). Liver function tests showed serum aspartate transaminase (AST) 52 IU/l, bilirubin 38 μ mol/l (2.2 μ g/100 ml). At laparotomy the main finding, apart from dilated bowel, was a nodular liver due to macronodular cirrhosis. It transpired that the man was drinking four pints (2.4 l) of beer and up to a further three measures of spirit a day. Postoperatively he developed increasing hepatic failure and died one month later.

Comment

Alcohol has numerous metabolic effects which may produce the syndrome of acute intestinal pseudo-obstruction. An increase in alcohol intake coupled with prolonged abstinence from food increases ketogenesis and a metabolic acidosis may occur, as in case I. Consequent disturbance of the acid-base equilibrium may inhibit gut motility. A similar mechanism may be implicated when pseudo-obstruction occurs in its reported associations of renal failure, pancreatitis, pneumonia, and congestive cardiac failure.¹ Alcohol may also act on gut hormones. Secretin and glucagon inhibit gastro-

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intestinal contractions.² Alcohol ingestion causes a rapid increase in plasma secretin, possibly by a direct action within the duodenal lumen.³ Chronic alcohol ingestion by producing hypoglycaemia induces a secondary hyperglucagonaemia. This occurs in hepatic cirrhosis of any aetiology.⁴ The resultant increases in these hormones may combine to inhibit gut motility and hence pseudo-obstruction. This syndrome may be drug-induced, and has been reported with tricyclic antidepressants and phenothiazines.⁵ In reporting these cases Milner emphasised that alcohol may worsen the obstructive features by potentiating the parasympatholytic action of the drugs. Nevertheless, in neither of our cases could drugs be implicated. The association of alcohol abuse and pseudo-obstruction and the possible mechanisms involved need further study.

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Kent and Canterbury Hospital, Canterbury CT1 3NG

J KARANI, BSC, MB, senior house officer in medicine
D VEALE, MB, senior house officer in medicine
M O RAKE, BSC, FRCP, consultant physician

Effect of depot medroxyprogesterone acetate on vaginal bleeding in the puerperium

If a woman becomes pregnant within two months of rubella vaccination there is a danger of fetal infection. Though the risk is probably small¹ a reliable contraceptive should be used. Depot medroxyprogesterone acetate (DMPA) has been recommended for this purpose in the puerperium.² Unfortunately, injectable progestogens may disrupt menstrual patterns and produce either irregular bleeding or amenorrhoea.³ This survey was undertaken to determine whether these side effects outweigh the convenience of giving DMPA to women requiring rubella vaccination in the puerperium.

Patients, methods, and results

Sixty patients who had puerperal rubella vaccination elected, after full discussion, to have a single injection of 150 mg DMPA. They were asked to record daily the amount of vaginal bleeding, to compare their blood loss with that after previous babies (multiparas) or menses before pregnancy (primi-

paras), and to attend a clinic or return the questionnaire after three months. Fifty-two patients (87%) (15 primiparas and 37 multiparas) completed questionnaires. They were matched with a control group of 52 vaccinated patients who received no hormonal treatment. The mean ($\pm$ SD) number of days of postpartum bleeding was 58.5 ± 18.5 in the treated patients and 20.8 ± 11.1 in the controls. This difference was significant ($P < 0.001$). Patients still bleeding at three months were treated with low-dose oestrogen/progestogen pills, which invariably stopped bleeding. Only one patient was so treated before three months. Thirty-three treated patients, but only two controls, had continuous bleeding for three months. In no case was the loss heavy and no patients developed anaemia (haemoglobin less than 10.0 g/dl). Six patients (five treated and one control) had a curettage. None had retained placental tissue. None of the 15 treated patients who had cyclical bleeding considered the loss heavy, compared with six controls. Three non-lactating patients in each group had amenorrhoea. Patients with continuous bleeding for the three months found it inconvenient and worrying. It usually made intercourse unacceptable.

Comment

Most patients given DMPA postpartum seem to have daily vaginal bleeding for at least three months, though treatment with oral contraceptives will correct this. Such intervention was required by 61% of patients in one series.² The low incidence of amenorrhoea in this series was probably related to the short period of observation.³ The menstrual disturbances associated with treatment with DMPA have to be set against its effectiveness as a contraceptive, its lack of inhibitory effect on lactation, and its freedom from the risk of inducing thromboembolism.⁴ Some patients may be prepared to tolerate the inconvenience as a price for safe, acceptable contraception.⁵

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Maternity Hospital, Wexford, Ireland

H M MURPHY, FRCSI, MRCOG, consultant obstetrician and gynaecologist

Clinically significant interaction between digoxin and quinidine

Previous reports^{1,2} have drawn attention to a rise in plasma digoxin concentration (PDC) when quinidine therapy was added to the medication and suggested that digoxin toxicity might ensue. In a retrospective study Leahey *et al*³ noted that such a rise in digoxin concentration might be responsible for an increase in the incidence of adverse reactions to digoxin. We report two cases in which quinidine given to digitalised patients caused digoxin toxicity. Blood for measuring digoxin and quinidine was drawn at least six hours after the last dose of the drugs. Digoxin was measured by radioimmunoassay and quinidine by a spectrofluorometric method.

Case reports

Case 1—A 69-year-old man with a history of ischaemic heart disease and myxoedema was admitted in left ventricular failure. He had been taking daily frusemide 160 mg, digoxin 0.125 mg, potassium supplements, and thyroxine 0.2 mg. The PDC was 1.1 nmol/l . His electrocardiogram (ECG) showed controlled atrial flutter-fibrillation. He responded to an increase in frusemide to 240 mg daily. Previous treatment was continued and on discharge the PDC was 1.0 nmol/l . Three weeks later he again went into left ventricular failure and his ECG showed fast atrial flutter-fibrillation. The PDC was again 1.0 nmol/l . Quinidine sulphate 250 mg 12-hourly was added to his regimen to control his dysrhythmia. He improved and was discharged, but was readmitted two weeks later with a five-day history of severe nausea and vomiting. The ECG showed atrial flutter-fibrillation with multiple ventricular premature beats. Plasma concentrations of digoxin and quinidine were 2.4 nmol/l and $10 \text{ } \mu\text{mol/l}$ respectively. Both drugs were withdrawn and his symptoms resolved. Subsequently he was maintained on digoxin 0.125 mg daily and the PDC after one week was 0.8 nmol/l .

Case 2—A 74-year-old woman had been admitted with an exacerbation of chronic obstructive airways disease and cor pulmonale. Her ECG showed atrial tachycardia with variable block. She was given prednisolone 3 mg, digoxin 0.125 mg, and frusemide 240 mg daily with potassium supplements. She developed large-bowel obstruction. Laparotomy revealed perforation of a sigmoid diverticulum, and a defunctioning transverse colostomy was performed. Postoperatively it was difficult to control her atrial tachycardia with digoxin alone although the PDC was 1.9 nmol/l on consecutive days. Quinidine sulphate 250 mg eight-hourly was added and her ECG reverted to sinus rhythm, but after five days multiple ventricular extrasystoles were noted. The plasma concentration of digoxin was 3.7 nmol/l and of quinidine $13.0 \text{ } \mu\text{mol/l}$. Digoxin was withdrawn and the extrasystoles resolved. She was eventually stabilised on digoxin 0.0625 mg daily and quinidine sulphate 250 mg 12-hourly, resulting in plasma drug concentrations of 1.2 nmol/l and $12.3 \text{ } \mu\text{mol/l}$ respectively.

Comment

In both cases the PDC rose sharply when quinidine was started. This caused symptoms and signs of digoxin toxicity which abated when digoxin was stopped. The conventional doses of quinidine used resulted in plasma drug concentrations within normal limits. In neither case could the rise in PDC be attributed to a deterioration in renal function, nor could other factors, such as compliance, be implicated. A number of changes in the pharmacokinetics of digoxin, including a reduction in its volume of distribution in the presence of quinidine, have recently been noted,⁴ and it has been shown that the most important effect of quinidine is to produce a two-fold decrease in the clearance of digoxin.⁵ In our cases routine measurement of the PDC brought this drug interaction to light, the symptoms and signs of digoxin toxicity correlating well with the plasma drug concentration. This suggests that measurement of the PDC may be particularly important when digoxin and quinidine are combined.

We thank Professor A F Lant for permission to publish details of patients under his care.

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Cardiac Drug Section, Poisons Unit, Guy's Hospital, London SE1

DAVID W HOLT, BSC, PHD, principal biochemist

ANGELA M HAYLER, BSC, biochemist

Department of Therapeutics, Westminster Hospital Medical School, St Stephen's Hospital, London SW10

MICHAEL E EDMONDS, BSC, MRCP, registrar

RICHARD FU ASHFORD, MA, MRCP, senior registrar

Death after complete detachment of a Bjork-Shiley prosthetic mitral valve

Dehiscence of prosthetic heart valves is a recognised complication of valve replacement,^{1,2} but total detachment has not been described. We report a fatal case of complete detachment of a Bjork-Shiley prosthesis six months after mitral valve replacement.

Case report

A 43-year-old schoolteacher underwent closed valvotomy for tight mitral stenosis in 1971. Because of increasing exertional dyspnoea, cardiac catheterisation was performed in October 1976 and showed severe mixed mitral valve disease. At operation a sclerotic, stenotic, and incompetent mitral valve was replaced with a Bjork-Shiley 31-mm prosthesis inserted with continuous 2/0 Polydek sutures. Her recovery was uneventful, routine bacterial cultures were sterile, and she was discharged 20 days after operation.