are in the habit of giving extra fluids to their infants right from birth.

Summary

Seventeen cases of "nocturnal" thirst fever in infants aged 3 to 10 months are described. The only presenting symptom was fever with a characteristic temperature pattern, being very high in the morning and gradually dropping to normal towards the evening. This particular temperature pattern is diagnostic. It helps in the early administration of the proper treatment—extra fluids—without recourse to unnecessary drugs or tedious investigations.

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Medical Memoranda

Phenindione-induced Haemorrhagic Ulcerative Colitis

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Adverse reactions to phenindione are not uncommon. Perkins (1962) reviewed 133 reported and three personal cases. The most common complication was a rash (100 cases); fever (34 cases) and diarrhoea (20 cases) also occurred frequently.

In reported cases the onset of diarrhoea, when specified, varied from one week (Tait, 1960) to three and a half months (Perkins, 1962) after the start of treatment. Usually the diarrhoea was mild, but sometimes it increased in severity if phenindione was continued. One patient developed frank steatorrhoea (Juel-Jensen, 1959). A mild latent steatorrhoea may have been present in this patient, as he excreted 10.8 g. of faecal fat in 24 hours after recovery. This patient developed dysentery-like stools with blood and mucus five days after starting phenindione on a subsequent occasion.

We record details of two patients with haemorrhagic ulcerative colitis, one occurring seven days, the other two days, after the start of a first course of phenindione. In neither case was there evidence of any underlying bowel disorder, nor was there any history of previous drug hypersensitivity.

CASE 1

A man aged 64 had two attacks of transient right hemiparesis. He was admitted to hospital on 28 January 1960 after the second episode. On 30 January he was started on phenindione 50 mg. twice daily. His bowels were opened once daily until 6 February, when he passed bright blood in his stools. He had never had any bowel symptoms before. His one-stage prothrombin time was 34 sec., control 13 sec. Later that day he collapsed with severe rectal bleeding, which persisted. He was given vitamin K₁ by injection and transfused with a total of 15 pints (8 litres) of blood. He continued to pass small quantities of blood. On 7 February he suddenly collapsed and died with a further severe haemorrhage.

A post-mortem examination was performed by Dr. R. I. K. Elliott. There was blood-stained fluid in the peritoneum. The colon was distended with blood and the colonic mucosa showed multiple, punched-out, circular ulcers. Some penetrated deeply and almost perforated the colon.

CASE 2

A man aged 64 was admitted to hospital on 8 November 1964 after a myocardial infarction. He had mild diabetes mellitus

controlled by diet. He was started on phenindione. Thirty-six hours later, after a total of 200 mg. phenindione, he passed a loose stool containing fresh blood and mucus. The one-stage (Quick) prothrombin time was 24 sec., control 12 sec. (16.5%). Sigmoidoscopy revealed a granular red congested mucous membrane, which bled on contact. The appearance was identical with that seen in ulcerative colitis. A rectal biopsy showed dilated blood-vessels with a dense cellular infiltration, mainly with polymorphs (Dr. R. I. K. Elliott).

Phenindione was stopped, and he was treated with codeine phosphate and a low-residue diet. He continued to pass blood and mucus in decreasing amounts for 48 hours, after which his stools became normal. A patch test to phenindione was negative. On 20 November 1964 he developed thrombophlebitis. He was treated with warfarin without side-effects.

COMMENT

Both these patients developed acute ulcerative colitis shortly after being given phenindione. The first patient died from recurrent massive haemorrhage and could only have been saved by total colectomy. The second patient recovered without specific treatment when phenindione was stopped. This patient resembled the man described by Juel-Jensen (1959), who also recovered when phenindione was stopped, except that our patient had never taken phenindione before.

This acute type of reaction seems to be uncommon. Most cases of diarrhoea due to phenindione are mild and do not progress to ulcerative colitis.

This report adds to the list of fatal reactions to phenindione. Though there have been fewer reports of side-effects from other oral anticoagulants, it cannot be concluded that they are safer than phenindione unless it is also known how many patients have received each type of drug.

We wish to thank Dr. W. A. Bourne for permission to report the second case.

A. R. TANSER,* M.B., M.R.C.P.,
Senior Medical Registrar,
Royal Sussex County Hospital.

E. C. B. KEAT, M.D., M.R.C.P.,
Consultant Physician,
Royal Sussex County Hospital.

* Present address: King's College Hospital, London.

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