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 7.0×10⁹/l (7000/mm³). Plasma electrolytes were chloride 80 mmol(mEq)/l, 7.6 × 10^{-/1} (7000/mm⁻). 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 (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 $9.6 \times 10^9/1$ (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 µmol/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 abstention 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 pseudoobstruction 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-

References

- ¹ Rich, E E, and Wilson, C H (editors) The Cambridge History of Europe IV, p 68. Cambridge, Cambridge University Press, 1967.
- ² Hayden, B, Archaeology, 1972, 4, 205.
 ³ Cameron, A, Classical Review, 1932, 46, 105.
- ⁴ Williamson, N, Population Reference Bureau Incorporated, 1978, 33, 1.
- ⁵ Boxer, C R, Mary and Misogyny, p 97. London, Duckworth, 1975.
 ⁶ Freeman, M R, Ver handlungen des XXXVIII, International American
- Congress, August 18, 1968, 12 bis Band II, Stuttgart-Munchen Rascovsky, A, et al, International Journal of Psychoanalysis, 1972, 53, 271. Amitai, E, Journal of Medical Ethics, 1976, 2, 8.
- Goodner, D A, Clinical Obstetrics and Gynaecology, 1976, 19, 973.
- ¹⁰ Hinman, L F, Clinical Obstetrics and Gynaecology, 1976, 19, 965.
- ¹¹ Dove, G, British Medical Journal, 1969, 3, 297.
- ¹² Quest, B, Thesis for LLB Degree, Leeds University, 1976.

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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 actiology.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.

- ² Rayford, P L, New England Journal of Medicine, 1976, 294, 1093.
- ³ Straus, E, Urbach, H J, and Yalow, R S, New England Journal of Medicine, 1975, 293, 1031.
- ⁴ Unger, R H, and Orci, L, *Physiological Reviews*, 1976, **56**, 778. ⁵ Milner, G, *Medical Journal of Australia*, 1969, **2**, 153.

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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-

¹ Lancet, 1979, 1, 535.