after the 41st week, against 27.4% of control cases.

The frequency of CPS was lower in boys born preterm. This lower incidence of CPS in premature low birthweight infants has been reported previously (e.g. Henderson, Brown, and Taylor, 1952; Wilson, 1960).

The higher birthweight in patients with pyloric stenosis has previously been commented on by Malmberg (1949) and by Shim et al. (1970); but this is the first demonstration that this higher birthweight is not due to longer gestation. The higher incidence of CPS in the higher birthweight group is consistent with the observation that CPS is more frequent in muscular men (Carter, 1961).

The family findings are presented in Table III, and in general correspond to the more precise information obtained by Carter and Evans (1969).

### Summary

The incidence of operated congenital pyloric stenosis was 1.46/1000 livebirths in Budapest, 1962–1967. In males, the frequency was significantly higher in infants with birthweight over 3500 g, particularly so when over 4000 g. This higher frequency cannot be explained by the longer mean gestational period.

### References


### Accidental Poisoning with Thyroid Extract Treated by Exchange Transfusion

Ingestion of large quantities of thyroid hormone may reproduce the symptoms of thyrotoxicosis (Levy and Gilger, 1957; Schottstaedt and Smoller, 1966; Funderburk and Spaulding, 1970). Two sisters were observed after accidental ingestion of large amounts of thyroid extract, and one was treated by exchange transfusion.

### Case Report

At 11.30 hours the mother of two girls aged 3 and 2 found them swallowing some tablets of thyroid extract. It was calculated that the 2 girls had swallowed 38 tablets of 50 mg or a total of 1900 mg. At 1300 hours gastric lavages were performed and the children were referred to hospital.

On admission at 1500 hours, the elder girl (Case 1, 12 kg) had a temperature of 37 °C; she was agitated and had a sinus tachycardia of 150/min with a blood pressure of 110/60 mmHg. No other symptoms were observed. The younger girl (Case 2, 11 kg) showed no clinical signs.

The white blood count in Case 1 was 17,000/mm³ with 72% neutrophils, and in Case 2 11,900/mm³ with 47% neutrophils. A mild hypokalaemia (3.1 mEq/l) was noted in both.

Gastric lavage was performed again and a laxative given. At 1900 hours we received the results of the serum thyroxine (T₄) and of the T₃ resin uptake (T₃-RU) of the blood drawn at the time of admission. The T₄ values were (Table) Case 1 20 μg I/100 ml, Case 2 12 μg I/100 ml.

Both children were treated with barbiturates and antipyretics. With the intention of hindering develop-
ment of thyrotoxicosis in Case 1, it was decided to perform transfusion. This was started at 2100 hours and completed twelve hours later when 3480 ml of blood had been exchanged. During the procedure the child showed a mild sinus tachycardia, and a few febrile peaks. T₄ and T₃RU were estimated every four hours (Table).

The results of the exchange transfusion were considered fairly good: T₄ fell from the initial 20 μg I/100 ml to 11.6 μg I/100 ml, and the T₃RU from 41% to 33% by the end of the transfusion.

These figures may be compared with Case 2, where without active treatment the T₄ level remained at 11-12 μg I/100 ml over the 34 hours after ingestion.

Next morning both girls showed mild agitation, slight fever, and tachycardia with occasional extrasystoles. Therefore, reserpine (0.5 mg, b.d., i.m.) and hydrocortisone (5 mg/kg per day, i.v.) were added to the treatment.

On the fourth day the clinical status of the sisters was practically normal; the administration of reserpine was stopped and hydrocortisone gradually withdrawn. The T₄ level returned to normal on the third day in Case 1 and on the fifth day in Case 2.

### Discussion

Although thyroid hormone intoxication is a not infrequent cause of poisoning in children, surprisingly few cases have been documented (Stangne and Henske, 1955). Jahr in 1936 described 3 cases with an apparently benign clinical course. The young boy described by Levy and Gilger (1957) was in acute distress for 12 hours immediately after ingestion of 3200 mg desiccated thyroid. Funderburk and Spaulding (1970) described a 3-year-old boy whose severe symptoms appeared much later, only between the third and the fifth days after ingestion of L-thyroxine. From the reported cases, children's reactions to thyroid hormone poisoning are unpredictable, and if symptoms appear at all, they may do so early or late. Children are said to tolerate thyroid intoxication much better than adults (Schottstaedt and Smoller, 1966).

A high serum level of T₄ was observed in one of our cases: this will only reflect the rate of absorption of the thyroid hormone but will not predict the clinical effects of the intoxication.

It is interesting to note that Case 1 had, at the end of the exchange transfusion, the same symptoms as her sister (Case 2). At that time the T₄ values of the two cases were almost identical. It is likely that the exchange transfusion did benefit the clinical course of this patient by decreasing her extrathyroidal thyroxine pool. Blood exchange is known to be an effective means of reducing the amount of thyroxine bound to plasma proteins (Ashkar et al., 1970). Peritoneal dialysis should also give good results (Herrmann et al., 1971). In both our cases, the clinical situation returned to normal on the fourth day of observation; this may have been related to the half-life of thyroxine which is between 3-9 and 5-9 days in children (Haddad, 1960).

We conclude that since it is difficult to predict the consequences of thyroid poisoning, as the duration, intensity, and delay in onset of symptoms are variable, active measures such as exchange transfusion or peritoneal dialysis should be considered.

### Summary

Two girls aged 2 and 3 ingested a total of 1900 mg thyroid extract. The older girl had the higher serum T₄ level and was treated by means of exchange transfusion.

### Table

<table>
<thead>
<tr>
<th>Time After Ingestion</th>
<th>Exchange Transfusion</th>
<th>3 (dy)</th>
<th>5 (dy)</th>
<th>7 (dy)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>T₄, μg I/100 ml</td>
<td>20.0</td>
<td>19.0</td>
<td>16.3</td>
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<tr>
<td></td>
<td>T₃, resin uptake (%)</td>
<td>37.0</td>
<td>41.0</td>
<td>39.0</td>
</tr>
<tr>
<td>Case 1</td>
<td></td>
<td>11.6</td>
<td>11.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.0</td>
<td>8.9</td>
<td>7.4</td>
</tr>
<tr>
<td></td>
<td>T₄, μg I/100 ml</td>
<td>12.0</td>
<td>11.6</td>
<td>12.2</td>
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<td>T₃, resin uptake (%)</td>
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<tr>
<td>Case 2</td>
<td></td>
<td>11.0</td>
<td>8.5</td>
<td>7.6</td>
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</tbody>
</table>

### References


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10w
Raised Immunoglobulin Levels and Thrombocytosis in Infantile Cortical Hyperostosis

Infantile cortical hyperostosis is characterized by swelling of soft tissues and cortical thickening of underlying bone, usually within the first 3 months of life. The mandible is almost always involved (Caffey, 1961). Fever is a common finding and pseudoparalysis has been observed in many patients (Gwinn and Barnes, 1967). Constant laboratory findings include a raised erythrocyte sedimentation rate, a raised serum alkaline phosphatase, and anaemia. Complete recovery is usual, though occasionally recurrences or death have been reported.

Case Report

A male infant, aged 9 weeks, was admitted on 12 February 1970 with a 7-day history of not moving his right arm. He had been a normal delivery at term after an uneventful pregnancy. His birthweight was 4·09 kg. He had progressed satisfactorily up to the onset of the recent illness. He was the youngest of 5 children; the elder sibs and the parents were healthy and showed no abnormal features. He was pale and had an unusually broad face, particularly the lower jaw. There was no obvious lesion visible to account for the lack of movement in the right arm. His weight on admission was 5·34 kg.

X-rays confirmed the suspicion that the patient had infantile cortical hyperostosis (Fig.). There was marked periosteal thickening of both mandibles, clavicles, scapulae, and of the anterior rib ends. Slight periosteal thickening along the shafts of both femora, tibiae, and humeri was also observed.

The following investigations were carried out: alkaline phosphatase 40 K-units %; serum calcium 9·1 mg/100 ml; serum phosphate 6·7 mg/100 ml; Wassermann reaction negative; uric acid 3·7 mg/100 ml; blood urea 16 mg/100 ml; serum iron 15 µg/100 ml; serum vitamin B12 500 pg/ml; serum folic acid 6·1 ng/ml; throat swab, no pathogenic growth; faeces, no pathogenic growth; urinalysis normal; osmotic fragility test, mean corpuscular fragility 0·44%; Coombs test negative; R.A. Latex test weak positive; Rose Waaler positive to 1/48; L.E. Latex serological test negative; antinuclear test negative; thyroid Latex tanned red cell agglutination test and complement-fixation tests for thyroid antibodies negative; parietal cell antibody test negative.

A summary of haematological investigations is shown in Table I. On admission the patient was anaemic and had an ESR of 44 mm/hr. The white cell count

<table>
<thead>
<tr>
<th>Date</th>
<th>Hb (g/100 ml)</th>
<th>WBC/mm³</th>
<th>Platelets/mm³</th>
</tr>
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<td>13 February 1970</td>
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<td>8,800</td>
<td>1,200,000</td>
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<tr>
<td>25 February 1970</td>
<td>8·7</td>
<td>13,100</td>
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</tr>
<tr>
<td>12 March 1970</td>
<td>7·9</td>
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<td></td>
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<tr>
<td>28 April 1970</td>
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<td>9,200</td>
<td>620,000</td>
</tr>
<tr>
<td>4 September 1970</td>
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<td>10,400</td>
<td>487,000</td>
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