Pre-Natal Causes:

Infections (toxoplasma, syphilis, rubella).

Hereditary causes (cranial anomalies, phenylketonuria, others).

Maternal irradiation.

Cretinism.

Unknown causes (mongolism, cerebral palsy, primary amentia).

Natal Causes:

Birth trauma, mainly anoxia.

Post-Natal Causes:

Kernicterus.

Degenerative cerebral diseases.

Infections (encephalitis, meningitis).

Head injury.

Lead and other poisons.

As a rule, the history and examination reveal the cause readily enough when one exists. But the majority of cases are due to developmental defect of the brain for no reason that we can yet understand, and it is therefore important, once again, not to let the parents blame themselves or anyone else for this all too common and seemingly natural event.

Having accepted the fact that their child is backward they will possibly next inquire whether he will have to be sent to a home. This question brings up the whole matter of future management, and the answer depends on the child's actual status. Let us consider three groups, and agree that children of less than mongoloid capacity—or, to put it another way, children whose mental age proves to be less than half their actual age—are best admitted to an institution at once for permanent care.

At the other end of the scale, children above mongoloid capacity are generally happiest in their own homes, provided that the family is a united one and living conditions are normal. They need love and understanding, of course, and in particular they need praise for their daily achievements, however simple. Too often we forget that backward children, as they grow older, are sensitive, and well aware of their lack of skill and success in nearly all the things that make up daily life. Training them for simple routine household jobs, such as laying a table, watering the garden or feeding the chickens, and frequently telling them how useful and helpful they are becoming, and how good it is to be able to rely on them, not only sweetens family life but makes these handicapped youngsters feel important, and gives them a sense of fitting into the only community they understand. Everything that we doctors can say and do to help parents accept these children as affectionate little helpers, and not as liabilities, is worth while. Everything that helps them develop independence and responsibility should be tried. There are all sorts of simple handicrafts that they can attempt. Older boys can grow vegetables in the garden, sell them to mother for pennies and spend the pennies in the shop. Girls can help with shopping and housework. They can watch for the postman and bring in the letters, and so on. And every day their parents ought to be able to find something for which to praise them. Discipline is necessary, of course. There must be rules in every home, and these must be kept; but scorn and criticism simply wilt them. Jenny, the little girl I mentioned earlier, was thawing nicely after a shy start, and had just whispered confidentially "I got a sore tooth, too", when her mother cut in coldly: "He's not a dentist, stupid." Jenny was seven, you may remember, and her mental age was three and a half. As childhood ends, there come the problems of fitting these children for adult life. Unfortunately I have no time to discuss this, except to say that the rehabilitation programme of the Commonwealth Social Services Department has plans for training the intellectually, as well as merely the physically, handicapped. In some parts of the world progress has been made in teaching simple routine manual tasks to subnormal adolescents, even down to the imbecile level.

So much for those below and those above mongoloid level. The third group involves mongoloid children themselves. Authorities have differed about their management, some urging that they be brought up at home and others that they be separated from their parents at birth and sent to an institution. My own views about them are between the two. There does certainly come a time, around the age of eight years, when their mental age is about four years, at which they need the companionship of others of their own kind and seem happiest in groups. From this stage onwards institutional care is right and proper. But in infancy they are like any other babies, save that they remain so for a longer time, and their needs are identical, and in early childhood they need the individual care and affection that only a mother can give, and definitely develop faster and further than those unfortunate enough to be in State homes.

I promised to tell you more about a child whose parents had been informed at her birth that she was mongoloid and that there was "no hope" for her. Just the other day a letter arrived, and I read:

> The dearest and most adorable child that ever gladdened a parent's heart died on June 17th. . . . Really, Doctor, she was as sunshine in a dark room and we all so dearly loved her. . . . At present our loss seems irreparable but we will always remember the love that she brought to our house.

It adds point to this letter to explain that it was written, not by the mother, but by the father, and that he was a husky member of a State police force.

Conclusion.

There is enormous scope, of course, in pædiatric illnesses, for medical science in all its aspects. But there is also a place, where children are concerned, for more than mere science, for children are complex little beings, and technical skill alone can never discover all their secrets. From the reports of those who knew him best, it was this other special quality, an understanding heart, that Harry Swift possessed. It was one of his greatest attributes, one of the factors in his success.

THE SYMPTOMATOLOGY, CLINICAL COURSE AND SUCCESSFUL TREATMENT OF PAPUAN ELAPINE SNAKE ENVENOMATION.

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THE Papuan and Australian venomous terrestrial snakes all belong to the family Elapidæ. The most venomous Papuan species, the taipan and the death adder, are common to both countries, while a third highly venomous and common Papuan snake, the Papuan black snake, is related to some common Australian venomous snakes (the red-bellied black snake and the king brown (mulga) snake).

Elapine snake venoms are complex mixtures of proteins with enzymic activities (Braganca and Quastel, 1953), and usually have their main toxic effects on the nervous system. After a serious elapine snake bite in man a characteristic clinical picture is produced, the main feature of which is a progressive paralysis of all the ocular, bulbar, jaw, facial and spinal muscles, with eventual death from respiratory obstruction or terminal peripheral failure of the diaphragm.

It has long been known that this paralysis, however severe it may be, is completely reversible if a "sublethal" dose of venom has been received, and it has always

been the hope of workers in this field that the paralysis would still prove to be completely reversible even after the injection of potentially lethal doses of venom, if the patient's life could be maintained during his period of respiratory insufficiency. Up till now there has been little, if any, clinical or experimental work to justify this hope.

We wish to record here our observations of 15 cases of elapine snake envenomation of varying severity, and to report successful treatment in potentially fatal cases of severely paralyzed patients, with antivenenes, tracheotomy and artificial respiration.

The patients were all Papuan natives admitted to the Port Moresby General Hospital over a ten-month period from November, 1959, to August, 1960, and were brought to hospital by road or aeroplane from within one hundred miles of Port Moresby. As many of the patients were too ill to give a history on admission (some being unable to speak), most of the histories were obtained through interpreters, retrospectively, after the patient recovered. As a consequence of this, the subjective sensations resulting from snake envenomation may be inadequately described. After the patient's admission to hospital, an hourly record of the pulse and blood pressure was kept throughout the first few days of the illness, and the progress of the condition was closely observed.

The main features of each case are summarized in Table I, and the clinical picture and treatment are described in more detail below.

Circumstances of the Bite.

With the exception of one, all the patients were bitten in the daytime. Each was barefooted at the time of the bite and was bitten on the foot or the leg. The bite occurred while the patient was walking through long, coarse grass, from one to several feet high, or along a bush track, from one to three feet wide, bordered by this grass. Three patients actually trod upon the snake. The other patients did not see the snake before it struck, and some never saw it at all. Only three patients thought that the snake grasped the bitten part between its jaws.

Clinical Picture.

Local Symptoms and Signs.

In the majority of cases there were no local symptoms or signs of any significance. Pain at the site of the bite was usually absent or mild. Severe pain in the whole of the bitten limb was complained of by only one patient. This man also had tender, enlarged inguinal lymph nodes, which were noticed within 12 hours of the bite. Slight œdema of the bitten part was present in four cases. A fifth patient, who had been bitten on the leg, had extensive swelling and œdema of the leg and foot, which persisted for 11 days. (In this case a tourniquet had been left on for four hours.)

General Symptoms.

Some symptoms or signs of systemic envenomation were always present within 12 hours of the bite, and often the first symptoms appeared within two or three hours. Vomiting was one of the earliest symptoms, the patients usually vomiting several times. Three patients also vomited small amounts of blood. After the vomiting ceased, three patients developed abdominal pain which lasted a few hours. (We have seen a patient suffering from severe snake bite admitted to hospital with a provisional diagnosis of acute appendicitis, owing to complaints of vomiting and abdominal pain associated with marked abdominal tenderness.) Six patients complained of mild headache, which lasted from a few hours to two days.

In addition, the patients complained of one or some of the following symptoms: drowsiness, blurred vision, double vision, inability to open the eyes, difficulty in speaking or swallowing or opening the mouth, general muscle weakness, heaviness of the bitten limb and the passing of blood-stained urine.

TABLE I.

Summary of the Clinical Features, Treatment and Results of 15 Cases of Elapine Snake Envenomation.

Case Number.	Approximate Age. (Years.)	Sex.	Symptoms and Signs.													Treatment.																Recovery Period. (Days.)	Residual Disability or Outcome.
			Headache.	Vomiting.	Abdominal Pain.	Visual Disturbance.	Paralysis[a] of: Eyelids.	Eye Movement.	Face Muscles.	Tongue.	Palate.	Jaw Muscles.	Head, Neck.	Intercostals.	Diaphragm.	Arms.	Legs.	Bleeding Tendency.	Antivenene.[b] 1/M.[c]: Tiger.	Taipan.	Papuan Black.	Death Adder.	1/V.[d]: Tiger.	Taipan	Papuan Black.	Death Adder.	Tracheotomy.	Artificial Respiration.	Prognosis without Treatment.		
I	17	M.	?	Yes.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	0	6				18 24			Yes.	Yes.	Death.	8	None.
II	20	M.	?	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+						18 18			Yes.	Yes.	Death.	5	None.
III	35	M.	No.	Yes.	No.	?	+	+	+	+	+	+	+	+	+	+	+	+						30 12			No.	No.	Survival.	6	None.
IV	25	M.	No.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+						12 36			Yes.	No.	Survival.	9	Death.
V	10	F.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	24	3							No.	Yes.	Survival.	—	None.
VI	30	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	24	6				18 30			Yes.	Yes.	Survival.	5	None.
VII	35	F.	No.	Yes.	Yes.	No.	+	+	+	+	+	+	+	+	+	+	+	+	30	9				30 30			No.	No.	Survival.	6	None.
VIII	13	M.	No.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	15					30 30			Yes.	No.	Survival.	5	None.
IX	25	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	6					30 36			No.	No.	Survival.	5	None.
X	14	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+									No.	No.	Survival.	1	None.
XI	40	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+									No.	No.	Survival.	8	None.
XII	20	F.	Yes.	Yes.	Yes.	?	+	+	+	+	+	+	+	+	+	+	+	+									Yes.	No.	Death.	62+	Yes.
XIII	25	M.	?	?	?	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	45	3				30 30			Yes.	Yes.	Death.	6	None.
XIV	17	M.	No.	Yes.	Yes.	No.	+	+	+	+	+	+	+	+	+	+	+	+	30	9				30 30			No.	No.	Survival.	6	None.
XV	25	M.	Yes.	Yes.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	6					30 36			Yes.	Yes.	Death.	21	None.

"?" = No deviation from normal. "+" to "+++" = Severe degree of involvement. [a] Thousands of units. [b] I/M = intramuscular administration. [c] I/V = intravenous administration.

General Signs.

Nervous System.

Bilateral ptosis was present in all cases. The paralysis of the eyelids was partial or complete, depending on the severity of the case, and was usually the earliest objective sign of snake envenomation to develop, appearing as early as two hours after the bite. This was followed by impaired ocular movements in a vertical or lateral direction. The ocular movements gradually became more restricted in the serious cases until no movements were detected at all. The eyes were fixed and central. The complete ptosis with the fixed central eyes presented quite a charactertistic clinical picture. The pupils were slightly to moderately dilated and always reacted to light.

In all but the mildest cases, the muscles of the jaw, the palate and the tongue were involved, usually after paralysis of the eyelids and the extrinsic eye muscles had commenced. The degree of their involvement varied from slight weakness, which was manifested by a nasal voice, slight difficulty in swallowing and inability to open the mouth completely or to protrude the tongue properly, to complete paralysis. In the last-mentioned circumstance, the patients were unable to speak or swallow at all, and secretions accumulated in the pharynx. The tongue was completely immobile on the floor of the mouth, and no movement of the palate could be detected. The patients were unable to open or close the mouth, the teeth being only a quarter of an inch apart. Sometimes the jaw was freely movable backwards and forwards by hand, and in the supine position, with gravity, it readily fell backwards, leading to respiratory obstruction. The patients with the most severe paralysis also had weakness or almost complete paralysis of all the muscles of the face. They were unable to wrinkle the forehead or move the lips or nose significantly. Complete paralysis of the muscles of the face developed late in the course of the illness.

A variable degree of involvement of the muscles of the neck, the trunk and the limbs was present in all but two cases. Usually the neck and trunk muscles were more extensively paralysed than the limb muscles. Often the patients were unable to move head or trunk at all. The limbs were symmetrically paralysed in nine cases, the proximal muscle groups always being more affected than the distal ones, and the paralysis was never complete. Even in the most severe cases, a finger or a hand, and some toes or a foot, could still be moved slightly. In three cases the muscle weakness of the limbs was more marked in the bitten limb.

Paralysis of the intercostal muscles usually occurred only after there was extensive paralysis of all the ocular and bulbar muscles. In the most severe cases the paralysis was complete. Respiration became entirely diaphragmatic in character. Progressive weakness of the diaphragm then occurred over a period of many hours, eventually terminating in complete failure of this muscle. Difficulty in breathing was thus a feature of all the serious cases, and was due to respiratory obstruction, or to paralysis of the intercostal muscles and the diaphragm. Observation of other patients indicates that death finally occurs from the effects of anoxia resulting from failure of the diaphragm, from respiratory obstruction or a combination of both these factors.

All the patients appeared drowsy on their admission to hospital, and some were cyanosed and occasionally restless. All but one patient remained cooperative in spite of severe paralysis, and appeared to hear and tried to obey commands. However, on recovery two of the most severely paralysed men (Cases I and XV) could not remember any details of the earlier part of their illness. One of these men (Case I) had had some injections of paraldehyde. One patient (Case XIII) appeared confused and restless throughout the earlier part of his illness, but this was atypical.

In five of our less serious cases, no abnormality in the deep tendon reflexes was detected. In the other cases, depending upon the severity of the limb paresis, there was a decreased reflex response or a complete absence of deep reflexes for some days.

Whenever possible, sensation (pain, light touch and, occasionally, vibration and joint sense) was tested. No generalized sensory changes were detected. In three patients there was some loss of pain and touch sensation over a small circumscribed area in the vicinity of the wound.

In cases in which muscle weakness alllowed it, muscle coordination was tested and no impairment was found. There was no disturbance of micturition.

Cardio-Vascular System.

There was singularly little involvement of the cardiovascular system. The blood pressure was usually well maintained (any significant alteration being related to inefficient ventilation of the patient or excessive bleeding from the tracheotomy wound, and responded quickly to treatment of the underlying cause). Transient tachycardia was observed in several cases before tracheotomy was performed, but once the anoxia was relieved the pulse rate returned to normal.

Urine.

Eleven patients had albuminuria, which persisted for three to four days. However, in some cases, the urine was obtained after the serum had been given. The centrifuged deposit of the urine sometimes contained up to 10 red cells or polymorphonuclear leucocytes per high-power field, with granular and hyaline casts occasionally being noted. One patient (Case V) passed heavily blood-stained urine after admission to hospital and another man (Case XIV) passed black urine. In these two cases these signs were the first objective evidence of envenomation, and they were followed by mild neurological signs.

Blood.

Some patients bled freely from the first-aid incisions and later from the tracheotomy wound, for one to three days. Venepuncture wounds in these patients were also noted to bleed excessively. Because of the presence of anoxia and recent first-aid trauma, together with the fact that blood counts were sometimes not obtained until after the serum had been given, no significance could be attached to changes in the white-cell count.

Cerebro-Spinal Fluid.

In two cases lumbar puncture was performed. The pressure was normal, and no abnormality was detected on microscopic or biochemical examination of the fluid.

Treatment.

First-aid treatment had been attempted in three-quarters of the cases. All the patients had been bitten many hours before admission to hospital. The shortest period between the bite and admission was four hours. The most urgent need on admission was the establishment of an adequate airway. A tracheotomy was performed under local anæsthesia in nine cases as soon as possible after admission. A cuffed tracheotomy tube (James tube) was inserted. Lives were sometimes saved by passing a cuffed endotracheal tube in the ward. Because of a shortage of trained staff throughout the 24 hours, we have felt it safer to err on the side of caution in performing an unnecessary tracheotomy, rather than have the patient develop obstructed breathing at night. The tracheotomy tube was usually left in for four to seven days. An anæsthetic machine (utilizing a semi-closed circuit with a soda-lime absorption unit) was used manually, mainly by semi-trained staff, to assist the patient's failing respiration in four cases. The longest period over which respiration had to be assisted was 84 hours.

When there was difficulty in swallowing, oral feeding was suspended and hydration was maintained by intravenous infusions of isotonic glucose-saline solutions. This usually had to be continued for three to seven days. Two patients, who bled freely from tracheotomy wounds, had to have blood transfusions.

The oral and tracheal secretions were sucked out as required. The patient's position was changed and the cuff of the tracheotomy tube was deflated every two hours. As a prophylactic measure antibiotics were given intramuscularly, and if required, intramuscular injections of paraldehyde were used to sedate the patient.

The Commonwealth Serum Laboratories have prepared three specific antivenenes—namely, taipan, death adder and Papuan black snake—for use in Papua. Australian tiger snake antivenene is also freely available, and is said to give some cross-protection in cases of bites by other venomous snakes. Antivenenes were given to all patients in quite large single doses intravenously (up to a total of 90,000 units). Some of the patients had already received small or large doses of antivenene (usually tiger snake) prior to admission to hospital.

Usually two or three types of antivenene were given to each patient in doses of 12,000 to 30,000 units each. If the snake was seen and it was a small one, death adder antivenene was included in the régime. If excessive bleeding was present, Papuan black snake antivenene was also given, but otherwise no set régime has been followed. Most commonly, taipan and tiger snake antivenenes were given together.

Untoward reactions to the serum were usually not serious; pyrexia, rigors, periorbital œdema, wheezing, coughing and pruritus were encountered. One patient died from acute pulmonary œdema, and it is thought that this was probably due to a reaction to the antivenene or to the excessive intravenous administration of fluids (Case V). Several injections of negostimine were given to two patients with severe paralysis without any beneficial effect.

Stage of Recovery.

In the mild cases, quick and complete recovery occurred, often within two to three days of the bite. In the most severe cases, one to three days after the bite the diaphragm movements became stronger and did not need assistance. The faintest flicker of eye movement was seen then or shortly before this (usually two to two and a half days after the bite), and within the next 12 to 24 hours definite eye movements were detectable. Completely normal eye movements were present within the following 24 hours. At the same time the ptosis decreased. The paralysed bulbar muscles improved more slowly, but the patient was able to swallow and speak within four to eight days of the bite. The weakness of the limb muscles diminished before that of the neck and trunk muscles. A complete recovery had usually occurred within five to 14 days of the bite. However, in one case the bulbar paralysis has persisted for six to ten weeks and improvement is still occurring. This patient has residual disability also from an upper motor neuron lesion affecting the left side of the face and the left hand (Case XIII).

DISCUSSION.

Species of Snake Inflicting the Bite.

Five venomous terrestrial snakes in Papua are capable of inflicting a fatal bite (Slater, 1956)— namely (i) the Papuan taipan (*Oxyuranus scutellatus canni* Slater); (ii) the Papuan black snake (*Pseudechis papuanus* Peters and Doria); (iii) the death adder (*Acanthophis antarcticus rugosus* Loveridge); (iv) the Papuan whip snake (*Demansia psammophis papuensis* Macleay); and (v) the small-eyed snake (*Micropechis ikaheka fasciatus* Fischer). As bites from only the rare, very large specimens of the last two species are likely to be lethal for man, fatal snake bite in Papua is due most commonly to a bite from a Papuan taipan, a Papuan black snake or a death adder. These three reptiles are all highly venomous and are not uncommonly encountered.

For its size (usually two and a half feet or less in length), the Australian death adder was regarded by Fairley (1929*c*) as the world's most deadly terrestrial snake. However, the taipan, because of its large size (up to 11 feet in length), its efficient biting apparatus and its production of a venom which is only slightly less toxic than that of the extremely potent Australian tiger snake venom (Morgan, 1956), is generally accepted as Australian's most dangerous snake (Kellaway and Williams, 1929; Morgan, 1956). The venoms of the Papuan taipan and the Papuan death adder are possibly very slightly more toxic than those of their Australian counterparts (Slater, 1960). Papuan black snake venom is much more toxic than the Australian black snake venom, but although this large Papuan snake has a high venom yield, the venom has only about one-eighth the potency of taipan venom (Slater, 1960). The taipan and the Papuan black snake are active by day, while the death adder is active by night (Kinghorn, 1956; Slater, 1956), and was regarded by Fairley (1929*c*) as the only Australian venomous snake to be encountered commonly at night.

In none of the cases reported above was the species of the snake causing the bite determined. This is a common experience of those dealing with snake bite clinically (Acton and Knowles, 1914; Clark, 1942; Ahuja and Singh, 1954), and raises considerable difficulties in connexion with antivenene treatment.

Factors Determining a Lethal Bite.

The presence or absence of symptoms following venomous snake-bite, and the rapidity with which symptoms develop and lead to death, are mainly dependent upon the amount of venom injected in relation to the size of the victim. For a given venom, the death time (the interval between the bite and death) is roughly proportional to the amount of venom injected (Acton and Knowles, 1914).

Apart from its size, many factors in the ecology of the snake, such as moulting, disease, recent feeding or biting and hibernation, influence the amount of venom available for injection at a given time. Individual variations in the venom yield also occur from time to time in the same snake for no apparent reason (Fairley, 1929*c*). Inefficient biting also may considerably reduce the amount of venom injected. A study of the biting apparatus of elapine snakes by various authors (Acton and Knowles, 1914; Fairley, 1929*b*) has shown that the largest amounts of venom are injected when the lower jaw is fixed, and a snake of this family bites at a mechanical disadvantage when it strikes at a flat surface such as the calf of the leg.

The larger the dose of venom received, the shorter the death time (Lamb and Hunter, 1904*b*; Acton and Knowles, 1914). If the venom is injected intravenously or if a massive dose is injected subcutaneously, death may occur within half an hour of the bite (Acton and Knowles, 1914; Kellaway, 1942). Such occurrences are very rare, and death most commonly occurs within two to 48 hours of a venomous terrestrial snake bite. In cases reported in Australian literature, death has more commonly occurred towards the end of this period (eight deaths in less than three hours of the bite and 62 deaths after more than 12 hours out of a total of 81 fatal cases—Fairley, 1929*a*).

In seven of the cases reported above, we believe that death almost certainly would have occurred without treatment. There is no doubt in our minds that these seven patients received a dose of venom that would have proved to be a lethal one without treatment. In none of these cases, without treatment, would the estimated death time have been under eight hours, so probably none of the patients received a massive dose of venom. These cases are quite comparable in severity to a number of fatal cases of snake bite reported in the Australian literature (Ferguson, 1926; Fairley, 1929*a*; Flecker, 1940; Kellaway, 1942; Benn, 1951). They are not as severe as the cases reported by Flecker (1944). The other patients in this series received a sublethal dose of venom, or a lethal dose that was modified by antivenene given prior to admission to hospital (tiger snake antivenene in small or large doses over a period of up to 36 hours), and recovery undoubtedly would have occurred without hospital treatment.

The Clinical Effects of the Venom.

Although the venoms of poisonous Australian snakes, like those of other elapine snakes, contain several toxic

factors such as enzymes (Porges, 1953), the clinical picture following envenomation is dominated by the effects of the neurotoxins, and death is usually due to the action of these toxins (Fairley, 1929*a*; Kellaway, 1942). This particularly applies to the venoms of the death adder and the taipan (Kellaway, 1929*c*; Kellaway and Williams, 1929). Because of this, it is usually clinically impossible to determine the species of the elapine snake causing the bite from the symptomatology produced (Lamb and Hunter, 1904*b*; Lamb and Hunter, 1906; Fairley, 1929*a*; Kellaway, 1942), and the symptoms described above resulting from bites of Papuan elapine snakes are very similar to the symptoms which follow bites by the cobra and the krait, the main venomous elapine snakes of India (Lamb and Hunter, 1904*a*, 1904*b*).

There has been no published work on the venom of the Papuan black snake, but clinical observations suggest that death from bites of this snake may sometimes be due to anticoagulant or hæmolytic effects of the venom. If these observations are substantiated, and work on the venoms of other snakes of the same genus suggests that they will be (Kellaway and Williams, 1929; Kellaway, 1930), clinical signs may be of some assistance in indicating that bites from this snake have occurred.

The main effect of elapine neurotoxins falls on the motor component of the nervous system, where they appear to have a curare-like action at the neuro-muscular junction, but they may also have some direct action on the muscle itself (Kellaway, 1932; Kellaway, Cherry and Williams, 1932). This action at the neuro-muscular junction is complex (Porges, 1953) and is uninfluenced by neostigmine.

Clinically, the susceptibility of the extrinsic ocular and eyelid muscles and the resistance of the diaphragm to the action of the neurotoxins would seem to support this theory of a curare-like action. Some features of interest in their action are the possibly long latent period before the effect is apparent, the long duration of their effect and the occasional maximal severity of their peripheral effect in the bitten limb. The drowsiness probably represents a central action of the neurotoxins, and in two of the severest cases the patients have had amnesia for the events of the first few days of their illness, although they had apparently remained conscious and cooperative.

The ocular and the bulbar muscles are usually most severely affected by the action of the neurotoxins. The fact that death may occur from involvement of the bulbar muscles has not been sufficiently stressed in the Australian literature. Death must occur quite commonly from this cause. The jaw may drop back; the tongue is paralysed and cannot move, and respiratory obstruction occurs in a patient who cannot move his head significantly. The patient cannot swallow, and secretions accumulate in the pharynx and may be drawn into the lungs and trachea. Fairley (1929*a*) commented on the fact that the tracheæ and bronchi of sheep were packed with food debris after death due to tiger snake venom. Also, owing to the paralysed intercostals and the weak diaphragm, bronchial secretions cannot be coughed up. With an airway obstructed by secretions or by the tongue, breathing becomes laboured, and the weak intercostal muscles and the diaphragm may cease working at a much earlier stage than they would have done had the anoxia been corrected. In some of our cases the improvement in the patient's general condition and respiratory movements after tracheotomy and the insertion of a cuffed tracheotomy tube was dramatic. However, in the most severe cases, even if an adequate airway is maintained, and oral secretions are prevented from being aspirated into the trachea and lungs while endobronchial secretions are removed, death will occur from paralysis of the intercostal muscles and diaphragm if artificial respiration is not resorted to. When respiration is assisted, complete failure of the diaphragm occurs many hours after the first onset of its weakness.

Once the effects of anoxia are removed, the lack of involvement of the cardio-vascular system by the venoms is to be noted. Many workers, experimenting with various elapine venoms in animals, have commented on the fact that the heart continues to beat strongly for some time after the cessation of respiration and before death (Lamb and Hunter, 1904*a*; Acton and Knowles, 1921; Fairley, 1929*a*; Kellaway, 1929*a*, 1929*b*, 1929*c*; Kellaway and Williams, 1929). However, Kellaway (1942) thought that the cytolytic action of Australian snake venoms led to the liberation of histamine, and that symptoms of cardio-vascular failure after bites of Australian snakes were due to this. However, a study of some such case reports suggests that chronic anoxia and carbon-dioxide retention may have been the cause of these cardio-vascular symptoms.

In several cases in this series, there has been a mild anticoagulant effect from the venoms, and in one case a mild hæmolytic effect. Albuminuria was an inconstant feature of envenomation, as was also microscopic hæmaturia.

The Reversibility of the Muscular Paralysis.

If the patient survives, the muscle paralysis is usually completely reversible, even if an apparently lethal dose of venom has been received. This fact needs to be stressed. Acton and Knowles (1921) state that in the case of cobra bites in animals, paralytic effects, however severe, are rapidly recovered from if the animal survives. The same observation has been made by Kellaway (1929*b*) in the case of bites from the Australian copperhead snake, and by Fairley (1929*a*) in the case of bites from the Australian tiger snake.

Clinically, muscles that were completely paralysed usually remained so for two to five days, and then recovery commenced, the process of recovery continuing for another two to 10 days. The usual course is for the paralysis to clear completely within five to 14 days of the bite without any residual disability. We believe that this represents the normal course of this peripheral effect of the venoms, in spite of the antivenenes administered. In one of our most severely ill patients, who was very anoxic on admission to hospital, the paralysis persisted much longer, and some residual disability will remain (Case XIII). A long duration of the paralysis following a bite from an Australian tiger snake has been noted (Fairley, 1929*a*). Krait bites are also sometimes followed by chronic neurological lesions (Acton and Knowles, 1921).

A question of great practical importance is this: Does the paralysis ever reach an irreversible stage? And is recovery still possible after the injection of massive doses of venom? Kellaway (1932) thought that the prolonged direct action of the lecithinase in Australian snake venoms, which alters the permeability of muscle cells, was difficult to reverse, and concluded that there was no evidence that it could be reversed within the times covered by his experiments. In none of his experiments was the artificial ventilation of the paralysed animal continued for longer than 30 hours, and the technical difficulties of maintaining life for such a long period in a small animal were stressed by him. As muscles may remain completely paralysed clinically for two days or more before they show signs of recovery, one concludes that these experiments probably were not continued long enough to show definitely that the paralysis was irreversible.

There is need to prove or disprove this point experimentally for the various elapine venoms by the use of modern techniques of artificial respiration, cuffed tracheotomy tubes and intravenous feeding in suitable animals. Working with rabbits, a small and possibly unsuitable animal, Silberberg (1954) attempted to do this in some measure by injecting three times the minimum lethal dose of tiger-snake venom subcutaneously into the animals and then artificially ventilating them through a tracheotomy tube when respiratory failure developed. He found that the rabbits all died from cardio-vascular failure within 11 hours of the commencement of artificial respiration. He attributed this cardio-vascular effect to the circulatory depressant action of the venom. We have

not observed any such effect in humans bitten by Papuan elapine snakes, and the need for further experimental work still exists.

If severe anoxia is present, some permanent neurological damage may result from this and from the effects of the venoms. The post-mortem changes found in the nervous system after death from snake bite are usually attributed to anoxia (Kellaway, Cherry and Williams, 1932).

The Treatment of Snake Bite.

First-aid treatment contributed little, if anything, to the successful management of our cases, and measures such as incision of the bitten area and wrongly used tourniquets often added to the patient's discomfort without giving any benefit. First-aid measures of proven value in animals are the immediate application of an effective tourniquet plus the excision or amputation of the bitten part or venesection of the bitten limb (Fairley, 1929*a*; Kellaway and Morgan, 1931). To be effective, these first aid measures must be applied immediately after the bite is inflicted. The likelihood of this occurring in practice is very remote. Except for the application of a tourniquet, these measures are too radical and impracticable for field use. This is particularly so when it is realized that it is sometimes difficult to decide whether a person has been bitten by a venomous snake or not, and completely impossible to decide initially whether a bite from a venomous snake is going to be a serious one.

First-aid treatment should be limited to the immediate application of a properly applied and properly used tourniquet (released every 20 minutes and discarded after two hours) and rest for the patient (probably impracticable suggestions for this country). It is perhaps of more importance to educate the people to recognize the first symptoms indicative of envenomation, and to realize the need for the urgent transport to hospital of patients with such early symptoms.

When the species of the snake responsible for the bite is unknown, correct treatment with specific antivenenes is difficult. Because of its slow rate of absorption and the large doses required, intramuscularly administered antivenene has no place in the treatment of snakebite. Very large doses of several types of antivenenes were given intravenously, after admission to hospital, to most of the patients described here. In addition, several of the patients had received intramuscular or intravenous doses of tiger-snake antivenene before their admission.

In only one case was there any dramatic reversal of neurological signs within a few hours of administering the serum. This man (Case XI) had a slight ptosis only and was admitted 15 hours after being bitten. In two cases (Case V and Case XIV) in which hæmaturia or hæmoglobinuria was present, these symptoms ceased after the administration of Papuan black-snake antivenene. The neurological signs in these two patients were not obviously influenced by this antivenene. In all the other cases no definite benefits were seen to follow the administration of antivenenes. In three of these cases (Case I, Case II and Case XV) the paralysis continued to progress in spite of the antivenene. In the other cases the antivenene may have prevented further paralysis developing.

The antivenene was given late in the course of the illness in most cases, often eight or more hours after the bite. Acton and Knowles (1921) thought that antivenene should still be of value if it was given within two-thirds of the death time and Ahuja and Singh (1954) state that, in the case of cobra bites, it is never too late to administer antivenene. In a case reported in the Australian literature, antivenene appears to have been of value although given more than 12 hours after the bite (Knyvett and Molphy, 1959). In the cases reported by Tisdall and Sewell (1931) and by Lester (1957), the attribution of the improvement to the antivenene, given late in the illness, was probably mistaken. On the whole, the observed benefits of antivenene treatment have been disappointing.

Tracheotomy has been widely used recently in a large number of different conditions in which mechanical ventilatory obstruction or secretional ventilatory obstruction exists (Nelson, 1958). These two indications for its use may exist in elapine snake bite. The successful use of tracheotomy in severe poliomyelitis suggested that it should be of value in snake bite (Reid, 1956). Knyvett and Molphy (1959) reported a case of severe snake bite in which a tracheotomy was performed. In this case it was performed very late in the course of the illness which was complicated by uræmia, and the patient died. Our observations have confirmed its value in the treatment of severe snake bite. Fortunately, although we are working with incompletely trained staff, no complications of tracheotomy have occurred.

Since the experiments of the workers of the Indian Snake Poison Commission (Richards, 1886), the importance of artificial respiration in prolonging life in cases of serious elapine snake bite has been well established. By inserting cannulæ into the tracheæ of dogs and artificially ventilating them, they were able to prolong for up to 41 hours the lives of dogs who had received lethal doses of cobra venom. However, they concluded that, although of great value in supporting the vital functions for some time, the measure was of doubtful value as a means of saving life. With our modern techniques, artificial respiration can undoubtedly prevent a fatal outcome in some cases of serious snake bite, but may have to be continued for two days or more.

The nursing care and medical treatment of the completely paralysed patient can be more easily carried out when the patient's breathing is assisted by some form of respirator or breathing apparatus attached to the tracheotomy tube. The patient must also be easily accessible so that he can be postured frequently. Because of this we think a tank respirator is to be avoided. We have found a manually operated rebreathing bag with a soda-lime absorption unit to be more satisfactory than an intermittent positive pressure breathing apparatus, and when used for such an ill and helpless patient it is probably much safer. Some simple apparatus involving the use of an oxygen cylinder, a Waters cannister and a rebreathing bag (Lassen, 1956) could probably be employed in rural hospitals for the treatment of this condition. So long as regular supervision is given by trained staff, intelligent semi-trained nursing staff can cope in an emergency with this apparatus and this method of treatment.

Conclusion.

Owing to the difficulties of transport in Papua, the cases reported in this paper may represent a selected group, as the most severe cases may never reach hospital. In these very severe cases other toxic factors in the venom may become of more significance and present new difficulties in treatment. However, we believe that, with modern treatment, the elapine snake victim should not die if he lives long enough to reach a hospital, and if the neurotoxic effects of the venom are the main factors in his illness.

Reports of Cases.

Case XIII.—A male Papuan, aged about 25 years, was admitted to hospital at 10.30 a.m. on May 31, 1960, in a semi-comatose condition. He had been bitten by a snake at 4 p.m. on May 30. During the night before his admission to hospital he had been given 15,000 units of antivenene (? tiger snake) by intermittent intravenous injections. On his admission, he was found to be semi-comatose, restless, cyanosed and sweating profusely. His jaw was completely relaxed and had fallen back so that the tongue obstructed his airway. Direct laryngoscopy was performed immediately, and the vocal chords were seen to be moving. A cuffed endotracheal tube was passed. After an adequate airway had been established, the cyanosis and sweating disappeared and the restlessness decreased. On more detailed examination then, he was found to respond to painful stimuli, but

not to the spoken word. Complete ptosis of both eyelids was present. The eyes were centrally fixed. The pupils were unequal, because of an old iritis affecting the right eye. The left pupil was dilated and reacted to light. There was no movement of any of the facial muscles. The jaw muscles were completely paralysed, and the mouth remained open. No movement of the tongue or palate was detected. The patient could not swallow, and secretions accumulated in the pharynx. He could move his head slightly from side to side, but could not raise it from the bed. He could move all his limbs, but they appeared to be generally weak. There was very little movement of the intercostal muscles, but the diaphragmatic movements appeared normal and the patient had quite an effective cough. All the deep reflexes were present. The superficial abdominal reflexes were absent, and the plantar response was flexor in both feet. The pulse rate was 156 per minute and the systolic blood pressure was 130 mm. of mercury. There was a small wound above the left ankle, which was bleeding slightly. Examination of the urine showed a very heavy deposit of albumin. The hæmoglobin value was 10·4 grammes per 100 ml.; moderately high polymorphonuclear leucocytosis was present. No malarial parasites were seen in thick or thin blood smears. The prothrombin time was 67% of normal. The serum electrolytes were normal. Examination of the centrifuged deposit of the urine showed no abnormality. One and a half hours after his admission to hospital, a tracheotomy was performed under local anæsthesia. The oral endotracheal tube was removed and a cuffed tracheotomy tube inserted. An intravenous infusion of a 4% dextrose and fifth-normal saline solution was commonced and continued for six days; 30,000 units of taipan antivenene and 30,000 units of tiger-snake antivenene were given by injection into the tubing of the infusion apparatus. Intramuscular injections of 500,000 units of crystalline penicillin were given every six hours for one week. Two and a half hours after his admission to hospital, the pulse rate was 114 per minute and the blood pressure was 110/80 mm. of mercury. The patient later had a rigor and his temperature rose to 104° F.; he remained restless throughout that day, requiring sedation with paraldehyde. The temperature gradually returned to normal over the next four days. There was no significant change in the patient's condition until the morning of June 2, 1960, when very slight lateral movements of the eyes and the slightest flicker of movement in the eyelids were detected. On the evening of that day restricted but definite eye movements were present. The patient was still restless and uncooperative. On June 3 slightly more movement of the eyelids was present, the eyes opening one-eighth of an inch and moving one-eighth of an inch laterally to the right. On June 4 the eyes opened one-quarter of an inch, full lateral movement to the right being present. By June 5 no ptosis was detected.

The subsequent progress of the patient can be summarized briefly. The patient initially had paralysis of conjugate movement of the eyes to the left and was unable to move his head to the left of the midline, but these signs disappeared by June 11. On June 6 it was apparent that there was slightly more weakness of the left side of the face, and that the left arm was weaker than the right. This left-sided weakness gradually decreased, but still persists. He gradually became less restless, and by June 6 was able to obey some commands, but he still appeared confused. He gradually became more rational and cooperative, and by the end of June was mentally normal. On June 8 he sat up with assistance, and he walked with assistance on June 20. Tube feeding by means of a gastric tube was commenced on June 6 and continued for two months. The tracheotomy tube was removed on June 9. After very slight initial movements of the lips, jaw, tongue and palate, very little improvement occurred in these muscles for two weeks, and then extremely slow but definite improvement started. By July 7 the patient was able to smile slightly, open his mouth about half an inch and protrude the tongue one-quarter of an inch beyond the teeth. His palate moved slightly, and he could now say "ah". On August 9 he was able to swallow normally, and tube feeding was suspended. He now could open his mouth for about one inch, and the tongue protruded one inch beyond the teeth. He was phonating better. On August 23 there was still only a moderate degree of palatal movement present. The movement of the jaw was still slightly restricted. He could not articulate clearly, and his speech was generally unintelligible. Apart from the upper motor neuron weakness of the left side of the face, the movements of the face appeared normal. On June 5 slight periorbital œdema was noted, and a transient erythematous rash and swelling of the lips developed on June 6. On July 12 he developed fever and acute polyarthritis affecting the right knee and the right proximal interphalangeal joints. Some joint swelling and pain from this condition still persist.

This patient had complete paralysis of all the ocular, facial and bulbar muscles. This led to respiratory obstruction and severe anoxia. He had a moderate degree of intercostal paralysis, but no detectable involvement of the diaphragm. The anoxia was possibly complicated by a cerebral vascular accident, and there were several manifestations of serum sickness. The recovery from the illness has been very slow, and residual disability is certain.

CASE XV.—A male Papuan, aged about 25 years, was admitted to hospital at 8.45 p.m. on August 5, 1960. He was unable to give any history, but an accompanying letter from the medical assistant in a rural hospital at Rigo indicated that the man had been bitten by a snake at approximately 12 noon that day. He had reported to the Rigo hospital at about 2.15 p.m., where he had been given 6000 units of tiger-snake antivenene and 9000 units of Papuan black-snake antivenene by intravenous injection before 3 p.m. He was then transferred by road to Port Moresby. After his recovery some additional history was obtained from the patient. While working in grass about a foot high, he was bitten above the left ankle by a black snake three feet long. The snake struck and let go immediately. After the bite he continued to work, but after some minutes he noticed a headache and then became dizzy. He became frightened, and ran about one hundred yards to his employer, who, about half an hour after the bite, applied a tourniquet a few inches below the knee and incised the wound. As the tourniquet was being applied the patient vomited some blood, and shortly after this he developed double vision. He felt sleepy and could not open his eyes. He remembers being brought to hospital but nothing after that. On examination the patient was restless and drowsy. He responded to some questions, but was uninterested. While being examined, he vomited a few millilitres of blood. Complete bilateral ptosis was present. The eyes were centrally fixed. The pupils were slightly dilated and reacted to light. The mouth could be opened to only half its normal extent, and the jaw was quite relaxed. The tongue could be protruded for about one inch beyond the teeth. There was a moderate degree of weakness of the palate. The patient appeared to be able to swallow, and his speech was slurred but intelligible. He was unable to lift his head from the pillow, but could move it from side to side. He could not sit up, but was able to roll his trunk from side to side and turn on to his side. There was generalized weakness of the limbs, affecting the proximal muscle groups more than the distal. The patient was unable to lift his legs from the bed. There was no movement of the intercostal muscles, and the diaphragm was weak. Pain sensation appeared normal. All the deep reflexes were present, and the plantar reflexes were flexor. There was a half-inch incised wound 4 in. above the anterior aspect of the left ankle, which was bleeding freely. There was slight œdema of the left foot. The temperature was 98° F. and the pulse rate was 90 per minute. The blood pressure was 140/90 mm. of mercury. A "grade 3" spleen was present. There was no albumin in the urine.

Papuan black-snake antivenene (21,000 units) was given intravenously soon after his admission to hospital. An intravenous infusion of a 4% dextrose and fifth-normal saline solution was commenced and continued for seven and a half days. Potassium chloride, in a dose of 8 grammes per day, was later added to this fluid. The patient's breathing became weaker and more obstructed, and an endotracheal tube was passed. A tracheotomy was then performed under local anæsthesia. A cuffed tracheotomy tube was inserted. A further 15,000 units of Papuan black-snake antivenene, 30,000 units of taipan antivenene, and 30,000 units of tiger-snake antivenene were given into the tubing of the infusion apparatus. Intramuscular injections of penicillin were commenced, but later the antibiotic was changed to "Chloromycetin". Four hours after the patient's admission to hospital the paralysis had progressed. The diaphragm was quite weak, but respiratory exchange was thought to be adequate. Bleeding continued from the leg wound and from the tracheotomy wound. One litre of blood was given during the night. By 9.30 a.m. on August 6, further extension of the paralysis had occurred. The diaphragm was very weak and breathing was assisted, the patient was cooperative, but he could not open his mouth at all. The tongue could not be protruded, but it could be moved slightly on the floor of the mouth. There was no movement of the palate. The patient could only wrinkle the forehead and could move the lips only slightly. His head movements were restricted to about half an inch on either side of the mid-line. He could not turn his trunk, but only twist his body slightly and ineffectually. The wrist and fingers could be flexed a little. The only movement in the lower limbs was in the

toes. The deep reflexes were absent. The œdema of the left foot was more marked. The urine contained a large amount of albumin. By 2 p.m. that day there were virtually no spontaneous respiratory movements, and at 5 p.m. there was no spontaneous breathing at all. However, by 12 o'clock midnight regular but very inadequate diaphragmatic movements had returned. The tracheotomy wound continued to ooze blood. On August 7 further extension of the paralysis in the limbs and face had occurred. The movements of the diaphragm remained weak, and were assisted all day. The patient could only raise the fingers and move the toes only slightly. There was only the slightest evidence of movement in the lips and forehead. However, in the eyes the first evidence of recovery was present. The patient could move his eyes about one-sixteenth to one-eighth of an inch laterally. Later this day the eyelids commenced to move, and they could be opened about one-eighth of an inch by evening, when slightly more movement of the tongue was evident also.

By August 8 further improvement was in evidence. The eyelids opened three-eighths of an inch, and the movements of the eyes were almost normal. The tongue could be protruded half an inch beyond the teeth. Breathing was still assisted, but the diaphragmatic movements had improved slightly. That night the eye movements were normal, and there was more movement at the wrists when gravity was eliminated. More movement of the lips and forehead was present. The patient tried to form words with his lips and to issue instructions. On August 9 the diaphragm movements were stronger, and artificial respiration was ceased that morning. There was only slight ptosis remaining in the right eye. The mouth could be opened about one inch, and the tongue could be protruded one inch beyond the teeth. Slight movement of the palate was present, and also weak movements of the lips, nose and forehead. The head was moved about two inches to either side of the mid-line, and more movements of the fingers, wrists and toes were present. That afternoon the patient was febrile and had a rapid respiratory rate. Although his chest was clinically clear, it was thought that he might be developing pneumonia and he was given regular intravenous doses of "Reverin" and his breathing was again assisted until the next morning. On August 10 he was still febrile. Radiological examination of the chest disclosed no abnormality. The tongue movements were normal. The movement of the jaw was still slightly restricted. There was more movement of the facial muscles, the wrists and the ankles. By August 11 the jaw movements were normal. However, the palate was still weak. He was still able to move his head from side to side and flex and extend his elbows and knees in the bed. The deep reflexes were still absent. By August 12 the facial movements were normal, but the palate was still weak. The arms could be raised above the head, and the hips, knees and ankles could be flexed and extended while on the bed. The deep reflexes were present. Later that day the patient could swallow fluid and sit up in bed with assistance. On August 13 the palate moved normally, and the patient was able to swallow. He could lift his extended legs off the bed. His temperature was normal. The tracheotomy tube was removed and the intravenous administration of fluids was ceased. On August 15 the patient was able to sit and stand without assistance, but weakness of the neck and proximal limb muscles still persisted. On August 24 he still had some weakness of the shoulder muscles and the muscles of the left leg, but he should recover without any residual disability.

SUMMARY.

1. The symptomatology, the signs, the clinical course and the treatment of 15 cases of Papuan elapine snake envenomation are described. Two cases are reported in full.

2. The main effect of the venoms of Papuan snakes is to produce motor paralysis of the ocular, bulbar, jaw, facial and spinal muscles. Significant sensory changes and primary circulatory abnormalities were not detected. A mild anticoagulant or a mild hæmolytic effect of the venoms was observed in a small number of cases.

3. The successful management of the most severe cases involved the use of tracheotomy, artificial respiration and antivenenes. Seven patients with severe paralysis, who probably would have died without attention, survived with this treatment.

4. The complete reversibility of the severe paralysis in potentially fatal cases of elapine snake envenomation and the importance of respiratory obstruction as a cause of death in this condition are stressed.

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Reviews.

Modern Gynæcology with Obstetrics for Nurses. By Winifred Hector and John Howkins, M.D., M.S. (London), F.R.C.S. (Eng.), F.R.C.O.G.; Second edition; 1960. London: William Heinemann Medical Books Ltd. 8¾" × 5½", pp. 236, with illustrations. Price: 17s. 6d. net (English).

MISS WINIFRED HECTOR is the principal tutor at St. Bartholomew's Hospital, London, and Mr. John Howkins is gynæcologist and obstetric surgeon. This small book is of special value to nurses in general training, because of the outstanding sections on nursing care, particularly in the gynæcological section. A sketchy review of the major gynæcological problems is given with an outline of some of the surgical procedures, but the nursing of the patients concerned is given in detail.

There are excellent sections describing the examination of the patient, the preparation of the instruments for ward examinations, the setting out of theatre instruments for operations, and detailed chapters on pre-operative treatment and post-operative care of patients in gynæcological wards. The nurse is warned of complications which may occur, and she is given a very clear plan of emergency treatment. There are one or two rather quaint details which seem to remain in nursing textbooks, but these do not in any way detract from the very up-to-date nature of this book.

The salient points are very well set out in the text with excellent printing and good paper, and the illustrations are line drawings which on first glance seem too simple. However, they emphasize the important points, and are on the whole very good.

As is characteristic of the English point of view, the gynæcological section takes up by far the greater part of the book. The section on obstetrics is very scanty, and leaves out many of the major problems, such as antepartum and post-partum hæmorrhage. The book is certainly not adequate as a textbook for nurses training in obstetric and gynæcological hospitals, but could be a very useful supplement to lectures for nurses undergoing their general training.

Lectures on Hæmatology. Edited by F. G. J. Hayhoe, M.A., M.D., M.R.C.P.; 1960. London: Cambridge University Press. 9¾" × 6", with illustrations. Price: 60s. net (English).

THESE lectures were delivered at a symposium on hæmatology held in December, 1959, at Cambridge, under the auspices of the University Post-Graduate Medical School. The audience comprised medical consultants, senior registrars and research workers having hæmatology as one of their major laboratory or clinical interests. The lecturers were well-known British hæmatologists, each speaking on a subject to which he has contributed considerable knowledge. The symposium could not have failed to be most stimulating to those fortunate enough to attend.

The editor and publishers deserve commendation for their efforts in producing this volume. Many reports of symposia are literal transcripts of the delivered papers, and often do not appear well in print. There is evidence here of considerable editing, so that each lecture is well written, remarkably free of typographical errors and liberally supplemented with tables and figures, and each has a good list of references. Each lecture reviews aspects of some expanding field of hæmatology. There are three lectures on anæmia, one on transfusion hazards, one on abnormal hæmoglobins, five on aspects of leukæmia and the myeloproliferative syndromes and three on hæmostasis.

The emphasis on leukæmia reflects the growing interest in this disease. Hayhoe stresses the value of cytochemistry in differentiating the three major varieties of acute leukæmia. Using the PAS reaction, Fuelgen reaction, Sudan black B staining and alkaline phosphatase and peroxidase reactions, he is able to classify most cases of blast-cell leukæmia. Correct classification may be important from the therapeutic viewpoint. Doll analyses various aspects of leukæmia, and concludes that there has been a real increase in incidence over the last thirty years. There is statistical evidence that leukæmia comprises several separate disease entities. Galton describes his experiences with drug treatment of chronic leukæmia. In a rather speculative paper, Kay discusses the present position of marrow transplantation in the treatment of leukæmia.

Quite a few provocative statements appear on these pages, and one feels that they were made deliberately to stimulate an experienced audience to participate in discussion. A transcript of the discussions that took place would have improved the volume, but would have added considerably to the editorial difficulties. For those interested in either the clinical or the laboratory aspects of hæmatology, the acquisition of this volume is the next best thing to attendance at the symposium.

Modern Trends in Diagnostic Radiology. (Third Series) edited by J. W. McLaren, M.A., M.R.C.P., F.F.R., D.M.R.E.; 1960. London: Butterworth & Co. (Publishers) Ltd. 9¾" × 6½", pp. 304, with many illustrations. Price: 102s. 6d.

THIS volume is the third of a series with this title, the last volume having appeared in 1953. It attempts, as the editor states in the introduction, to "record our knowledge of the advances made in the last seven years". The various chapters are written by British and Continental authors, all of international repute in radiology. Those chapters which have been translated into English have lost little in clarity or ease in reading.

Modern equipment, including various methods of image intensification, three-phase generators, electronic printers and xeroradiography, is discussed, and this section offers an interesting glimpse of the radiology department of the future. A chapter on protection gives a comprehensive summary of present knowledge of radiation hazards and the technical means whereby individual dosage and population gonad dose may be minimized, and the chapter on radiology in obstetrics is a reasoned and balanced view of a subject jolted badly out of perspective by the Stewart preliminary report. It is a pity that these two chapters, which could be read with interest and benefit by all who operate X-ray equipment and all who practise obstetrics, should be part of a volume otherwise written fairly exclusively for radiologists.

Chapters on functional studies of the heart, œsophagus and urinary tract, and on the use of radiology in research, offer a fascinating suggestion that the horizons of radiology may be receding more rapidly than ever. The present usefulness of the various methods of operative radiology, including selective renal arteriography, peripheral arteriography, pelvic venography and cavography and splenoportography, is discussed and the technique in each case is summarized. Other chapters include a review of newer methods of investigation and interpretation of findings in the lungs, the biliary tract and the kidneys, in hypertension, in the bones and soft tissues and in pædiatric radiology.

As the volume contains chapters from a number of different authors, it is inevitable that the standard varies slightly from chapter to chapter; but it is generally of a high order throughout. X-ray appearances are constantly related to pathology and disturbance of function, and there