no special intervention is required. If ventilation is inadequate from the start or if arterial PO_2 deteriorates more aggressive treatment is indicated. Mechanical ventilation with positive-end-expiratory-pressure is required,²¹⁻²³ titrating the amount of pressure to produce the minimum amount of intrapulmonary shunt.²¹ The use of corticosteroids is not routinely recommended, although their place in the patient with true secondary drowning has not been studied in any large series.

Recognising the possibility of this syndrome of secondary drowning is a major factor in management. An analysis of this and other series shows that a major high-risk group comprises young children who have almost drowned in salt or polluted water, in whom spontaneous respiration has not occurred for at least five to 10 minutes after rescue, but who appear to improve rapidly thereafter. Although it is not always done, all near-drowned victims must be admitted to hospital for observation, irrespective of their apparent relative wellbeing within several hours after rescue. Respiratory deterioration after apparent post-rescue wellbeing can occur rapidly.

With the recent increase in survival rates after near-drowning²⁴ more cases of this post-immersion respiratory distress syndrome are being encountered.²⁵ This review suggests that rescuers and clinicians should expect primary lung function to deteriorate within four hours of rescue in about one in 20 survivors of drowning accidents. Provided that the syndrome is anticipated, recognised, and treated vigorously the prognosis (generally excellent in childhood near-drowning¹⁵) should remain optimistic.

References

- Fuller RH. Drowning and the postimmersion syndrome: a clinicopathologic study. *Milit Med* 1963;**128**:22-36.
- Edmonds C. A salt water aspiration syndrome. *Milit Med* 1970;**135**:779-85.
- van Haeringen JR, Kleine JW, Sluiter HJ. Drowning. *Lancet* 1972;iii:880.
- Maestracci P, Grimaud D. Les oedèmes pulmonaires des noyés. *Ann Anesthesiol Franc* 1975;16 (spécial II et III):101-10.
- Saule H. Sekundäres Ertrinken. *Klin Paediatr* 1975;**187**:346-9.
- Modell JH. *The pathophysiology and treatment of drowning and near-drowning*. Springfield, Illinois: Charles C Thomas, 1971:9.
- Segarra F, Redding RA. Modern concepts about drowning. *Can Med Assoc J* 1974;**110**:1057-62.
- Clarke EB, Niggemann EH. Near-drowning. *Heart Lung* 1975;**4**:946-55.
- Miles S. Drowning. *Br Med J* 1968;iii:597-600.
- Golden FStC, Rivers JF. The immersion incident. *Anaesthesia* 1975;**30**:364-73.
- Haglund P, Fauarel-Garrigues JC, Nicod J, Lobera A. Biological disturbances during drowning in sea water. *Resuscitation* 1974;**3**:121-7.
- Pearn JH, Nixon J. Prevention of childhood drowning accidents. *Med J Aust* 1977;**1**:616-8.
- Pearn JH, Nixon J. Swimming pool immersion accidents. An analysis from the Brisbane Drowning Study. *Med J Aust* 1977;**1**:432-7.
- Patrick M, Bint M, Pearn JH. Salt water drowning and near-drowning accidents involving children. A five-year total population study in South-east Queensland. *Med J Aust* 1979;**1**:61-4.
- Pearn J. Survivors of childhood freshwater immersion accidents: neurologic and psychometric studies. *Lancet* 1977;ii:7-9.
- Wong FM, Grace WJ. Sudden death after near-drowning. *JAMA* 1963;**186**:724-6.
- den Otter G. Low-pressure aspiration of fresh water and sea water in the non-anoxic dog. An experimental study of the pathophysiology of drowning. *Forensic Sci* 1973;**2**:305-16.
- Gerya YF, Yankovsky VD. Use of artificial circulation in resuscitation of drowned dogs. *Resuscitation* 1976;**5**:145-52.
- Giammona ST, Modell JH. Drowning by total immersion. Effects on pulmonary surfactant of distilled water, isotonic saline and sea water. *Am J Dis Child* 1967;**114**:612-6.
- Modell JH, Calderwood HW, Ruiz BC, Downs JB, Chapman R. Effects of ventilatory patterns on arterial oxygenation after near-drowning in sea water. *Anesthesiology* 1974;**40**:376-84.
- Modell JH. Near drowning. *Int Anesthesiol Clin* 1977;**15**:107.
- Marshall LF, Smith RW, Shapiro HM. The outcome with aggressive treatment in severe head injuries. Part II. Acute and chronic barbiturate administration in the management of head injury. *J Neurol Sci* 1979;**50**:26-30.
- Prefaut C, Ramonatxo M, Boyer R, Chardon G. Human gas exchange during water immersion. *Respir Physiol* 1978;**34**:307-18.
- Pearn JH. Survival rates after serious immersion accidents in childhood. *Resuscitation* 1978;**6**:271-8.
- Eggink WF, Bruining HA. Respiratory distress syndrome caused by near- or secondary drowning and treatment by positive end-expiratory pressure ventilation. *Neth J Med* 1977;**20**:162-7.

(Accepted 18 September 1980)

SHORT REPORTS

Injection abscesses in a diabetic due to *Mycobacterium chelonae* var *abscessus*

Most diabetics do not sterilise their hypodermic syringes before each insulin injection. They use glass syringes, sterilise them at intervals, and keep them in disinfectant. We report the case of an insulin-dependent diabetic who stored her glass syringe in disinfectant which was inadvertently inactivated and who developed multiple injection abscesses caused by *Mycobacterium chelonae*, var *abscessus*.

Case report

The patient, a 24-year-old English woman whose diabetes was diagnosed in 1968, first noticed a lesion on the upper lateral aspect of her right thigh in October 1979. At this time she was using both thighs as insulin injection sites. The lesion was deeply subcutaneous, painful, indurated, roughly 2 cm × 3 cm in size, and the overlying skin was warm and discoloured, breaking down after two weeks with a central discharging sinus. Routine culture of sinus pus did not detect any recognised bacterial pathogens. A course of flucloxacillin was ineffectual. A similar lesion developed after two weeks on the opposite thigh, and culture of pus taken from it yielded an anaerobic coccus (*Peptococcus* spp). She was treated with metronidazole for five weeks with no perceptible effect upon the lesions. In November 1979 each of them had extended deep satellite lesions into the surrounding subcutaneous tissues, and one of these broke through to the surface, discharging buff-coloured creamy pus. In February 1980, after five weeks' incubation at 30°C, a Lowenstein Jensen culture of pus draining from the

original abscess on the right thigh yielded a growth of an atypical mycobacterium. The organism was identified by Dulwich Regional Tuberculosis Laboratory and by Dr J L Stanford of the Middlesex Hospital as *M chelonae*, var *abscessus*, and was subsequently shown to be resistant in vitro to isoniazid, PAS, ethambutol, rifampicin, and co-trimoxazole but sensitive to erythromycin. At about the time the organism was isolated the patient developed two further inflamed lesions, one on each buttock close to the ischial tuberosities in areas remote from any insulin injection sites. Treatment was begun with a combination of oral erythromycin and co-trimoxazole and the lesions have steadily resolved since.

Comment

M chelonae is a rare human pathogen. When it is pathogenic it may cause injection abscesses¹ with a prolonged incubation period of up to several months or, less commonly, severe generalised infections in immunosuppressed patients.² Probably our patient's initial abscess resulted from organisms introduced by an insulin injection. We cannot be sure if the second abscess was caused in the same way, since infected lesions may develop at sites of trauma during a period of *M chelonae* septicaemia, and this could account for the third and fourth abscesses. Our patient, normally fastidious in her injection technique, used a non-disposable syringe and disposable needles. Each needle was used for two injections. The syringe was boiled weekly and stored, with the needle, in hypochlorite. Unfortunately, in her twice-weekly preparation of hypochlorite she often used boiling water, which would have driven off the chlorine and rendered the solution inactive. She now uses a new disposable needle and syringe for each injection.

One must accept that if a syringe and needle are reused without

sterilisation there is a risk of infection. Diabetics commonly keep their syringes in spirit although similar practices have been abandoned in hospital, where sterile instruments are now stored dry. It would seem logical for diabetics to store their syringes—glass or plastic—in the same way. Thirty patients reported by Greenough *et al*³ used a single plastic syringe, stored dry in a domestic refrigerator, for up to two months without infection. Nevertheless, using a new needle, as these patients did, does not eliminate the risk of infection since the syringe nozzle may become infected when an infected needle is removed.⁴ The ideal solution would be for diabetics to use a new sterilised syringe and needle for each injection. But the bacteriological risk is clearly small and is not the only consideration. It must be balanced against the cost of meeting it.

¹ Inman PM, Beck A, Brown AE, Stanford JL. Outbreak of injection abscesses due to *Mycobacterium abscessus*. *Arch Dermatol* 1969;100:141-7.

² Tice PM, Solomon RJ. Disseminated *Mycobacterium chelonae* infection: response to sulfonamides. *Am Rev Respir Dis* 1979;120:197-201.

³ Greenough A, Cockcroft PM, Bloom A. Disposable syringes for insulin injection. *Br Med J* 1979;ii:1467-8.

⁴ Evans RJ, Spooner ETC. A possible mode of transfer of infection by syringes used for mass inoculation. *Br Med J* 1950;iii:185-8.

(Accepted 14 August 1980)

Department of Clinical Bacteriology and Virology and Unit for Metabolic Medicine, Guy's Hospital, London SE1 9RT

P G JACKSON, MRCP, senior registrar

H KEEN, MD, FRCP, professor

C J NOBLE, MSC, MB, senior registrar

N A SIMMONS, FRCPATH, consultant microbiologist

Phenylbutazone overdose: abnormal metabolism associated with hepatic and renal damage

Phenylbutazone poisoning is uncommon and apparently has not been reported in the United Kingdom. We report the case of a patient with very abnormal phenylbutazone metabolism in whom hepatic and renal damage occurred.

Case report

A previously healthy 17-year-old girl was admitted 16 hours after allegedly taking 40 phenylbutazone tablets (8 g). She did not drink alcohol or take any drugs regularly and the tablets had belonged to her mother. The patient initially fell asleep but woke feeling very unwell with severe nausea. On examination she was drowsy with nystagmus. Intravenous fluids were started because of persistent vomiting. The next morning she developed a sinus tachycardia (120/min) and was hyperventilating with a respiratory alkalosis (arterial H^+ 32 nmol/l (pH 7.49), P_{CO_2} 2.9 kPa (22 mm Hg), and bicarbonate 17 mmol(mEq)/l). Malaise, drowsiness, and vomiting continued for three days, when jaundice appeared with biochemical evidence of liver damage (figure). The maximum plasma bilirubin concentration, alanine aminotransferase activity, and alkaline phosphatase concentration were 125 μ mol/l (7.4 mg/100 ml), 471 U/l, and 136 U/l on the 4th, 6th, and 8th days respectively. Tests for hepatitis B surface antigen and antibody were negative and there was no evidence of haemolysis or other haematological toxicity. The plasma albumin concentration remained within normal limits and there was no clinical evidence of fluid retention. Despite adequate fluid replacement there was early but transient impairment of renal function with proteinuria, a reduction in creatinine clearance to 34 ml/min, and a raised plasma urea concentration (figure).

The patient subsequently improved and was discharged on the sixth day, although liver function tests were still abnormal at follow-up nine days later. Serial plasma concentrations of phenylbutazone and its metabolites oxyphenbutazone and γ -hydroxyphenylbutazone were measured simultaneously by high performance liquid chromatography.¹ The initial plasma phenylbutazone concentration was grossly raised at 670 μ g/ml and the half life, normally about three days, was very short at 23 hours. In contrast to the pattern of metabolites in plasma after therapeutic doses of phenylbutazone² the relative proportions of oxyphenbutazone and γ -hydroxyphenylbutazone were reversed, with the former disappearing rapidly and high concentrations of the latter, which rapidly exceeded those of the parent drug (figure).

Comment

The clinical features in this patient resembled those described in the few previous reports of phenylbutazone intoxication, although jaundice does not always occur. Convulsions and gastrointestinal perforation have also been reported.³⁻⁵ Treatment is of necessity symptomatic. Phenylbutazone is highly bound to plasma proteins and extensively metabolised.² Thus forced alkaline diuresis, dialysis, and haemoperfusion are unlikely to enhance elimination significantly.

Phenylbutazone metabolism was very abnormal in our patient. Despite liver damage the plasma half life was much shorter than

Serial plasma concentrations of phenylbutazone, γ -hydroxyphenylbutazone, oxyphenbutazone, urea, bilirubin, and alanine aminotransferase after phenylbutazone overdose.

Conversion: SI to traditional units—plasma bilirubin: 1 μ mol/l $\approx$ 0.059 mg/100 ml. Urea: 1 mmol/l $\approx$ 6 mg/100 ml.

previously described² and the concentrations of γ -hydroxyphenylbutazone were extraordinarily high. This metabolite normally accounts for under 5% of the total drug and metabolites in plasma after a single therapeutic dose of phenylbutazone, although a somewhat higher percentage has been reported after longer-term administration.² In four other cases of minor overdose of phenylbutazone we found that γ -hydroxyphenylbutazone, oxyphenbutazone, and phenylbutazone accounted on average for 3%, 14%, and 83% respectively of the total in plasma, and the maximum for γ -hydroxyphenylbutazone was 6%. Thus in our patient the pattern of metabolites was completely reversed. Possible explanations include greatly increased production of γ -hydroxyphenylbutazone, concentration-dependent saturation of aromatic hydroxylation, and impaired renal excretion of the γ -hydroxy metabolite. The kinetics, however, were not consistent with saturation, and increased γ -hydroxylation seems the most likely cause in view of the unusually short half life of the parent drug. The hepatotoxicity of phenylbutazone may be mediated through metabolic activation (as with paracetamol), and we are tempted to postulate a relationship between abnormal metabolism of the drug in this patient and subsequent liver damage.