scored according to the method shown in the Table, a modification of the method used by Pierson et al. (1974).

Results

Pulses, respiratory rates, and clinical scores are shown in the Figure. No child showed any significant deterioration after starting treatment, and the condition of all children improved steadily after one or two hours. No statistically significant difference (Student's t test) between the aminophylline and salbutamol groups was found, except for pulse rates at 18 and 24 hours, when salbutamol appeared to cause a relative tachycardia.

Discussion

Clinical signs improved in all children from starting treatment, and pulse and respiratory rates improved after 2 hours. All children were unequivocally better at 6 hours. Their improvement during this period can be largely attributed to either salbutamol or aminophylline. Although the children received hydrocortisone after 2 hours, it is unlikely that this drug began to have any effect for several hours (Collins et al., 1970, 1975). Furthermore our data give no indication that there is a precise time at which recovery is accelerated after the administration of hydrocortisone. The results are similar to those of Pierson et al. (1974) who carried out a study in which hydrocortisone appeared to ameliorate hypoxaemia immediately, but did not affect any other parameters in this way.

Salbutamol caused a tachycardia seen at 18 and 24 hours, but the clinical significance of this is trivial. No patient suffered any cardiac arrhythmia.

Adder bites in children

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SUMMARY 16 children with adder bites were admitted to hospital in Southampton in the years 1969–77. Two children were severely poisoned; these 2, and a 3rd, fulfilled suggested criteria for antivenom administration but they recovered without it. Three children had no treatment, and 9 children no analgesia. Prolonged morbidity was not seen in these children. Careful observation in hospital was the most important factor in management, with early clinical improvement obviating the need for antivenom.

Children are less likely to suffer long-term morbidity after snake bites than adults, but can suffer no less severely from the immediate consequences of the venom. Experience in Southampton shows that snake bites to children are not uncommon, although Reid (1976) suggested they were rare. Controversy still exists about the use of antivenom, particularly in children. The experience with children bitten by Vipera berus, and admitted to hospital in Southampton is reviewed.

Patients

A retrospective study was made of all children, aged 14 years or younger, admitted to hospital in Southampton with a diagnosis of adder bite during the 9 years 1969–77. The diagnosis was accepted if the observed signs were compatible, whether or not a snake was seen. Postdischarge morbidity was ascertained by writing to the families; most of the children were visitors to the area, making follow-up difficult.

During the period 1969–77, 16 children (14 of whom were boys) were diagnosed on clinical grounds as having suffered a snake bite (a snake was not seen in 2 cases), and admitted to hospital. They ranged in age from 14 months to 14 years. All but...
one incident occurred within the New Forest area of Hampshire in the months June, July, or August. The one exception was a boy who kept snakes for a hobby and was bitten at home in April (Case 15). Eight children were bitten on the leg or foot, 7 were bitten on the hand, and one on his ear as he lay in the grass.

**Results**

Details of the clinical findings and subsequent course are given in the Table.

**Immediate effects.** Pain from the infliction of the bite was the first symptom in most children, but one child complained initially of a painful, swollen foot (Case 9). The 14-month-old baby (Case 2) was alleged by his parents to have lost consciousness for 5 minutes shortly after the bite, but this may have been a breath-holding attack. Vomiting occurred in 4 children: in two within a few minutes (Cases 6 and 13), and in Cases 8 and 15 within 30 minutes of the bite.

**Features on admission.** Most of the children were seen in hospital within an hour of the bite, with the exception of Case 12 who sought help the next day. Those in whom envenomation had occurred displayed the characteristic signs as shown in the Table. Six children were thought to be only mildly poisoned, pain and swelling being slight. Another 5 were moderately severely poisoned, with swelling of the bitten limb. Only 2 were severely poisoned; the history of one is given below (Case 8).

It was thought envenomation did not occur in 3 children (Cases 2, 10, and 11), although characteristic fang marks were easily identifiable in each case. The following case reports illustrate the course of events in 2 children.

**Case reports**

**Case 13.** A 9-year-old girl vomited within minutes of being bitten on the right ankle. When seen in casualty department an hour later ankle oedema was noted. No systemic signs were noted; her BP was 90/65 mmHg. She needed pethidine to control severe pain. Within 2 hours bruising covered the

Table **Details of 16 children admitted to hospital with adder bites**

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Bite Date</th>
<th>Bite Site</th>
<th>Effects</th>
<th>Drugs*</th>
<th>Days in hospital</th>
<th>Severity of poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>9</td>
<td>7/1969</td>
<td>L. foot</td>
<td>Pain +++, oedema, bruising, adenitis +++, infection</td>
<td>Promethazine, papaveretum, codeine, paracetamol, erythromycin</td>
<td>8</td>
<td>Moderate</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>14/12</td>
<td>8/1969</td>
<td>L. ear</td>
<td>Bite mark, loss of consciousness</td>
<td>None</td>
<td>2</td>
<td>Nil</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>8</td>
<td>8/1969</td>
<td>R. ankle</td>
<td>Oedema, bruising, adenitis +</td>
<td>Promethazine</td>
<td>6</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>8</td>
<td>6/1973</td>
<td>R. hand</td>
<td>Oedema, bruising</td>
<td>Aspirin, hydrocortisone</td>
<td>2</td>
<td>Mild</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>5</td>
<td>6/1974</td>
<td>L. finger</td>
<td>Oedema</td>
<td>None</td>
<td>1</td>
<td>Mild</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>10</td>
<td>7/1974</td>
<td>L. finger</td>
<td>Oedema, vomiting</td>
<td>Hydrocortisone, promethazine, chlorpheniramine, aspirin</td>
<td>6</td>
<td>Moderate</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>11</td>
<td>7/1974</td>
<td>R. thumb</td>
<td>Oedema, haemorrhagic blister</td>
<td>Hydrocortisone, promethazine, chlorpheniramine, ampicillin, paracetamol</td>
<td>9</td>
<td>Severe</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>3½</td>
<td>6/1975</td>
<td>R. foot</td>
<td>Oedema, bruising, lymphangitis, vomiting, pain, hypertension</td>
<td>Hydrocortisone, promethazine, chlorpheniramine, betadine, pentazocine</td>
<td>3</td>
<td>Mild</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>4</td>
<td>7/1975</td>
<td>R. ankle</td>
<td>Oedema, bruising, adenitis ++, blister</td>
<td>Hydrocortisone</td>
<td>1</td>
<td>Nil</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>9</td>
<td>7/1975</td>
<td>L. calf</td>
<td>Oedema, bruising, lymphangitis, vomiting, pain, hypertension</td>
<td>Hydrocortisone, chlorpheniramine</td>
<td>1</td>
<td>Nil</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>5</td>
<td>7/1975</td>
<td>R. ankle</td>
<td>Bite marks</td>
<td>None</td>
<td>2</td>
<td>Mild</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>6</td>
<td>7/1975</td>
<td>L. ankle</td>
<td>Oedema</td>
<td>Hydrocortisone</td>
<td>3</td>
<td>Moderate</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>9</td>
<td>7/1976</td>
<td>R. ankle</td>
<td>Vomiting +++, pain +++, oedema, bruising</td>
<td>Chlorpheniramine, betadine, phenoxymethylpenicillin</td>
<td>2</td>
<td>Mild</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>7</td>
<td>8/1976</td>
<td>R. finger</td>
<td>Oedema, bruising, vomiting, pain +++, oedema +++, moderate hypotension</td>
<td>None</td>
<td>3</td>
<td>Severe</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>14</td>
<td>4/1977</td>
<td>R. hand</td>
<td>Oedema</td>
<td>Hydrocortisone, promethazine, chlorpheniramine, betadine, phenoxymethylpenicillin</td>
<td>8</td>
<td>Moderate</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>13</td>
<td>8/1977</td>
<td>R. hand</td>
<td>Oedema</td>
<td>Chlorpheniramine</td>
<td>8</td>
<td>Moderate</td>
</tr>
</tbody>
</table>

*All children received tetanus toxoid.
dorsum of the foot and ankle. Investigations showed a raised total white blood count (17·0 × 10⁹/ℓ); a slightly raised blood urea (8 mmol/l; 48·2 mg/100 ml); slight acidosis (HCO₃ 20 mmol/l); a raised creatine phosphokinase (CPK 253 IU/l, normal <80 IU/l). She was treated with chlorpheniramine and penicillin V and the leg was kept in a raised position. 14 hours later oedema had spread to the thigh. Throughout, her BP remained stable. Her recovery was uneventful and she was discharged after 3 days.

Case 8. A 3½ year-old boy became pale, lethargic, and vomited 30 minutes after being bitten on the right foot. When seen in hospital less than an hour later, the foot was oedematous. He was given hydrocortisone and transferred to Southampton. 90 minutes after the bite he was still pale and vomiting, with a recorded systolic BP of 50 mmHg. Bruising covered the foot, and inguinal nodes were already very tender. He was still hypotensive (45/20 mmHg) 4½ hours after the bite. Chlorpromazine was given to control vomiting, and chlorpheniramine for the rapidly developing oedema. Severe pain required pethidine initially, then pentazocine. Six hours after the bite his BP had returned to normal. At 16 hours swelling had reached the inguinal region. Thereafter his recovery was slow but uneventful, and he was discharged on the 8th day.

Treatment. All the children received tetanus toxoid; in 3 children this was the only medication. Pain was severe enough in 3 children to require narcotic analgesics, but 9 required no analgesia. The other drugs given are shown in the Table.

In no case was Zagreb antivenom used, although it has been available in Southampton since 1972.

Sequelae. All children made a complete recovery. The average length of stay in hospital was 3·4 days (range 1–9). Most children were virtually symptom-free at discharge, but Cases 13 and 14 were in discomfort for a further week, and Case 7 was lethargic for about 2 weeks.

Discussion

Experience gained by hospital admission of 16 children bitten by adders in one small area of the country during the last 9 years adds to our knowledge about their management. Although these 16 comprise all the children who presented at hospital in Southampton, it is not possible to comment on children who managed without medical help, or who presented at their local hospitals; one child in this series (Case 12) presented the day after being bitten.

In this series most of the children suffered no more than acute discomfort in a swollen, bruised limb. Three children were probably not poisoned; about 30% of snake bites fail to envenom (Henderson and Dujon, 1973).

In moderate poisoning it is likely that no treatment is required other than symptomatic therapy, usually analgesics and antiemetics. The use of antihistamines can be limited to those in whom angioneurotic-type oedema appears (Reid, 1976); it did not occur in the present series. Prednisone is of no value in the treatment of the more severe Malayan viper bite (Reid et al., 1963).

Three children gave cause for anxiety (Cases 8, 13, and 15), particularly the 2 boys with hypotension. All 3 had early indications for the administration of Zagreb antivenom (Reid, 1976; D.A. Warrell, 1976, personal communication), but they recovered without it. Reid’s indications include persistent or recurrent hypotension, a leucocytosis >20·0 × 10⁹/l, metabolic acidosis, ECG changes, raised CPK values, and in adults, the presence of massive swelling within 2 hours of the bite. Warrell would also include neurological abnormalities, bleeding diatheses, and massive swelling within 4 to 6 hours, in children as well as adults.

Transient hypotension lasting 4 to 12 hours is common in severe venom poisoning: it lasted 6 hours in Case 8 and 5 in Case 15, and did not recur. Had the condition of either of them deteriorated or the hypotension persisted after 12 hours, antivenom would probably have been given. Case 13 had investigation abnormalities. From the criteria of Reid (1976), these indicated that antivenom should have been given, but as the patient was in a reasonably good clinical state, it was withheld pending further observation.

No child developed early massive swelling. Reid’s reason for this being an indication for antivenom in adults is to reduce the period of morbidity, which can last several months. Prolonged morbidity is not a problem with children and these data confirm this. Warrell adds that early massive swelling indicates a large envenomation, and therefore an increased potential for cardiotoxicity; hence he includes children. This, however, he bases on his experience with puff-adders; it is not a reported problem with Vipera berus.

Conclusions

Some risk is inherent in many forms of treatment, particularly if the material is immunogenic. Zagreb antivenom, being a foreign protein, is in this category. Nevertheless it has been shown to combine efficacy with safety in monkeys (Theakston and
Reid, 1976), and there is no reason to believe it is less safe in humans. Antivenom has not yet been used in Southampton although 3 children might well have received it if the criteria (Reid, 1976; D. A. Warrell, 1976, personal communication) had been wholly accepted. However early clinical improvement showed that it was not necessary.

When there is a successful outcome one can accept that the clinical decisions were correct; the difficulty lies in deducing future management. Guidelines for the administration of Zagreb antivenom are reasonable, but if acted upon too early in children, may lead to unnecessary administration. In Southampton a policy of symptomatic treatment in hospital and careful observation has been safe. Zagreb antivenom remains available for the serious cases that do not show early improvement.

I thank Professor I. C. S. Normand, Dr M. Radford, and Dr C. F. Speirs for their advice and criticism.

References


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Coeliac disease in identical twins

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SUMMARY Coeliac disease occurred at the same age in MZ twins. The diagnosis was confirmed by histology of the small intestine, rapid response to a gluten-free diet, and relapse after reintroduction of gluten.

While the familial incidence of coeliac disease is incontestable, some workers emphasise the importance of inheritance (Anderson et al., 1972; McNeish and Anderson, 1974), and others the environment (Hoffman et al., 1966; Walker-Smith, 1973). We here report coeliac disease with identical age of onset in a pair of monozygotic twins. This is the only well documented report of concordant monozygotic twins with genetic study, apart from that of David and Ajdukiewicz (1975) which included one set of adult female twins who were 'probably monozygotic'.

Case reports

White twin boys were born at term, without complications, and with a single placenta. Twin 1 weighed 2.6

Fig. 1 Twins aged 2 years 8 months.