view expressed by the authors I believe it is possible to explain clinical features of NMS without implicating the hypothalamus. The hypothalamus was originally suggested as a contributory cause of the hyperthermia, peripheral heat production being important too. This argument was supported by suggesting that other signs such as sweating, tachycardia, hypotension and altered consciousness (signs of 'disturbed autonomic function') were also of hypothalamic origin. However the severity of illness in these cases is sufficient to explain most features described in the literature, other than the muscular rigidity, akinesia and hyperthermia. The hypothesis suggested here depends on the assumption that hypothalamic disturbance plays a primary role in NMS. If endorphins are involved it may not be correct to assume that the hypothalamus is affected.

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Spontaneous rupture of the bladder in a patient with cor pulmonale presenting as acute abdominal emergency

Sir,

Spontaneous rupture of the bladder is usually associated with malignant disease, schistosomiasis (Jenkinson 1981; Powell *et al.*, 1983) and anatomical abnormalities (Cumes & Kessler, 1979). Acute spontaneous rupture of the bladder in the absence of these abnormalities and urinary retention has not been reported previously. The following case report describes its occurrence in a very common medical condition.

A 60 year old Chinese male with cor pulmonale on salbutamol, theophylline, frusemide, slow-K and low dose prednisone regularly was admitted in respiratory failure. He also complained of frequency and nocturia for 4 years. He was given intravenous aminophylline, antibiotics and chest physiotherapy. Rectal examination showed a moderately enlarged prostate. The daily urine output varied between 2 to 3 litres, as did the intake.

He complained of sudden severe lower abdominal pain on the seventh day of admission, accompanied by signs of peritonitis. Bladder catheterization yielded 1.5 litres of blood-stained urine. At laparotomy 2 litres of urine was present in the peritoneal cavity, and a 3.5 cm tear was seen at the dome of the bladder. An intravenous urogram was normal. Cystoscopy showed a moderately enlarged prostate, with trabeculated bladder mucosa. A biopsy of the latter showed nonspecific chronic cystitis, and a biopsy of the prostate showed benign nodular hyperplasia.

Spontaneous rupture of the bladder in this patient was totally unexpected in the absence of any obvious urinary retention. Perhaps the combinaton of aminophylline in relaxing the bladder smooth muscle, a reduced elasticity due to nonspecific chronic cystitis, and raised intra-abdominal pressure in chronic obstructive airways disease all contributed to rupture of a bladder which would normally cope with the diuresis even in the presence of moderate prostatic hypertrophy.

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