and that control of the hyperthyroidism resulted in amelioration of bronchospasm. In fact, the patient required no treatment at all when she developed hypothyroidism as a result of radio-iodine therapy. Although replacement therapy did lead to broncho-
spasm, episodes were mild and infrequent and easily relieved by bronchodilators.

The precise mechanism whereby hypothyroidism aggravates the asthmatic state is not known. It is possible that a decrease in levels of adenosine triphosphate, which has been observed in hyperthyroidism, results in a decrease in intracellular levels of cyclic 3', 5'-adenosine monophosphate (cAMP), thereby predisposing to bronchospasm in susceptible individuals. However, results of studies on cAMP levels in hyperthyroidism do not support such a hypothesis. Decreased catecholamine levels (which have been observed in hyperthyroidism) may also explain the exacerbation of bronchospasm in these patients. However, other studies on the relationship between catecholamines and hyperthyroidism have not corroborated these results. According to Bush et al., a likely mechanism for the association between thyroid disease and the severity of asthma relates to altered corticosteroid metabolism. Hyperthyroidism appears to increase the conversion of hydrocortisone to its inactive 11-ketonic derivative, and in this way may aggravate bronchospasm, whereas the reverse is true for hypothyroidism. Reduced breakdown of prostaglandins, which has been demonstrated in rats made hyperthyroid with thyroxine, could account for the increased bronchospasm seen in our patient. On examination of the studies showing that histamine is released from the thyroid mast cells of rats and mice in response to thyroid-stimulating hormone, it could be postulated that aggravation of the asthmatic state in association with hyperthyroidism is due to histamine release.

In conclusion, regardless of the mechanism involved in the relationship between hyperthyroidism and asthma, recognition of the interactions between the two diseases occurring in the same patient is essential for the proper management of these disorders.

REFERENCES

Spontaneous rupture of the bladder
A case report

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Summary
A case of spontaneous rupture of the bladder is described. The patient was a 35-year-old woman presenting with an unusual clinical pattern of peri­

Spontaneous rupture of the bladder is rare. The diagnosis should be considered in patients who present with minor abdominal pain and an inability or a decreased ability to void urine, diarrhoea, abdominal distension, cough, pneumonia, post-cathe­

Case report
A 35-year-old woman was admitted to Coronation Hospital, Johannesburg, with a 4-day history of continuous diffuse abdominal pain, vomiting, diarrhoea, coughing and difficulty in voiding urine. She denied having gynaecological or urological disease, an excessive alcohol intake or trauma. On examination the patient appeared ill and was dehydrated. The abdomen was slightly distended and tender, with increased bowel sounds. The haemoglobin concentration was 10 g/dl, haematocrit 33,5 and white blood cell count 15 x 10^9/L, the sodium level was 119 mmol/L, potassium 4,9 mmol/L, urea 58,9 mmol/L, creatinine 300
\[ \text{\( \mu \text{mol/l} \) and amylase 804 IU. Blood gas analysis showed metabolic acidosis, with a pH of 7.2. An abdominal radiograph showed a few fluid levels. The patient was catheterized and 50 ml of cloudy urine was drained. A diagnosis of gastro-enteritis, pre-renal failure and dehydration was considered. Two days later, after intravenous administration of saline solution, the patient had improved; the electrolyte levels were normal as was the urine output, but a cloudy haematuria appeared. In the radiology department 500 ml of urine drained spontaneously during performance of erect abdominal radiography. A cystogram showed that the bladder had a leak (Fig. 1).}

\[ \text{Fig. 1. Cystogram showing a leak from the bladder.} \]

On surgical exploration free, cloudy blood was revealed; multiple erosions and microhaemorrhages covered the serosa of the bowel, and a few light adhesions were found. There was a tear about 8 cm long with necrotic edges in the fundus of the bladder, which was sutured in two layers. A bladder biopsy specimen showed necrotic tissue only. The patient was discharged in good condition, passing urine normally.

**Discussion**

Rupture of a normal bladder after trauma is well documented, but spontaneous rupture is rare. The first case was reported by Peisus. In 1979 Shumaker reported 115 cases of bladder rupture, of which only 4 (3.4\%) were spontaneous. Eighty-four cases have been reported in the literature to date,\(^1\) and a similar rare case has previously been described by one of the authors of this article (Z. E. H.). The usual history is that of sudden, diffuse lower abdomen pain which becomes continuous. Extraperitoneal rupture may have an insidious onset with less pain than that associated with intraperitoneal rupture. In 10\% of cases there is no definite history and absence of abdominal pain.\(^4\) The presence of sterile urine in the abdominal cavity for 7 days causes an inflammatory reaction of the viscera, and these are covered by fibrin as well as granulation tissue. Accumulation and stasis of urine in the intraperitoneal space causes reabsorption of urea, creatinine and potassium, and serosal damage, adhesions and partial bowel obstruction. A bladder tear does not prevent the evacuation of urine through the urethra, especially in the erect position as in our case. The urine irritated the bowel, resulting in diarrhoea, multiple superficial erosions and serosal microhaemorrhages.

In our patient serum urea, creatinine and potassium levels and the blood pH settled to normal levels 6 - 48 hours after catheterization of the intraperitoneal cavity.\(^5\) Suarez and Firi\(^4\) have described the presence of haemorrhagic necrosis on pathological examination, but Mitchell\(^6\) writes: 'Extravasated sterile urine is probably rapidly absorbed and therefore poses no threat'. High blood urea, bicarbonate and potassium levels are described in many reports, while all the patients who sustained intraperitoneal bladder rupture and had symptoms of longer than 24 hours' duration had mild-to-moderate azotaemia and acidosis with secondary urinary reabsorption.\(^2,9\)

It is generally accepted that operative intervention is indicated in cases of traumatic rupture of the bladder, but some authors have suggested conservative treatment in a limited number of cases of extraperitoneal rupture.\(^8,10,11\) The mortality rate has been high in some series; Stone\(^7\) reported a mortality rate of 46\%, and Bastable\(^8\) one of 47\%. In these series death was attributed to the underlying disease, haemorrhage or infection of extravasated urine.

Peritonitis due to spontaneous rupture is associated with a mortality rate of 80\% in undiagnosed cases;\(^1\) mortality during the first 12 hours is 10\%, after 24 hours 25\% and thereafter 50\%.\(^1,5\)

Thompson et al.\(^1\) believe that the majority of cases of spontaneous rupture are attributable to disease of the bladder; bladder rupture is more frequent in patients with intermittent cystitis. Higson and Smith's study\(^12\) showed that prolonged bladder distension in patients with predisposing factors such as cystitis, detrusor instability, ischaemic nerve damage, reduced bladder capacity or post-irradiation bladder fibrosis could lead to bladder rupture. Chronic alcoholism, and particularly ephedrine and alcohol abuse, lead to spontaneous rupture.\(^8,12-15\)

We believe that the rupture of the bladder in our case resulted from heavy alcohol intake.

In most cases the site of rupture is the junction of the posterior and superior wall (as in our case). This is said to be the weakest part of the bladder, being covered only by peritoneum and not having fascial or bony protection.\(^11\) The size of the tear can vary between 1 and 8 cm.\(^2,7\) An extended rupture can be compared to a 'collapsed dam'. The bladder space and abdominal cavity act as a bypass, making voiding of urine difficult or impossible.

**REFERENCES**