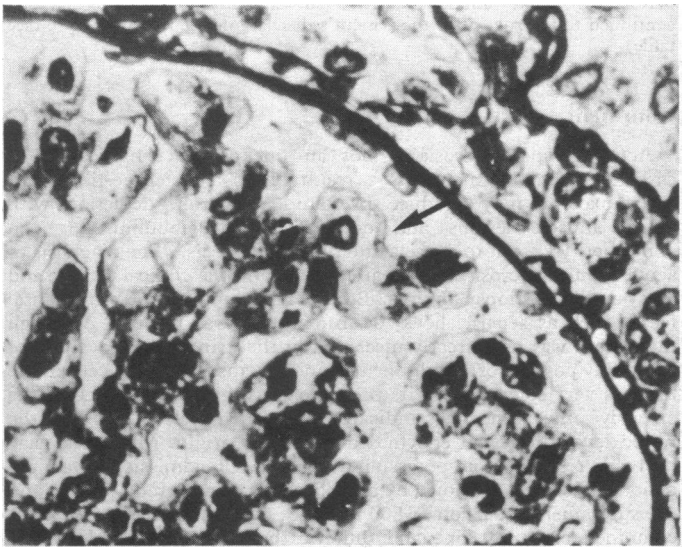
Discussion

The diagnosis of rheumatoid arthritis and MCGN was made after SLE with lupus nephropathy had been excluded by the absence of LE cells, antinuclear factor, and the usual clinical features. The selective depression of C_3 was also more characteristic of MCGN than SLE.

We wondered whether the rheumatoid arthritis was related to the MCGN. The sequence of events in this case suggested that the rheumatoid arthritis preceded the renal disease. It is also uncommon for isolated MCGN to occur at this patient's age. Thus the concurrence of the two diseases in this patient seemed more than coincidental. If there was an association, how might this be explained?



Section of glomerulus showing increased mesangial cell proliferation, thickening of capillary basement membrane, and double-contour appearance (arrowed). (Periodic acid-silver stain × 5280.)

The renal changes usually associated with rheumatoid arthritis are renal amyloidosis and analgesic nephropathy. Experiments have shown, however, that rheumatoid serum may aggravate experimental nephritis.[3] Rheumatoid factor may also contribute to the immune-complex formation in the glomerulus.[4] Furthermore, depression of total haemolytic complement and individual complements—particularly C4 and sometimes C3—occurs in some patients with rheumatoid arthritis. Such patients tend to have a high concentration of rheumatoid factor, severe rheumatoid disease, and a high incidence of recurrent infections.[5]

The deposition of immune complexes in the glomeruli with consequent activation of complements has been implicated in the pathogenesis of MCGN.[1] Possibly a similar mechanism may have been operative in this case. Alternatively, the hypocomplementaemia may have preceded rather than followed the formation of the immune complexes. Complement deficiency may cause a defect in immunological defence that results in persistent infection and chronic immune-complex formation and finally leads to MCGN.[2]

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Non-accidental immersion in bath-water: another aspect of child abuse

New acts are constantly being added to the tragic repertoire that comprises the child abuse syndrome. Deliberate poisoning has recently been added to the well-known forms of acute physical trauma (fractures, dislocations, lacerations, contusions, intracranial haematomata, burns), emotional trauma, deprivation, and pathological neglect.[1] During an extensive total population study of immersion accidents affecting children[2] a survey of bathtub drownings and near-drownings showed that some of these were deliberately produced by a battering parent.

Incidents

The cases of deliberate immersion that have come to our notice have all occurred in household bathtubs. In each case gross social disorders were pre-existent in the home. Features of these non-accidental immersions, and some differences distinguishing them from bathtub drownings in general, are shown in the table. In one of the cases we encountered the child (who survived the immersion incident) subsequently suffered an unexplained fractured skull at the age of 22 months. In another case we discovered later that a child who was deliberately immersed in the bath at the age of $3\frac{1}{2}$ years was already known to the child protection agencies because of neighbours' reports of child abuse. Another child suspected of being immersed (a handicapped infant) died 12 hours after presentation to hospital from cerebral anoxia. Of the two children described who survived the near-drowning incident one was severely brain damaged as a result.

Common characteristics of children and families in cases of accidental and non-accident immersion in the bath

	Accidental immersions	Non-accidental injury
Age (months)	Usually 9-15*	Usually 15-30
Social class	Usually IV or V	Probably all classes
Pre-accident morbidity:		
Parents	Usually normal	Full sociopathology of parents inflicting non-accidental injury
Child	Usually normal; often youngest in large family; epilepsy or convulsions may be present	May be handicapped; often eldest child of small sibship
Precipitating cause	Household routine upset; child left in bath with other children	Acute parental stress, often domestic altercations
Circumstances	Often many children in bath: older ones get out leaving youngest to drown	Single child in bath; incident occurs at unusual time of day

*Data from Brisbane drowning study.[2]

The modus operandi seems to be deliberate immersion until unconsciousness ensues. At this point panic or remorse causes the child to be extracted from the water and help summoned. In the cases we have encountered it was the mother (or de facto wife) who was thought to have been responsible. In all cases the child was unconscious and apparently dead when taken from the water.

Comment

Unlike lacerations, fractures, and head injuries,[3] but like punitive head shaking and whiplash injuries[4] and deliberate poisoning[1] non-fatal immersion accidents leave no pathognomonic stigmata. It is unfortunate that these more subtle forms of child abuse have been emphasised in only the last three years, and further more sophisticated types of battering, at present unrecognised, will probably also come to light.

An older infant or toddler is usually bathed at the end of the day, when a mother is tired and when stress and frustration are usually maximal. It is not surprising that this scenario is the occasion when a disturbed parent finally acts out the temptation and impulses that are usually chronically present, but barely submerged, in this group of families at risk.

Near-drowning in the bath has been ascribed to child neglect in at least one report[5]; but neglect (as opposed to a positive act of wilful child-directed violence) is difficult to define in the context of children's drowning accidents. The parents who get drunk while supervising

infants at swimming pools and parents of infants who wilfully refuse to isolate, by a safety barrier, a water hazard in the home may be considered neglectful. We think that the psychodynamics here, however, are quite different from those where the parent makes a conscious act of aggression using the full bathtub as the agent.

It would be fruitful to investigate retrospectively hospital admissions for pneumonia and acute respiratory symptoms of children who are known to have been abused in other ways. It is important to include non-accidental injury in the differential diagnosis of atypical childhood bathtub immersions; all children presenting as such should be identified in the appropriate "at-risk" register in the admission room or casualty department.

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Rhabdomyolysis and acute tubular necrosis associated with carbenoxolone and diuretic treatment

Carbenoxolone sodium has been used for several years for treating gastric ulcers. Several side effects have been reported, including salt and water retention, hypertension, and potassium depletion.[1] We report here another complication: acute tubular necrosis.

Case report

A gastric ulcer was diagnosed in this 61-year-old man who had been admitted on 11 November 1975 for haematemesis. His blood pressure was 160/100 mm Hg. Measurement of serum electrolytes and renal function and urine analysis showed no abnormalities. Carbenoxolone 300 mg/day was prescribed.

Early in December chlorthalidone 50 mg every other day was added to control ankle oedema. Carbenoxolone was continued at the same dose. No potassium supplement was given. At the end of December the patient complained of progressive muscle weakness, mental confusion, and disorientation. Drug treatment was continued.

The patient was readmitted on 8 January 1976 for rapidly progressive oliguria. He had an ataxic gait and profound muscle weakness of the four limbs. Deep tendon reflexes were diminished. There was no other neurological abnormality. Blood pressure was 160/90 mm Hg. The results of laboratory investigations are shown in the table. Definite hypokalaemic alkalosis was associated with severe renal failure. Serum transaminase and creatinine phosphokinase concentrations were strikingly high. 200 ml of dark brown urine was obtained by bladder catheterisation. Despite the absence of red and white blood cells in the sediment, the orthotolidine test was positive. Urinary sodium (27 mmol (mEq)/l) and urea (1287 mmol (7·75 g)/l) levels were compatible with acute tubular necrosis.

The electrocardiogram was typical of hypokalaemia with multiple ventricular ectopics. Intravenous pyelography disclosed normal sized kidneys. An early, faint, stable nephrogram typical of acute tubular necrosis was obtained. Electromyography disclosed muscle fibre lesions and a mild decrease of motor nerve conduction velocity.

Muscle biopsy confirmed the existence of focal muscle necrosis. A renal biopsy performed 12 days after admission showed flattened tubular cells with numerous mitoses and mild interstitial oedema without cellular infiltrate—a picture suggestive of recovering acute tubular necrosis. No immunoglobulins, complement, or fibrinogen were detected. The patient retained 800 mmol (mEq) of potassium during the first five days. Muscular abnormalities subsided promptly. Anuria persisted for 10 days, so six haemodialyses were performed. Renal function returned subsequently towards normal and continued to improve after the patient's discharge on 7 February 1976 (see table).

Comment

Acute tubular necrosis is not an unexpected complication of carbenoxolone treatment. As a result of its aldosterone-like effect carbenoxolone may produce profound potassium depletion with severe muscle lesions and occasionally myoglobulinuria,[2–4] an abnormality known to cause acute tubular necrosis.[5] The diagnosis of acute tubular necrosis in our patient relied on the clinical picture and *x*-ray and renal biopsy findings. Besides myoglobinuria no other causal factor such as sepsis, shock, or intoxication was present. Although myoglobin was not directly measured in the urine, the association of a strongly positive orthotolidine test and the absence of red cells in the urine is very suggestive of its presence.[5] Myoglobinuria reflected the extensive muscle necrosis indicated by both muscle biopsy and the raised levels of serum enzymes of muscular origin. Muscle damage probably resulted from potassium depletion. On admission potassium deficit was severe, as shown by hypokalaemia despite renal failure and the subsequent retention of 800 mmol of potassium. It resulted from combined carbenoxolone and diuretic therapy. Carbenoxolone was given at high doses for too long (seven weeks). Furthermore, carbenoxolone's kaliuretic properties were greatly enhanced by the addition of a diuretic without potassium supplementation.[1]

This case not only illustrates a new complication of carbenoxolone but also provides a new example of acute tubular necrosis produced by non-traumatic rhabdomyolysis, a condition diagnosed with increasing frequency.[5]

Requests for reprints should be sent to Professor C van Ypersele.

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Serum levels

	Urea (mmol/l)	Creatinine (μmol/l)	Potassium (mmol/l)	Total CO_2 (mmol/l)	Creatinine* phosphokinase (IU/l)	Serum aspartate† aminotransferase (IU/l)
8 January	48·1	1193	1·4	36		550
10 January	53·1	1370	2·2	40	4400	400
4 February	19·6	301	3·8	26	26	18
3 March	11·0	177	4·6	25	31	12
19 May	11·6	168	4·9	24	35	20

*Normal range: 3-65 IU/l. †Normal range: 6-30 IU/l.
Conversion: SI to traditional units—Urea: 1 mmol/l ≈ 6 mg/100 ml. Creatinine: 1 μmol/l ≈ 0·0113 mg/100 ml. Potassium: 1 mmol/l = 1 mEq/l. CO^2: 1 mmol/l = 1 mEq/l.