

The mean age of patients with gastric carcinoma is much higher than that of those with peptic ulceration. Because of fewer numbers of surviving sibs, the accumulation of sibship data is more difficult. Excesses of group A were noted when the propositi were compared with the unaffected sibs and with the population controls. An excess of A was also found in the propositi of the 16 sibships segregating for group A. None of these differences were of statistical significance. All that

TABLE V.—Analysis of the Chance of the Propositus Having Group O in the Sibships Segregating for Blood Group O After Sibs of the Sex Opposite that of the Propositus are Removed

	No. of Sibships	Group O Observed	Group O Expected	Variance
Gastric ulcer ..	32	19	15.8166	7.7051
Duodenal ,,*	65	35	29.9413	15.2293
Combined data	97	54	45.7579	22.9344

From which we obtain:

	One-tail Test	Two-tail Test
Gastric ulcer:	$\frac{19-15.8166}{\sqrt{7.7051}} = \frac{3.1834}{2.776} = 1.15$	0.10 < P
Duodenal ,,	$\frac{35-29.9413}{\sqrt{15.2293}} = \frac{5.0587}{3.902} = 1.30$	0.05 < P < 0.10
Combined data:	$\frac{54-45.7579}{\sqrt{22.9344}} = \frac{8.2421}{4.789} = 1.72$	0.02 < P < 0.05

* Buckwalter *et al.* (1960).

may be said is that these data are consistent with the causal explanation for the association of blood group and carcinoma of the stomach. The most important reason for publishing these data is to make them available for the use of other investigators in evaluating their findings.

Conclusions

Evidence is presented of an increased frequency of blood group O in gastric ulcer patients, in the siblings of gastric ulcer patients, and in the patients as compared with their siblings.

These data have been combined with the previously published duodenal ulcer findings, which show similar increases in O frequency. Additional support for a causal explanation of the association of the ABO blood groups with peptic ulceration is provided.

The findings in the gastric carcinoma sibship study, based on fewer data, are less convincing, but show an excess of blood group A in patients, their siblings, and in the patients compared with their siblings.

The original manuscript was reviewed by Dr. J. A. Fraser Roberts. The reader has thereby been provided with a more critically reasoned and precisely stated report of our findings. We are fully responsible for the shortcomings of this report.

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Medical Memoranda

Case of Adder-bite with Thrombosis of the Saphenous Vein

The noxious effects of adder-bite are usually insignificant compared with the fear and alarm engendered by such an occurrence. Nevertheless, venom from *Vipera berus* may place the life of a child in jeopardy, as described below.

CASE HISTORY

At 7 p.m. on June 21, 1960, a healthy 5-year-old boy living in a remote rural area of Argyll complained of a painful and swollen left ankle. It was discoloured and flecked with blood on the medial aspect. The boy's statement that his ankle had been injured "in a door" was believed. One hour later he began to vomit, and this continued intermittently for 24 hours. By 10 p.m. the discoloration had spread from the ankle to mid-thigh, and the child was delirious and in considerable pain.

Next day he was seen by a local practitioner, who sent him to the West Highland Hospital, Oban, some 30 miles (48 km.) distant. At this point he admitted having been bitten by a "big worm" while engaged with his brother in the strictly forbidden sport of trying to throw a snake on to the roof of the cottage. The boy was given 100 mg. of hydrocortisone and transferred by ambulance to the Royal Hospital for Sick Children, Glasgow, where he was admitted at 5 p.m. on June 22. Prior to his arrival antivenin had been obtained from Edinburgh.

On admission he was profoundly shocked and restless, with a very striking and persistent head-shaking movement. He was mentally confused and markedly dehydrated. The radial pulses were not palpable. The blood-pressure could not be recorded, and the heart sounds were rapid and of very poor quality.

The entire left leg was grossly swollen, the skin being tense, shining, mottled dark blue, purple, and yellow, with a sharp line of demarcation just below the inguinal ligament. Neither the femoral nor any other arterial pulse could be felt in the left leg or foot. Two small puncture wounds were visible on the medial aspect of the ankle, and the long saphenous vein was thrombosed.

Resuscitation and treatment for shock were carried out promptly in the form of intravenous infusion with injection of promethazine hydrochloride and adrenaline. Sedation was given by the use of intramuscular paraldehyde. The general condition improved rapidly and the systolic blood-pressure reached 130 mm. Hg. Intramuscular penicillin and oral tetracycline were started and continued. A small incision was made at the area of the bite, and 5 ml. of anti-venin (Pasteur Institute E.R. serum) infiltrated locally. A further 5 ml. was injected intramuscularly into the opposite thigh.

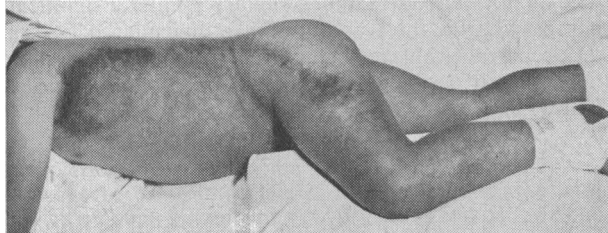
Next morning (June 23) he was no longer shocked and was hydrated. Mental confusion persisted, with repetitive shaking of the head, and he appeared to be in great pain. The affected leg was enormously swollen with no palpable arterial pulsation present. Two injections of hyaluronidase were given into the thigh, but these were followed by only a minor transient diminution in the circumference of the limb.

By June 24 the discoloured area had crept above the inguinal ligament and the leg was cold. The boy was still confused, and his blood urea had risen to 83 mg./100 ml. Seventy-two hours after the adder-bite (and after much consideration) an injection of 5,000 units of chymotrypsin was given intramuscularly into the opposite thigh. This resulted in three things: (a) a brisk general reaction causing mild shock occurred 30 minutes after the injection and lasted one hour; (b) the area of inflammatory swelling and tissue discoloration spread from the left iliac fossa to the left

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axilla (see photograph); and (c) swelling and tension in the left leg decreased, and in 12 hours the femoral pulse was palpable.

From this time onward the state of the leg improved. However, the general condition of the boy deteriorated sharply; the abdominal and thoracic walls on the left side were now swollen and acutely painful, and were held immobile. On June 28 an extensive left-sided pneumonia



Photograph taken 12 hours after an injection of chymotrypsin.

developed, apparently hypostatic in type, despite the efforts which had been made to alter the patient's position frequently so far as the pain would permit, and despite the prophylactic treatment with penicillin and tetracycline. The pneumonia responded fairly quickly to chloramphenicol, and by July 8 the boy was up, running about, and threatening destruction to the adder population of Argyll.

DISCUSSION

The bite of the adder (*Vipera berus*) is seldom dangerous to the adult, and is usually just reward for interfering with this shy and non-aggressive reptile. The average adult receiving such a bite will suffer immediate pain, swelling, and discoloration locally, and perhaps transient giddiness. Only if vomiting and progressive shock appear over a period of hours does the use of antivenin locally or otherwise appear to be desirable. Analgesic and antihistaminic treatment is recommended (Morton, 1960). Death, when it occurs, usually takes place 6 to 60 hours after envenomization (Walker, 1945).

In the present instance delay aggravated the initial severity of the bite as judged by extensive local and systemic reaction and the involvement of the saphenous vein in a small child. Once the severe shock from which the boy suffered had been overcome the principal concern was for his leg, which was little improved by the injections of antivenin or of hyaluronidase. Tryptic extracts were tried as a last resort.

It has been stated (U.S. Dispensatory, 1955) that the venom of viperines is irreversibly combined with tissues within a few hours of a bite. Injection of chymotrypsin produced a reaction which strongly suggested that venom may have been released and was still active. No further antivenin was given, because further profound shock did not occur, and the risk of anaphylaxis following a second injection of antivenin seemed considerable.

Prior to May, 1961, the last recorded death from snake-bite in Great Britain occurred in 1941, and the last death from reaction to antivenin serum in 1957 (Morton, 1960).

Having been called upon to deal with this acute emergency at short notice has made us aware of the following important points brought out by the lucid contributions of Morton (1960), Birch (1959), and Manson-Bahr (1957): (1) When adder-bite occurs one should not incise locally and attempt to suck out venom or apply a tourniquet. (2) When envenomization has been considerable, vomiting followed by shock is likely to occur within a few hours of the bite. Resuscitative measures and treatment with antihistamines initially,

followed by antivenin if necessary (and available), are then indicated. Anaphylaxis as a possible complication must be anticipated. (3) The condition is always apt to be more dangerous in a child.

Enthusiasm should be tempered with reason when treatment is given. It seems a simple and obvious precaution that antivenin should be available locally in rural areas of Scotland where *Vipera berus* is present.

GAVIN C. ARNEIL, M.D., Ph.D., F.R.F.P.S.,
M.R.C.P., D.C.H.,
Paediatrician,

JOHN C. MACLAURIN, M.B., M.R.C.P.Ed.,
Lecturer in Child Health,
Department of Child Health, University of Glasgow,
and the Royal Hospital for Sick Children, Glasgow.

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A Second Chinese Family with Haemoglobin Q

Haemoglobin Q was discovered in a Chinese man in Singapore (Vella, Wells, Ager, and Lehmann, 1958) who was suffering from a haemoglobinopathy, haemoglobin Q being associated with haemoglobin H. The same haemoglobin was then found in his mother, in whom it was present as a simple trait (A + Q).

Haemoglobin Q has not been found since, and we therefore consider it worth while recording a second observation made in Singapore in 1960. On this occasion the haemoglobin Q was accidentally found in a 9-year-old Chinese boy who was admitted to hospital with anaemia, fever, and jaundice. As a haemoglobinopathy was one of the possible diagnoses the haemoglobin was examined and found, on paper electrophoresis at pH 8.6, to consist of two barely separable fractions—the front one in the position of haemoglobin A

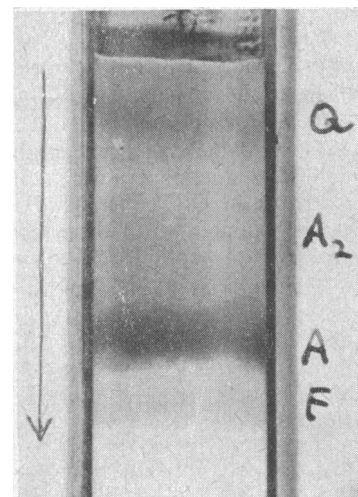


FIG. 1.—Chromatography on IRC 50 resin at pH 6 of the child's haemoglobin, showing haemoglobins F, A, A₂, and Q.

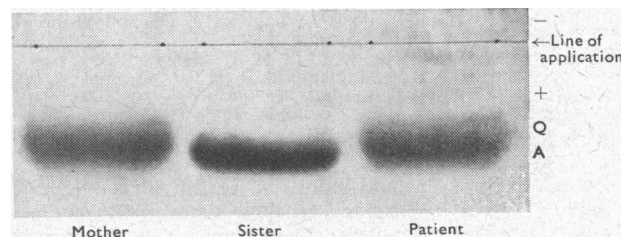


FIG. 2.—Paper electrophoresis barbiturate buffer, pH 8.6, of the haemoglobin of the patient (A + Q), his sister (A only), and his mother (A + Q).