Blegen and Aas (1950), Heller and Jacobson (1950), and Werkö *et al.* (1952), and in many of the cases confirm that changes in renal circulation in patients with valvular heart disease occur long before the onset of congestive heart failure. After operation the renal blood flow and renal plasma flow often increased, but did not return to the normal range in any of the patients, and only in Case 12 did the glomerular filtration rate reach normal levels.

The clinical state following valvotomy improved in all except two patients (Cases 6 and 7), but this improvement was not always associated with one in the renal circulation, nor was it consistently accompanied by a change in the diuretic response to water ingestion. In the group with congestive heart failure (patients in grade 4), one patient (Case 13) improved to such an extent—grade 4 to grade 1—that she was only slightly disabled after operation and her renal blood and plasma flows increased greatly. In Cases 5, 6, and 10 there was less improvement in the clinical state, and this was associated with much smaller changes in renal circulation (Table I). The fifth patient with congestive heart failure (Case 7) showed a dramatic increase in her renal blood and plasma flows after valvotomy, but despite this she improved only slightly at first and then relapsed into congestive heart failure. Although all 10 patients without heart failure improved clinically after operation, only two (Cases 4 and 12) showed an improvement in renal circulation, and in one (Case 11) the renal blood and plasma flows and glomerular filtration rate decreased. These results are in accordance with those of Werkö *et al.* (1955) and Judson *et al.* (1955), who reported variable changes in renal blood flow after valvotomy.

Before operation all five patients whose response to the ingestion of 1 litre of water was tested excreted less than 702 ml. of urine in two hours (Table II), which was the lowest value found in normal fasting subjects by Taylor (1955). In all but one patient the maximal rate of diuresis was also below normal, but the time of the maximal urine flow was not delayed beyond the normal range given by Taylor (1955), except in one patient (Table II). After operation there was an improvement in the diuretic response in three patients. The maximal rate of diuresis increased to within normal limits in two patients, and in one of these the two-hourly urine volume also reached normal limits. In all patients maximal diuresis occurred within the normal time range (Table II). There was no apparent correlation between the improvement in diuretic response and changes in the renal circulation, since the patients whose two-hourly urine volumes and maximal rates of diuresis increased showed no significant change in renal blood flow or glomerular filtration rate, and Case 2, whose glomerular filtration rate increased, had a diminished diuretic response after operation. These results are in keeping with those of Judson *et al.* (1955), who reported that post-operative increases in water excretion could not be consistently correlated with any specific change in renal function.

Although these results show that sometimes there is a marked improvement in the renal circulation following mitral valvotomy, the series is too small to allow these changes to be correlated with recovery from or persistence of congestive heart failure. It is obvious that further studies are required, especially in patients with congestive heart failure who recover completely after operation, before it is possible to decide whether improvement in the renal circulation as a result of the operation is directly related to an increase in the capacity to excrete salt and water and the disappearance of congestive heart failure.

Summary

The renal circulation was studied before and after mitral valvotomy in 15 patients, and in five of these the diuretic response to water ingestion was also tested.

Five patients had congestive heart failure prior to operation; six others were severely and four moderately disabled.

Before operation the renal circulation was impaired in all patients and the diuretic response was abnormal in the five patients in whom it was studied.

After operation one patient died with congestive heart failure, another was unchanged, and the remaining 13 showed improvement in their clinical state. The renal circulation improved in five patients, was further reduced in four, and showed little change in six. In three of the five patients studied the diuretic response improved, and in one it became more abnormal.

From this small series there was no obvious correlation between changes in the renal circulation, diuretic response, and the clinical state, although one patient with congestive heart failure who was greatly improved showed a considerable increase in renal blood and plasma flows after operation. The results indicated a need for further study, especially in patients with congestive heart failure prior to operation.

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SEA-SNAKE BITES

BY

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Sea-snakes are the most abundant and venomous of the world's reptiles. They inhabit the shores of the Indian and Pacific Oceans, ranging from the Persian Gulf to south Japan, the coasts of tropical Australia, and the southern Pacific islands. In this vast triangle all fishermen know the sea-snake and its hall-mark, the flat rudder-like tail. Water-snakes, also common along these shores, have round tapering tails (see Fig. 1). They are harmless—a fact familiar to the fishermen.

The frequency of particular genera and species of the sea-snake family (Hydrophiidae) varies in different parts of the world, but, generally speaking, the commonest species is *Enhydrina schistosa* (common sea-snake). The adult is 3–4 feet (0.9–1.2 metres) long, about 1–2 inches (2.5–5 cm.) thick, a uniform grey above and whitish below. It is easily recognized by the deep cleft in its chin (see Fig. 2). Young ones have dark-grey bars. Most other species are banded. Some species of *Hydrophis* grow to a length of 9 feet (2.7 metres), but are relatively slender, with small heads. The genus *Astrotia* are the most massive, being up to 4 inches (10 cm.) thick. *Pelamis platurus* has the widest distribution, and has been noted off the western coasts of tropical America and the

shores of south-east Africa (Smith, 1943). The genus *Laticauda* is amphibious, but the rest of the sea-snakes are purely marine.

Sea-Snake Venom

Rogers (1902–3) found the average yield per bite of the common sea-snake (*E. schistosa*) to be 9.4 mg. This was about one-twenty-fifth of the yield from a cobra, but it was much more potent. The fatal dose for a 70-kg. man was estimated as 3.5 mg., or about one-third of an average bite. *P. platurus* was equally toxic, *Hydrophis* species less so. Fraser and Elliot (1905) confirmed the virulence of *Enhydrina*, and found *Lapemis curtus* toxicity similar to that of the cobra. Smith (1926) found *L. hardwickii* extremely toxic for guinea-pigs. Smith and Hindle (1931) reported the venom of *Laticauda colubrina* to be twice as toxic as cobra for mice, but thought the yield too small to kill healthy human adults. Rogers (1903–4) and Fraser and Elliot (1905) demonstrated a neurotoxic effect mainly on the respiratory centre but also on the motor nerve-ends. Unlike cobra venom, there was no direct action on the vasomotor centre or heart and no vasoconstriction, haemolysis, or effect on the coagulation (in concentrations likely to be present in human beings). After a latent period of half to several hours, depending on the dose, progressive paresis, dyspnoea, and ptosis ensued. Respiration finally ceased, although the heart continued to beat for two to three minutes. Thus the Hydrophiidae (true sea-snakes) are almost purely neurotoxic, in contrast to the other two poisonous snake families, the Elapidae being chiefly neurotoxic but slightly haemotoxic, the Viperidae mainly haemotoxic. Cobra antivenene was ineffective against *Enhydrina* or *Lapemis* poisoning. Richards (1873) kept a dog alive for over 24 hours by tracheotomy and bellows. Prior to this it was moribund. Venom was probably excreted in the dog's urine. Further investigations on sea-snake venom will be reported in due course.

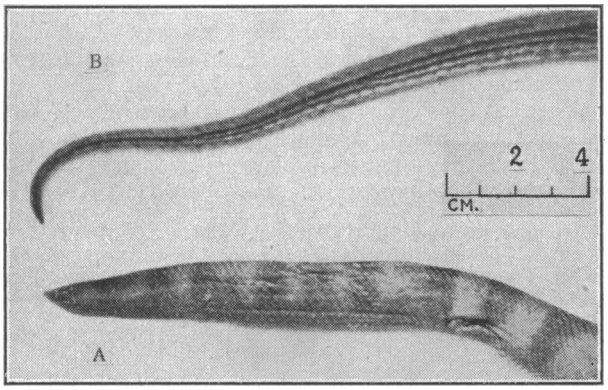

FIG. 1.—A, The flat oar-like tail of a poisonous sea-snake. B, The rounded tapering tail of a harmless water-snake. Both inhabit coastal waters.

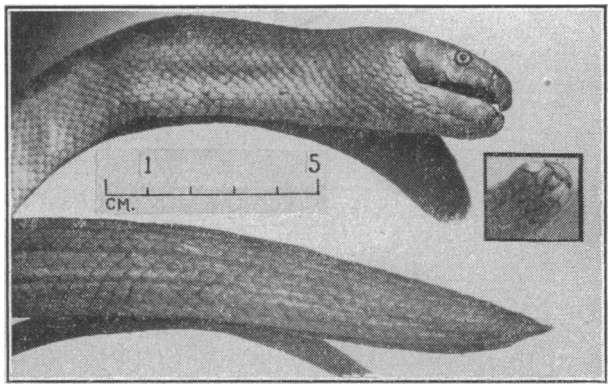

FIG. 2.—Head and tail of a young adult *Enhydrina schistosa*, the common sea-snake (3 feet—0.9 metre—long). Eyes are small and rounded; valvular nostrils on top of snout. Inset is a view of the chin showing the characteristic cleft.

Sea-Snake Poisoning

Records are rare and usually inadequate. The literature describing seven fatal cases (Cantor, 1841; Richards, 1886; Fayrer, 1874; Forné, 1888; Buckland, 1879; Fossen, 1940; Bokma, 1941) and two recoveries (Peal, 1903; Bokma, 1942) will be reviewed in a paper published elsewhere, together with an account of a series of cases personally observed or investigated in a current survey. From these a clear clinical picture emerges.

Victims are mostly fishermen bitten through some accident such as handling nets, sorting fish, treading on a sea-snake, etc. They usually know that it was a sea-snake and are aware of the significance. People bitten whilst bathing, paddling, etc., often do not see the snake or, if they do, think it is a fish. After the initial prick there is no pain or reaction at the site of the bite, the appearance of which is described below under "Diagnosis." A delay of minutes to several hours, usually about one hour, then elapses before further symptoms ensue. Thus the victim continues activities promoting spread of the venom. He may not even connect the bite incident with his illness.

The symptoms are due to bulbar and motor paresis, which is usually generalized from the start but may begin in the legs and ascend, rapidly within an hour or two, to involve the trunk, arm, and neck muscles. During the first few hours the complaint may be "aching," "stiffness," or considerable pain on moving rather than actual weakness. The paresis is usually flaccid, with depressed or absent tendon reflexes, but may be spastic, with hyperreflexia initially. Bulbar palsy may precede, accompany, or follow the peripheral paresis. Jaw stiffness due to trismus is the outstanding feature and is easily overlooked. Ptosis is another early and important sign. It is often mistaken for drowsiness (the patient is usually mentally clear until respiratory failure is advanced). Thirst, burning or dryness of the throat, a general feeling of coldness, and increased sweating are common early symptoms, some of which may be due to fright, but more probably are bulbar manifestations. The pupils are usually dilated. Dysarthria and dysphagia, mainly due to the trismus, are also common although not invariable. Vomiting, nasal regurgitation, and ocular and facial palsy may occur.

The paresis progresses gradually or rapidly, the patient being unable to sit up unaided, then incapable of lifting the heels or head off the bed, and finally lies quite inert. This total paralysis, combined with ptosis, may give a deceptively peaceful appearance. Muscles are extremely tender, although objective sensory involvement is absent or minimal. As trismus increases, only the end of the tongue, eventually not even the tip, can be protruded beyond the teeth margin. There is no clinical evidence of cerebral, cerebellar, or extrapyramidal involvement, and sphincter disturbance is uncommon. In fatal cases respiratory paralysis with terminal hypertension and cyanosis usher in death, which may take place a few hours or several days after the bite. Occasionally, little or no peripheral paresis occurs and the patient dies from pure bulbar palsy, often with convulsions. Failing vision is a fatal sign to fishermen. Cerebrospinal fluid is normal, albuminuria usual, and haemoglobinuria common. The peripheral blood is normal apart from a leucocytosis and some haemoconcentration. Except for the urine findings, there is no clinical evidence of direct cardiovascular involvement by the venom.

A Fatal Case

This is a representative fatal case from my series. A preliminary report has already been made (Reid, 1956).

A 26-year-old Chinese man was bitten at 3 p.m. on October 24, 1954, whilst bathing off Penang Island. He was walking in mud about 20 feet from the shore, the water up to his chest, when he thought he had been bitten by a crab. He looked at his foot but saw nothing wrong with it. No pain or swelling was felt after the initial prick. He continued bathing. Half an hour after the bite he had difficulty in talking owing to a dry feeling in the throat. He returned home. One hour after the bite he had general aching pains in the proximal limbs, with some stiffness and pain across the lumbar region. Two hours later he was unable to sit up unaided or open his mouth properly. Three hours later he noticed difficulty in swallowing. He was unable to sleep, and 12 hours later felt numbness of the fingers though not of the toes.

He was admitted to hospital on October 25, and I first saw him at 10 a.m.—that is, 19 hours after the bite. Two pairs of fang marks, each 7 mm. apart, were seen below the right external malleolus. There was no swelling or discoloration of surrounding skin. He was mentally clear. There was well-marked ptosis and trismus (maximal jaw opening, 2 cm.). The masseters were tender, but spasm was not observed. The eye and facial movements were full and there was no nystagmus. He was able to talk, to cough quite forcibly, and to swallow water, the latter with some difficulty owing to the trismus. The pupils were dilated and reacted to light ; abdominal and cremasteric reflexes were present but no plantar responses were obtained. All muscles were tender. There was generalized paresis, with increased tone and tendon reflexes. He was able to move the limbs in bed but unable to lift them or the head off the bed. There was no objective loss of light touch, pain, or vibration sense. The blood pressure was 130/85 and the remaining physical examination was negative. The urine was normal except for a trace of albumin. The Hess test, bleeding-time, coagulation time, platelet count, and prothrombin time were normal ; Hb 100%, and W.B.C. 16,000. He had been given 10 ml. of polyvalent antivenene intravenously and 5 ml. into the bite area on admission. On the basis that the venom action was similar to that of curare, 1.5 mg. of neostigmine was given intravenously at 10.45 a.m. Abdominal pain and vomiting followed, but there was no change in the paresis. In the afternoon his temperature rose to 101° F. (38.3° C.) and the pulse rate to 120 (probably a serum reaction). Thereafter the temperature varied between 98 and 99° F. (36.7 and 37.2° C.) and the pulse rate between 95 and 110.

Early next morning (36 hours after the bite) haemoglobinuria was apparent and continued until his death. The paresis was now more marked, the tone was diminished, and tendon reflexes were absent. Trismus had increased (maximal jaw opening 1 cm.) and there was difficulty in looking upwards. Breathing and coughing were normal. In the afternoon a marked increase in sweating developed. The E.C.G. (14 leads) was normal ; Hb 110% ; W.B.C., 20,000 ; plasma bilirubin, 1.2 mg. per 100 ml. ; Kahn test negative ; and spectroscopy showed a mixture of pigments in the urine, chiefly methaemoglobin.

Early on October 27 (60 hours after the bite) difficulty with breathing first appeared. He could cough fairly well; B.P. 128/75, Hb 108%, W.B.C. 26,000. A Bragg-Paul pulsator was started and he was nursed in the prone position. At 11 a.m. the paresis was quite flaccid, with slight hand and foot movements only. Breathing became embarrassed within a minute of stopping the respirator. Sweating continued to be profuse ; B.P. 130/80. At 2.45 p.m. the B.P. was 145/100 ; there was no cyanosis or evidence of respiratory obstruction. At 3.15 p.m. cyanosis was first noted. At 4.30 p.m. the B.P. was 159/90 and pulse rate 120. At 7.45 p.m. the pulse was imperceptible, heart sounds were faint, there was no response to questions, and the pupils were dilated but reacted to light. He died at 8.20 p.m., 77 hours after the bite.

In addition to polyvalent antivenene he had received cortisone, 100 mg. twice daily, penicillin, intravenous glucose-saline, and milk by gastric tube. Although haemoglobinuria continued till the end, the plasma bilirubin just before death was 1 mg. per 100 ml. and the urinary output had remained satisfactory. The Schumm and the Coombs tests were both negative.

A Mild Case

Less severe cases will not develop respiratory insufficiency. Trismus, ptosis, and peripheral paresis resolve rapidly within a few days, occasionally taking a few weeks. The milder cases only develop " weakness " or " stiffness," often confined to the lower limbs, for a few hours or days. Tendon reflexes are depressed or absent. Usually, there are minimal bulbar symptoms as well and albuminuria. The following is a representative mild case.

A Chinese girl aged 16 was sorting fish on the shore at Batu Muang, a small Penang fishing village, at 8 a.m. on March 25, 1955, when she accidentally trod on a sea-snake, which bit her left ankle. It was an *E. schistosa*, 2 feet (0.6 m.) long. She felt no pain after the bite. She was given herbal medicine and brought to hospital at 12 noon. Her only complaint was " stiffness " in both legs and back, which started half an hour after the bite. Below the left medial malleolus were fang marks 8 mm. apart. On admission the knee- and ankle-jerks were sluggish, but no other abnormality was observed; in particular no objective paresis or bulbar signs were present. The urine contained a trace of albumin. Cortisone, 250 mg. intramuscularly, and polyvalent antivenene, 20 ml. intravenously, were given. She had a brisk anaphylactic reaction requiring three injections of adrenaline.

At 3 p.m. the knee- and ankle-jerks were more sluggish, maximal jaw opening was 4 cm. (5 cm. at noon), the urine contained a heavy cloud of albumin; Hb 94%, and W.B.C. 15,000 (P. 80%, L. 16%, M. 1%, E. 3%). Next morning she was feeling well; the maximal jaw opening was again 5 cm., the tendon reflexes were brisker and the W.B.C. 16,000 ; there was no spectroscopic evidence of haemoglobinuria, and the urine deposit, Hb, blood urea, serum bilirubin, and plasma prothrombin time were all within normal limits. She was discharged on March 30, when the tendon reflexes were significantly brisker than on the 26th Albuminuria subsequently diminished, and was absent by April 17.

If recovery takes place it is complete. No permanent after-effects such as occur in poliomyelitis were known to the many fishermen whom I have questioned.

Diagnosis

When there is a " sea-snake scare," as recently happened in Penang, the majority of cases coming to hospital thinking they may be sea-snake victims turn out to be something else. During the last 3½ years, 17 cases of fish sting, mostly by catfish, have been admitted to Penang General Hospital, compared with nine cases of sea-snake bite and one case of eel bite and jelly-fish sting (and 28 cases of land-snake bite). On the other hand, if the doctor is unacquainted with the features of sea-snake poisoning he may miss the diagnosis entirely, even though the patient claims to have been bitten in the sea.

The minimum criteria of sea-snake bites are that (1) the patient was in the sea or river-mouth, sorting fishing-nets, etc., (2) felt no pain after the initial prick, and (3) bears fang marks at the site of the bite. The bite may be felt as a slight " brush." Sometimes the patient is quite unaware of a bite until the fang marks are seen. They are usually around the ankle—one or more pairs of circular dots as though made by a hypodermic needle. The pattern varies according to the number of teeth penetrating as well as the fangs, which are small in sea-snakes, rarely exceeding 4 mm. long. Behind them is a maxillary row of teeth and two internal palatine rows—that is, four parallel rows in the upper jaw. Most of my cases have had four dots (see Fig. 3), although two is common and single or multiple

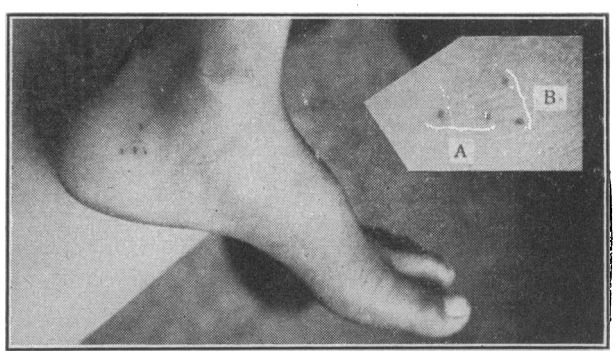

FIG. 3.—Sea-snake bite, typical marks; inconspicuous, no local reaction or tenderness. The dots represent two bites (fang marks A and B in the inset).

dots may occur. Fang marks are slightly bigger than the teeth marks, usually 5–10 mm. apart, and there is no swelling, bruising, or tenderness around them (unless, of course, local "treatment" has been given). Sometimes the fangs or teeth break off and remain embedded. Fishermen draw a hair over the site to extract them as a diagnostic measure. Pin-head oozing of blood stops within a minute or two. The bite marks are often inconspicuous, and a good light may be needed to see them.

These three points—the sea, absence of pain, and fang marks—justify a presumptive diagnosis of sea-snake bite, and treatment should be given. The diagnosis is certain if the snake has been seen to have a flat tail or there is evidence of sea-snake poisoning, such as leg paresis, trismus, or ptosis. The delay between the bite and the onset of poisoning symptoms may be deceptive. Although usually half to one hour, periods of up to six hours have been recorded. If, however, more than six hours have elapsed since the bite and there is no evidence of poisoning, either it is not a sea-snake bite or no venom has been injected. Fish stings are easily distinguished, as they are always extremely painful and tender (see Fig. 4).

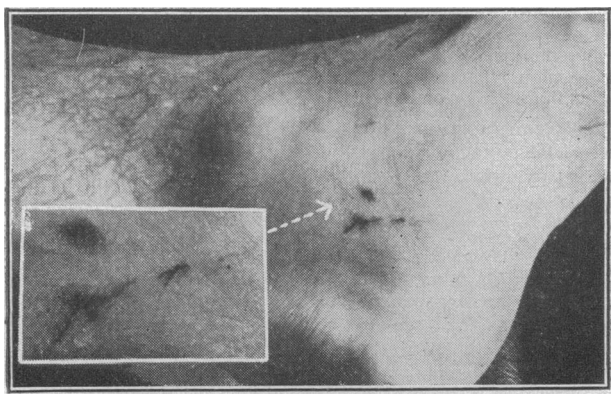

FIG. 4.—Sting mark from a catfish (*Plotosus:* "ikan sembilang"). This is always very painful, and the mark is ragged, linear, and tender. It is not always as conspicuous as in this case.

Treatment

From evidence which will be reported elsewhere, it is improbable that first-aid and local treatment are of any benefit except from the psychological viewpoint. Mainly for this latter reason, the following first-aid measures are recommended: (1) avoid *all* exertion, especially with the limb bitten (the most important measure); (2) apply a tourniquet to the thigh (in leg bites) or arm above the elbow (in upper-limb bites), releasing it every 30 minutes; (3) leave the fang marks alone; and (4) if possible, kill the snake by one sharp blow on the neck (avoid the head) and, after wriggling has stopped, lift by the tail into a container and bring to the doctor.

Specific antivenene is not yet available for sea-snake poisoning. A polyvalent antiserum containing a krait fraction should be used, as, of the Elapidae, krait poisoning (Ahuja and Singe, 1954) bears the closest resemblance to that of sea-snakes. In the admission room 20 ml. is slowly injected intravenously, preceded by cortisone to prevent serum reactions, which are frequent and may be severe. Adrenaline is indicated if, as commonly happens, the reaction occurs immediately, before the cortisone is acting. I have not found parenteral antihistamines of help. The problem of antivenene dosage and therapy is discussed later.

Feeding by gastric tube, preferably "polythene," which is less irritating than rubber, will usually be necessary on account of the trismus and, if present, dysphagia. The fluid and electrolyte balance may need careful watching, note being made of vomit and increased sweating on the output chart. The posture should be frequently changed and antibiotics given to prevent or control infection. Morphine derivatives, on account of their depressive effects on respiration, must not be given. If a sedative is required, a barbiturate or intramuscular paraldehyde is suitable.

If respiratory insufficiency develops it may be due to (1) damage to the respiratory centre by the venom; (2) peripheral paralysis of respiratory muscles; and (3) obstruction of the airway by secretions, food, or vomit. The management of such cases, similar to that of bulbospinal poliomyelitis, toxic polyneuritis (Russell, 1955), and tetanus (Shackleton, 1954), is difficult and complex (Astrup *et al.*, 1954). The patient should be nursed prone, with the foot of the bed raised to drain secretions. Intubation, tracheotomy, and artificial respiration by intermittent positive pressure may all be required, involving intensive team-work between the medical and nursing staff. Cuirass respirators are inefficient and tank respirators have too many disadvantages (Astrup *et al.*, 1954).

Discussion

A great many problems remain to be solved, including various biological features of the sea-snakes, many aspects of the venom (such as yield and toxicity of different species in varying circumstances, rate and route of absorption, rate of fixation, its nature, site, and mode of action, excretion, and fate in the body), the effectiveness of a tourniquet, other local measures, and prolonged artificial respiration; and, most important, the question of specific antivenene. The latter should be effective against all the common species of sea-snakes, and could be combined with hyaluronidase to enable injection at peripheral dispensaries. In some river-mouth villages, active immunization might be the best solution. Detailed discussion of these matters will be postponed until the results of current research are available. At this stage it would seem profitable to consider three problems—namely, the incidence of sea-snake bites, the safety of bathing, and the outcome when people are bitten.

What is the Frequency?

It is generally assumed that sea-snake bites are extremely rare. Certainly, records are scarce. The three main reasons for this are: (1) the timidity of the snake; (2) the victim is usually a fisherman in some remote village: he very rarely comes to responsible medical authorities (Smith, 1926); and (3) for fear of offending the sea-snakes and other reasons, fishermen are reluctant to talk about incidents, especially fatal ones. The casual inquirer would probably be told that none occurred. Although the seas around northern Australia abound with sea-snakes, there are no known instances there of poisoning (Gray, 1930; H. Flecker, 1953, personal communication). Smith (1926) quotes a pearl-fisher who told him divers were often bitten, although symptoms were slight or absent. P. D. G. Pugh (1953, personal communication) was unable to confirm this from the many divers he questioned. In Gato, a small islet of the Philippines, enough sea-snakes breed to maintain a supply of 10,000 skins, at least 1 metre long, a year indefinitely (Herre and Rabor, 1949). Before the war 30,000 were taken each year. Despite this abundance, no cases have been recorded from the vast forces in that area during the second world war (personal communications from the Departments of Navy, Army, Air Force, U.S.A., 1955). Bokma (1941), in Java, found only six fatal cases over 20 years. There is a conspicuous dearth of reports in the Indian medical literature. Reviewing world mortality of snake bite, Swaroop and Grab (1954) produced few facts about sea-snake victims except that fishermen were occasionally bitten.

However, Rogers (1902–3) said that deaths were not rare among oyster fishers along the Madras coast. Peal (1903) was told that three or four fishermen were bitten each year in a fishing village on the Orissa coast and 25% died. Hospital statistics give virtually no help in assessing incidence, because at present the victims rarely go to hospital. An exception is Kuala Perlis, a village of about 1,200 fishermen in northern Malaya, from which 30 cases of sea-snake bite

were admitted in 1953 and 29 in 1954 (A. A. Wahab, 1955. personal communication). Smith (1926), writing of the Gulf of Siam, says, "There is hardly a village that cannot tell you of its fatalities." The preliminary findings of the survey of fishing villages already mentioned fully confirm this statement. Kuala Muda, a river-mouth 60 miles south of Kuala Perlis, has 10–15 cases each year amongst 750 fishermen (two fatal cases in 1954 and 1955).

In eight small villages (averaging 200 fishermen each) around Penang island I have so far personally interviewed the victims or close witnesses in 43 cases (8 fatal) occurring during the last 10 years. For varying reasons this is a considerable underestimate. Villages vary, but sea-snake bite is certainly not rare, especially at river-mouths. I believe that with improved transport, diminishing prejudice against hospitals, etc., many more cases amongst fishermen will come to light, as has already happened in north-west Malaya. This area contains a minute fraction of Asian fishing villages. Unless it is exceptional in the incidence of sea-snake bite (and there seems to be no reason why this should be so), the yearly toll amongst Asian fishing folk must be considerable, the mortality far from negligible.

Is it Safe to Bathe?

Sea-snakes, though very poisonous, are inoffensive and bite only under considerable provocation (Wall, 1909; Smith, 1926; Herre, 1942; Tweedie, 1953). However, two bathers were fatally bitten on the same afternoon in 1954, both ironically enough near "Lover's Isle," a famous beauty spot of Penang and (formerly) a favourite bathing-place. This posed the question, is it safe to bathe along the vast coastline frequented by sea-snakes? Millions must have previously bathed or paddled without harm. The risk is obviously infinitesimal. However, this statistic would be of small solace to a victim and not very reassuring to anxious mothers who are aware of the existence of sea-snakes.

Of 68 cases recorded or personally investigated 15 were bitten (7 fatally) whilst bathing, wading, or paddling for pleasure. Of the remaining 53 victims, 36 (11 fatal cases) were fishermen bitten at work, in 13 the circumstances of the bite (5 fatal) were closely connected with the fishermen's occupation, and 4 non-fishermen were fatally bitten when they grabbed the snake. As the vast majority of victims are fishermen not observed by doctors, these figures mean little except that a sea-snake bite whilst bathing is a possibility, albeit remote. For the fisherman it is an occupational hazard which many accept with apparent nonchalance, although fully conversant with the implications.

Are there any special factors that make bathing dangerous? Little is known about the breeding habits of sea-snakes, although at such times they are said to be more active and aggressive (Herre, 1942). Many fishermen believe that altered salinity makes them attack people. This theory is supported by the fact that certain villages at larger river-mouths have a much higher incidence of bites, especially among bathers (8 of the 15 cited above were bathing, etc., in a large river-mouth). However, the latter may merely be due to sea-snakes being commoner around river-mouths (Smith, 1926). In captivity there is no doubt about the reluctance of the sea-snake to bite. I have observed a number of different species in glass tanks and have repeatedly tried to goad them into biting a plastic doll, fish, etc., both in the water and on the floor, without any success, provided they are not held. My present opinion is that these bathing cases are unfortunate coincidences, and it is just as safe to bathe now as always. But it would be wise to avoid river-mouths.

The Outcome

The outcome of a bite depends essentially on the amount of venom injected. Only a minority of those bitten develop severe symptoms or die. I have been accidentally bitten twice by sea-snakes, and dogs have been experimentally bitten with little or no evidence of poisoning.

It would thus be difficult to assess the effectiveness of antivenene in human victims. So far it has not proved possible to make specific antivenene, although the problem is being pursued. The mortality in cases of krait-poisoning treated by polyvalent but not specific antivenene is 77.1% in India (Ahuja and Singh, 1954). Similarly, polyvalent antiserum is not likely to be effective in sea-snake poisoning except in large amounts (preliminary findings suggest that antivenene with a krait fraction has limited neutralizing power against sea-snake venom). Little progress seems to have been made during the last 30 years in refining and concentrating antivenenes. Discussing this problem in 1921, Acton and Knowles pointed out that, for a "large" bite, over 700 ml. of antivenene would be required within a quarter of an hour. Polyvalent antivenenes available to-day are feeble affairs, 1 ml. neutralizing less than 1 mg. of venom. Specific antivenenes are not much more potent.

Theoretically, to counteract a "large" bite from the commoner venomous snakes an enormous dose of antivenene would still be necessary. As with diphtheria, the sooner the antiserum is circulating the more effective it is likely to be. Dividing and delaying the dose, as is often done, is irrational; but a pint (570 ml.) of antivenene by rapid intravenous drip would probably kill the patient. Even with 10 ml., reactions have often been very severe despite cortisone, etc. One is therefore reluctant to "push" intravenous polyvalent antivenene dosage. Subcutaneous injection with hyaluronidase may solve this problem when a specific antiserum becomes available. It will then be more important to decide whether the victim has received a "large" bite or not. Points indicating this are: (1) the snake has been seen to be fat—for example, thicker than a man's thumb; (2) fang marks more than 10 mm. apart; (3) rapid evolvement of poisoning symptoms—for example, several features within half an hour; and (4) haemoglobinuria and failing vision. With one or more of these criteria, admittedly far from infallible, a large dose of antivenene would be indicated.

Experimentally, Rogers (1902–3) noted a similarity to tetanus toxin, which Wright (1954) found to be rapidly fixed to cells of guinea-pig brain stems. More than half the dose injected was fixed within two hours. Presumably sea-snake neurotoxin reaches the brain stem much more quickly than tetanus toxin. If a lethal dose is injected and fixed like tetanus toxin the outlook would appear to be grim. However, Richards (1873) kept his dog alive for over 24 hours by artificial respiration. Using similar methods, Lassen (1953) reduced the mortality of bulbospinal poliomyelitis from 94% to less than 40%, and Shackleton (1954) showed that cases of tetanus hitherto deemed inevitably fatal could be saved. As permanent after-effects of sea-snake poisoning are unknown, severe effects are usually short-lived, and there is experimental evidence that venom is excreted in the urine, it is possible that modern methods of artificial respiration by intermittent positive pressure will save many severe cases. With a specific antivenene the mortality should be even further reduced.

Summary

The clinical picture, diagnosis, and treatment of sea-snake poisoning are outlined.

Further problems are mentioned. The incidence and outcome of sea-snake bites and the safety of bathing are discussed. Cases amongst bathers occur, but are extremely rare except in river-mouths. In contrast, the incidence in fishermen and their kin is much higher than the scanty literature would indicate. A specific antivenene, adequately concentrated and refined, is urgently needed for these cases.

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OBSTETRIC INDICATIONS FOR THE USE OF PUDENDAL NERVE BLOCK ANALGESIA

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The problem of anaesthesia during the second stage of labour is complicated by the fact that the safety of both mother and foetus has to be considered. Anaesthesia often has to be induced in an unprepared patient in whom the foetus is showing signs of distress.

The technique of pudendal nerve block is simple and safe, and it avoids many of the dangers and difficulties of inhalation, extradural, and spinal anaesthesia. It produces analgesia of the lower vagina, the vulva, a varying area of surrounding skin, and some relaxation of the pelvic floor. Excluding those patients who are mentally unsuited for local analgesic techniques, safe and reliable analgesia can be produced for a number of obstetric manœuvres with a minimum risk to the mother and her yet unborn infant.

Anaesthetic Mortality and Morbidity

The present low maternal death rate due to anaesthesia should not lead to complacency, as there are fewer greater tragedies to a family than the loss of its mother. Inhalation anaesthesia is the most common form of anaesthesia used during the second stage of labour. Its complications are the inhalation of vomit or regurgitated stomach contents, depression of the foetal respiratory centre, and relaxation of the uterus leading to post-partum haemorrhage. Spinal and extradural techniques need even more specialized experience, and there is always the risk of infection and permanent spinal cord or nerve damage. Both of these techniques produce varying degrees of hypotension.

Inhalation of vomit has been reported as a cause of death by many workers, and consideration must be paid to the asthmatic type of aspiration pneumonitis caused by the inhalation of acid stomach contents. Mendelson (1946) described 60 cases giving rise to two fatalities; these occurred among 44,016 cases.

The following authors have also described deaths due to aspiration of stomach contents. The incidence is higher than in prepared surgical cases, but often anaesthesia in obstetric practice is administered as an emergency, and it is well known that the emptying time of the stomach in women in labour is delayed. Eastman (1941) reported 51 deaths; an incidence of 1 in 40,000 anaesthesias. Dieckmann (1945) reported 45 cases in 46,000 deliveries (2 deaths; an incidence of 1 in 23,000 anaesthesias. Gilliatt (1949), reviewing the causes of death among 55 patients, found that 3 were due to inhalation of vomit; an incidence of 1 in 11,382 anaesthesias. Merrill and Hingson (1951) reported 59 deaths; an incidence of 1 in 44,135 anaesthesias. Parker (1954) reported 5 deaths; an incidence of 1 in 3,708 anaesthesias. Hall (1940) and Willcox (1949) have also reported five and two deaths respectively.

The variation between the British and American figures is explained by the more frequent use of general anaesthesia during the second stage of labour in American hospitals.

Local analgesics rarely give rise to death, and when death does occur the following factors are usually involved: (1) large amounts of agent, (2) intravenous injection, and (3) indiosyncrasy. The first two factors are controllable and preventable; the third can be minimized by interrogation about previous idiosyncrasy, and the symptoms can be controlled by the administration of a rapidly acting barbiturate. Idiosyncrasy is very rare.

The toxic effects of lignocaine have been discussed in the correspondence columns of the *British Medical Journal* (1955). There is no doubt that lignocaine can be dangerous, but not if the maximum dosage is kept well below 1 g. in a single dose. Many of the cases mentioned as having had toxic reactions have one thing in common—namely, that 2% lignocaine had been used. Lock and Greiss (1955) report a death following the injection of 40 ml. of 2% lignocaine for pudendal nerve block in a secundipara with mitral stenosis. She died seven minutes after the injection. Dutton (1955) reports a case in which convulsions occurred after 80 ml. of 1% lignocaine had been used. Therefore safety lies in weak solutions of lignocaine, used in the minimum quantity necessary for effective analgesia.

The Pudendal Nerve and its Branches

The pudendal nerve arises from the second, third, and fourth anterior sacral trunks, which form a single trunk 0.5 to 1 cm. proximal to the ischial spine. The nerve trunk courses through the greater sciatic foramen inferior to the pyriformis muscle, passing posterior to the ischial spine—between the sacrospinous ligament anteriorly and the sacrotuberous ligament posteriorly. As the nerve passes the inferior tip of the ischial spine it enters the pudendal canal. The nerve lies medial to the pudendal vessels. It divides into three main branches: the inferior haemorrhoidal nerve; the dorsal nerve of the clitoris, and the perineal nerve.

Inferior Haemorrhoidal Nerve.—Klink (1953) in a series of 85 dissections found that in 50% of cases this nerve did not arise from the pudendal nerve but as a separate branch from the fourth anterior sacral primary ramus. In these cases the nerve did not lie in the pudendal canal but passed through the sacrospinous ligament 0.5 to 1 cm. inferomedial to the pudendal nerve. This nerve supplies the mucosa of the lower part of the anal canal, the external anal sphincter, and the skin anterior and lateral to the anus.

The dorsal nerve of the clitoris runs between the layers of the urogenital diaphragm, terminating in the filaments supplying the clitoris.

The perineal nerve arises above the inferior border of the ischial tuberosity and divides into superficial and deep branches. The former constitutes the main nerve supply of the superficial structures of the vulva and perineum, and also anastomoses with the perineal branches of the posterior