In Crohn's disease the mean plasma propranolol levels were significantly raised at all sampling times apart from the first. The area under the curve was also significantly greater than that of the controls ($P=0.01-0.001$). The values in Crohn's disease showed a much greater spread than those of the controls or the patients with coeliac disease. They fell into two groups: high values and those nearer control values. The high values seemed to relate to a rise in the erythrocyte sedimentation rate. Except in one patient who had a slightly lowered plasma albumin concentration, no abnormalities in liver function were found. The relevance of these findings to multiple dosing as used clinically is to be investigated.

We thank Professor O L Wade for his support and encouragement.

¹ Parsons, R L, *et al*, *Gut*, 1976, **17**, 139.

² Parsons, R L, and Paddock, G M, *Journal of Antimicrobial Chemotherapy*, 1975, **1**, 59.

³ Shand, D G, Nuckolls, E M, and Oates, J A, *Clinical Pharmacology and Therapeutics*, 1970, **11**, 112.

University of Birmingham, Birmingham B15 2TH

R E SCHNEIDER, MD, FRCPD, senior research fellow, department of medicine

JANET BABB, BA, research technician, department of therapeutics and clinical pharmacology

HILARY BISHOP, research technician, department of medicine

M MITCHARD, PHD, MSc, senior lecturer, department of therapeutics and clinical pharmacology

Queen Elizabeth Hospital, Birmingham B15 2TH

A M HOARE, MB, MRCP, senior registrar

C F HAWKINS, MD, FRCP, consultant physician

Comment

Cardiac arrhythmias have been implicated as the major cause of death in patients with myocardial infarction; ventricular fibrillation has been present in 90% of these patients. So far intravenous lignocaine has been the most widely used antiarrhythmic agent for a variety of ventricular arrhythmias.

Our report illustrates a case of recurrent ventricular fibrillation after myocardial infarction that was resistant to intravenous lignocaine given at a rate of 2 mg/min over many days but which responded to oral disopyramide.

Disopyramide is one of the recent antiarrhythmic agents found to be effective in various arrhythmias, both atrial and ventricular.²⁻⁴ It is said to possess both parasympatholytic and local anaesthetic properties, being as active as lignocaine in this latter respect.⁵ The drug's apparent effectiveness is probably due to its ability to prolong the refractory period of the atria and the ventricles.²⁻⁴ Supraventricular or ventricular tachycardias are thought to be due to re-entry mechanisms initiated by atrial or ventricular premature beats. Suppression of these premature beats by disopyramide in addition to prolongation of the atrial or ventricular effective refractory period should be a valuable effect for the termination of such tachycardias. Oral disopyramide may therefore be a useful alternative to intravenous lignocaine in preventing life-threatening ventricular tachyarrhythmias after myocardial infarction.

¹ Jennings, G, *et al*, *Lancet*, 1976, **1**, 51.

² Katz, M J, *et al*, *Clinical Therapeutic Research*, 1963, **5**, 343.

³ Desruelles, J, *et al*, *Therapie*, 1967, **22**, 937.

⁴ Vismara, L A, Mason, D T, and Amsterdam, E A, *Clinical Pharmacology and Therapeutics*, 1974, **16**, 330.

⁵ Baines, M W, *et al*, *Journal of International Medical Research*, 1976, **4**, suppl No 1, 5.

Mount Vernon Hospital, Northwood, Middlesex HA6 2RN

T D I M S DE LANEROLLE, MD, MRCP, registrar in general medicine

V EDMUNDS, MD, FRCP, consultant physician

A H WILCOX, MB, BCHIR, senior house officer in general medicine

Disopyramide in a case of recurrent ventricular fibrillation

Disopyramide (4-di-isopropylamino-2-phenyl-2-(2-pyridyl) butyramide phosphate) given prophylactically after myocardial infarction has been known to cause a significant reduction in ventricular arrhythmias.¹

We describe a patient with recurrent ventricular fibrillation after myocardial infarction that was resistant to intravenous lignocaine but responded to oral disopyramide.

Case report

A 65-year-old welfare officer was admitted with a six-hour history of central chest pain. For a week before admission he had had twinges of pain in the same area on exercise. There was no other significant history. He was a non-smoker. His mother died of coronary heart disease at the age of 62.

On admission he looked fit and was in sinus rhythm with a pulse of 64/min and a blood pressure of 130/90 mm Hg. He had no evidence of heart failure. An electrocardiogram showed an antero-septal and lateral infarction. The chest x-ray picture showed an enlarged cardiac shadow with clear lung fields. Cardiac enzymes—aspartate transaminase and hydroxybutyrate dehydrogenase—were 230 IU/l (normal 9-35) and 1660 IU/l (normal 50-325) respectively 24 hours after admission. After three days in bed he was gradually mobilised. On the 14th day, when he was fully mobilised, he suddenly collapsed. He was pulseless, with an unrecordable blood pressure; he was in ventricular fibrillation. He was restored to sinus rhythm with a DC shock of 200 joules, followed by a bolus of 100 mg lignocaine intravenously and an infusion of lignocaine at 3 mg/min, reduced to 2 mg/min four hours later. In spite of the intravenous lignocaine and boluses of intravenous phenytoin and procainamide, further cardiac arrests occurred at 12-24 hour intervals, with ventricular fibrillation shown on the monitor. By the 28th day his condition had deteriorated as the periods between arrests had decreased to one to two hours. His lungs remained clear on 80 mg of frusemide daily.

After 29 incidents requiring DC conversion, he was started on disopyramide, 200 mg four times a day, and lignocaine was tailed off. His last arrest (30th) was an hour after starting the drug.

He made an uneventful recovery and was discharged three months after admission. During his stay he was on prophylactic subcutaneous heparin. At one stage he developed acute urinary retention, a known complication of disopyramide. He remained on the drug, 100 mg four times a day, for 12 weeks after discharge.

Cervical pregnancy managed by local excision

Cervical pregnancy is rare, the incidence recorded by Dees¹ being 1 in 18 000 pregnancies. It is often associated with considerable vaginal bleeding which usually necessitates emergency hysterectomy. In the case described below treatment was by local excision.

Case report

A 26-year-old married primigravida with 12 weeks amenorrhoea was admitted as an emergency, complaining of painless vaginal bleeding for about 24 hours. Three weeks before admission she had had a similar, but smaller painless vaginal haemorrhage, which had lasted 18 hours but for which she had not sought medical advice. Symptoms suggestive of early pregnancy had been present but had diminished during the two weeks before admission.

Her general condition was good; abdominal examination showed no obvious abnormality apart from very slight suprapubic tenderness. Vaginal examination confirmed that she was bleeding briskly; findings on digital examination suggested that there were products of conception within the cervical canal, and it was also noted that the uterus was only slightly enlarged, with no masses in the appendages. Speculum examination confirmed the presence of a mass resembling products of conception in the cervical os, but attempted removal with sponge-holding forceps simply caused further bleeding. The patient was therefore transferred immediately to the operating theatre.

At operation, an intracervical mass, about 4 cm in diameter was found; it was visible through the partly dilated external os, and was adherent to the thinned-out cervix. There was heavy bleeding from a punctum on this mass.

After clamping each side of the cervix to occlude the lateral cervical blood vessels, the intact internal os was dilated using Hegar's dilators and the uterine cavity curetted. The mass was carefully dissected free from the

cervix, which, being friable, was torn in several places. Finally, two lateral cervical sutures were inserted and the clamps removed, the torn cervix was repaired with catgut sutures and the vagina firmly packed. One litre of blood was transfused during the procedure.

Postoperatively, the patient made good progress and she was finally discharged from hospital on the seventh postoperative day. She was very well when seen six weeks later and the cervix had completely healed. Histology of the curettings showed endometrium with an appreciable Arias-Stella reaction and the presence of trophoblastic tissue was confirmed in the cervical mass.

Discussion

This case history conforms to the strict criteria proposed by Duckman² for the diagnosis of cervical pregnancy, in that there was—“(1) a dilated, thin-walled cervical canal containing histological evidence of products of conception; (2) a patulous external os; (3) a small, firm uterine body with a normal sized internal os, resting on top of a dilated cervix.”

The main difficulty in the management of a cervical pregnancy is the decision when to perform a hysterectomy, both Mortimer³ and Dodek⁴ suggesting that the procedure is indicated when the gestational age is beyond eight weeks. Nevertheless, in describing a similar case to that presented above, Flanagan⁵ proposed that local excision was feasible owing to the decidual necrosis and thrombosis of the end vessels in the placenta after the previous death of the pregnancy, an event which had undoubtedly occurred.

There is insufficient evidence on the likelihood of complications developing in a subsequent pregnancy, and the risk of a further cervical pregnancy is unknown. Cervical incompetence should not occur, since by definition the internal os has not been affected.

¹ Dees, H C, *Southern Medical Journal*, 1966, **59**, 900.

² Duckman, S, *American Journal of Obstetrics and Gynecology*, 1951, **62**, 1381.

³ Mortimer, C, and Aitken, D, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1968, **75**, 741.

⁴ Dodek, S M, *Southern Medical Journal*, 1965, **58**, 167.

⁵ Flanagan, J F, and Walsh, C R, *Obstetrics and Gynecology*, 1954, **4**, 511.

Department of Obstetrics and Gynaecology, University Hospital of South Manchester, Manchester M20 8LR

M J WHITTLE, MB, CHB, clinical tutor

Inflammatory bowel disease in West Indians

Inflammatory bowel disease has not been reported in West Indians. We wish to report here five cases of “colitis” in West Indian patients presenting in the past 18 months. Failure to consider this condition because of their race led to a delay in diagnosis in two of our patients.

Patients and comment

Details of the patients are given in the table. Rectal biopsies from the three patients with Crohn's disease showed transmural inflammation and

Details of patients with inflammatory bowel disease

Patient	Sex	Birthplace	Age at onset of symptoms	Years in UK before symptoms	Symptoms	Findings of barium enema	Diagnosis
1	M	UK	10	10	Diarrhoea. Abdominal pain. Weight loss	Marked ulceration of distal transverse and descending colon	Crohn's colitis
2	F	UK	10	10	Perineal ulceration	Fissures and ulcers in rectum. Irregular mucosal pattern in sigmoid colon	Crohn's colitis
3	M	Jamaica	28	12	“Bloody” diarrhoea. Weight loss. Fever	Gross mucosal destruction with ulcers and pseudopolyps throughout colon	Crohn's colitis
4	F	Jamaica	42	6	“Bloody” diarrhoea	Fine mucosal ulceration in the rectum and sigmoid colon	Ulcerative colitis
5	M	Jamaica	34	7	“Bloody” diarrhoea	Normal	Ulcerative colitis

granulomata formation. In patient No 3 this was confirmed on examination of a subsequent colectomy specimen. Patient No 2 displays many interesting features other than the fact that she is a West Indian child and these are reported elsewhere.¹

The rectal biopsies of the two remaining patients showed intense cellular infiltration of the lamina propria and they are thought to be suffering from ulcerative colitis. Because one of them (No 5) had normal appearances on barium enema examination a colonoscopy was performed. A mild total colitis was noted and biopsies from various parts of the colon displayed depletion of goblet cells and distortion of the crypts in addition to inflammatory cells.

All five patients showed normal features on barium meal follow-through examination.

Discussion

All five patients described here are West Indians of African stock. Inflammatory bowel disease has not been reported in these people, which is perhaps surprising as both ulcerative colitis and Crohn's disease are seen in the American negro, although admittedly less commonly than in corresponding white populations.² The American negro has of course been in the United States as long a time as his Caucasian countryman, whereas the West Indian in Britain is a relative newcomer. There are a few reports of inflammatory bowel disease in the South African Bantu.^{3,4} The problem of reaching such a diagnosis in areas of high parasitic infestation has been well documented by Sobel and Schamroth.⁵

As the incidence of Crohn's disease in Britain is increasing,⁵ probably we will see more of this disease in our West Indian population. It is of particular interest that the three patients with undoubted Crohn's disease are young, two being children who were born in the UK and the third a man in his twenties who had lived in Britain for 12 years before the onset of symptoms. Thus there has been adequate time for exposure to environmental factors, one of which may be a slow virus.

Failure to consider inflammatory bowel disease led to a delay in diagnosis in two of the patients reported here. Diagnosis is not usually a problem in ulcerative colitis, but it may be in Crohn's disease, which may present in various ways and sometimes without symptoms referable to the gastrointestinal tract, as shown by one of our patients (No 2). Hence, clearly the doctor must consider this disease when he is presented with patients with such symptoms as unexplained fever, arthralgia, or loss of weight, even though they are of West Indian extraction.

We thank Mr G C Brentnall and Dr A S E Fowle, who each referred a patient to us, and Mr H E Lockhart-Mummery for allowing us to report on one of his patients. We also acknowledge the financial support of the Joint Research Board of St Bartholomew's Hospital, London.

¹ Wallace, S, and Walker-Smith, J A, *Acta Paediatrica Scandinavica*, in press.

² Acheson, E D, *Gut*, 1960, **1**, 291.

³ Sobel, J D, and Schamroth, L, *Gut*, 1970, **11**, 760.

⁴ Giraud, R M A, Luke, I, and Schamman, S, *South African Medical Journal*, 1969, **43**, 610.

⁵ Miller, D S, Keighley, A C, and Langman, M J S, *Lancet*, 1974, **2**, 691.

Department of Gastroenterology, St Bartholomew's Hospital, London EC1A 7BE

D P O'DONOGHUE, MB, MRCP, honorary senior registrar

M L CLARK, MD, FRCP, senior lecturer in medicine and honorary consultant physician