avidly protein bound persists in the tissues. We could not implicate the subcutaneous injection techniques in the patients (Cases 3-9) who developed indurated pruritic transient lesions within hours of injection. Contact sensitization to cresols is recognized,⁵ and we suspect that all local and one of the systemic reactions seen in our patients resulted from hypersensitivity to the chlorocresol preservative. Intradermal skin testing with chlorocresol-preserved heparin gave immediate responses, whereas in only one (Case 1) of the tests with preservative-free heparin (Weddel) was there a reaction.

We suggest that in cases of apparent heparin sensitivity treatment may be able to continue without adverse reaction after cautious challenge with chlorocresol-free heparin.

We are grateful to Professor J. Richmond for helpful criticism, and to Mrs. E. C. Baxter (Weddel Pharmaceuticals Ltd.) for useful information.

¹ Moser, R. H., *Clinical Pharmacology and Therapeutics*, 1968, 9, 255.

² Richmond, J., personal communication, 1975.

³ O'Toole, R. D., *Annals of Internal Medicine*, 1973, 79, 759.

⁴ Hume, M., Smith-Petersen, M., and Fremont-Smith, P., *Lancet*, 1974, 1, 261.

⁵ Fisher, A. A., *Contact Dermatitis*, 2nd edn., p. 374. Philadelphia, Lea and Febiger, 1972.

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Deglutition Syncope

Syncope provoked by swallowing is rare according to the few cases reported. Probably this condition is not recognized and passes unnoticed. A salient feature of the reported cases is that in most of them disorders of the oesophagus could be shown. These ranged from diverticula¹ to strictures,² spasm, and carcinoma.³ In most cases syncope occurred mainly as a result of atrioventricular heart block, but sinus bradycardia and sinus arrest have also been reported,³ and in one case¹ both sinoatrial and atrioventricular block as well as signs of other disturbances of the sinus node were recorded.

We report a case of what we suggest be termed deglutition syncope characterized by repetitive syncopes during swallowing due to heart block.

Case Report

A 43-year-old man with a past history of rheumatic fever during childhood had for several years suffered from suddenly occurring retrosternal pain, which occurred mainly during meals but also spontaneously. On many occasions he had fainted, but usually the pain was accompanied only with dizziness. Drinking of carbonated beverages regularly provoked such attacks, but no other food could be pointed out as of particular significance. The general examination, including x-ray examination and endoscopy of the oesophagus, was uneventful except for a left anterior hemiblock in the electrocardiogram (E.C.G.). He was monitored in the ward, and on a couple of occasions a marked variation of the heart rate was observed. Over a short period of time the heart rate could vary from 44 to 80 beats/min, showing chaotic atrial activity. A provocation test letting the patient drink carbonated water resulted in a conspicuous sinus arrhythmia and bradycardia (see fig.).

E.C.G. telemetry while swallowing, showing changing P-wave configuration and frequency slowing from 70 to 20 beats/min.

E.C.G. registration with an oesophageal lead produced atrioventricular blockade. Because of these defects of the conduction system of the heart we decided to implant an on-demand pacemaker. A control later on with inhibition of the implanted pacemaker and with the patient simultaneously drinking carbonated water showed the occurrence of an asystole lasting 3.7 seconds.

Comment

Local changes of the oesophagus have been considered the cause of an enhanced sensitivity of the nervous receptors so that a vagovagal reflex might be put at work with resultant heart block. Such disorders, however, are not always demonstrable, as in this report, and they alone are hardly responsible for deglutition syncope or similar conditions. The prime factor in this condition is probably a disease of the conduction system of the heart. This is supposed to exist at a subclinical level, but can be triggered by nervous reflexes originating in the oesophagus. This also applied to our patient, who according to current views suffered from the sick-sinus syndrome but who in addition displayed an atrioventricular blockade during stimulation of the nervous receptors of the oesophagus.

Deglutition can also provoke tachyarrhythmias,^{2,4} and in one reported case² swallowing initially produced a tachycardia of 140 beats/min. This suddenly changed into a sinus bradycardia of 45 beats/min and syncope, but asystole did not ensue. By definition a sick sinus produces both tachyarrhythmias and bradyarrhythmias.

It is important to recognize deglutition syncope since such syncopes may be life-threatening and easily can be prevented by insertion of a pacemaker.

¹ James, A. H., *Lancet*, 1958, 1, 771.

² Tolman, K. G., and Ashworth, W. D., *Digestive Diseases*, 1971, 16, 1026.

³ Tomlinson, I. W., and Fox, K. M., *British Medical Journal*, 1975, 2, 315.

⁴ Bajaj, S. C., et al., *Gastroenterology*, 1972, 62, 632.

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Septic Abortion Associated with a Lippes Loop

Septic abortion associated with an intrauterine contraceptive device (I.U.D.) has been well documented in the United States.¹ The Dalkon Shield has been mainly concerned with this complication^{2,3} and the Food and Drug Administration suspended sales of it in the U.S.A. in June 1974. Subsequently the F.D.A. has reported 219 septic abortions and 13 deaths in association with this device. These serious complications have not been confined exclusively to the Dalkon Shield, however, and this paper reports a case of septic abortion with a Lippes Loop.

Case Report

A 26-year-old woman at 21 weeks gestation in her third pregnancy was admitted with a history of pyrexia and rigors for 24 hours and abdominal pain and vaginal bleeding of increasing severity for eight hours. She had had a Lippes Loop I.U.D. inserted two years earlier, six months after a normal pregnancy. Her first pregnancy had been terminated at 10 weeks. The current pregnancy had been uneventful up to the time of admission and she denied interference.

On admission she was severely distressed and pale. Her temperature was 38.5°C; the blood pressure was 110/70 mm Hg and the pulse rate 130 beats/min. There was moderate lower abdominal tenderness and the uterus, the size of which was compatible with the stated period of gestation, was contracting strongly every five minutes. Vaginal bleeding was moderate; membranes bulged through the cervix, which was 2-cm dilated. The haemoglobin was 9.6 g/dl and the total leucocyte count $12.3 \times 10^9/l$. Blood cultures subsequently grew *Proteus* spp. She was transfused two pints (1.1 l) of blood. Ampicillin treatment, started before admission, was continued by intramuscular injection. She spontaneously aborted two hours after admission.

The liquor was offensive and the placental swab subsequently grew *Proteus* spp. and *Escherichia coli*. The I.U.D. was expelled with the fetus.

Two hours after delivery the blood pressure fell to 70/50 mm Hg, despite the blood transfusion. She was centrally cyanosed; the pulse was rapid and feeble. The central venous pressure (C.V.P.) was plus 12 cm water and the urine output for the preceeding 12 hours had been only 400 ml. The antibiotic was changed to intravenous cephaloridine together with intravenous hydrocortisone. The patient's condition then rapidly improved. The blood pressure, C.V.P., and urinary output all returned to normal levels after 10 hours. Cephaloridine was continued for eight days. She was discharged home, well, nine days after admission.

Discussion

Pregnancy associated with an I.U.D. is a potentially serious condition. If the pregnancy is to continue the F.D.A. recommends that the device should be removed if the strings are visible. Removing the device may decrease the incidence of spontaneous abortion.⁴ Furthermore, the insidious onset of symptoms is impressive² and the incidence of other complications is also increased.⁵ There is a great need for more information on I.U.D.s, and a registry system for I.U.D.s such as is being started in the U.S.A. should be considered in Britain.

The value of C.V.P. monitoring in a hypotensive patient is further emphasized here. A C.V.P. reading slightly above normal established that the cause of hypotension was "pump failure" rather than under-transfusion. It was thus the septicaemia which was treated urgently rather than the further administration of intravenous fluids.

I thank Mr. S. Madden for his assistance and permission to publish this case.

¹ U.S. Food and Drug Administration, Press Conference on I.U.D.s and Supporting Documents, Rockville, Maryland, 20852 20 December 1974.

² Christian, C. D., *American Journal of Obstetrics and Gynecology*, 1974, **119**, 441.

³ Hurt, W. G., *Obstetrics and Gynecology*, 1974, **44**, 491.

⁴ Alvoir, G. T., jun., *Obstetrics and Gynecology*, 1973, **41**, 894.

⁵ Stevens, J. D., and Fraser, I. S., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1974, **81**, 282.

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Pancreatitis in the Hard Water Syndrome

If dialysis fluid used in haemodialysis is made up of hard water unpleasant complications may develop. They include headache, sweating, tachycardia, hypertension, vomiting, and sometimes gastrointestinal haemorrhage. These have been attributed to a rise in plasma calcium and magnesium concentrations.¹ Therefore in hard water areas the tap water required for preparing dialysis fluid should be softened or deionized. Failure of the softening or deionizing system may result in severe hypercalcaemia causing the hard water syndrome.

Case Report

A 23-year-old girl with terminal renal failure due to chronic pyelonephritis was established on home haemodialysis in December 1973. Warm, single-pass dialysis fluid was prepared in a Dylade proportionater by mixing softened water and a concentrated solution of salts.* The usual dialysis fluid calcium concentration was 1.88 mmol/l (3.75 mEq/l) and magnesium 0.5 mmol/l (1 mEq/l). Water was softened by a Permutit domestic water softener, which the patient was instructed to regenerate once a week. Access to blood was via a forearm arteriovenous fistula. For nine months she dialysed thrice weekly without trouble. On 23 October 1974 she was admitted urgently to hospital within a few hours of completing dialysis. She complained of severe headache, nausea, vomiting, central abdominal pain, generalized weakness, and sweating. These symptoms had developed while dialysing. During the penultimate dialysis she had experienced similar though less severe symptoms.

On examination she was afebrile, perspiring, and restless; pulse rate 88/min, regular; extremities warm; B.P. 210/130 mm Hg. Her arteriovenous

*Renalyte; McCarthy's.

Graph showing high plasma amylase associated with high plasma calcium and subsequent parallel reduction in both measurements. D represents haemodialysis.

Conversion: SI to Traditional Units—Creatinine: 1 mmol/l $\approx$ 0.0113 mg/100 ml. Calcium: 1 mmol/l $\approx$ 4 mg/100 ml. Phosphate: 1 mmol/l $\approx$ 3.1 mg/100 ml.

fistula had thrombosed. There were epigastric guarding and rebound tenderness, normal bowel sounds, and no abdominal distension or free fluid. All tendon reflexes were exaggerated. Fundi normal. Haemoglobin was 6.1 g/dl, white blood count $8.8 \times 10^9/l$, blood urea 11.6 mmol/l (70 mg/100 ml), serum creatinine 530 mmol/l (6.0 mg/100 ml), sodium 128 mmol/l, potassium 5.5 mmol/l, bicarbonate 36 mmol/l, calcium 4.9 mmol/l (19.6 mg/100 ml), serum phosphate 1.3 mmol/l (4.1 mg/100 ml), alkaline phosphate 42 IU/l, protein 72 g/l, albumin 39 g/l, amylase 2838 Somogyi units/100 ml, serum alanine aminotransferase 12 IU/l, bilirubin 12.0 mmol/l (0.7 mg/100 ml). Magnesium was not measured. E.C.G. showed much shortened QTc intervals. Erect and supine abdominal radiographs showed gaseous distension, no fluid levels, and no pancreatic calcification.

The patient admitted that during the past three to four weeks she had neglected to check the hardness of the water before dialysis and had not regenerated the water softener. Probably, therefore, she had been exposed to a high dialysis fluid calcium concentration during at least two dialysis periods. When checked, the calcium concentration of the unsoftened tap water was 3.1 mmol/l (12.3 mg/100 ml) and the magnesium 0.4 mmol/l (1.0 mg/100 ml). She was dialysed in hospital for the next week using a leg Scribner shunt. Plasma calcium and amylase levels rapidly fell to normal and the symptoms resolved over the next three to four days (see fig.). Barium meal examination and a cholecystogram were subsequently found to be normal.

Discussion

The symptoms caused by the hard water syndrome have been attributed to hypercalcaemia and hypermagnesaemia.¹ Mixer *et al.*² reported pancreatitis in 11 cases of hyperparathyroidism and reviewed 51 previous cases from the literature. Hypercalcaemia was present in nearly all their cases though it was not always a reliable clue to pancreatitis. Hypercalcaemia might induce hypertension, and Drukker³ considered this to be a possible cause of the headaches, nausea, and vomiting. In hypercalcaemia associated with hyperparathyroidism intravascular coagulation has been noted and microthrombi have been seen in pancreatic and renal vessels.⁴ Perhaps the clotting in our patient's AV fistula was related to a state of hypercoagulability. The effect of hypercalcaemia on the conversion of trypsinogen to trypsin may also be important.⁵

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¹ Freeman, R. M., Lawton, R. C., and Chamberlain, M. A., *New England Journal of Medicine*, 1967, **276**, 1113.

² Mixer, C. G., jun., Keynes, W. M., and Cope, O., *New England Journal of Medicine*, 1962, **266**, 265.

³ Drukker, W., *Proceedings of the European Dialysis and Transplant Association*, 1968, **5**, 284.

⁴ Baer, L., and Neu, H. C., *Annals of Internal Medicine*, 1966, **64**, 1062.

⁵ Haverback, B. J., *et al.*, *American Journal of Medicine*, 1960, **29**, 424.

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