Unreviewed Reports

Bullous eruption associated with cinoxacin and long wave ultraviolet A light

Five days after completing a two week course of cinoxacin 500 mg twice daily for a urinary tract infection a 27 year old woman, who had been using a domestic ultraviolet A "sunlamp" for a month, developed large bullae on the lower legs and smaller blisters on the dorsal surface of the hands. New lesions continued to develop for six weeks after she stopped using the lamp. Investigations to find a cause for her bullous eruption were negative, and it was attributed to cinoxacin, which is structurally related to nalidixic acid, which has been reported to cause a similar eruption.[1]—ALLAN HIGHET, York District Hospital, York YO3 7HE. *(Accepted 24 January 1986)*

1 Ramsay CA, Obreshkova E. Photosensitivity from nalidixic acid. *Br J Dermatol* 1974;**91**:523-8.

Increase of circulating atrial natriuretic peptide in acute pulmonary embolism

Atrial natriuretic peptide (ANP) is released in response to increased atrial pressure and diameter.[1] In support of this observation we report on a patient with pulmonary embolism who presented with dyspnoea, tachycardia, hypotension, and an arterial Po_2 of 5·9 kPa (44 mm Hg). Pulmonary artery pressure was 78/34 (mean 48) mm Hg. Heparin was started, and angiography showed a 50% occlusion of the pulmonary vasculature. Two days later pulmonary and arterial pressures and heart rate were normal and arterial Po_2 8·7 kPa (65 mm Hg). Plasma concentrations of ANP were 345 pg/ml at the onset and 151 pg/ml 48 hours later (normal 42 (SD 3)). The patient went into negative fluid balance immediately before pulmonary embolism was diagnosed, showing that the concentration of ANP had reached an effective level.—J F E MANN, R E LANG, *et al*, Departments of Internal Medicine and Pharmacology, University of Heidelberg, D-6900 Heidelberg, West Germany. *(Accepted 27 January 1986)*

1 Lang RE, Thoelken H, Ganten D, Luft FC, Ruskoaho H, Unger T. Atrial natriuretic factor—a circulating hormone stimulated by volume loading. *Nature* 1985;**314**:264-6.

Overdose of chlormezanone: a new clinical picture

A 32 year old man ingested 11 g of chlormezanone. Three hours later he was comatose with dilated pupils, hot dry skin, flaccidity, and absent reflexes. He then developed brisk reflexes, ankle clonus, an extensor plantar response, and adduction of the legs with fasciculation of quadriceps. Five hours after ingestion his condition alternated between coma and excitement provoked by minor disturbances. His symptoms settled gradually over 12 hours, although the hypertonicity persisted for 30 hours. This clinical picture—dominated by anticholinergic effects—is very different from that described in previous reports[1] and may be due to the high dose ingested.—B W KIRKHAM, J B EDELMAN, King George Hospital, Ilford, Essex 1G2 7RL. *(Accepted 27 January 1986)*

1 Armstrong D, Braithwaite RA, Vale JA. Chlormezanone poisoning. *Br Med J* 1983;**286**:845-6.

Irreversible renal transplant failure after enalapril therapy

Reversible renal failure and renal artery occlusion may occur if patients with bilateral or unilateral renal artery stenosis are given angiotensin converting inhibitors. A 45 year old man received a renal transplant in 1980. Five years later he presented with left ventricular failure secondary to hypertension and ischaemic heart disease. Serum creatinine concentration was 141 μmol/l (1·6 mg/100 ml). He was given 5 mg enalapril and immediately became anuric owing to complete occlusion of the renal artery of the transplanted kidney. Exploration showed a fresh thrombosis distal to a stenosis. Angiotensin converting inhibitors should be used with caution in patients who may have transplant artery stenosis.—A R BROWN, P F WILLIAMS, Renal Unit, Addenbrooke's Hospital, Cambridge CB2 2QQ. *(Accepted 27 January 1986)*

Aortogastric fistula after operation for bleeding gastric ulcer

A 74 year old man had a bleeding benign posterior gastric ulcer underrun. Eighteen months later he developed further haematemesis, which did not respond to conservative management. At laparotomy a fistula had formed between the aorta and the stomach through the ulcer, probably due to apposition of the two organs by the previous operation. He recovered well after the fistula was closed and the ulcer removed by partial gastrectomy. Aortodigestive fistulas, of which this is an unusual cause, are often due to diseases of or operations on the aorta.[1] They are potentially fatal and must be recognised and treated promptly.—DAVID SELLU, JOHN LYNN, Department of Surgery, Ealing Hospital, Southall, Middlesex UB1 3HW. *(Accepted 30 January 1986)*

1 Champion MC, Sullivan SN, Coles JC, Goldbach M, Watson WC. Aortoenteric fistulae: incidence, presentation, recognition and management. *Ann Surg* 1982;**195**:314-7.

Pancreatitis associated with sulphasalazine

A 22 year old woman developed diarrhoea and abdominal pain. Sulphasalazine, 500 mg four times daily, alleviated her symptoms. Epigastric pain recurred, however, and she was referred to hospital. The only abnormal findings were raised urine and serum amylase concentrations. Sulphasalazine was stopped and her symptoms resolved. Amylase concentrations returned to normal within 12 days. She was rechallenged with sulphasalazine, and within 24 hours amylase concentrations and the amylase:creatinine clearance ratio increased, reverting to normal two days after the drug was stopped. The patient remained asymptomatic during this period. There has been only one previous report implicating sulphasalazine as a cause of pancreatitis.[1]—H SURYAPRANATA, H DE VRIES, *et al*, Department of Internal Medicine I, University Hospital, Rotterdam, The Netherlands. *(Accepted 31 January 1986)*

1 Das KM, Eastwood MA, McManus JPA, Sircus W. Adverse reactions during salicylazosulfapyridine therapy and the relation with drug metabolism and acetylator phenotype. *N Engl J Med* 1973;**289**:491-5.

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