are similar regulations in many parts of the USA, Canada, and South Africa.

No such legislation exists in this country; it has been stated that the number of deaths is comparatively small and that it is difficult to justify safety regulations that affect private property. Accidents that leave a child dead or severely handicapped, however, are a tragedy, particularly when they most probably could have been prevented by simple safety measures. Although these deaths are a relatively small proportion of the 50 or so children aged under 5 who are drowned each year,⁵ they are preventable; it is surely not unreasonable, therefore, to require the owners of swimming pools, who are usually relatively wealthy, to ensure that they are safely enclosed.

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Multiple hepatic abscesses complicating continuous ambulatory peritoneal dialysis

Peritonitis is common during continuous ambulatory peritoneal dialysis^{1,2} and may prove lethal.³ We report a case of relapsing peritonitis complicated by multiple hepatic abscesses.

Case report

A 41-year-old man had been receiving haemodialysis from 1970 to 1977. Continuous ambulatory peritoneal dialysis was started in March 1980 for terminal renal failure due to chronic rejection causing nephrotic syndrome. During the first nine months of continuous ambulatory peritoneal dialysis three episodes of peritonitis were recorded (overall incidence at this unit—one episode every 16 patient-months). After 11 months the patient was admitted to hospital for peritonitis; culture grew no pathogens. Continuous peritoneal lavage with cefalexin, in association with parenteral cefalexin and tobramycin, resulted in clinical improvement and after three days continuous ambulatory peritoneal dialysis was started again with the same antibiotic treatment. Despite the persistence of slight fever, antibiotics were stopped on the 14th day. Three days later, epigastric pain returned, peritoneal effluent became cloudy, and culture was positive for *Staphylococcus albus*, sensitive to cephalosporins. Intraperitoneal and parenteral treatment were given in accordance with "antibiogram," but despite initial relief abdominal pain reappeared after 11 days, localised in the right upper quadrant, radiating to the shoulder; palpation elicited extreme pain at the hepatic border, localised at the intersection of the outer edge of the rectus abdominis. Peritoneal fluid was purulent with a high leucocyte count; intramuscular tobramycin was started. Abdominal echotomography gave negative results.

Nineteen days after operation the patient underwent exploratory laparotomy. A corpuscolate fluid was found in the peritoneal cavity and a small area (8 × 3 cm) of swelling and reddening with microabscesses was found at the lower side of the right hepatic lobe, along the posterior edge (segments V, VII, VIII). Exploration puncture gave scant bloody fluid, but culture was positive for *Staph albus*. The abscesses were too small (1-4 mm in

diameter) to warrant surgical drainage, so the Tenckhoff catheter was removed and replaced by a stylet catheter. Antibiotic treatment included intraperitoneal and intravenous vancomycin and intravenous amikacin for three weeks, and erythromycin orally for four weeks. From the second postoperative day the patient underwent haemodialysis through an external femoral bypass. Thirteen days after operation hepatic scintigraphy showed no alteration attributable to abscess and the patient was discharged without antibiotic treatment. Twelve months after discharge the patient is undergoing haemodialysis and is rehabilitated to grade A 1 (code of the European Dialysis and Transplant Association).

Comment

Gram-positive micro-organisms, particularly *Staph albus*, are the most frequent causal agent of peritonitis during continuous ambulatory peritoneal dialysis.^{1,2} Generally, these agents are responsive to cephalosporins, which are used as initial treatment until the results of bacteriological culture are available.² The case described here illustrates that peritonitis due to *Staph albus* may lead to severe infectious complications, such as hepatic abscess. This patient presented predisposing factors such as nephrotic syndrome and a long period of immunosuppressive treatment before starting continuous ambulatory peritoneal dialysis. We believe that antibiotics were discontinued too soon, before the septic focus in the liver had been eradicated. Staphylococci are not usually the cause of hepatic abscesses.⁴ In the case described micro-organisms presumably reached the liver through the portal vein, though no such occurrence has been reported in patients undergoing continuous ambulatory peritoneal dialysis.

We recommend exploratory laparotomy for making a prompt diagnosis when other methods fail to show the cause of a persisting peritonitis.⁵ This may be of considerable help in establishing appropriate treatment for diseases such as hepatic abscesses which have a poor prognosis. Multiple hepatic abscesses do not lend themselves to adequate surgical drainage, and, though they can sometimes be evacuated by syringe, an aggressive schedule of specific antibiotic treatment maintained for a sufficiently long period remains the best possible way of achieving a cure.

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Myxoedema coma induced by beta-adrenoreceptor-blocking agent

Although the pathogenesis of myxoedema coma is uncertain, precipitating factors such as exposure to cold, infection, administration of central nervous system depressants, alveolar hypoventilation, and hyponatraemia are reported to predispose to its development.¹ Myxoedema coma induced by administration of beta-adrenoreceptor-blocking agents has not, however, been reported. We describe a patient with myxoedema coma induced by a beta-blocker who recovered consciousness after withdrawal of the drug.

Time course of laboratory findings

	Normal range	17 Dec 1981 (before beta-blocker given)	2 Feb 1982 (on admission)	7 Feb 1982 (after withdrawal of beta-blocker)	15 Feb 1982 (after withdrawal of beta-blocker)	15 April 1982 (after treatment with thyroxine)
Total cholesterol (mmol/l)	3.3-6.4	9.9	10.7	10.0	8.4	4.3
Triglyceride (mmol/l)	0.3-1.7	2.5	11.9	1.2	1.5	1.5
Creatinine phosphokinase (mmol/s/l)	0.7-2.6		17.5	9.9	4.1	0.9
Serum glutamic-oxaloacetic transaminase (μ mol/s/l)	0.10-0.30	0.42	0.50	0.33	0.31	0.14
Cardiothoracic ratio (%)		58	66	54	54	48

Conversion: SI to traditional units—Total cholesterol: 1 mmol/l $\approx$ 39 mg/100 ml. Triglyceride: 1 mmol/l $\approx$ 90 mg/100 ml. Creatine phosphokinase: 1 mmol/s/l $\approx$ 60 U/ml. Serum glutamic-oxaloacetic transaminase: 1 μ mol/s/l $\approx$ 133 U/ml.

Case report

A 65-year-old woman was admitted unconscious in February 1982. Until the age of 50 her health had been good, but she had subsequently suffered from constipation, cold intolerance, and retarded thinking. In the year before admission she had gained 10 kg in weight. In December 1981 her doctor had prescribed bufetolol hydrochloride,² a non-cardioselective beta-blocker, 15 mg three times daily for hypertension (186/110 mm Hg). Examination had shown raised serum cholesterol and triglyceride concentrations and raised serum glutamic-oxaloacetic transaminase activity. Electrocardiography showed a low voltage with sinus rhythm (heart rate 70 beats/min) and chest radiography a slight increase in cardiothoracic ratio. In January the cardiothoracic ratio was 63% and she complained of general fatigue; two hours before admission she became comatose.

On admission she was semiconscious with temperature 35.5°C, blood pressure 160/105 mm Hg, and respiration 14 breaths/min. Her skin was dry, and facial oedema was noted. Neurological signs were not evident. Plasma electrolyte concentrations were sodium 141 mmol(mEq)/l, potassium 3.2 mmol(mEq)/l, and chloride 101 mmol(mEq)/l. Plasma glucose concentration was 7.2 mmol/l (129 mg/100 ml) and blood urea nitrogen 1.8 mmol/l (11 mg/100 ml). Creatine phosphokinase activity and triglyceride concentrations were greatly increased.

The table shows the time course of plasma lipid concentrations, enzyme activity, and cardiothoracic ratio. The blood picture was not characteristic of inflammation. Blood-gas analysis showed pH 7.35, oxygen pressure 11.3 kPa (84.5 mm Hg), carbon dioxide pressure 5.2 kPa (39.2 mm Hg), and bicarbonate concentrations 21.3 mmol(mEq)/l. Computed tomography of the brain and electroencephalography showed no abnormality. Chest radiography showed distinct cardiac enlargement and pulmonary congestion, and electrocardiography showed a low voltage with regular sinus rhythm (heart rate 76 beats/min). Thyroid function was: triiodothyronine concentration 1.0 nmol/l (0.69 ng/ml) (normal range 1.3-2.9 nmol/l; 0.85-1.9 ng/ml); thyroxine 7.7 nmol/l (0.6 μ g/100 ml) (normal range 59-141 nmol/l; 4.6-11.0 μ g/100 ml); thyroid-stimulating hormone 1736×10^{-3} IU/l (normal range $0.6-0 \times 10^{-3}$ IU/l); basal metabolic rate (measured at fourth hospital day) -17%, and antimicrosomal antibody 1/25 600 dilutions. Six hours after admission she gradually regained consciousness.

We followed up this patient without any special medication as she was fully conscious and was able to walk when hypothyroidism was diagnosed. Her condition was considered to be attributable to ill-advised administration of a beta-blocker. On the sixth hospital day radiography showed no pulmonary congestion and the heart size was reduced. Enzyme and lipid values decreased until the sixth hospital day. Plasma lipid values thereafter remained constant, although creatine phosphokinase activity gradually decreased until the 14th hospital day. She was then treated with thyroxine and her condition returned to normal.

Comment

Central nervous system depressants such as sedatives³ or narcotics⁴ cause myxoedema coma in patients with unrecognised hypothyroidism. We could find no previously reported precipitating factors except cardiac failure in our patient.

Myxoedema coma induced by a beta-blocker has not previously been reported. In our patient, however, there was clearly a relation between the beta-blocker and the myxoedema coma as after withdrawal of the drug she became conscious, pulmonary congestion and cardiac enlargement disappeared, and the raised plasma lipid and enzyme values returned to normal without treatment. The mechanisms must presumably be related to the action of beta-blockers on cardiac function, depression of the central nervous system (not prominent in this case judging from the respiratory state), or the effect of further peripheral blocking of beta-receptors on the reduced beta-adrenergic responsiveness in hypothyroid states.⁵

Careful screening for the presence of unrecognised hypothyroidism should be undertaken before the administration of beta-blocker to prevent the development of myxoedema coma.

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MEDICINES APPROPRIATED TO THE BRAIN—Before we treat of medicines appropriated to the brain, it is requisite that we describe what the nature and affection of the brain is. The brain which is the seat of apprehension, judgment, and memory, the original of sense and motion, is by nature temperate, and if so, then you will grant me that it may easily be afflicted both by heat and cold, and it is indeed more subject to affliction by either of them, than any other part of the body, for if it be afflicted by heat, sense and reason, it is immoderately moved, if by cold, they languish, and are dulled, to pass by other symptoms which invade the head, if the brain be altered from its proper temper. Also this is peculiar to the brain, that it is delighted or offended by smells, sights, and sounds, but I shall meddle no further with these here, because they are not medicines.

Cephalical Medicines may be found out from the affections of the brain itself. The brain is usually oppressed with moisture in such afflictions; therefore give such medicines as very gently warm, cleanse, cut, and dry: but withal, let them be such as are appropriated to the head, such as physicians say (by an hidden quality) strengthen the brain. Again, if you consider the situation of the brain, you shall find it placed in the highest part of the body, therefore it is easily afflicted with hot vapours: this punishes a man with watching and headache, as the former did with sottishness and sleepiness, in such cases use such *Cephalecs* as gently cool the brain. To make *Cephalecs* of *Narcotics*, or stupifying medicines, is not my intent, for I am confident they are inimical both to the brain and senses. Of these, and such medicines as also purge the brain, I shall speak by and by. To return to my purpose. Some *Cephalics* purge the brain, some heat it, some cool it, some strengthen it; but how they perform this office peculiarly to the brain, most physicians confess they could neither comprehend by reason, nor describe by precepts, only thus, they do it by an hidden quality, either by strengthening the brain, thereby descending it from diseases, or by a certain antipathy between them and the diseases incident to the brain. Lastly, For the use of *Cephalics*, observe, if the brain be much afflicted, you cannot well strengthen it before you have purged it, neither can you well purge the brain before you have cleansed the rest of the body, it is so subject to receive the vapours up to it; give cooling *Cephalics* when the brain is too hot, and hot *Cephalics* when it is too cold. Beware of using cooling medicines to the brain when the crisis of a disease is near: how that time may be known, I shall (God assisting me) instruct you hereafter, let it suffice now, that according as the disease afflicting your head is, so let your remedy be. (Nicholas Culpeper (1616-54) *The Complete Herbal*, 1850.)