abnormality. The table shows that all subjects had raised serum TSH and low T₄ concentrations. Cases 1-5 had immeasurable or low serum T_g values compatible with depressed or absent thyroid function. Cases 6-10 had normal or high T_g values, but in each case serum T₃ concentration was normal (in one case, high). This implies the presence of functioning thyroid tissue and raises a query about the nature and degree of their hypothyroidism.

Results in children with suspected hypothyroidism

Case No	Age	Serum T ₄ (nmol/l)	Serum T ₃ (nmol/l)	TSH (mU/l)	T _g (μg/l)
1	12 months	30	1.7	250	10
2	9 days	6	1.6	250	12
3	5 years*	12	1.5	75	Undetectable
4	9 years	<10	1.4	170	Undetectable
5	3 days	10	0.8	150	7
6	8 months	7	3.4	290	Undetectable
7	6 years	60	5.4	84	240
8	5½ years	18	3.3	>200	120
9	9 days	16	3.2	265	43
10	1 month	30	2.2	780	50
		49		>40	66
Normal range:		60-150	1.6-3.4	<5	10-130

*Two separate samples on same child. Sera kindly supplied by Dr R Illig, Kinderspital, Zurich, and Dr D B Grant, Hospital for Sick Children, Great Ormond Street, London.

Conversion: SI to traditional units—T₄: 1 nmol/l ≈ 0.078 μg/100 ml. T₃: 1 nmol/l ≈ 0.65 ng/ml.

Thyroxine is usually given promptly when hypothyroidism is suspected in the hope of preventing mental deterioration. We do not question the correctness of this approach, but in view of our demonstration of functioning thyroid tissue in five out of 10 suspect patients, we wish to emphasise the need for later full re-evaluation of the diagnosis. Serum thyroglobulin assay appears to provide a useful and reliable indication of overall thyroid secretory capacity.

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Acute poisoning with Distalgesic

Although the dangers of dextropropoxyphene have been emphasised,^{1,2} it is still widely prescribed, particularly in preparations such as Distalgesic in which it is combined with paracetamol. We report a case of acute poisoning with this preparation.

Case report

A 15-year-old girl, apparently well a few minutes previously, was found on the floor unconscious and cyanosed. A friend with first-aid knowledge found her pulseless and apnoeic and, after a blow on the chest, began mouth-to-mouth artificial respiration, which produced a palpable radial pulse and spontaneous respiratory efforts. The girl was taken within 20 minutes to hospital. During the journey she had convulsive movements. On arrival these stopped and she had a cardiorespiratory arrest. Full resuscitative measures were instituted, including endotracheal intubation and intermittent positive-pressure ventilation, intravenous sodium bicarbonate, electrical defibrillation, and, when an ECG showed asystole, intravenous adrenaline. After resuscitation the ECG showed sinus rhythm but her blood pressure was unrecordable. She remained deeply unconscious with fixed, dilated pupils and areflexia. She made only minimal respiratory gasping efforts, so intermittent positive-pressure ventilation was continued. When transferred to the intensive therapy unit she did not improve, despite adequate

ventilation of both lungs. Chest x-ray examination was normal. Urinary catheterisation showed virtual anuria over the next three hours, and her central venous pressure was 30 cm H₂O (2.94 kPa). The ECG at this stage (figure) showed first-degree heart block, complete right bundle-branch block, and extreme axis deviation.

Above: ECG in patient with acute dextropropoxyphene poisoning. Below: ECG in same patient 3 hours after start of naloxone treatment.

Four hours after initial presentation the patient's mother reported that about 50 tablets of Distalgesic were missing. Naloxone 1.2 mg intravenously produced an immediate increase in respiratory efforts (although insufficient to allow withdrawal of artificial ventilation) and a palpable pulse associated with a rise in blood pressure to 90/50 mm Hg. The central venous pressure dropped to 11 cm H₂O (1.0 kPa). Within an hour 200 ml urine was voided. The ECG returned virtually to normal within three hours (figure) and was completely normal the next day. Although naloxone 0.8 mg was continued subcutaneously every four hours, intermittent positive-pressure ventilation was required for 20 hours. She remained unconscious for 70 hours. Her plasma paracetamol concentration four hours after admission was 2300 μmol/l (345 mg/l). A subsequent calculated plasma paracetamol half life of eight hours indicated a high risk of liver damage. Cysteamine was given intravenously and liver function tests were monitored over the next 10 days. There was a slight rise in serum alanine transaminase (serum ALT; GPT) concentration, but this later returned to normal. She was discharged home physically normal after 10 days.

Comment

The clinical features were typical of acute poisoning with dextropropoxyphene, although the cardiovascular depression (with pronounced electrocardiographic changes) was greater than usual. The dramatic improvement after naloxone suggests a direct toxic effect of dextropropoxyphene. Without such an effective antidote she would almost certainly have died. Probably cysteamine treatment prevented severe liver damage secondary to poisoning with paracetamol. Although naloxone is known as an effective antagonist of the respiratory depression caused by dextropropoxyphene,³ this case shows that it can also antagonise the cardiac toxicity. The continuing popularity of preparations containing dextropropoxyphene is difficult to understand. There are many simpler, cheaper, and potentially less toxic preparations with probably greater therapeutic value.⁴

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