formulation as blue-coloured, sweet-tasting tablets, and in the fact that it is prescribed for migraine, a relatively non-serious disease. Dixarit is probably regarded as a non-dangerous drug, being stored in places accessible to children, while, by contrast, the antihypertensive Catapres is treated with appropriate respect. We hope that this article will alert doctors prescribing Dixarit so that they can issue appropriate warnings to parents to keep this drug out of the reach of young children.

When the size of this problem became known, the manufacturers alerted pharmacists through a letter published in the *Pharmaceutical Journal*,⁵ and, more recently, a warning has been incorporated in the label on the standard package for Dixarit. As yet, we have detected no decrease in the rate of new cases, possibly because the new labels did not reach all retailers until late 1977. Probably, however, further measures will be needed, and safety packaging is being considered.

We thank those doctors who replied to our questionnaire and thus made possible this survey. We are also grateful to Dr J W Bell and Dr D Segal of Boehringer Ingelheim Ltd Bracknell, UK for their advice and co-operation.

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Myositis and acute respiratory infection

Acute viral infections are commonly accompanied by myalgia. Electromyographic examinations in these cases have failed to show pathological findings.¹ We describe one patient with positive EMG findings in association with acute viral infection.

Case report

The patient was a 31-year-old man, who one week after the onset of non-productive cough, mild fever, and tiredness developed coarse muscle twitching in his fingers. Within one week the twitches had affected several other muscles in the fingers and toes, and he also complained of paraesthesiae in the thenar eminences and mild muscle pains in the hands and feet. His respiratory symptoms subsided after two weeks and the mild fever after three weeks, although muscle twitches persisted for four months. No abnormal signs whatsoever were found on neurological examination. During the first three weeks after the onset of the disease the blood sedimentation rate was 2-4 mm in the first hour, and the blood leucocyte count $3.4-3.8 \times 10^9/l$. There was an insignificant rise in the serum antibody titre of adenoviruses (1/16-1/64) but no other changes in the tested viral antibody titres. The serum electrolytes and transaminase concentrations were normal. Three and four weeks after the start of the disease the serum creatine kinase concentrations were 140 and 41 U/l (at 37°C) respectively. Two months from the onset the blood sedimentation rate was 2 mm in first hour, and the blood leucocyte count $7.6 \times 10^9/l$. The ECG and chest x-ray appearances were normal.

Electromyographic findings—The first EMG (taken three weeks after the initial symptoms) was abnormal. The proximal muscles of the arms, predominantly both the biceps, showed insertion activity of prolonged duration and abundant abnormal spontaneous activity as fibrillation potentials and positive sharp waves. Bizarre high-frequency potentials were also present. The motor unit action potentials were normal. A normal interference pattern was detectable with maximal effort. The distal muscles of the arms showed less spontaneous activity, and in the legs only the extensor digitorum brevis showed a few fibrillation potentials. The motor conduction velocities in the median and peroneal nerves were normal, and no fasciculation potentials were found. On repeat EMG examination one month later the pathological findings were fewer: prolonged insertion activity persisted, but spontaneous activity had diminished appreciably. The motor unit action potentials remained normal.

Comment

This patient's illness was considered to be an atypical myositis associated with viral infection, although the virus was not identified. In 1957 Lundberg¹ described an epidemic of 74 cases of an influenza-like syndrome which was followed in three to five days by severe myalgia of both calves, which remitted spontaneously within one week. The same type of myositis has been reported as being caused by influenza B virus,² A2 Hong Kong virus,³ and influenza A virus.⁴ Nearly all of these patients have been children. The present case differed clinically in the course of the illness: at the time of the patient's illness, moreover, there was no epidemic of influenza in Finland.

The results of EMG examination were normal in the three cases examined by Lundberg.¹ In the present case the findings were clearly abnormal, although the presence of normal motor unit potentials made them different from those classically described in myositis, most probably because of the acute nature of the disease. No EMG findings of this kind have been reported in conjunction with acute respiratory infection. Hence we believe that the muscular symptoms which accompany acute viral respiratory infection may be partly attributable to inflammatory irritation of the muscle.

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Digoxin prescribing: an evaluation of clinical judgment

When prescribing digoxin most doctors rely on intuition. We report the variation in mean steady state serum digoxin concentration after intuitive dosing in a large group of patients. Comparison is made with the outcome expected had other prescribing methods been used.

Patients, methods, and results

A total of 129 patients in the Manchester region (67 men, 62 women; mean (± 1 SD) age 59 ± 12 , weight 62 ± 13 kg, creatinine clearance 60 ± 23 ml/min) receiving maintenance digoxin were studied. Of these, 110 were outpatients and 19 were acute medical admissions. The daily dose (0.27 ± 0.11 mg) had been selected previously by a general practitioner or a hospital doctor. We prescribed this as Lanoxin (Wellcome Medical Division Ltd, UK and Ireland Region), to be taken at 1000 and made every effort to encourage compliance. After allowing three weeks to attain "steady state" conditions blood samples for digoxin radioimmunoassay were taken on an average of four visits per patient. Results were expressed as estimated mean steady state concentration.¹

The above results were compared with those expected had alternative prescribing methods been used. The alternatives considered were a fixed daily dose of 0.25 mg (method A); a one-compartment model² (method B), whose predictive value ranked higher than that of other pharmacokinetic models studied¹; and the score shown in the figure (method C). The expected concentrations were calculated on the assumption that a proportional relationship exists between the dose given to an individual and the digoxin concentration he achieves. Excluded from the comparison were five patients who had not been receiving Lanoxin and ten who were found to be intolerant of digoxin at serum concentrations below 1.28 nmol/l (1.00 µg/l).

The mean serum digoxin concentration achieved by intuitive dosing (1.94 nmol/l (1.5 µg/l)) was similar to that expected for method A (2.05 nmol/l (1.6 µg/l)), method B (1.73 nmol/l (1.4 µg/l)), and method C (1.78 nmol/l (1.4 µg/l)). The variance in the logarithm of the concentrations achieved, 4.62×10^{-2} (a $\log_{10}$ transformation being used to approach a normal distribution), was not different ($P > 0.7$) from that expected using A, 4.33×10^{-2} , but differed ($P < 0.0001$) from that expected using B, $2.38 \times$

10^{-2} , or C, 1.61×10^{-2} (Pitman's test for comparison of correlated variances).³ Expressing the findings in practical terms, we found that 55.2% of 114 patients given an intuitive dose achieved concentrations within the desired range, 1.28 to 2.56 nmol/l (1.00 to 2.00 µg/l), as compared with an expected 52.6% using method A, 59.7% using B, and 76.3% using C. Methods B and C would have the additional advantage of fewer patients exceeding this range (method B 11.4% and C 7.9% as against intuitive dosing 21.1% and A 20.2%).

Start with a score of zero		The table shows the maintenance dose corresponding to a given score	
For a patient		Total score	Tablets prescribed
not in hospital	add 17	0-13	One 0.0625 mg daily
male	add 7	14-24	One 0.0625 mg/two 0.0625 mg on alternate days
aged less than 70 years	add 8	25-37	One 0.125 mg daily
of weight 55 to 64 kg	add 5	38-49	Three 0.0625 mg daily
of weight 65 to 74 kg	add 10	50-61	One 0.25 mg daily
of weight 75 kg and above	add 15	62-73	Three 0.125 mg daily
not housebound or confined to wheelchair	add 12	74-85	Two 0.25 mg daily*
in sinus rhythm	add 4	86-100	Three 0.25 mg daily*
not receiving diuretic therapy	add 4	*Doses should be divided between two or three administration times to avoid early gastrointestinal symptoms.	
with a serum creatinine below 70 µmol/l	add 33		
with a serum creatinine 70 to 99 µmol/l	add 11		
with a serum creatinine 100 to 199 µmol/l	add 4		
Total score for the patient			

A score for prescribing digoxin. This was derived¹ from data on the 129 patients described

Comment

We have presumed that no doctor intended to exceed a mean steady state serum digoxin concentration of 2.56 nmol/l (2 µg/l).⁴ If heart failure or atrial fibrillation remains uncontrolled at this concentration, a diuretic in the first case or a beta-blocker in the second should be added rather than risk cardiotoxicity by increasing the dose of digoxin. Concentrations below 1.28 nmol/l (1 µg/l) may be beneficial in heart failure and are indicated in those with slow heart rates or symptoms of toxicity at higher concentrations.⁵ Such patients were excluded from the comparison.

Our results show that doctors with access to patients and their case records were unable to select a dose to give a concentration between 1.28 and 2.56 nmol/l (1-2 µg/l) any more frequently than if they had simply prescribed 0.25 mg daily to all. The relative value of a conventional pharmacokinetic model in prescribing digoxin and the potential of an empirically derived score are shown. A multicentre trial to evaluate the score in other groups of patients is in progress.

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Management of embolised central venous catheters

The infraclavicular subclavian vein has become the favoured site for inserting central venous lines for pressure monitoring, drug administration, and parenteral feeding since its introduction in 1952.¹ With the increasing use of central lines has developed the problem of polyethylene catheter embolism.²⁻⁵ The infraclavicular approach is easy to learn and quick to perform because of the constancy of the subclavian vein anatomy. Operators should be aware of the potential for catheter embolism especially when polyethylene catheters are inserted infraclavicularly through a metal needle. This report follows the referral of five patients to this department in a period of six months with catheter fracture or embolism and describes the commonest technique of removal.

Patients and management

Three patients were referred within a week of open heart surgery. Each had an infraclavicular subclavian venous catheter inserted preoperatively. Two patients had catheters inserted after major abdominal surgery. A standard approach between the clavicle and the first rib was employed in each case. A needle catheter intravenous placement unit* was used in which, after percutaneous insertion of a 14 gauge needle into the vein, a polyethylene catheter was passed through the needle which was then withdrawn leaving the catheter in situ. At the time of removal some three to ten days later the catheters had fractured and either embolised (2) or remained fixed in the subclavian vein (3).

A Seldinger puncture of the femoral vein was performed under local anaesthetic. The venotomy was dilated through a 8 mm skin incision with a 9F gauge dilator. A 9F gauge grey Kifa catheter† (110 cm long, internal diameter 2.80 mm) was then advanced under fluoroscopic control over a Teflon guide wire to the inferior vena cava. The catheter was manufactured without a tapered tip. A 260 cm long spring guide wire‡ (0.635 mm diameter) was then doubled over and reshaped so that on insertion through the distal end of the catheter a snare would unfold^{2,3} (figure). The snare was then manipulated over the distal catheter fragment and drawn tight. The Kifa catheter, snare, and catheter fragment were then withdrawn as an entity through the puncture site and pressure applied for ten minutes. All fragments were easily removed with less than 15 minutes of snare manipulation required.

Kifa catheter and snare assembly.

Comment

Polyethylene catheter insertion through a hollow needle carries the risk of severing the catheter at time of insertion. Often the catheter is just "scored" and after lying in the blood for a few days it becomes brittle and is likely to fracture on withdrawal.^{4,5} Catheter fracture is even more likely using a hollow needle introducer via the infraclavicular approach as the catheter makes a sharp bend at the point of entry to the subclavian vein.⁵

Many cases of catheter embolism have been followed with no complication but mortality rates up to 40% have been reported for unremoved emboli, especially in ill patients.⁴ Early deaths are related to arrhythmias, especially with right ventricular emboli. Thrombosis, clot embolism, cardiovascular perforation, and infection are less frequent. The high-risk sites for embolic fragments are the right

*Bard I-Cath, Bard International Ltd, Sunderland, England.

†Kifa, Stockholm, Sweden.

‡William Cook, Denmark.