

The Poisonous Terrestrial Snakes of our
British Indian Dominions (including
Ceylon) and how to recognize them,

WITH SYMPTOMS OF SNAKE POISONING
AND TREATMENT.

By

Major F. WALL, I.M.S., C.M.Z.S.

Third Edition, Fourth Thousand.

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PUBLISHED BY THE BOMBAY NATURAL HISTORY SOCIETY.

1913.

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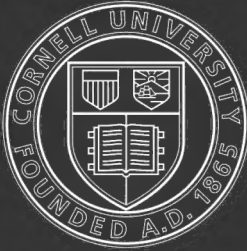
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POISONOUS SNAKES.

PRESS COMMENTS ON THE FIRST EDITION.

“This book meets a longfelt want, in affording a ready means of identifying a dead snake.”

“Invaluable to medical men in India.”

“So far. as the venomous kinds are concerned a very little practice with Major Wall’s key will enable any one to discover the species with ease and certainty.”—*Country Queries and Notes*, May 1908.

“The book is arranged in the most lucid manner, and ought to be included in the library of every Anglo-Indian. It reveals a large amount of patient research work, and is interesting throughout.”—*The Pioneer*, 9th February 1908.

“Of great value to the medical profession as well as to the naturalist.”—Review, *Times of India*.

“The book is well adapted to enable medical men and others to identify with the minimum of trouble any of the Indian poisonous land snakes.”—*Madras Mail*.

“A valuable contribution to the literature of the Thanatophidia of India.”—*Advocate of India*.

“The object of Major Wall’s little book is to point out characters of scaling by which, in his opinion, any one should be able to arrive at the name of any poisonous snake To many, the system proposed in this book will be of real utility. . . . We wish Major Wall’s little work a wide circulation in India.”—G. A. B. *Country Life*, April 1908.

PREFACE.

Unpretentious, and concise as this little work is, it has met with a measure of appreciation greater than the author ever anticipated when contemplating its compilation. The first edition of 1,500 copies published in 1908 sold out within three months, and the second edition of 1,500 copies is now exhausted. The Secretary of the Bombay Natural History Society now contemplates the production of a third edition, and has asked me to revise the previous matter, amplify it, and bring it up to date.

I find there is little to add to what has been previously written. As far as I am aware no new poisonous snake has been described during the last five years within our Indian Dominions, but the existence of a viper hitherto undescribed has been discovered in Waziristan by Major O. A. Smith. As it seems possible this viper may be found hereafter within British territory, I have given a description in this edition, and have christened the snake provisionally (?) *Pseudocerastes bicornis*.

The favourable reception given to my earlier work has encouraged me to add a second and third part to the present edition in which I propose to deal with the highly important subject of ophitoxæmia and the treatment of snake poisoning.

It is to be regretted that I am unable, with very few exceptions, to add to my original remarks on the effect of snake poisons. Our complete ignorance as to whether many of the snakes referred to are fatal, over and above being poisonous, must have struck all readers of the previous editions; and it was hoped that many who realized

where they could fill in gaps, and extend our knowledge would be induced to report cases coming under their notice. Many large employers of labour, planters especially, must frequently get snake-bite casualties among their coolies. To take examples, we have no single record of symptoms of the poisoning of the banded krait (*Bungarus fasciatus*), common as this snake is in Assam and Burmah. Again, snakes so common as the black kraits (*B. lividus* and *niger*) in Assam, as *Callophis maclellandi* in the Assam Hills and Eastern Himalayas, as the pit vipers *Lachesis macrolepis*, *anamallensis* and *strigatus* in the Southern Indian Hills, and *L. trigonocephalus* in Ceylon should furnish many records which would be received with appreciation by the author, or by the Secretary of the Bombay Natural History Society. Any information, however meagre, is worth reporting, and may prove useful and even a badly mutilated snake is capable of identification in competent hands.

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AN APPEAL.

The author will be very pleased to identify, and return any snakes sent to him, and would be most grateful for any information, however meagre, his readers may be able to give him of snake-bite fatalities, especially where the offender has been killed. No matter how mutilated the snake may be it is of value. He feels certain that much valuable information concerning snakes, the poison of which we are entirely ignorant, is being lost to Science each year. Specimens should be addressed to the Honorary Secretary, *Bombay Natural History Society*, 6, Apollo Street, Bombay.

PART I.

THE IDENTIFICATION OF INDIAN
LAND SNAKES.

THE POISONOUS TERRESTRIAL SNAKES OF OUR BRITISH INDIAN DOMINIONS AND HOW TO RECOGNISE THEM.

*(Reprinted from papers which appeared in the Bombay Natural
History Society's Journal by special request of the Inspector-
General of Civil Hospitals, Central Provinces, and others.)*

BY MAJOR F. WALL, I.M.S., C.M.Z.S.

INTRODUCTORY REMARKS.

During the last decade a vast advancement in our knowledge of snake venoms has been acquired, both in the province of toxicology and in the all-important one of therapeutics.

Whilst many observers have been engaged in the intricate, laborious, and minute researches connected with the investigation of the toxic properties of various venoms, very little, if any, advance has been achieved in that equally important and sister branch of the subject which deals with the identification of snakes, and especially with the distinction of the poisonous from the non-poisonous varieties.

In the treatment of snake-bite these two fields, though very distinct, are mutually interdependent. It is of little use to have the knowledge derived from one set of investigators at one's finger's ends, and its fruits—*viz.*, antivenene—to hand in all our hospitals, if the medical attendant is incompetent to recognise a poisonous

snake. It is only this knowledge in conjunction with the other that can make rational treatment possible, by teaching him when to withhold antivenene, and when to administer it.

It is to meet the unsatisfactory state of our knowledge on the subject of the identification of snakes that these papers have been contemplated, in the hope that they may bring this part of the subject up to a standard approaching that to which we have arrived in the study of snake venoms. Fully appreciating the already over voluminous and ever-increasing subjects which the profession of medicine embraces I have endeavoured to make the subject as practical as possible to the oriental practitioner by avoiding technicalities, or, where this cannot be done, explaining them with the aid of outline drawings, by which means I hope to bring the matter of identification within the easy grasp of hospital assistants and assistant surgeons, as well as medical officers.

In Volume XIV of the Bombay Natural History Society's Journal I wrote a paper on the distinguishing characters between poisonous and non-poisonous snakes, and appended a key in which I attempted to frame easy rules for their separation. This key far from satisfied me at the time, its length and complexity detracting from its practical value; however, in spite of its shortcomings, it has been favorably received, and I have been repeatedly asked for spare copies till my stock is exhausted. Recently the Inspector-General of Civil Hospitals in the Central Provinces wrote asking if he might circulate this paper in his Province, and the compliment conveyed in this request has caused me to revise it. Since its publication, in 1901, I have examined many hundreds of snakes collected by myself and others as well as large collections in various institutions, including the British Museum, and I am, therefore, now better qualified to deal with this subject. As a result I find that I can simplify and curtail the original key so as to considerably enhance its practical utility.

The good reception accorded to this first brief paper has prompted me to extend my remarks, so that in the present paper I propose to deal in detail with every known poisonous land snake within our Indian Possessions. The easy identification of these is my first object, and one which I hope to assist by means of outline drawings,

but I hope to do more, and to incorporate with each species a few remarks so as to make the paper useful to the medical profession as well as to the naturalist.

The abbreviations marked on the shields in the outline figures attached to these papers are the same throughout, and read as follows :—

An.	Anal.	Prf.	Præfrontal.
A.S.	Anterior sublinguals.	P.S.	Posterior sublinguals.
C.	Costals.	R.	Rostral.
F.	Frontal.	S.	Supraocular.
Int.	Internasal.	Sc.	Subcaudal.
L.	Loreal.	Sl.	Supraloreal.
M.	Mental.	So.	Subocular.
N.	Nasal.	Sub.	Sublingual.
Oc.	Occipital.	T.	Temporal.
Pa.	Parietal.	V.	Ventrals.
Po.	Postocular.	Val.	Vertebrals.
Pra.	Præocular.	Arabic numerals—Supralabials.	

Roman numerals—Infralabials.

With reference to *midbody* the point indicated is midway between the snout and the anus or vent (a transverse slit in the hinder part of the belly, see fig. 9). *Anterior* with reference to scales indicates a point 2 head lengths behind the head; *posterior* similarly implies a point 2 head lengths in front of the vent.

The conception of a poisonous snake, as alluded to hereafter, demands some remarks on the classification of these reptiles.

Mr. Boulenger considers the *Ophidia* (snakes) a Suborder of the Order *Squamata* (which includes lizards and chameleons). He divides snakes into nine families based on osteological peculiarities which can only be made apparent by the minutest and most careful dissection or disintegration of the soft tissues, and hence are of far too complicated a character for the general enquirer to readily investigate or comprehend. I venture to think the same end may be equally well attained by attention to external characters alone. The recommendation for such a method is obvious, since it enables the enquirer to ascertain at a glance the requisite points by an examination of the creature as it lies dead before him. Without

disturbing Mr. Boulenger's classification, which is the accepted one, I divide them as follows:—

TAILS NOT MARKEDLY COMPRESSED.

(i.e., not flattened like an eel's—see fig. 1 B and C.)

FAMILY.

A—VENTRALS

ABSENT.

Snakes in which the belly and back are clothed with identical scales (see fig. 2).

“—VENTRALS NARROW.

Snakes with the belly covered with transverse plates (ventrals) which, however, do not extend completely across the belly, so that when the specimen is laid on its back the whole of the last costal row, or even many costal rows, are visible on each side (see figs. 3 and 4).

C—VENTRALS BROAD.

Snakes with the belly shields stretching so far across as to permit only part of the last costal row to be seen on each side when the specimen is laid on its back (see fig. 5).

- 1 Typhlopidae.
- 2 Glauconiidae.

Small blind snakes worm-like, and living beneath the ground.

} **HARMLESS.**

- 3 Boidae.
- 4 Ilysiidae.
- 5 Uropeltidae.
- 6 Xenopeltidae.
- 7 Colubridae.
- (Sub-family Homalopsinæ).

} **HARMLESS.**

- 7 Colubridae (except the Sub-families Homalopsinæ and Hydrophiinæ).
- 8 Amblycephalidae.
- 9 Viperidae.

INCLUDES HARMLESS AND POISONOUS VARIETIES.

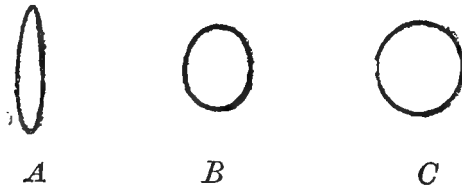
HARMLESS. POISONOUS.

TAILS COMPRESSED.

(i.e., flattened like an eel's—see fig. 1 A).

Sea snakes. Family Colubridae. Sub-family Hydrophiinæ. **POISONOUS.**

Fig. 1.



A.—Highly compressed tail typical of the sea snakes (Hydrophiinæ . Poisonous. B. and C.—Slightly compressed and round tails of landsnakes (including fresh water forms) seen in both harmless and poisonous species.

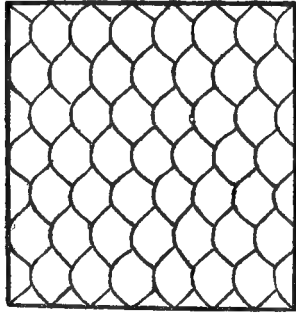


FIG. 2.—Belly of Typhlops ($\times 5$)

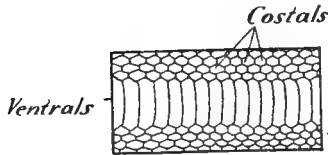


FIG. 3.—Belly of *Hipistes hydrinus* (nat. size).

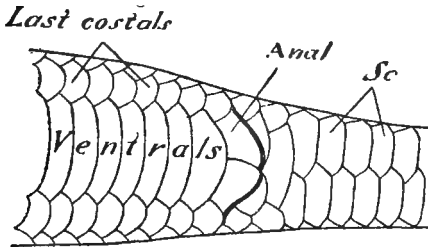


FIG. 4.—*Xenopeltis unicolor*.

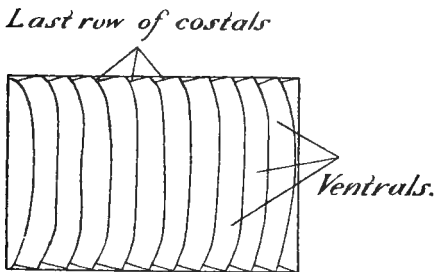


FIG. 5.—Belly of Russell's viper.

THE POISONOUS SNAKES OF INDIA.

A glance at this simple key will enable the enquirer to isolate two large groups of harmless snakes, by an inspection of the belly shields alone, and a third group of poisonous snakes by the conformation of the tail (sea snakes).

It is a somewhat difficult matter to decide where to draw the line between the so-called non-poisonous and the poisonous varieties. To begin with, all the *viperine* snakes are poisonous, and from investigations conducted by Alcock and Rogers* in Calcutta in 1902, it appears probable that all *colubrine* snakes contain in their saliva a toxic element identical with that to which the poisons of the cobras, kraits, and other deadly colubrines owe their lethal properties. If this is so, strictly speaking, *all* colubrines are poisonous, and their various salivas merely differ in degrees of toxicity.

The *Colubridæ* are divided into three groups: (1) *Aglypha* characterised by the absence of a poison fang, (2) *Opisthoglypha*, snakes furnished with a specialised tooth in the form of a grooved fang situated at the back of the maxilla (upper jaw bone), and (3) *Proteroglypha*, snakes endowed with a specialised grooved tooth (fang) in the front of the maxilla. It is to the third group that I reserve the term "poisonous," purely as a term of convenience however, for although all the snakes whose bite is known to prove fatal to man fall into this category many of the group are known to produce baneful effects usually falling short of death, whilst the effects of many others remain in obscurity.

The difficulty in laying down hard-and-fast rules by which to distinguish the poisonous varieties and separate them one and all from their non-poisonous allies may be appreciated from the fact that there are no less than 330 species already known within our limits, of which 69 are poisonous. Of these 69 species, 40 are terrestrial, 29 marine. All the poisonous species fall into one of the following 5 groups with one solitary exception, *viz.*, *Azemiops feae*, the existence of which may be ignored for all practical purposes since only one specimen is known. It was found in the Kachin Hills, Burma.

* Proceedings of the Royal Society, 1902, p. 446.

Key to distinguish the Poisonous Snakes.

- | | | | | |
|----|--|---|---|-------------------------|
| 1. | Tail compressed (<i>i.e.</i> , flattened like an eel's) (<i>see</i> Fig. 1A). Snout and crown covered with large plate-like shields (<i>see</i> Fig. 6). | Sea snakes (29 species). (<i>see</i> p. 8.) | } | Poisonous colubers. |
| 2. | Tail round (<i>see</i> Fig. 1 C). Median row of scales down the back distinctly enlarged (<i>see</i> Fig. 7). Only 4 infralabial shields, the 4th largest (<i>see</i> I to IV, Fig. 8). | Kraits (11 species). (<i>see</i> p. 9.) | | |
| 3. | Tail round (<i>see</i> Fig. 1 C). 3rd supralabial touching the nasal shield and the eye (<i>see</i> Fig. 12).* | Cobras and coral snakes (9 species). (<i>see</i> p. 23.) | | |
| 4. | Tail round (<i>see</i> Fig. 1 C). A conspicuous opening in the side of the face between the eye and the nostril (<i>see</i> Fig. 24 B). Vertebrae not enlarged.† | Pit vipers (13 species). (<i>see</i> p. 39.) | } | Vipers (all poisonous). |
| 5. | Tail round (<i>see</i> Fig. 1 C). Snout and crown covered with small scales as on back of body (<i>see</i> Fig. 37). Only part of the last row of costals visible on either side of the ventrals when the specimen is laid on its back (<i>see</i> Fig. 5 and contrast with Figs. 3 and 4). | Pitless vipers (6 species). (<i>see</i> p. 54.) | | |

A specimen which cannot be brought into one of these five groups is harmless, except *Azemiops feav*, which may be known from all other snakes by having 17 rows of scales in midbody, and 6 supralabials, the third of which only touches the eye.

* Since writing this I have seen two specimens of *Callophis maccllellandi*, in which the 3rd supralabial just failed to touch the nasals, and as the contact between these shields in many of the species *Callophis* and *Doliophis* is often very small, it is probable that the same departure from the normal may be met with in certain individuals of other species of these genera. For this reason when the 3rd just fails to touch the nasal, I give an alternative method of diagnosis as follows. 3rd supralabial touching the eye, and a suture running from the nostril to the 2nd supralabial. In the genus *Naja* the contact of the 3rd supralabial with the nasal is invariable, and this alternative rule therefore is not intended to apply.

† One harmless snake has a loreal pit, the very rare *Elachistodon westermanni*, but in this the vertebrae are enlarged. Only three examples are known, all from Bengal (Rungpore, Purneah and Jalpaiguri).

GROUP 1.—SEA SNAKES.

Identification.—*Tail compressed** (i.e., flattened like an eel's—see fig. 1A). *Snout and crown covered with large plate-like shield* (see fig. 6).

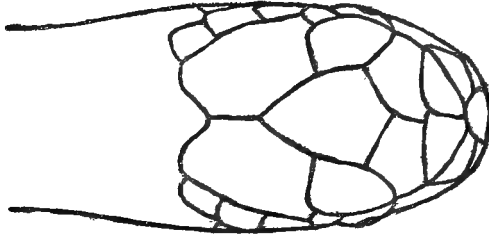


FIG. 6.—*Platurus laticaudatus* (× 4).

The sea snakes (*Hydrophiinae*) are all reputed highly venomous. Investigation by Rogers† shows that the venom of our commonest species (*Enhydryna valakadym*) is eight times more potent than that of the binocellate cobra! There are many published records of fatalities owing to bites from sea snakes, but the name of the offender is rarely, if ever, given, so that our knowledge of the venoms of this family of snakes is extremely meagre,—in fact, we have no certain knowledge of any one of them with the exception quoted above. The recognition of many of the species is extremely perplexing, and in consequence the confusion in terminology is great. Even our best books are very disappointing, and fail to make the recognition of many of them possible.

* Only one harmless snake has a compressed tail, viz., *Chersydrus granulatus*, an aquatic species found in rivers and seas. In this the snout and crown are covered with small scales only.

† "The Lancet," February 6th, 1904.

GROUP 2.—THE KRAITS (*BUNGARUS*).

Identification.—(1) Tail round. (2) Median row of scales down the back distinctly enlarged (see fig. 7). (3) Only 4 infralabial shields, the 4th largest (see I to IV, fig. 8*).

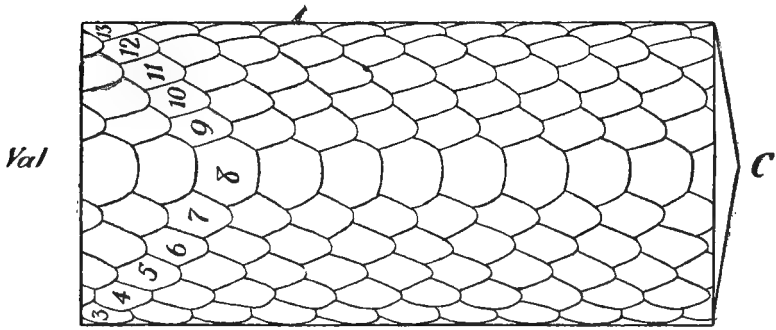


FIG. 7.—Back of Common Krait (*Bungarus caeruleus*) ($\times 2$).

Val=Vertebrales.

C=Costals.

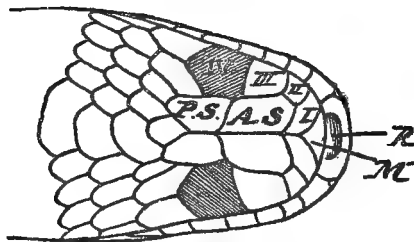
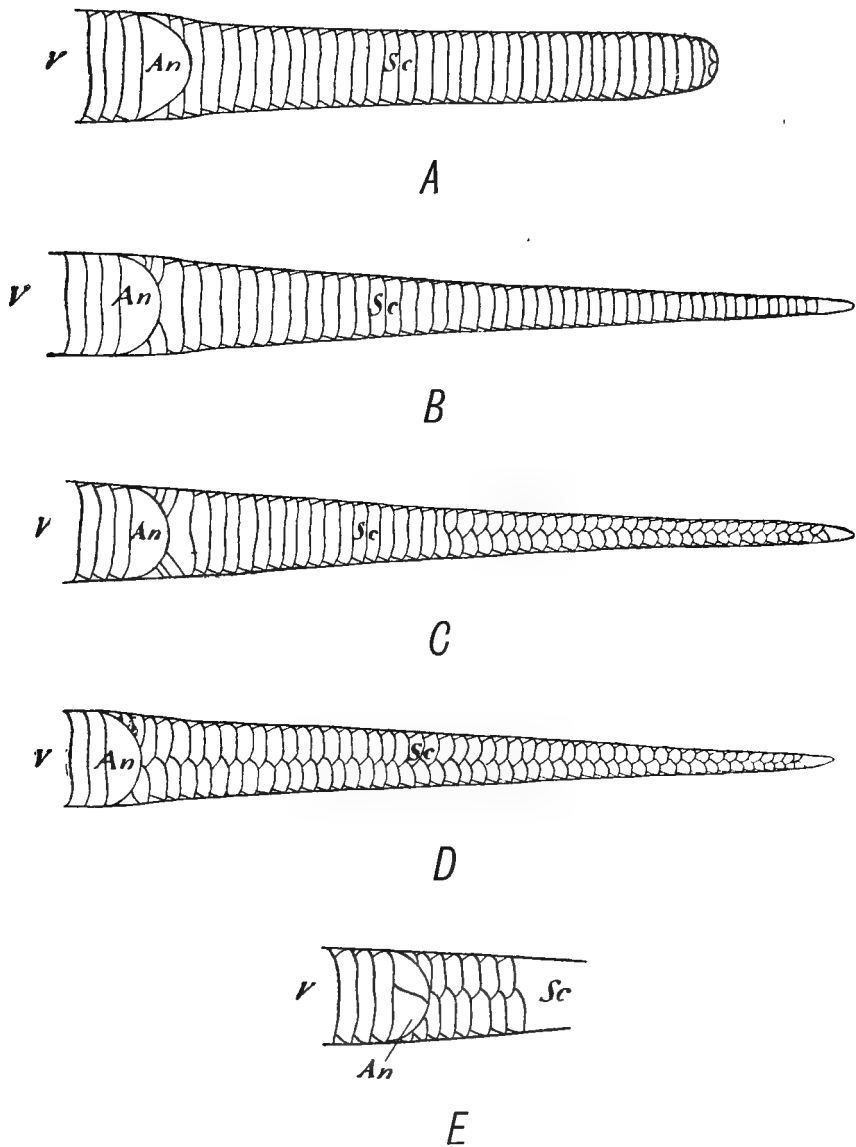


FIG. 8.—Chin shields of *Bungarus caeruleus* ($\times 1\frac{1}{2}$).

* With reference to this latter point, care must be taken not to count the first median shield which is called the mental (M.). Again, the last shield along the border of the lower lip which touches the posterior sublinguals (P.S.) is invariably to be considered the last infralabial.



V.=Ventrals. An=Anal. Sc.=Subcaudals.

FIG. 9.

- A. *Bungarus fasciatus* } —Subcaudals all entire.
 B. " *cæruleus* }
 C. " *flaviceps*.—Subcaudals entire at base, divided at tip of tail.
 D. *Naia tripudians*.—Subcaudals all divided.
 E. *Hemibungarus nigrescens*.—Anal divided.

The first essential point in the identification of a krait is to find the enlarged vertebral row of scales. The enlargement is very obvious, and without this the specimen *cannot* be a krait. Unfortunately, however, for our purpose this distinction is not absolutely confined to the kraits, since a few harmless snakes are similarly distinguished, *viz.*, the genera *Dipsadomorphus*, *Dendrophis*, and *Dendrelaphis*, some species of *Amblycephalus*, *Xenelaphis hexagonotus*, and *Elachistodon westermanni*, and it is due to this fact that other supplementary characters are necessary to formulate a rigid rule.

Supplementary generic characters.—Other important characters to be observed in the scale arrangement of kraits, but not necessarily peculiar to them, are as follows :—The *nasal* shield touches the 1st and 2nd supralabials, but never the 3rd. *Loreal* absent, so that only two scales intervene between the eye and the nostril. *Temporal*, a single shield touching the 5th and 6th supralabials. *Supralabials* 7, the 3rd and 4th touching the eye. *Posterior Sublinguals* touch the 4th infralabial shield (rarely 3rd also). The 4th *infralabial* is the largest of the series, and touches only 2 scales behind. The *costals* are the same number in the whole length of the body, except in some specimens of *sindanus* and *walli*. *Anal* entire. *Subcaudals* entire throughout, or in some species only at the base, the remaining shields being divided. The *iris* is black in all species except *B. fasciatus*, in which the pupillary edge is thinly margined golden, and the pupil which is round in form is only discernible during life in this one species.

The shields on the heads of all kraits are so closely similar in number and form that with the exception of the 2nd supralabial they are of no assistance in separating the species. The numbers of rows of scales over the back, however, vary from 13 to 19, and the vertebral row varies in breadth in some of the species. The colour, too, is very distinctive in all the species, and habitat is of great importance.

11 of the 12 known members of the genus occur within our Indian limits. Two are common, *viz.*, *Bungarus caeruleus* and *B. fasciatus* but the rest are local and uncommon, some being specially rare.

KEY TO THE KRAITS.

SCALES IN 13 ROWS IN MIDBODY (see Fig. 7) *Bungarus flaviceps*.

SCALES IN 15 ROWS IN MIDBODY (see Fig. 7).

A—SOME OR ALL THE SHIELDS BENEATH
THE TAIL DIVIDED (see Fig. 9, C and D). ,, *bungaroides*.

B—ALL THE SHIELDS BENEATH THE
TAIL ENTIRE (see Fig. 9, A and B).

(a) Vertebrae narrow, longer than broad, not as
broad as the last costal row. ,, *lividus*.

(b) Vertebrae broader than long in midbody.

(a¹) 2nd supralabial as broad as 1st and 3rd. . . . ,, *cæruleus*.

(b¹) 2nd supralabial narrower than 3rd and often
than 1st.

(a²) Subcaudals 23 to 40.

(a³) Tail tapering. Banded black and
white. Peculiar to Ceylon ,, *ceylonicus*.

(b³) Tail blunt, finger-like. Banded
black and yellow.
Habitat.—Orissa, Assam, Burma to
China ,, *fasciatus*.

(b²) Subcaudals 42 to 57.

(a³) 11 to 14 white bands on body,
2 to 3 on tail ,, *magnima-*
culatus.

(b³) 31 to 48 white bands on body, 11
to 13 on tail ,, *multicinctus*.

(c³) No bands. Uniform black above . . ,, *niger*.

SCALES IN 17 OR 19 ROWS IN MIDBODY (see
Fig. 7).

(A)—BODY COMPRESSED. ,, *walli*.

(B)—BODY ROUND ,, *sindanus*.

Scheme for Identification of the Kraits.

Name of species.	Costals.				Ventrals.	Subcaudals.	Some subcaudals divided at tip of tail.	2nd supralabial narrower than 3rd.	Body compressed.	Number of bands or bars on body.	Number of bands or bars on tail.	Habitat.
	2 headlengths behind the head.	Midbody.	2 headlengths before the anus.	Vertebrae in midbody broader than long.								
<i>flaviceps</i> ..	13	13	13	yes	193—226	42—54	yes	?	?	None.		Tenasserim, Cochin, China, Malay Peninsula and Archipelago.
<i>bungaroides</i> ..	15	15	15	yes	220—237	44—51	yes	yes	no	Many equidistant white chevrons or lines.		Eastern Himalayas, Khasi Hills, Cachar.
<i>lividus</i> ..	15	15	15	no	209—215	35—42	no	yes	no	None.		Brahmaputra Basin, South of Himalayas.
<i>caeruleus</i> ..	15	15	15	yes	200—218	38—50	no	no	no	Many white lines in pairs.		Indus Basin, Ganges Basin, Peninsula, India and Ceylon.
<i>ceylonicus</i> ..	15	15	15	yes	219—235	32—40	no	yes	no	15 to 20 2 to 5		Ceylon.
<i>fasciatus</i> ..	15	15	15	yes	200—234	23—39	no	yes	no	Complete white bands.		Brahmaputra Basin, South of Himalayas, Mahanadi Basin, Irrawaddy Basin, Tenasserim, Indo-China, South China, Malay Peninsula and Archipelago.
<i>magnimaculatus</i> .	15	15	15	yes	218—229	42—49	no	yes	no	11 to 14 2 to 3		Irrawaddy Basin.
<i>multicinctus</i> ..	15	15	15	yes	194—218	45—53	no	yes	no	31 to 48 11 to 13		Lower Brahmaputra Basin, Irrawaddy Basin, Andamans, South China to Formosa.
<i>niger</i> ..	15	15	15	yes	216—231	47—57	no	yes	no	None.		Brahmaputra Basin, South of Himalayas.
<i>sindanus</i> ..	17	17 or 19	17	yes	201—237	43—52	no	no	no	Many white lines in pairs.		Rajputana, Sind, Baluchistan, Punjab.
<i>walli</i> ..	17	17 or 19	17	yes	192—207	46—55	no	yes	yes	Many equidistant white beaded lines.		Ganges Basin.
<i>candidus</i> *	15	15	15	yes	210—222	40—50	no	?	no	20 to 25 7 to 9		Malay Peninsula and Archipelago.

* Does not occur within our limits.

BUNGARUS FLAVICEPS—The Yellow-headed Krait.

Identification.—It is the only one of the genus with the scales arranged in 13 rows.

Supplementary characters.—The vertebral scales are as broad as long, or even broader in the middle of the body. The subcaudals are entire at the base, and divided towards the tip of the tail (*see* fig. 9 C).

Distribution.—This rare snake belongs to the Malayan fauna, but extends through the Malay Peninsula as far north as Tenasserim, where it encroaches upon our Burmese Province.

Poison.—Nothing seems to be known about the effects of its poison.

Dimensions.—Grows to 6 feet and over.

Colour.—I quote from Boulenger* :—“ Black above, with or without a yellow vertebral line, two outer rows of scales black and yellow; head red or yellow; tail and sometimes posterior part of body orange red.”

BUNGARUS BUNGAROIDES—The North-Eastern Hill Krait.

Identification.—It is the only krait with scales in 15 rows that has any shields beneath the tail divided. In all the others these shields are entire throughout (*see* fig. 9).

Supplementary characters.—The vertebral scales are as broad as long or rather broader in the posterior part of the body.

Distribution.—This is a very rare species, and a very local one. Hitherto it has only been recorded from the Himalayas in the vicinity of Darjeeling, the Khasi Hills in Assam and N. Cachar.

Poison.—Nothing known.

Dimensions.—Grows to 3 feet.

Colour.—Black with white linear crossbars, the most anterior of which are chevron-shaped.

* Cat. Snakes, Brit. Mus., Vol. III, p. 371.

BUNGARUS LIVIDUS.—The Lesser Black Krait.

Identification.—Its uniform black colour taken with the slight enlargement of the vertebral row in which the scales are longer than broad at midbody make its identity easy. Both ventrals and subcaudals are fewer than in *niger*.

Distribution.—A rare snake. Of 4 specimens in the British Museum, 3 are from Assam, and 1 from India, the precise locality of which is not noted. I have lately had 5 specimens from the Jalpaiguri District and two from Tindharia, E. Himalayas (2,800 feet). Another mentioned by Sclater from Saidpur is probably of this species, but I failed to find the specimen in the Indian Museum.

Poison.—Mr. A. E. Lloyd submitted for my identification a specimen of this snake 3 feet 2 inches in length that bit a cooly woman below the ankle one night on his estate in Assam. She succumbed after the lapse of some hours.

Dimensions.—Somewhat uncertain owing to its confusion hitherto with *B. niger*. The largest of 12 I have seen is 3 feet and 5 inches.

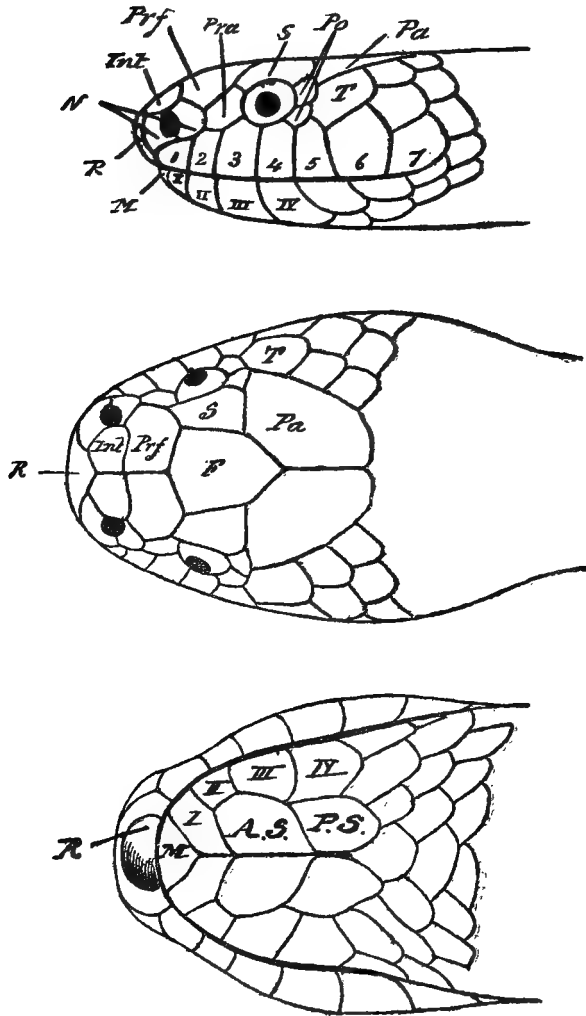
Colour.—Uniform black above, white below with more or less dark mottling at the base of the ventrals and subcaudals.

BUNGARUS FASCIATUS.—The Banded Krait.

The "Raj Samp" and "Sankni" of Bengal. Fayrer* says it is called "Koclea Krait" in the North-West. I presume he means N.-W. Bengal, for it does not exist in N.-W. India. According to Russell it is called "Bungarum pamah" on the Coromandel Coast. In Burmah it is known as "Gnandawja," "Ngan-wa," "Ngan-than-kwin-syut," "Nat-mywe," and "Mywe-min."

Identification.—The alternate bands of yellow and black are sufficiently distinctive. The one snake which bears some superficial resemblance to it is *Lycodon fasciatus*, a harmless snake known from the Assam and Burmese Hills. This latter is much smaller, the bands more numerous and their outlines very irregular unlike the banded krait. Moreover, the scale characters mentioned as peculiar to the kraits are all absent in the *Lycodon*.

* *Thanatophidia*, p. 11.

FIG. 10.—*Bungarus fasciatus* ($\times 2$).

Supplementary characters.—The vertebral row is more enlarged than in any others of the genus, the scales being considerably broader than long. The back is ridged along the spine, and the tail is blunt, and finger-like (see fig. 9 A).

Distribution.—From Southern China and the Malayan Subregion, it extends through Tenasserim to the Basins of the Irrawaddy and the Brahmaputra, South of the Himalayas. It is only known from Peninsula India in the North-East as far South as the Basin of

the Mahanadi River.* North of the Ganges the most Western limit I know of is Bettiah (N.-W. Behar).

Poison.—(See Part II, page 95.)

Dimensions.—Six feet is a very unusual length, but Major O. A. Smith has recorded one seven feet long (Bomb. N. H. Journl., Vol. XXI, page 284).

Colour.—Alternately and completely banded black and yellow.

BUNGARUS MAGNIMACULATUS.—The Burmese Krait.

Identification.—It has broader and fewer bands than any other krait.

From *candidus* with which it has been confused it is known by its colour, and habitat.

From *ceruleus* it is known from the larger number of ventrals (218-229), the narrow 2nd supralabial, colour and habitat.

From *multicinctus* it is known by the larger number of ventrals, colour and more restricted habitat.

Distribution.—At present known from a very restricted area within the basin of the Irrawaddy.† It is the only krait peculiar to Burma.

Poison.—Nothing known.

Dimensions.—Grows to 4 feet $3\frac{1}{2}$ inches.

* There is one specimen in the British Museum presented by Colonel Beddome, and labelled from the Anamallays. This is the sole record of this snake in Peninsula India outside the limits specified above.

The accuracy of Beddome's record is shattered by the following facts. He records no less than 7 other species from Southern India, not known otherwise from this area. These are *Tropidonotus parallelus*, *T. subminiatus*, *T. himalayanus*, *Lycodon jara*, *Simotes splendidus*, *S. octolineatus* and *Dendrelaphis caudolineatus*. All of these snakes are known otherwise from areas in which *Bungarus fasciatus* occurs. Now it is certain that Beddome received snakes from Burma and Tenasserim because there are specimens in the British and Indian Museums presented by him from those areas, viz., *Simotes cruentatus*, *S. violaceus*, *S. cyclurus* and *Dipsadomorphus hexagonotus*. From these facts it would appear that specimens from Burma and Tenasserim including one of *Bungarus fasciatus* had been mixed up with his Southern Indian collections.

In proof of these statements, *vide* Boulenger's Catalogue of Snakes 1893 to 1896 and Slater's list of Snakes in the Indian Museum, Journal Asiatic Society of Bengal, Vol. LX, 1891.

† Monywa, Meiktila, (Wall and Evans. Bomb. Nat. Hist. Journl., Vol. XII, p. 611) and Meiktila (Slater, Journl. Asiatic Soc., Bengal, Vol. LX, p. 245.) Hmawbi, Myingyan, and Shwebo District (Evans.—*In epistola.*)

Colour.—Black with 11 to 14 light bands on the body and 2 or 3 on the tail. These bands are white streaked with black lines in the length of the snake. Belly quite white.

BUNGARUS MULTICINCTUS.—The Many Banded Krait.

Identification.—This species has more bands than any other krait.

From *cæruleus* it is distinguished by the narrow 2nd supralabial, its colour, and habitat, from *candidus* by colour, and habitat, and from *magnimaculatus* by the fewer ventrals, colour, and habitat.

Distribution.—Rare in Burmah. Evans and I obtained one from Insein, another dubiously from Rangoon, and there is a specimen in the British Museum from Toungoo. Two specimens in the Indian Museum are labelled Purneah. Occurs also in the Andamans, Southern China, Hainan, and Formosa.

Poison.—Nothing known.

Dimensions.—3 feet 8 inches is the largest measurement I know.

Colour.—Black with from 31 to 48 pure white bands on the body, 11 to 13 on the tail. Belly white.

BUNGARUS NIGER.—The Greater Black Krait.

Identification.—Quite black or blue-black above, with the vertebrae broader than long in the middle of the body. The ventrals and subcaudals are more numerous than in *lividus*.

Distribution.—I obtained seven specimens in Dibrugarh and one from Sadiya, Assam, and have lately received four from the Eastern Himalayas, Tindharia 2,800 feet, and Pashok 2,000 to 4,500 feet, Sibsagar and Garo Hills (Sclater.)*

Poison.—Nothing known.

Dimensions.—My largest specimen was 4 feet and half an inch.

* Sclater (in the Journl., Asiat. Soc., Bengal, Vol. LX., p. 246) mentions 3 specimens under the title *Bungarus lividus*. He notes that two of these have the vertebrae broader than long. These, I have examined. Those from Sibsagar and the Garo Hills are *B. niger*. The third specimen from Saidpore (Dinapur District) is probably the true *lividus*, but I failed to find it.

Colour.—Uniform black above. Belly white with more or less dark mottling at the base of the posterior ventrals and subcaudals.

BUNGARUS GEYLONICUS.—The Ceylon Krait or Karawala.

Identification.—The bands are complete. It is the common Krait of Ceylon.

Supplementary characters.—The vertebral row is unusually large, the breadth of the scales considerably exceeds the length, and in this respect it almost compares with *B. fasciatus*.

Distribution.—Peculiar to Ceylon.

Poison.—The only cases of bites from this snake known to me are reported by Mr. E. E. Green (Spol. Zeylan. April 1908, p. 103) and Dr. Willey (Spol. Zeylan. April 1906, p. 228). In the former record a cooly was bitten in the left foot at 4 a. m. At 5-30 a. m. he was sleepy and drowsiness increased till 10 a. m. When dosed with whisky shortly afterwards swallowing was difficult and vomiting ensued. He walked about till his legs refused to move. At 2 p. m. he was feverish and insensible and he died at 4 p. m.

In Dr. Willey's case a Malay woman bitten in Colombo succumbed within 12 hours.

Dimensions.—Grows to 3 feet and over.

Colour.—Glistening black with white cross bars. Belly white banded blackish. Young, with head white, spotted black.

BUNGARUS CÆRULEUS.—The Common Krait.

(Synonym—*B. arcuatus*).

The "Karait" and "Dhomum chitti" or "chitti" of Bengal; "Valla pamboo" of Malabar. "Katto virian" and "Anali" of Madras. The "Godi nagera" of Mysore according to Rice, and the "Gedi paragoodoo" and "Pakta poola" of the Coromandel Coast (Russell). "Kowriya" or "Chit kowriya" are names given in the Punjab (Major O. A. Smith), and "Kandor" in Bengal about Kalna, so Mr. Muir tells me.

* Gunther and other Authors have wrongly used this as the Singhalese name for *Ancistrodon hypnale*. Ferguson, Willey, and others have, however, shown this mistake.

Identification.—The linear white arches, taken with 15 scale rows and the undivided subcaudals (*see* fig. B) suffice to declare its identity. One important feature for those to note who in spite of all precautions persist in trying to identify their specimens by colour and markings instead of by conformation and relationship of shields, is the fact that in all the snakes which resemble this species in colour, *viz.*, *Lycodon aulicus* (certain varieties) and *L. striatus*, together with *Dryocalamus nympha*, *D. gracilis* and *D. davisoni*, the white cross bars are most evident in the anterior part of the body, and gradually fade posteriorly till they are often lost. It is characteristic of this krait, however, that the white bars are most distinct posteriorly, and often fade away anteriorly—in fact, the anterior one-third or one-half of the body is frequently without marks in adults.

Supplementary characters.—In the vertebral row the scales are about as broad as long in the middle of the body (*see* fig. 7). The 2nd supralabial is peculiar in being as broad as the 3rd.

Distribution.—Throughout the Indus Valley, the Ganges Valley, Peninsula India, and Ceylon. Though essentially a snake of the plains I have obtained it in Almora at an altitude of 5,400 feet, and have other records exceeding 5,000 feet. It is very rare in Ceylon.* It is the only Krait found in Peninsula India South of the Ganges Basin.

Poison.—(*See* Part II, p. 89).

* There are 5 specimens of this snake in the British Museum, presented by Cantor from the Malay Peninsula. I think there are good grounds to discredit the accuracy of this record. It is noteworthy that six other Indian Snakes are recorded from the Malay Peninsula on the sole authority of Cantor, *viz.*, *Typhlops bothriorhynchus*, *Polyodontophis sagittarius*, *Xenochrophis cerasogaster*, *Zamenis fasciolatus*, *Helicops schistosus* and *Hypsirhina sieboldi*. All of these snakes are known from Bengal but not as far East as Burma. Now it is certain that Cantor received snakes from Bengal because specimens of the following species are given in his name from Bengal to the British Museum, *viz.*, *Polyodontophis sagittarius*, *Xenochrophis cerasogaster*, *Lycodon jara*, and *Hypsirhina enhydris*. Under these circumstances one cannot escape the conviction that the snakes above enumerated together with *Bungarus caruleus* were received by him from Bengal and inadvertently mixed with his Malayan collection. Further Boulenger has cast doubts on the locality of a specimen of *Dryophis mycterizans* presented to the British Museum by the same collector labelled from Assam. In support of these statements, *vide* Boulenger's Catalogue of Snakes in the British Museum, 1893 to 1896.

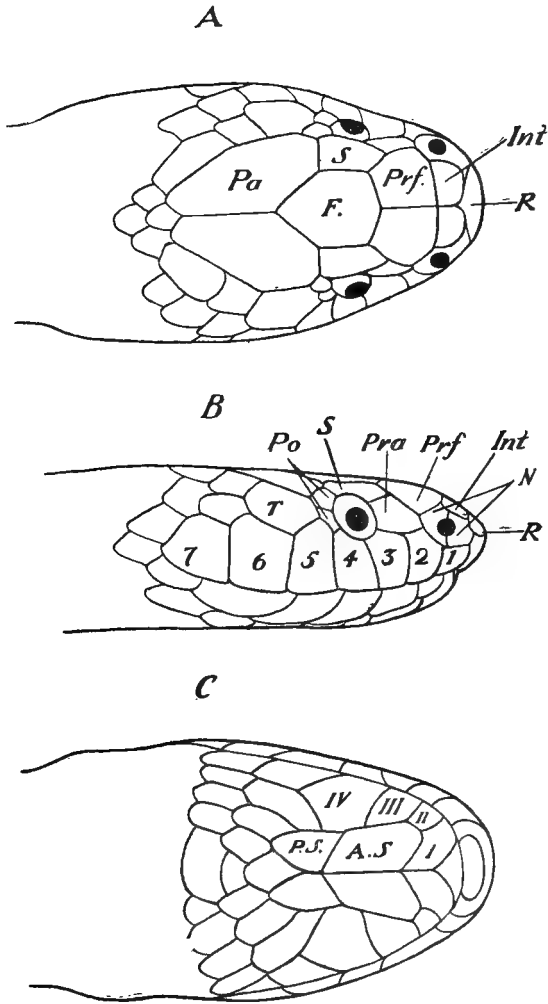


FIG. 11.—*Bungarus caeruleus*.

Dimensions.—Specimens over 4 feet are rare. Captain Paterson, I.M.S., wrote to me of one he killed in Lahore measuring 4 feet 6 inches. Major O. A. Smith reported one (Bom. N. H. Jourl., Vol. XXI, page 284) which was 4 feet $6\frac{3}{4}$ inches in length. Dr. Annandale speaking of this same specimen which was killed in Hazaribagh says, in spirit it had shrunk to 4 feet $4\frac{1}{2}$ inches. I have measured a dry skin of a specimen killed at Bannu, N. W. Frontier, which was 4 feet 6 inches.

Colour.—Glistening black with linear, white arches thrown in pairs across the back, sometimes more or less absent in front. Belly white.

BUNGARUS SINDANUS—The Sind Krait.*

Called "Pee-un" by the natives of Upper Sind.

Identification.—The scales over the back are in 17 (rarely 19) rows in midbody, the first three supralabials are sub-equally broad, and the body is round in section.

Supplementary characters.—The vertebrae are as broad, or broader than long in midbody. The subcaudals are entire. (Except in the type specimen where a few of the last are divided.)

Distribution.—Rajputana, Sind, Baluchistan, Punjab.

Poison.—Nothing known.

Dimensions.—Grows to 6 feet.

Colour.—Black with white cross bars most evident posteriorly and usually paired.

It is extremely like the common krait, (*cæruleus*) in its markings.

BUNGARUS WALLI—Wall's Krait.†

Identification.—Scales in 17 or 19 rows, the vertebral scales as broad or broader than long, and a distinctly compressed body.

* In a note to be published shortly in the *Bombay Natural History Journal*, I have given good reasons, I think, to doubt whether *sindanus* is entitled to rank as a species apart from *cæruleus*. I think specimens of kraits from Indore with 17 scale rows will prove to be *cæruleus*, since I see no tendency toward compression of the body as far as I can judge from the spirit specimens submitted to me.

† Though I am aware that Dr. Annandale (*Proc. As. Soc. Bengal New Series*, Vol. VII, No. 7, 1911.) thinks that this form is not different from *sindanus* (Boulenger), I adhere to my original opinion. The marked compression of the body in *walli* proclaims it a very distinct snake from *sindanus*. I have not examined *sindanus* in life and the compression of a snake's body is often a very difficult matter to ascertain after distortion in spirit, but Major Ward giving me details of 5 specimens that I consider *sindanus* from Fort Sandeman remarks "the body is not compressed."

In snakes such as *Zamenis mucosus*, and *korros*, etc., which have a markedly compressed body one never sees an individual exception in this feature, any more than one finds, exceptions in the conformation of a snake's body which like *Bungarus cæruleus* is round

Supplementary characters.—The 2nd supralabial decidedly narrower than the 1st and 3rd, ventrals 192 to 207.

Distribution.—The Ganges basin (Fyzabad, Gaya, Midnapore, Purneah).

Dimensions.—The largest record I know is 4 feet $11\frac{3}{4}$ inches.

Colour.—Mercurial-black with equidistant white bars formed of roundish spots. These are not arranged in pairs as in *cæruleus*, and *sindanus*. The tail is more or less sullied with plumbeous beneath, especially towards the tip, unlike *cæruleus*, and *sindanus*.

GROUP 3.—COBRAS AND CORAL SNAKES.*

Identification.—(1) Tail round. (2) The 3rd supralabial shield touches the nasal, and the eye (see fig. 12).†

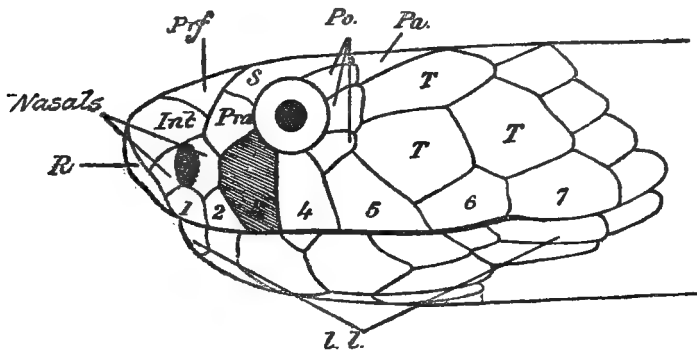


Fig. 12.—*Naia tripudians* ($\times 1\frac{1}{2}$).

This second feature alone separates the members of this group from all other snakes (see footnote, page 6). The group comprises 4 genera, and includes 9 species.

* The name coral snake is applied to a South American poisonous species, *Elap-coralinus*. I use the title here for those snakes which are allied to the above and to which I think the term singularly appropriate, since most of them have bellies adorned with a most beautiful colouring resembling pink coral. This, however, disappears after a day or two's immersion in spirit.

† I am only aware of one harmless snake in which the 3rd supralabial touches the nasal shield, viz., *Xenopeltis unicolor*, and in this case it fails to touch the eye. (See fig. 13.)

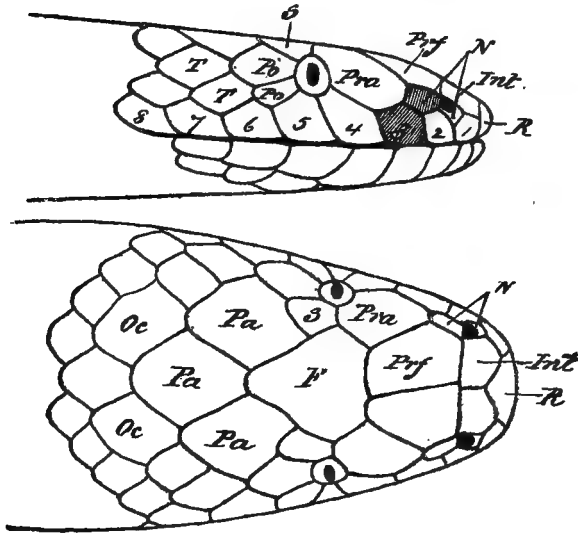


FIG. 13.—*Xenopeltis unicolor* (× 2).

Key to the identification of the Species.

ANAL ENTIRE. (See An. Fig. 9.)

TEMPORAL TOUCHES 5th AND 6th SUPRALABIALS ONLY. (see T., Fig. 15 B.)

Internasal not touching præocular. (See Int. and Pra., Fig. 15 B.)

- Belly uniform red..... *Doliophis bivirgatus.*
- Belly barred with black..... *Doliophis intestinalis.*

Internasal touching præocular. (See Int. and Pra., Fig. 16B.)..... *Naia tripudians.*

TEMPORAL TOUCHES 5th, 6th, AND 7th, SUPRALABIALS. (See T., Fig. 18A.)

- Subcaudals at base of tail entire. (See Sc., Fig. 9C.)..... *Naia bungarus.*
- Subcaudals divided throughout. (See Sc., Fig. 9D.)..... *Callophis bibroni.*

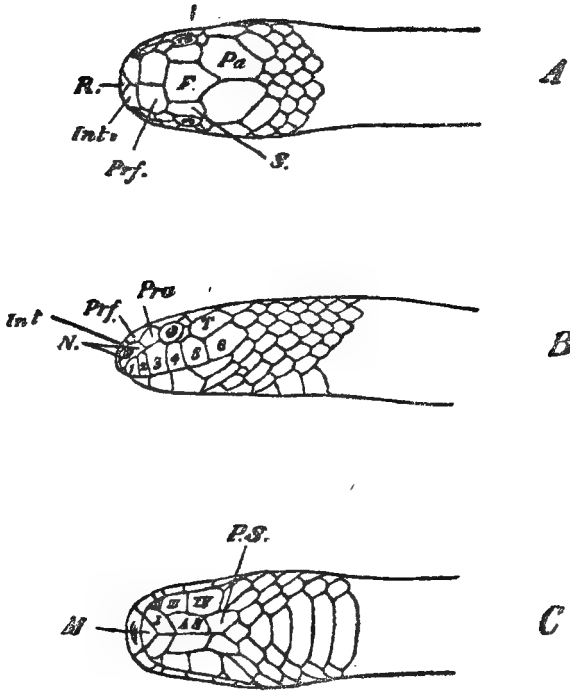
ANAL DIVIDED. (See An. Fig. 9.)

TEMPORAL TOUCHES 5th AND 6th SUPRALABIALS. (See T., Fig. 20 B.)

- Supralabials 6. (See Fig. 21 B.)..... *Callophis trimaculatus.*
- Supralabials 7. (See Fig. 20 B.)..... *Callophis maclellandi.*

TEMPORAL TOUCHES 5th, 6th, AND 7th SUPRALABIALS. (See T., Fig. 22B.)

- Tail with 2 black bands..... *Callophis maculiceps.*
- Tail with no band..... *Hemihungarus nigræscens.*

DOLIOPHIS BIVIRGATUS—The White-striped Coral Snake.FIG. 15.—*Doliophis bivirgatus*. (× 1).

Identification.—This and the next species agree in having only 6 supralabials, and the anal shield entire, which characters serve to distinguish them from all the rest of the group. The belly in this snake is uniform red in colour.

Supplementary characters.—*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular, and frontal. *Temporal.*—One, which touches the 5th and 6th supralabials. *Supralabials* 6. *Anterior sublinguals* touch the 1st, 3rd and the 4th only of the infralabials. *Posterior sublinguals* touch the 4th infralabial only. *Infralabials.*—The 4th is the largest of the series, and touches 2 scales behind. *Scales* are 13 in the whole body. *Anal* entire. *Subcaudals* divided throughout.

Distribution.—This Malayan form extends into our Burmese territory, where, however, it is rare.

Poison.—Nothing is known about it. The poison glands in this and the next are peculiar; unlike all our other poisonous snakes, instead of being confined to the temple they extend back into the abdominal cavity as far as the heart.

Dimensions.—Grows to 5 feet.

Colour.—Blackish above with two or four white lines down the back. Head and tail red. Belly red.

DOLIOPHIS INTESTINALIS.—The Belted Coral Snake.

Identification.—Like the last it has only 6 supralabial shields, and the anal is entire, but the belly is barred with black.

Supplementary characters.—*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular and frontal. *Temporal.*—One, which touches the 5th and 6th supralabials. *Supralabials* 6. *Anterior sublinguals* touch the 1st, 3rd and 4th infralabials. *Posterior sublinguals* touch the 4th infralabial. *Infralabials.*—The 4th is the largest of the series, and touches 2 scales behind. *Scales* are 13 in whole length of body. *Anal* entire. *Subcaudals* divided throughout.

Distribution.—This like the last belongs to the Malayan fauna, but is said to extend into Burmah.

Poison.—Nothing known as far as I am aware, though it appears to be fairly common in parts of the Malayan region.

Dimensions.—Grows to 2 feet.

Colour.—Boulenger* says: “Brown or blackish above, with darker or lighter longitudinal streaks; tail pink or red beneath; belly with black crossbars.”

NAIA TRIPUDIANS.—The Cobra.

Vernacular names.—According to Fayerer the spectacled or binocellate cobra is called “gokurrah” about Calcutta and the monocellate variety, which exhibits a single spot on the hood subject to much variation in size and shape, the “keautiah.” Both names appear

* Cat. Snakes, British Museum, Vol. III, p. 402.

to receive further qualification according to variations in colour. I have never heard these names in other parts of India, and it is probable that they are peculiar to Bengal. The former is the common variety in the Indian Peninsula, in which region the latter is decidedly rare according to my experience, but the converse holds good in Burmah and further East. In Bengal the distribution of the two forms seems to overlap, and both are common. In Bengal I have heard "Nag samp" and "Kala samp" as frequently in use as in other parts of India. In Madras it is called by the Tamils "Nalla Pāmbū," and on the Malabar Coast is known as "Sairpoon" and "Moorookan." In Mysore it is the "Nagara havoo," and according to Russell "Nagoo" on the Coromandel Coast. It is the "Mwè howk" of the Burmese.

Identification.—I have no doubt that to most people living in India, the recognition of a cobra seems a very simple thing, and this is true as a rule. If the snake is seen alive at close quarters with the hood expanded, its identification will hardly admit of a doubt. Still it must be remembered that the hamadryad expands its hood to an almost equal degree, and that certain harmless snakes, especially the Keelbacks (*Tropidonoti*, and their allies), erect themselves, and flatten the neck, though to a lesser degree. The spectacle mark on the hood of the binocellate cobra, and the oval spot surrounded by an ellipse on the hood of the monocellate or Burmese variety, are both of them quite distinctive of this species, and if constant would make diagnosis invariably easy. Many cobras, however, have these marks so modified or obscured that most people unfamiliar with this subject, would fail to recognise them if reliance is placed on these alone.

After death the hood is obliterated, and if the creature is stiff cannot be readily demonstrated, and I have frequently under these conditions known people express surprise when told that a specimen is a cobra, shake their heads, and think they know better. Again, I have seen the loose skin about the neck of a harmless snake pulled out, and a hood claimed where none existed, so that one must admit that in a few cases, at least, the cobra is not recognised, and sometimes a harmless snake is mistaken for it.

Nicholson's footnote on page 159 of his work on Indian snakes is a striking corroboration of my own experience. He says: "I have seen an Englishman, considered rather an authority on snakes, declared that a *Ptyas mucosus* (now *Zamenis mucosus*) just brought to me was a cobra; he even pointed out the poison-fangs." So long as people continue to be guided by these faulty characters in diagnosis, mistakes are sure to occur.

Now there are one or two very distinctive peculiarities about the scales of a cobra which if looked for should place its identity beyond question. These are as follows:—

*The præocular shield touches the internasal** (See Pra. and Int., fig. 16 B). In only two other snakes is this relationship to be found, viz., in *Xylophis perroteti*, a small harmless snake peculiar to the hills of Southern India, and the rare *Amblycephalus monticola*. In both the third supralabial shield does not touch the nasal.

Between the 4th and 5th infralabial shield a small wedge-shaped scale occurs, the "cuneate" (see fig. 16 B). Sometimes a second or even a third similar scale borders the lower lip. This scale may easily be overlooked, lying partly or wholly concealed, as it may do, by the overlapping of the upper lip, so that the mouth should be opened when looking for it. It occurs in no other land snake. I have never even observed it in the hamadryad, but it is seen in a few species of sea-snakes. A head is rarely so broken that one or other of these points cannot be made out on one side. If, however, the head is mutilated beyond recognition there is one feature about the scales over the back of a cobra which is peculiar to itself. It is the concavity in the arms of the bracket-shaped pattern which these form, and which I have shown by thickened lines in fig. 17. Beside this, I have placed another drawing to illustrate what is seen in other snakes, the pattern forming a chevron. This

* This is a very easy point to determine if it is remembered that the shields immediately behind the rostral (in land colubrids) are called internasals, and the shields touching the front of eye the præoculars. In the instances where the præfrontal shield touches the eye as in Fig. 19, it is obvious that this shield from its size and position has a prior claim to be considered a præfrontal, and in such a case the præocular is said to be absent.

is perhaps rather a nice point which may require a practised eye to

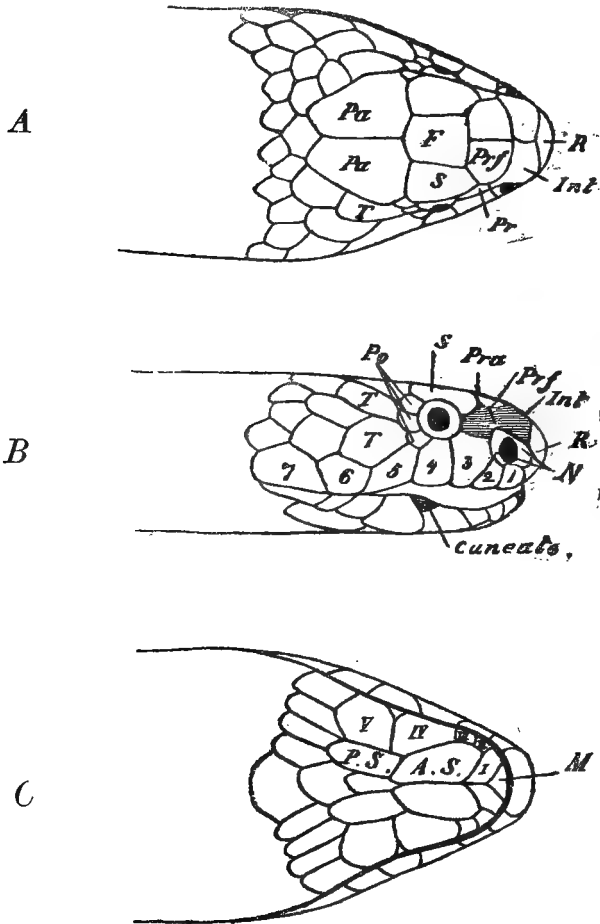


FIG. 16.—*Naia tripudians* (nat. size).

determine positively, but to an observant enquirer there should be little difficulty, and with proper care the character is a very valuable one.

Supplementary characters.—*Præfrontals* touch the internasal, præocular, supraocular, and frontal. *Temporals* 2, the lower touching the 5th and 6th supralabials. *Supralabials* 7. *Anterior sublinguals* touch 4 infralabials. *Posterior sublinguals* touch the 4th and 5th infralabials. *Infralabials.*—The 4th and 5th are the largest of the series, and about subequal. *Scales.*—2 heads length behind the

head 19-27; midbody 19 to 27; 2 heads length in front of the

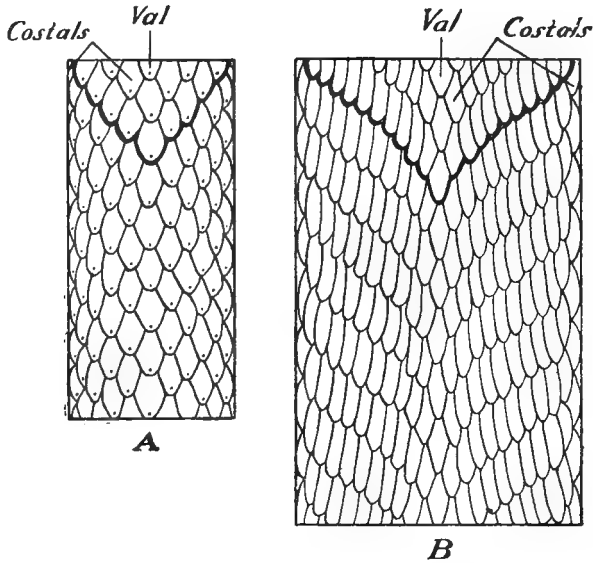


FIG. 17—A, Scales on back of *Lycodon aulicus*.
B. " " *Naia tripudians*.

vent 15 usually (rarely 13 or 17). *Anal* entire. *Subcaudals* divided throughout.

Distribution.—It occurs in one or other of its many colour varieties throughout the whole of our Indian possessions from Burmah in the east to Sind in the west and from the Himalayas to Ceylon and is always a fairly common snake. It is an inhabitant of the plains, but it has been recorded at altitudes up to 6,000 feet.

Poison.—Undoubtedly fatal to man, but by no means every case of cobra bite necessarily proves fatal; on the contrary a percentage hard to determine, but believed by Lamb to be about 30 per cent. of all cases, escapes with moderate or very severe symptoms, the dose injected being less than the lethal. (See part II, page 76.)

Dimensions.—Specimens over 6 feet in length are very uncommon. The largest measurements known to me are both 6 feet 7 inches. One was reported in the Bombay Natural History Journal (Vol. XXI, p. 718), from Shamshirnagar, and the other is in the possession of Sir Thomas Lipton. It was killed in Colombo,

and is now set up, in his residence at Ossidge. I measured it as now set up, but it is stated to have been 7 feet long in life.

Colour.—Very variable. It may be any shade, from buff or wheat colour to olivaceous, brown, or tarry black and even foliage green (N. Siam). These hues are uniform, or more or less variegated. The hood may be without marks, or adorned with a spectacle-like device, or an oval spot surrounded by an ellipse or various modifications of these.

NAIA BUNGARUS—The Hamadryad or King Cobra.

Identification.—A pair of large shields are *in contact with one another*, behind the parietals (see Oc., fig. 18), and this alone will serve to distinguish this from every other snake.* Even if the head is badly mutilated I think this feature will be made out. In case, however, the point is dubious, the snake will be known by the existence of the following 2 characters which must co-exist. The shields under the base of the tail are entire, whilst those towards the extremity are divided, and the vertebral row of scales is similar in size and shape to the adjacent rows.

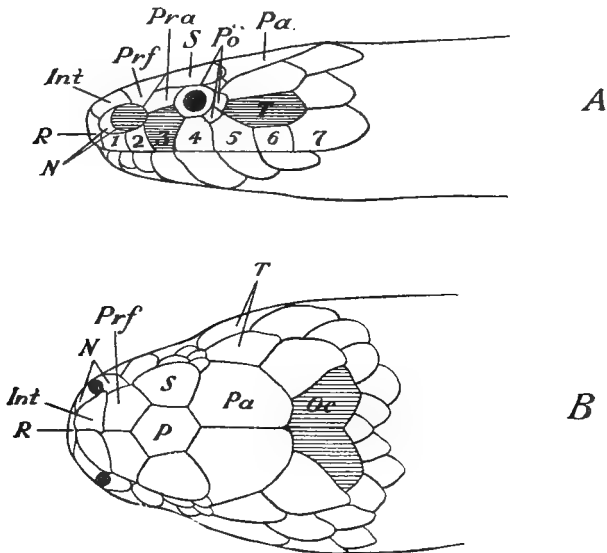


FIG. 18.—*Naia bungarus* ($\frac{2}{3}$ nat. size).

* In almost every other snake the parietals are succeeded by small scales, and in the rare exceptions where occipitals are present, they do not touch one another (see Oc., fig. 13).

Supplementary characters.--*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular, and frontal. *Temporals* 2, the lower touching the 5th, 6th and 7th supralabials. *Supralabials* 7. *Anterior sublinguals* touch 4 infralabials. *Posterior sublinguals* touch the 4th and 5th infralabials. *Infralabials.*—The 5th is the largest of the series and touches 2 scales behind. *Scales.*—2 heads lengths from head 17 (rarely 15) mid-body 15, 2 heads lengths in front of vent 15. *Anal* entire.

Distribution.—It is found throughout our Indian domains (with the exception of Ceylon, and I believe Western Rajpootana, Sind, and the Punjab?) in suitable localities, that is, in jungles or their vicinity. It occurs in hilly regions up to an altitude of 7,000 feet at least and in the plains in their near vicinity.

Poison.—(See Part II, p. 87.)

Dimensions.—The largest record I am aware of is that reported by Phipson.* The snake which was captured in the Konkan measured 15 feet 5 inches. Another of exactly similar length was reported in the "Pioneer" September 4th, 1896, from Travancore by Lieut. V. H. Branson, 28th Madras Infantry.

Colour.—Young are jet black with white or yellow conspicuous cross bars or chevrons on the body and tail. The head is crossed by 4 similar bars, usually complete, sometimes interrupted.

Adults vary a good deal. They may be yellow, olive-green, olive-brown, blackish-brown, or black, usually with more or less distinct yellowish or whitish cross bars or chevrons on the body, which are narrower than the intervals. Light specimens are often more or less variegated with black in the hinder part of the body and tail. Often, too, the shields on the head and scales on the neck are bordered with black, but the crossbars seen in the young are absent. The belly may be nearly uniform, mottled, or barred, but the throat is usually uniformly light-yellowish or cream coloured.

*Bombay Nat. Hist. Journl., Vol. II., p. 245.

CALLOPHIS BIBRONI—Bibron's Coral Snake.

Identification.—It may be told from all others of this group by the fact that the præfrontal shield touches the 3rd supralabial. (Prf. and 3, fig. 19 B.)

Supplementary characters.—Præfrontals touch the internasal, posterior nasal, 3rd supralabial, eye, supraocular and frontal. *Temporal* 1 touching the 5th, 6th and 7th supralabials (and sometimes the 4th also). *Supralabials* 7. *Anterior sublinguals* touch the 1st, the 3rd and the 4th infralabials. *Posterior sublinguals* touch the 4th infralabial.

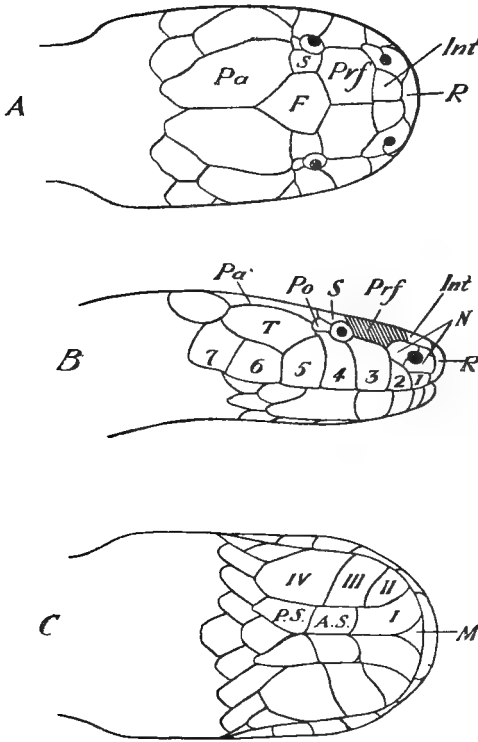


FIG. 19.—*Callophis bibroni* (× 3).

Infralabials.—The 4th is the largest of the series, and touches 3 scales behind. *Scales* are 13 in the whole body. *Anal* entire. *Subcaudals* divided throughout.

Distribution.—A rare species recorded only from the Western Ghats of India.

Poison.—Nothing known.

Dimensions.—Grows to 2 feet and over.

Colour.—Boulenger says*: “Cherry-red to dark purplish brown above, red beneath, with black transverse bands which are sometimes continuous across the belly; anterior part of head black above.”

* Cat. Snakes, Brit. Mus. Vol. III, p. 99.

CALLOPHIS MACCLELLANDI—Maclelland's Coral Snake.*Identification.*—

From others of the group it can be distinguished by the following 3 characters co-existing:—The anal shield divided (as in fig. 9 E); supralabials 7; and a single temporal touching only the 5th and 6th supralabials. (See fig. 20 B.)

Supplementary characters.—*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular, frontal. *Temporal 1* touching the 5th and 6th supralabials. *Supralabials* 7. *Anterior sublinguals* touch 4 infralabials.

Posterior sublinguals touch the 4th infralabial only. *Infralabials*.—The 4th is the largest of the series, and touches 2 scales behind. *Scales* 13 in whole body. *Anal* divided. *Subcaudals* divided throughout.

Distribution.—Ranges from the Himalayas as far west as Kasauli, Nepal and Sikkim through Assam, and Burmah to Southern China and Formosa. Common in the Khasi Hills about Shillong.

Poison.—Nothing known.

Dimensions.—My largest specimen is 2 feet 7½ inches.

Colour.—There are four very distinct colour varieties.

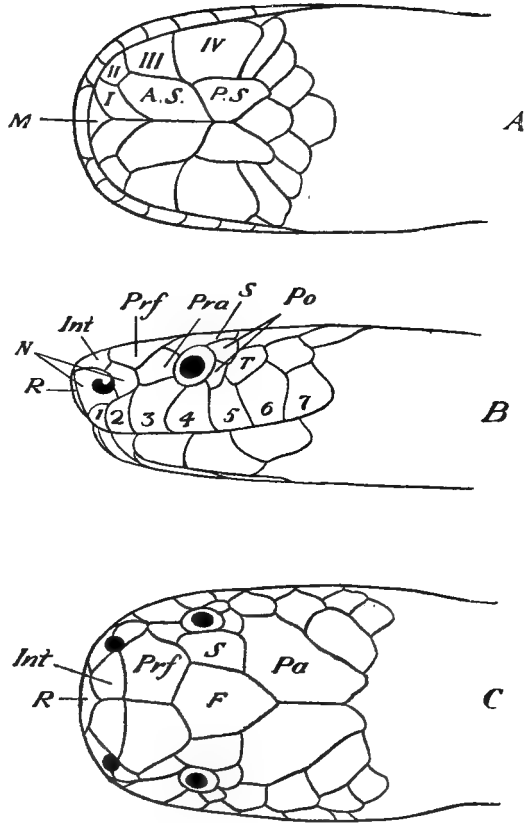


FIG. 20. - *Callophis macclellandi* ($\times 3$).

Variety A, forma typica.—Cherry-red above with from 16 to 26 black bands on the body and 3 to 4 on the tail. These bands are narrow, completely surround the belly and are outlined more or less distinctly with buff or yellow. The belly is sulphur yellow, and a large black irregularly-shaped blotch occurs between each band. It is very common in the Khasi Hills about Shillong. It has been recorded from Burma by Evans and myself who obtained a specimen from the Pegu Yomas. I have had a specimen from Mogok, Ruby mines, and a specimen from Pegu is in the British Museum. It extends into Southern China and Formosa.

Variety B, univirgatus.—Cherry-red, or brown with 23 to 32 black bands, many of which are incomplete, especially in midbody over the spine. A black streak runs down the spine. Otherwise this variety is like the last. It is known from the Eastern Himalayas. (Nepal and the vicinity of Darjeeling.)

Variety C, gorei.—This has no black bands and no vertebral stripe. It is cherry-red above with about 30 small, black, vertebral spots. Some of these are rather broader than long. Belly pale yellow with irregularly-shaped, median, black ventral spots, smaller than those seen in the other two forms. I received two specimens of this new colour variety from Mr. C. Gore whose name I attach to it. Both were obtained at Jeypore (Assam) at the foot of an outlier from the Naga Hills and more recently one from Manipur.

Variety D, nigriventer.—Known from a single specimen from Kasauli described by me. It differs from *univirgatus* in having no trace of black rings and in having a broad black continuous stripe along the middle of the body. The only form known from the Western Himalayas.

In all four varieties the head is black with a very well defined, enamel-white band across the head.

CALLOPHIS TRIMACULATUS—The Slender Coral Snake.

Identification.—Differs from others of this group in combining the 2 following characters. The anal shield is divided (*see* fig. 9 E) and there are 6 supralabials.

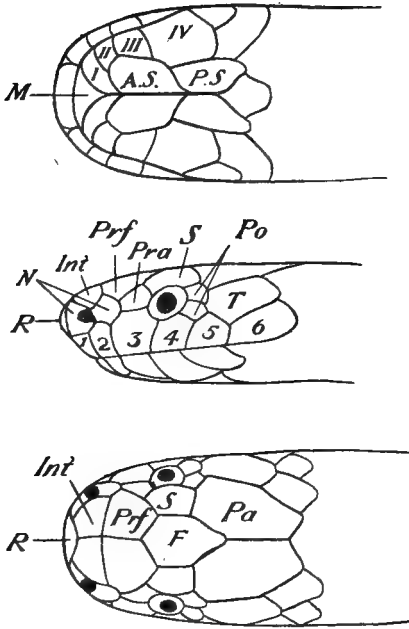


FIG. 21.—*Callophis trimaculatus* ($\times 6$).
from Ceylon*, S. India, Deccan, Kanara, Bengal, and Burmah.

Poison.—Nothing known.

Dimensions.—Of very slender form. Grows to 13 inches.

Colour.—Light yellowish-brown. Head and neck black. Tail with two black rings. Belly coral pink.

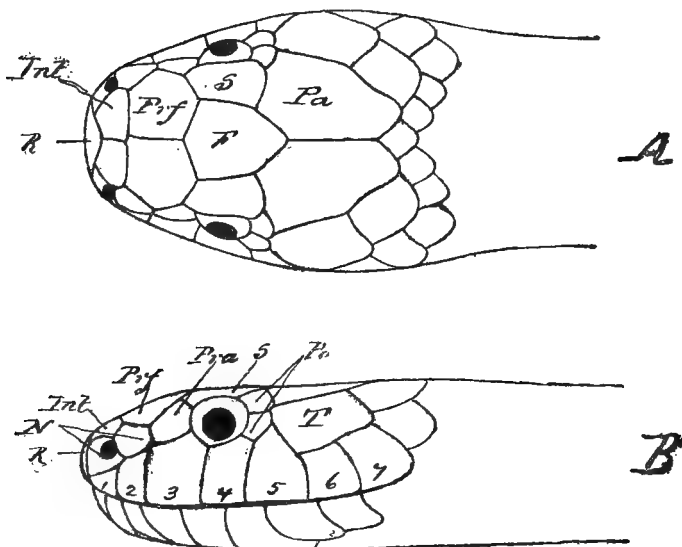
CALLOPHIS MACULIGEPS—The Small-spotted Coral Snake.

Identification.—This and the next differ from others of this group in having the anal shield divided (as in fig. 9 E) and the temporal shield touching the 5th, 6th and 7th supralabials. The habitat will separate one from the other.

* I examined one in the Colombo Museum from Tissamaharana, 20 miles N. E. of Hambantota.

Supplementary characters.—*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular, and frontal. *Temporal* 1, touching the 5th and 6th supralabials. *Supralabials* 6. *Anterior sublinguals* touch 4 infra-labials. *Posterior sublinguals* touch the 4th infra-labial. *Infralabials.*—The 4th is the largest of the series, and touches 2 scales behind. *Scales* in 13 rows in whole body. *Anal* divided. *Subcaudals* divided throughout.

Distribution.—An uncommon snake recorded

FIG. 22—*Callophis maculiceps* ($\times 6$).

Supplementary characters.—*Præfrontals* touch the internasal, posterior nasal, præocular, supraocular, and frontal. *Temporal*—One; touching the 5th, 6th and 7th supralabials. *Supralabials* 7. *Anterior sublinguals* touch 4 infralabials. *Posterior sublinguals* touch the 4th infralabial. *Infralabials*.—The 5th, rarely 4th, is the largest of the series, and touches 2 scales behind. *Scales* 13 in whole body. *Anal* divided. *Subcaudals* divided throughout.

Distribution.—An uncommon snake restricted to the Burmese area of our British-Indian Territory.

Poison.—Nothing known.

Dimensions.—Grows to $1\frac{1}{2}$ feet.

Colour.—Head and neck black. Body yellowish-brown above, with a series of small black dots on each side of the spine. Two black bands on the tail—one basal, the other subterminal. Belly coral pink. Tail dappled black, and grey beneath.

HEMIBUNGARUS NIGRESCENS—The Common Indian Coral Snake.

Identification.—Like the last this differs from others of this group, in that the anal shield is divided, and the temporal touches the 5th, 6th and 7th supralabials. Its habitat will distinguish it.

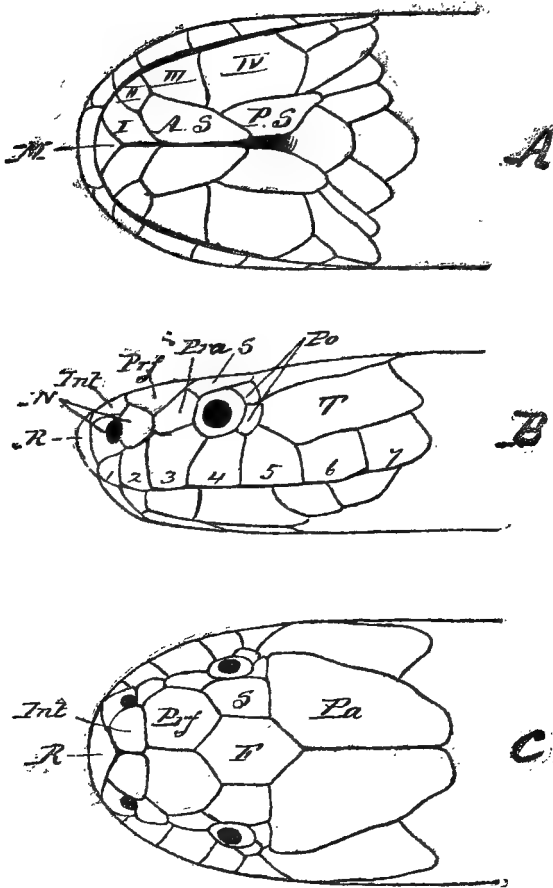


FIG. 23.—*Hemibungarus nigrescens* (×3).

including the Nilgiris and Anamallays. Also from the Ganjam Hills.

Poison.—Nothing known.

Dimensions.—It grows to 4 feet.

Colour.—Head and neck black except for a yellowish oblique occipital streak. Dorsally purplish-brown, reddish-brown, or red, with 3 or 5 longitudinal series of spots which in some specimens are confluent, and form lines. Belly uniform red.

Supplementary characters.—*Præ-frontals* touch the internasal, posterior nasal, præocular, supraocular, and frontal. *Supralabials* 7. *Anterior sublinguals* touch 4 infralabials. *Posterior sublinguals* touch the 4th infralabial. *Infralabials*.—The 4th is the largest of the series, and touches 2 scales behind. *Scales* in 13 rows in whole of body.

Anal divided. *Subcaudals* divided throughout.

Distribution.—It is a hill species confined to the Hills of Western India, from Wynad to Travancore

GROUP 4.—THE PIT-VIPERS.

Identification.—(1) *Tail round.* (2) *A conspicuous opening in the side of the face between the eye and the nostril (the loreal pit) (see Fig. 24 B).*

This very distinctive character is peculiar to this sub-family of vipers. In spite of the fact that many members of this sub-family (*Crotalinæ*) attain formidable proportions, and almost all are endowed with remarkably large poison fangs, the numerous accounts of bites inflicted by them to be found in scientific and other journals concur in showing that death is an exceedingly rare event. My own experience, supported by that of many of my friends, who have favoured me with letters on this subject, entirely confirms the foregoing. A painful and swollen condition locally and a very variable degree of constitutional disturbance lasting in some instances for weeks passes on to complete recovery.

These snakes are nearly all exclusively confined to hilly regions at altitudes ranging between 1,500 to 10,000 feet. The characters of the shields, and scales upon which the classification of nearly allied ophidian forms is so largely based, are subject to very great inconsistency in the members of this group, so much so that it is with the greatest difficulty one can frame a lucid and really practical key to identify the various species. I have, however, examined and re-examined most critically all the specimens in the British Museum, and have only made allusion to those peculiarities which are most constant, and which seem to me of real practical use in identification.

*Key for identification of Pit-Vipers.***HEAD WITH LARGE SHIELDS** (*see Figs. 24 to 27*).

Scales midbody in 21 or 23 rows (*see Fig. 7*) *Ancistrodon himalayanus.*

Scales midbody in 17 rows

(a) Supraocular as broad as frontal, longer than parietals *Ancistrodon hyznale.*

(b) Supraocular broader than frontal, shorter than parietals ,, *millardi.*

Scales midbody in 15 or 14 rows *Lachesis macrolepis.*

HEAD COVERED WITH SMALL SCALES (*see* Fig. 28).

SUPRAOCULARS UNDIVIDED (*see* S. Fig. 26).

- 2ND SUPRALABIAL DISTINCT FROM LOREAL PIT (*see* Fig. 28). *Lachesis strigatus.*
- 2ND SUPRALABIAL WITH A FURROW IN ITS UPPER PART DIRECTED INTO LOREAL PIT (*see* Fig. 27).
MANY SMALL SUBOCULAR SCALES (*see* Fig. 29) „ *monticola.*
AN ELONGATE SUBOCULAR SHIELD (*see* So, Fig. 26).
SCALES 21 or 19 IN POSTERIOR BODY (*see* Fig. 7).
 Nasal and 1st supralabial partially or completely united (*see* Fig. 30).
Scales in midbody 29 rows (*see* Fig. 7) „ *cantoris.*
 „ „ „ 27 to 23 rows „ *purpureomaculatus.*
 Nasal and 1st supralabial distinct (*see* Fig. 33) „ *mucrosquamatus.*
SCALES 17 or 15 IN POSTERIOR BODY (*see* Fig. 7).
 Supralabials 7 or 8 (*see* Fig. 33) „ *jerdoni.*
 „ 9 to 12 (*see* Fig. 34) „ *gramineus.*
- SUPRAOCULARS DIVIDED (*see* S. Fig. 35)
 Subocular touching 3rd supralabial (*see* So, Fig. 35) „ *trigonocephalus.*
 „ not „ „ „ (*see* So, Fig. 36) „ *anamallensis.*

ANCISTRODON HIMALAYANUS—The Common Himalayan Viper.

Identification.—The top of the head has the shields in front enlarged, and the scales in the middle of the body are arranged in 21 to 23 rows. These combined characters will distinguish this from the other pit-vipers, and even if the head is badly mutilated short of dissolution, I think the enlarged head shields will be generally clearly recognized.

Distribution.—It is confined to the Himalayan region, including the Khasi Hills of Assam, at altitudes between 5,000 to 12,000 feet, and is exceedingly common in some localities (Chitral, Kashmir and the whole Western Himalayas).

Poison.—(*See* Part II, page 117.)

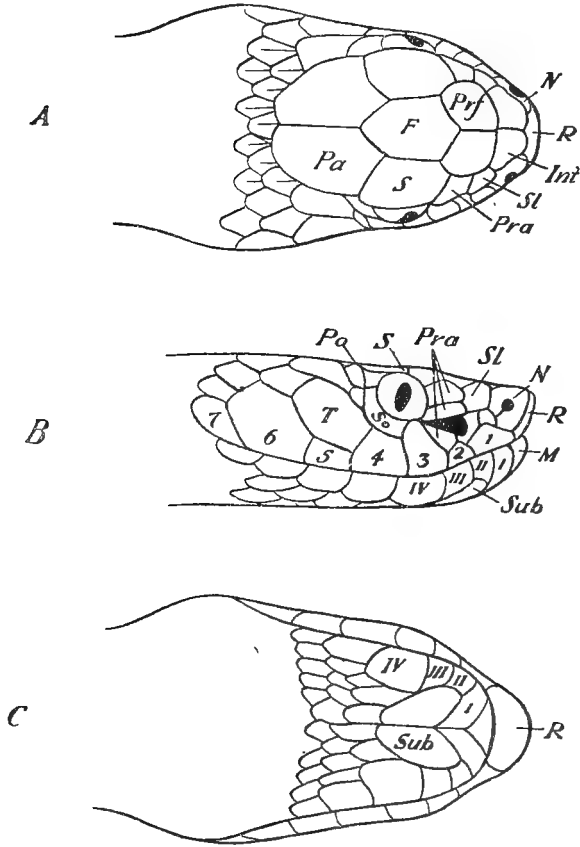


FIG. 24.—*Ancistrodon himalayanus* ($\times 2$).

Dimensions.—Specimens over two feet are uncommon, the largest I know is that reported by Dr. Stoliczka which measured 2 feet 10 inches. (Journl. As. Soc. Bengal, XXXIX, p. 226.)

Colour.—Brown of various hues, sometimes nearly uniform, especially in light specimens, but more often mottled or variegated so as to form bars, or a nondescript carpet-like pattern. Belly peppered blackish and red, on a whitish ground.

ANCISTRODON HYPNALE—The Hump-nosed Viper.

Identification.—Like the last this species has large shields on the front of the head, but differs in the scales, numbering 17, in the

middle of the body, and both these characters will usually be detected even in a badly mutilated specimen. The frontal shield along a line connecting the centres of the eyes is subequal to the breadth of the supraoculars. The supraoculars are from three-fourths to four-fifths the length of the parietals. Ventrals 116 to 131. Subcaudals 24 to 37. The boss on the snout is much higher than in the next species, and is covered with smaller and more numerous scales (8 to 12).

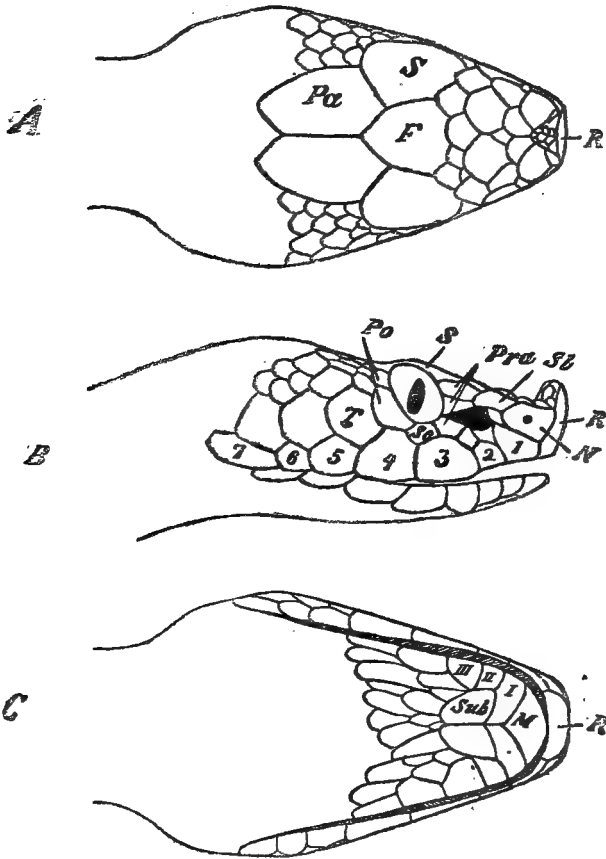


FIG. 25.—*Ancistrodon hypnale* ($\times 3$).

Distribution.—The Hills of Ceylon. It occurs at altitudes varying from 3,000 to 6,000 feet and is not uncommon in many parts. It is a very common snake in some of the hilly districts in Ceylon (Hakgalla).

Poison.—Writers are not consistent upon this question. Tennent* says emphatically that a fatal issue does sometimes occur, but not invariably. Gunther† says it is exceptionally fatal to man, and then not before the lapse of some days. Dr. Davy knew a dog bitten by one recover after severe symptoms in 48 hours, but a fowl bitten by the same snake the next day succumbed after 4 days. These effects on small animals serve to show that the poison is not very virulent. Mr. Drummond Hay has written to me of two cases of bite, both in cooly women. One bitten on the ankle did not suffer in the slightest once she had recovered from her fright, but whether she was treated or not I am unable to say. The other bitten in the hand became unconscious and he thought when he saw her the same night would die, but with the aid of stimulants had recovered by the next day. Ferguson‡ mentions the self-related facts of a Mr. A. F. Sanderson who was bitten by one. The seat of injury was the little toe. Pain was so acute as to prevent sleep, and the limb swelled to the knee for 2 or 3 days, but he recovered. He treated himself by ligature above the knee, cross cuts locally with the application of carbolic acid, and strong potations of brandy.

Dimensions.—Grows to 18 inches, but I have known females adult at $11\frac{1}{2}$ inches, as shown by pregnancy.

Colour.—The prevailing colour is brown, variously mottled or variegated, but a longitudinal series of largish oval dark spots on each side of the back is a constant characteristic. The belly is finely mottled.

ANCISTRODON MILLARDI§—Millard's Viper.

Identification.—Shields on the top of the head enlarged, scales at midbody 17, supraoculars decidedly broader than the frontal, and as long or longer than the parietals. Ventrals 136 to 152. Subcaudals 30 to 44. The boss on the snout is not so pronounced as in the last, and is covered with larger and fewer scales (4 to 6).

* Nat. Hist. of Ceylon, p. 296.

† Rept. Brit. Ind., p. 395.

‡ Bom. Nat. Hist. Journ. Vol. X, p. 9.

§ Described in the Bom. Nat. Hist. Journal, Vol. XVIII, page 792

Distribution.—The 5 specimens I have seen are from Carwar on the West Coast and Castle Rock on the top of the Western Ghats. Specimens in the British Museum from Ceylon and the Hills of Western India (Belgaum and Anamallays), judging from the numbers of the ventrals and subcaudals, appear to agree.

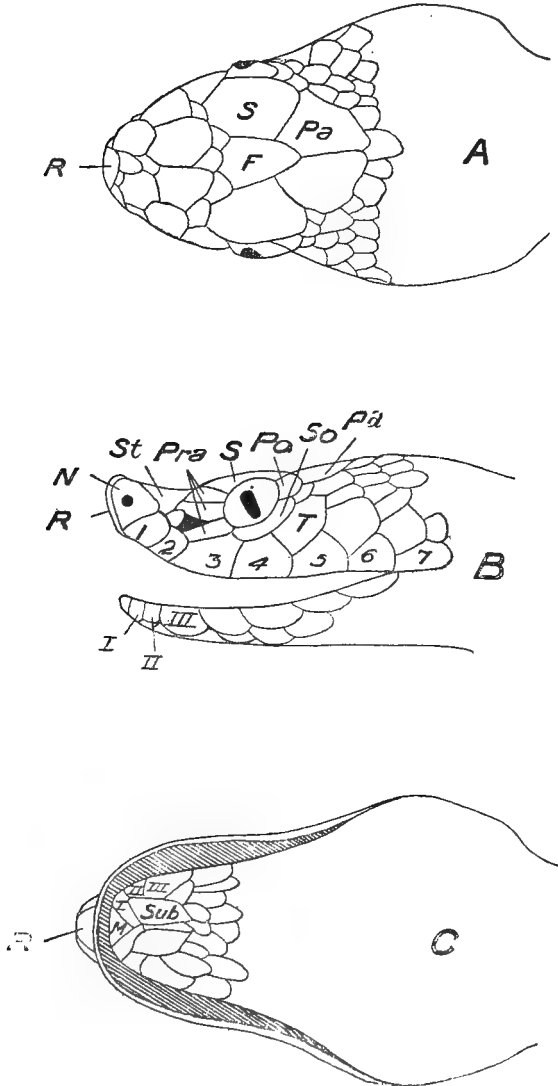


FIG. 26. *Ancistrodon millardi* (x3).

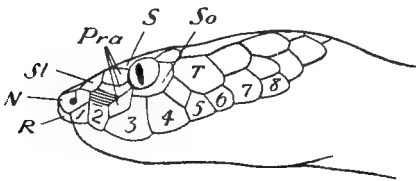
Poison.—Nothing known.

Dimensions.—About a foot or a little over.

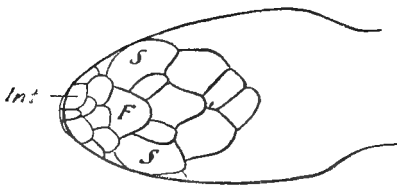
Colour.—Brown of varying shades and mottlings. A series of ovate dark costal spots are always more or less in evidence. Very like *hypnale* in general appearance.

LACHESIS MACROLEPIS—The Large-scaled Viper.

Identification.—One very distinctive feature makes the recognition



of this snake a very simple matter. The scales of the last row along the body are smaller than any of the other rows. In all other British Indian snakes the scales in this row are subequal to, or much larger than, those lying above.



Distribution.—Confined to the Pulney, Shevaroy, and Anamallay Hills of Southern India, where it is plentiful at altitudes vary-

FIG. 27.—*Lachesis macrolepiss* (nat. size).

ing from 2,000 to 7,000 feet.

Poison.—Jerdon* knew several cases of bite from this species, but none proved fatal. The Rev. F. Castets has informed me that he once caused a fresh adult to bite a jackal, but the jackal did not seem to mind, and suffered no ill-effects.

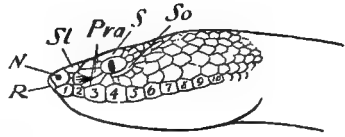
Dimensions.—Grows to 2 feet.

Colour.—Uniform bright foliage-green above, lighter beneath. A well-defined white or yellow line runs down the flanks. Sometimes a blackish supercilium; blackish marks along the spine, and blackish rings round the tail, but these rapidly fade in spirit. Rarely specimens are met with uniform olive-brown in colour.

* Journal, Asiatic Soc., Bengal, Vol. XXXII, p. 521

LACHESIS STRIGATUS—The Horse-shoe Viper.

Identification.—This is the only species in which the 2nd labial shield is entirely distinct from the loreal pit (see Figs. 27 and 28), and this alone will suffice to establish its identity.



Supplementary characters.—*Internasals.*—No scales are sufficiently enlarged to deserve the name. *Supraocular.*—A single shield.

Nasal.—Not united to 1st labial, one or more minute scales are intercalated between it and the furrowed shield forming the inner wall of the loreal bit. *Subocular.*—Not touching the 3rd labial. *Scales.*—Anterior usually 21 (rarely 19); midbody usually 21 (rarely 23); posterior usually 15 (rarely 17).

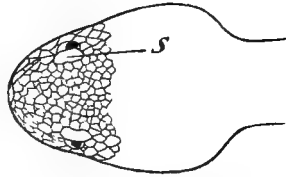


FIG. 28.—*Lachesis strigatus*
(nat. size).

Distribution.—The Western Ghats and the Nilgiri, Anamallay, Shevaroy, and Pulney Hills of Southern India, at altitudes from 3,000 to 8,000 feet. Gray mentions it as common about Ootacamund, and Jerdon as not uncommon in the wooded parts of the Nilgiris; but judging from the paucity of specimens in museums, and the written testimony of friends, it appears to me an uncommon snake everywhere.

Poison.—Jerdon* mentions being bitten by one. A ligature speedily applied, followed by suction, warded off any ill-effects, but the skin round the bite blackened in a minute or two, detached itself, and came off in his mouth during suction.

Dimensions.—Grows to $1\frac{1}{2}$ feet.

Colour.—The prevailing colour is brown, mottled darker to form an irregular coarse variegation. A pale buff or yellowish horse-shoe mark on the nape. A dark streak behind the eye. Beneath light-coloured mottled with darker hues.

*Journal, Asiatic Soc. Bengal, Vol. XXII, p. 525.

LACHESIS MONTICOLA—The Large-spotted Viper.

Identification.—This is the only species that has no subocular shield, and this character will serve to diagnose it.

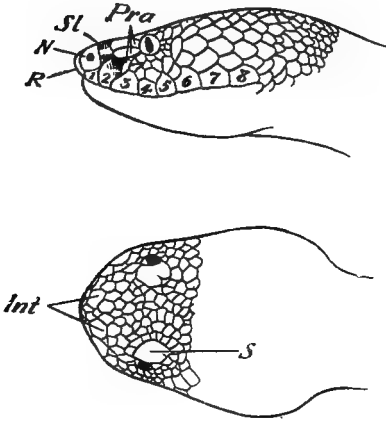


FIG. 29.—*Lachesis monticola* (nat. size).
 anterior usually 23 (rarely 25);
 midbody 23 (rarely 21 or 25); posterior 19 (rarely 21).

Distribution.—The Himalayan region (from 2,000 to 8,000 feet), including hills of Assam, Burmah and Yunnan. Colonel Waddell, I.M.S., in his book "Among the Himalayas" (p. 240) mentions it as common in Bhotan. I found it common in the Khasi Hills (Shillong), and in the Eastern Himalayas (below Darjeeling).

Poison.—(See Part II, page 119.)

Dimensions.—Grows to 3 feet.

Colour.—Light brown or buff with large irregularly squarish patches or spots of black on the middle of the back, and a coarse mottling of these two hues in the flanks. Crown dark-brown with a buff V-bordered dark-brown below. Belly yellowish, uniform in front, obscurely spotted or mottled behind.

LACHESIS CANTORIS—Cantor's Viper.

Identification.—Most easily identified by the rows of scales in the middle of the body numbering 29.

Supplementary characters.—*Internasals.*—A pair separated by one small scale. *Supraocular.*—A single shield. *Nasal* partially or completely united with 1st labial; no minute scales intercalated

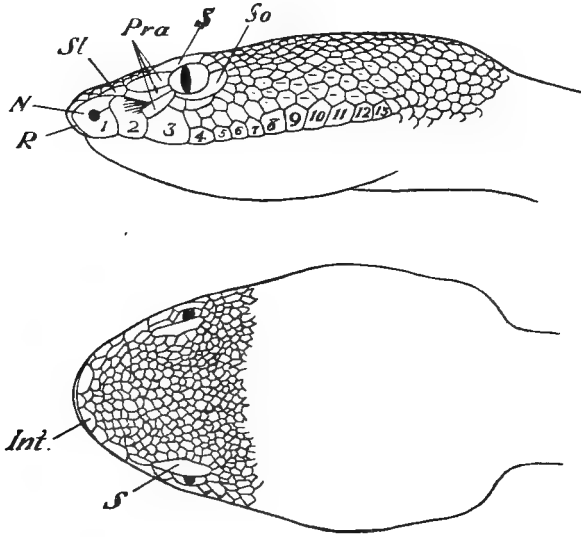


FIG. 30.—*Lachesis cantoris* (nat. size).

Note confluence of nasal (N) and 1st supralabial (1).

between it and the 2nd labial. *Subocular* not touching the 3rd labial. *2nd labial* furrowed in its upper half, and forming the inner wall of the loreal pit. *Scales.*—Anterior, 27; midbody, 29; posterior, 21.

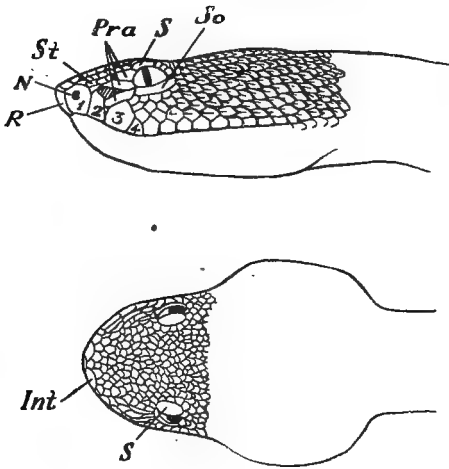
Distribution.—Peculiar to the Andaman and Nicobar Islands.

Poison.—Stoliczka remarks on the small size of the poison gland even in specimens 3 to 4 feet long, and both he and Dr. Rink who visited the insular groups above mentioned, where they found this snake extremely abundant, elicited information from the natives showing that they (the natives) did not regard the bite as fatal.

Colour.—There are two varieties, the one bright green or dull greenish with dark spots, often arranged alternately in five longitudinal series; the other light, or dark brown, spotted with pale greenish. Usually a well-defined white line runs along the flanks, and the head has frequently a pale lateral streak. Belly whitish or greenish, uniform or mottled.

LACHESIS PURPUREOMACULATUS.—Gray's Viper.*

Identification.—The nasal shield more or less united with the 1st



labial, and the scales in the posterior part of the body numbering 19, when taken together will distinguish this from all the rest of the group.

Supplementary characters.—*Internasals.*—A pair in contact with one another, or more usually separated by one small scale. *Supraocular.*—A single shield.

FIG. 31.—*Lachesis purpureomaculatus*. (nat. size.) *Nasal.*—Partially or completely united with the 1st labial; one or more minute scales intercalated between it and the 2nd labial. *Subocular.*—Not in contact with 3rd labial. *2nd labial* with a furrow in its upper part directed into the loreal pit. *Scales.*—Anterior, 23 to 25; midbody usually 25 (rarely 23 or 27); posterior, 19.

Distribution.—Bengal, the Himalayas from probably the Sutlej in the West, Assam, and Burmah, Andamans, and Nicobars. In India it is not met with in the plains, but in Burmah occurs in hills and plains alike. Has been and is frequently confused with *L. gramineus*.

Poison.—Stoliczka's observations with regard to the opinion of the natives in the Andamans and Nicobars show that it is not regarded as fatal to man.

Dimensions.—Grows to 4 feet.

Colour.—Three varieties are met with: (A) uniform foliage green; (B) uniform purplish-brown, or purplish-black; (C) variegated, purplish-brown and green. Usually a well-defined white or yellow flank line. Beneath uniform greenish or whitish with sometimes obscure mottling.

*As it is convenient to give English names to snakes, and Gray was the first to describe this viper, I take the liberty of calling it by his name. It is meet, too, that the work of our great herpetologists should be thus memorialised.

LACHESIS MUCROSQUAMATUS—The Formosan Viper.

Identification.—The scales in the posterior part of the body number 21 or 19, the nasal not united to the 1st labial, and the presence of a subocular establish the diagnosis, but all three characters must co-exist.

Supplementary characters. *Internasals.*—A pair separated by from 2 to 4 small scales. *Supraocular.*—A single shield. *Nasal*—Not united with the 1st labial, one or more minute scales intercalated between it and the 2nd labial. *Subocular*

not touching the 3rd labial. *2nd labial* with a furrow in its upper part directed into the loreal pit. *Scales.*—Anterior, 25 to 27; midbody, 23 to 27; posterior, 19 to 21.

Distribution.—Naga Hills, Assam; also Formosa.

Poison.—Nothing known.

Dimensions.—Grows to 3½ feet.

Colour.—Brownish with 3 longitudinal series of blackish spots, the vertebral series being the largest. Belly mottled brownish and white, or uniform whitish.

LACHESIS JERDONI—Jerdon's Viper.

Identification.—The subocular touching the 3rd labial together with 7 to 8 supralabials make diagnosis certain.

Supplementary characters.

Internasals.—A pair, separated by from 1 to 3 small scales.

Supraocular.—A single shield preceded by an enlarged shield peculiar to this species. *Nasal* not united with 1st labial; small scales may or may not be intercalated between it and the

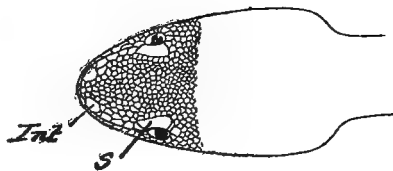
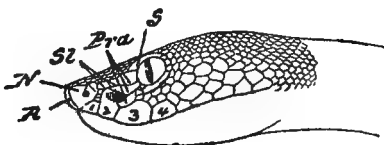


FIG. 32.—*Lachesis mucrosquamatus*.
(nat. size).

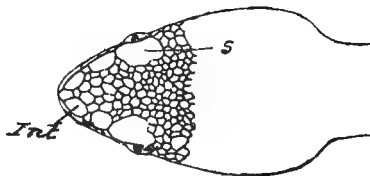
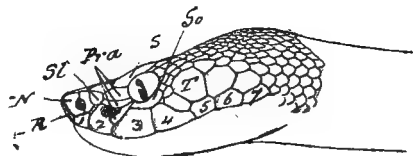


FIG. 33.—*Lachesis jerdoni* (nat. size).

2nd labial. *Subocular* touches the 3rd labial. *2nd labial* with a furrow in its upper part directed into the loreal pit. *Scales*.—Anterior, 21 usually (rarely 23); midbody, 21 (rarely 19); posterior, 17 (rarely 15).

Distribution.—Khasi Hills, Assam, Thibet.

Poison.—Nothing known.

Dimensions.—Grows to $2\frac{1}{2}$ feet.

Colour.—Variegated greenish and black; head black, ornamented with yellow; belly mottled greenish and black.

LACHESIS GRAMINEUS—The Common Green Viper or Bamboo Snake.

Identification.—Scales 15 in the posterior part of the body, supraocular a single shield, supra-labials 9 to 12, the 2nd furrowed in its upper half, if co-existing will serve to identify it.

Supplementary characters.

Internasals.—A pair, in contact, or separated by one or two small scales.

Supraoculars.—A single shield.

Nasal.—Sometimes united with 1st

labial, sometimes distinct; small scales may or may not be intercalated between it and the 2nd labial. *Subocular* may or may not touch the 3rd labial. *2nd labial* with a furrow in its upper part directed into the loreal pit. *Scales*.—Anterior, 21; midbody, 21; posterior, 15.

Distribution.—Much the most plentiful and the most widely distributed of our Indian Pit-Vipers. From the Malayan region it extends through Burma, including the Andamans and Nicobars, to the Himalayan region probably as far west as the Sutlej River.

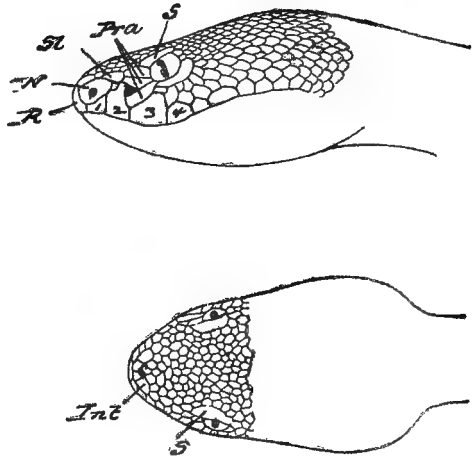


FIG. 34.—*Lachesis gramineus* (nat. size)
(Variety from Western Ghats.)

It is found in the Eastern Ghats, Western Ghats, Nilgiris and other hills in the Peninsula of India. It does not occur in the plains of India, but affects an altitude of from 1,500 to 6,000 feet. East of Calcutta it occurs in the plains and hills alike.

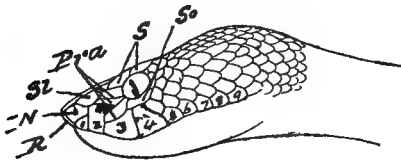
Poison.—The bite is rarely, if ever, fatal, but severe local effects and constitutional disturbances are usually attendant. There is abundant evidence to substantiate this assertion among Europeans and natives alike (*See* illustrative case, Part II, page 122).

Dimensions.—Grows to $3\frac{1}{2}$ feet.

Colours.—Usually vivid foliage-green. More rarely yellowish, or olivaceous or brown, sometimes obscurely streaked or barred with black. A well-defined white or yellow flank line usually. Belly whitish, plumbeous-greenish, uniform or indistinctly mottled.

LACHESIS TRIGONOCEPHALUS—The Green Tic.

Identification.—The supraocular shield divided, and the subocular touching the 3rd labial, if found co-existing, serve to fix its identity.



Supplementary characters.—*Internasals.*—A pair in contact with one another. *Supraocular* divided. *Nasal* not united with 1st labial; no small scales intercalated between it and the 2nd labial. *Subocular* touches the 3rd labial. *2nd labial* with a furrow in its upper part directed into the loreal pit.

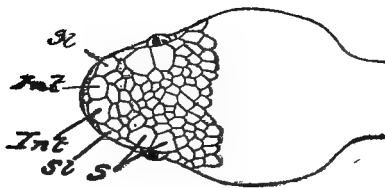


FIG. 35.—*Lachesis trigonocephalus*
(nat. size).

Scales.—Anterior, 17 or 19; midbody 17 or 19; posterior, 13 or 15.

Distribution.—Peculiar to Ceylon, where it is common in many parts of the hills. It is known to the planters as the Green Tic Polonga.

Poison.—Mr. Drummond Hay has informed me in a letter that he once had a Eurasian conductor bitten by a full-grown one in

his presence. The bitten hand swelled up at once, but by evening had much reduced, and the following day the swelling had almost entirely disappeared.

Dimensions.—Grows to $2\frac{1}{2}$ feet.

Colour.—Foliage-green, uniform, or with black blotchings. A black streak behind the eye. Belly uniform greenish or yellowish.

LACHESIS ANAMALLENSIS—The Anamallay Viper.

Identification.—Supraocular divided, and co-existing with this, a subocular not touching the 3rd labial.

Supplementary characters.—*Internasals*.—A pair separated by a small scale. *Supraocular* divided. *Nasal* not united with 1st labial; small scales may or may not be intercalated between it and the 2nd labial. *Subocular*.—Not touching the 3rd labial. 2nd labial, with a furrow in its upper part directed into the loreal pit. *Scales*.—Anterior, 21; midbody, usually 21 (rarely 19); posterior, 15 or 17.

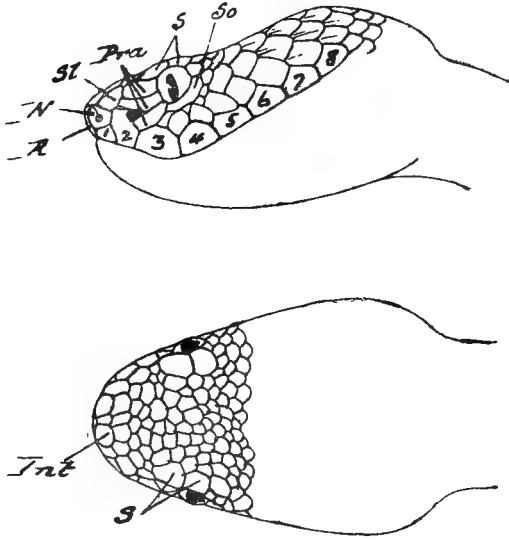


FIG. 36.—*Lachesis anamallensis* (nat. size).

Distribution.—Confined to the Western Ghats and hilly regions south of the Krishna River, where it is quite common, at altitudes ranging between 2,000 to 7,000 feet.*

Poison.—Rarely, if ever, fatal as far as we know (see Part II, page 123). Jerdon has known several cases of bite, but none proved fatal.

Dimensions.—Grows to $3\frac{1}{2}$ feet.

* The specimen from Cuttack (No. 4122 in the Indian Museum, Sclater. J. A. S. Bengal, Vol. LX) is in my opinion *L. gramineus*.

Colour.—Greenish variegated with blackish, or dark blackish-green. Boulenger says olive, yellowish or reddish-brown. Flanks coarsely dappled with buff. Belly greenish or yellowish.

GROUP 5—PITLESS VIPERS.

Identification.—(1) Tail round. (2) Snout and crown covered with small scales similar to those on the back of the body* (see fig. 37). (3) Only a part of the last row of costals is visible on either side of the ventrals when the specimen is laid on to its back (see fig. 5). (4) No loreal pit.

This group includes 6 species referable to 4 genera. They may be identified as follows :—

- A. Shields beneath tail similar to those beneath belly (see SC., Fig. 9 B)..... *Echis carinata*.
- B. Shields beneath tail divided (see SC., Fig. 9 D).
- (a) Ventrals with 2 ridges (see V, Fig. 37)..... *Eristocophis mcmahoni*.
- (b) Ventrals not ridged.
- (a¹) No horn above eye.
- Scales 27 to 33 in midbody 3 chains of large spots, one along spine, and one on each side .. *Vipera russelli*.
- Scales 23 to 27, 1 chain of spots along spine, none on the sides.. ,, *lebetina*.
- (b¹) A horn-like appendage above the eye. Scales 23 to 25 in midbody..... *Pseudocerastes persicus*.
- (c¹) Two horns above the eye. Scales 21 in midbody ,, *bicornis*.

ECHIS CARINATA—The Saw-scaled Viper.

The “Kuppur” of Sind. “Phoorsa” of the Bombay Presidency “Afai” about Delhi. The “Kallu havoo” of Mysore. “Kattu virian” about Madras; and the “Horatta pam,” according to Russell, on the Coromandel Coast. Dr. C. A. Owen tells me it is called “phissi” in the Jhelum District.

Identification.—The undivided state of the shields beneath the tail will admit of no confusion with others of this group.

Supplementary characters.—*Supraocular* not divided. *Nasal* touches the rostral and the 1st supralabial. *Eye*.—Diameter exceeds

* A few harmless snakes have the snout covered with small scales—for instance the Genus *Eryx*. *Hipistes* too may be included with these. In all these, two or often many more rows of costals are visible from beneath (see Fig. 3).

its distance to the nostril, and is greater than its distance to the

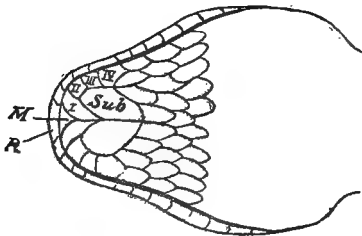
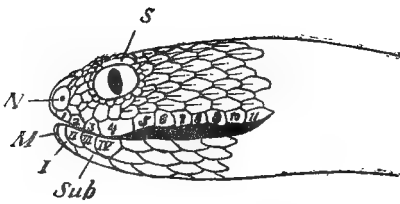
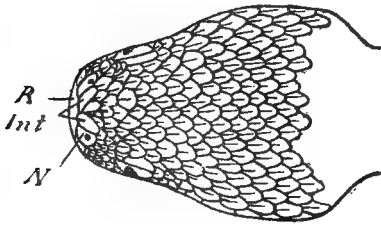


Fig 37.—*Echis carinata* ($\times 2\frac{1}{2}$).

where it is frequently very common. Jerdon remarks it is common throughout the Carnatic. I have found it especially so about Trichinopoly. I believe it does not occur in the narrow tract between the Hills and the Malabar Coast, South of Karwar. To the North-East its limits are not exactly known; if it occurs in Bengal it is scarce and only to the South of the Ganges. To the North-West it extends through Rajpootana, the Punjab, Sind and Baluchistan to Transcaucasia and is extremely abundant in these parts. Some idea of its prodigious numbers was furnished by Vidal.* He says that in the Ratnagiri District alone during 6 years Government rewards were paid on an average of 225,721 phoorsas per annum!

edge of the lip, 2 rows of scales between it and the supralabials. *Supralabials*.—The 4th is! the largest of the series (rarely, the 3rd). *Sublinguals* touch 3 or 4 infralabials, and 2 small scales behind. *Infralabials* 4 (rarely 3), the 4th touching 2 scales behind. *Scales* in midbody 27 to 37. *Ventrals* not ridged laterally. *Subcaudals* undivided. During life its peculiar habit of throwing its body into a double coil, inflating itself, and then rubbing one coil against the other so as to produce a sound closely resembling hissing, will in itself proclaim its identity.

Distribution.—It occurs in the North-East of Ceylon and throughout a large area of the Indian Peninsula from Cape Comorin to the Ganges, but being a desert form preferring an arid sandy soil, it is distributed chiefly in isolated patches

* Journal, Bombay Natural History Soc., Vol. V., p. 64.

Later he remarks that when the Government reward was raised tentatively from six pies to two annas per head, 115,921 were paid for in 8 days (December 2nd to 10th, 1862). Again Candy in the same Journal (page 85) says that in Ratnagiri, in August and September, the Mhars go out with long sticks to which forks are attached and catch them in thousands for Government rewards. It is an inhabitant of the plains, and becomes progressively scarcer at altitudes ranging up to 5,000 feet. 5,700 feet is the highest I know of. Nicholson shows† that of 1,225 poisonous snakes collected in the vicinity of Bangalore (circa 3,000 feet) upon which Government rewards were paid in the year 1873, only one proved to be an *Echis*.

Poison.—Very conflicting opinions have been expressed regarding the virulence of *Echis* poison. It is asserted by many that death is an extremely rare sequel to its bite, but I think there can be no doubt that fatalities are much more frequent than many suppose. Vidal, whose paper in the Bombay Natural History Journal‡ is a most valuable contribution to the literature on this species, states that he found records of 62 fatal cases treated in the Civil Hospital at Ratnagiri in the year 1878. He estimated that about 20 per cent. of the cases of *Echis* bite proved fatal, and remarks that the poison is slow, death occurring on an average in 4½ days, but that some cases lingered on for 20 days. He says later that the *Echis* is a far more potent factor than any other venomous species in swelling the mortality of the Bombay Presidency. He substantiates this assertion by the very significant observation that in *Echis*-ridden tracts the mortality from snake-bite far exceeds that in districts where this snake is comparatively scarce. In a table compiled from official returns for 8 years (1878 to 1885), for the districts of the Bombay Presidency, he shows that in the districts of Hyderabad, Thar and Parkar, Karachi (Sind) and Ratnagiri where the *Echis* abounds, one man in 5,000 dies per annum from snake-bite, whereas in the districts of Bijapur, Nasik, Ahmednagar and Sholapur, where this snake is rare or absent, only one man in 100,000 dies from snake-bite. Murray§ says "this little viper is very venomous; although the action of its poison is not quite so quick as that of a cobra, it is equally as potent, and numerous deaths annually occur from its bite." Dr. Inlach, Civil

† "Indian Snakes," p. 173.

‡ Vol. V., p. 64.

§ "Reptilia of Sind," p. 57.

Surgeon at Shikarpur* (Sind), says, "A reference to police returns will show that in by far the greatest majority of cases serious injury and death have been caused by the bite of this species." Again he avers "the Kuppur is without exception the most deadly poisonous snake in Sind." Mr. Millard has informed me by letter of the case of an attendant in the Bombay Natural History Society's Rooms who, in October 1903, was bitten by an *Echis* in the temple. He was taken off at once to hospital, admitted that he felt no fear, but in spite of prompt treatment died 24 hours afterwards.

In Delhi, in 1897, I knew, and many times saw, a famous snake-catcher called Kallan bring his week's bag to the Civil Hospital where he extracted the poison of cobras, kraits and "afais" for the Civil Surgeon (Major Dennys, I.M.S.), who sent it on to the Government of India. The poison collected, he conveyed his specimens to the Deputy Commissioner for the Government rewards. Each head had to be chopped off, and when later he was counting these out for the satisfaction of an official before payment, one *Echis* head fastened itself on to his finger. The dose of poison under the circumstances must have been very small nevertheless most alarming symptoms rapidly supervened and Major Dennys told me that when he visited the man that night he expected he would die, so grave was his condition. He, however, recovered. One must not allow oneself to be misguided by the many records in which dogs and other small animals have not succumbed to the bite of this snake, and infer that man would probably be even less effected. One can find numerous instances of small animals not succumbing to the effects of bites of cobras and Russell's vipers, though we know how fatal these poisons usually are. This matter is dealt with more fully in Part II (p. 109 *et. seq.*)

Dimensions.—Mr. Colan writing from Jodhpore has told me of a specimen measuring 2 feet 6 inches, but anything over 2 feet is unusual.

Colour.—Various shades from sandy to dark cedar. A more or less distinct pale sinuous flank line always present. A pale mark on the crown somewhat resembling the imprint of a bird's foot. Belly uniform whitish, or dotted with light brown or dark spots.

ERISTOCOPHIS MCGMAHONI—McMahon's Viper.

Identification.—The ventral shields are ridged, on either side unlike other species of this group, and this is the best means of diagnosis (*see fig. 37 C.*).

* Trans. of the Bomb. Med. and Phys. Soc., Vol III., p. 80.

Supplementary characters.—*Supraocular* absent, replaced by small

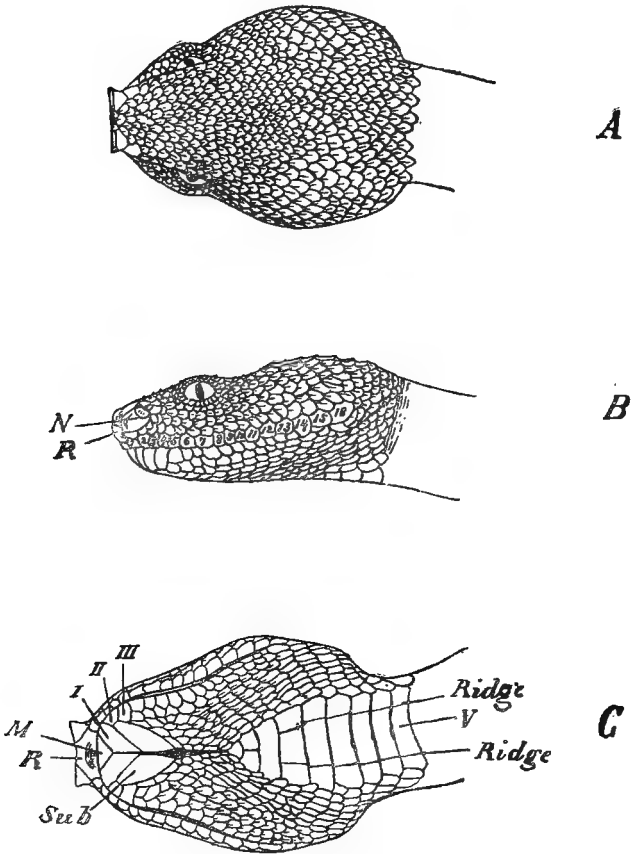


Fig. 38.—*Eristocophis McMahonii* (nat. size).

scales. *Nasal* does not touch the rostral, nor the 1st supralabial. *Eye*.—Diameter less than the distance between eye and nostril; about half the distance to the labial margin; 5 or 6 rows of small scales between it and supralabials. 4th *Supralabial* not enlarged. *Sublinguals* touch 3 *infralabials*, and 3 small scales behind. *Infralabials* 3, the 3rd touching 3 scales behind. *Scales* in the middle of the body 23 to 27. *Ventrals* ridged laterally. *Subcaudals* divided.

Distribution.—Very little is known on this point. Baluchistan, where it was discovered by Captain McMahon* when delimiting the Afghan Baluch border, is probably the fringe of its distribution,

* Now Sir A. H. McMahon.

and it is probably only to be found at this corner of our Indian possessions. It is a desert form inhabiting sandy tracts.

Poison.—Nothing is known.

Dimensions.—The largest specimen was about 2 feet.

Colour.—Reddish sandy brown, with white edged dark-brown spots along the back.

VIPERA RUSSELLI—Russell's Viper, the Chain Viper, the Daboia.

The "Tic polonga" of Ceylon. "Kanardi virian" of Tamils in Madras. "Mandali" of Malabar. "Mandalatha havu," and according to Rice "Kolaku mandala" of Mysore. The "Bora," "Chundra bora," "Siah chunder amaitar," and "Jessur" of Bengal according to Fayrer. The "Katuka rekula poda" of Russell (Coromandel coast?). The "Gunnus" of Bombay. The "Chitar" of Guzerat according to Mosse. The "Khad Chitra" of Dantra District in the Bombay Presidency according to Fenton. I am told the "Korail" of Sind. The "Mwe-bwe" of Burmah. Probably also the "Cobra monil" of some natives as suggested by Jerdon; literally "necklace snake" in Portuguese, and like other names dating from the Portuguese occupation of India, such as "biscobra," its significance has become obscured, and surrounded with mystery by the native mind.

Identification.—The sublinguals touching 4 or 5 infralabials, the subcaudals divided, and the three series of large dorsal spots when occurring in the same specimen will establish the diagnosis.

Supplementary characters.—*Supraocular* a single shield. *Nasal* touches the rostral and the 1st supralabial. *Eye*—Diameter exceeds distance of eye to nostril, and is subequal to its distance to the labial margin in the adult; 2 or 3 rows of scales between it and the labial margin. 4th *Supralabial* the largest of the series. *Sublinguals* touch 4 or 5 infralabials and 2 scales behind. *Infralabials* 5 large normally, the 5th touching 2 scales behind. *Scales* in mid-body 27 to 33. *Ventrals* not ridged laterally. *Subcaudals* divided.

Distribution.—Ceylon. Peninsula India from Cape Comorin to the Ganges. It is, I believe, not found to the North of this River,* and though common in the Irrawady Basin is not known from the Brahmaputra Basin. To the West it extends throughout the Indus Basin from North Sind to the Himalayas. It is chiefly an

* I am aware of the record by Selater of Purneah, but this solitary record, I think, calls for confirmation.

inhabitant of the plains, but is common in some localities from 2,000 to 4,000 feet and has been met with at altitudes up to 6,000

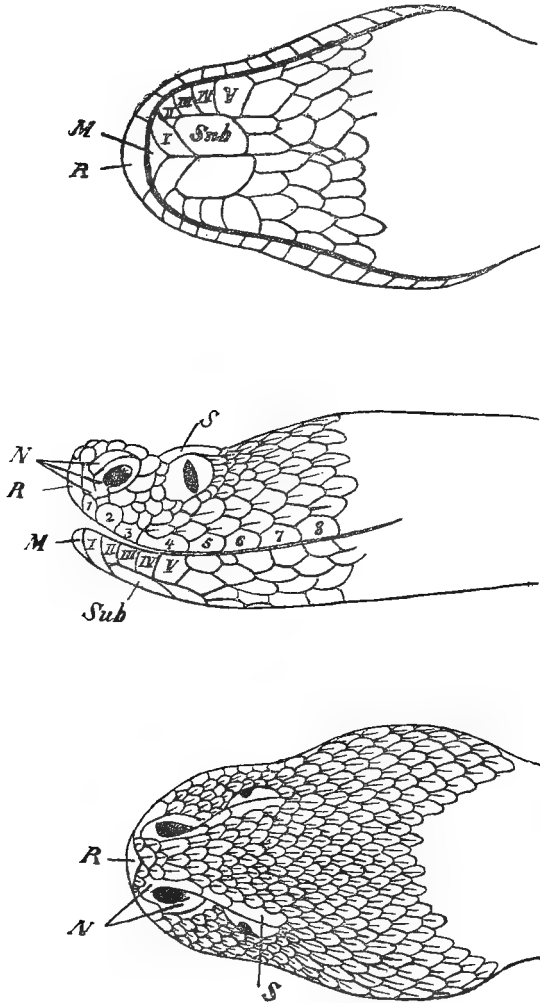


FIG. 39.—*Vipera russelli* (nat. size).

and 7,000 feet.* In most parts it is quite a common snake, but is especially so in certain localities. *Fayrer*† says it is very common in the Punjab and that at Umritsar in 1866 as many as 471 specimens were brought in for Government rewards in one day!

* Kashmir 6,000 ft. (*Stoliczka*). Nilgiris 6,000 ft. (*Henderson*, private letter). Pulneys 6,300 ft. (*Revd. Father Gombert*, private letter). Pulneys 7,000 ft. (*Henderson*, private letter). Hakgalla, Ceylon, 5,700 ft. (I have received specimens from *Mr. Nock*).

† "*Thanatophidia*," p. 55.

Mr. Millard tells me it is common near Bombay. Mr. Henderson in a private letter says it is fairly common at Kodai Kanal in the Pulneys. Father Gombert, S. J., in a private letter makes the same remark with regard to the Pulneys. Stoliczka* says it is very common in the south portion of the Kulu Valley. I have found it common at Trichinopoly and Cannanore, and Ferguson† says it is common in the low country at Travancore. Tennent‡ says that at Trincomalee, Ceylon, in 1858, the Judge's house was so infested with this species that his family had to quit their quarters, and Bassett Smith§ also remarks on the number of this species in the same place. Evans and I found it common in most parts of Burmah, and in certain parts of that Province it is so numerous that the natives wear grass shoes made with "uppers" when busy in the crops as a protection against this snake, notably at Mahlaing, Magwe, and Myo-thit in Upper Burmah. Theobald¶ remarks on the commonness of the species in the Tharrawaddy District in Lower Burmah, and above Rangoon. On the other hand, Nicholson|| shows it is uncommon in the vicinity of Bangalore, where only 2 were brought in for Government rewards out of 1,225 poisonous snakes in the year 1873. Again Murray** says it is not common in Upper Sind. Blanford†† makes the same remark of S.-E. Berar, and Mr. Miller writes me it is rare about Darjeeling. He has only known one, *viz.*, at Kurseong, 4,600 feet, in many years.

Poison.—Frequently fatal to man (*see* Part II, page 99 *et. seq.*)

Dimensions.—Grows to 5½ feet, but specimens over 5 feet are very exceptional.

Colour.—Buff, or light brown with 3 longitudinal series of large spots along the back. These usually consist of three zones, a central one of the same colour as the ground, a narrow dark zone, skirted by a still narrower white or buff zone. Some of these spots in the median series often confluent. The spots in the lateral rows are often broken at their lowermost outline. Head ornamented with large dark marks, and a conspicuous pink or salmon V with its apex on the snout. Belly whitish with dark semilunar scattered spots.

* Journl., Asiatic Soc. of Bengal, Vol. XXXIX, p. 226.

† Journl., Bomb. Nat. Hist. Soc., Vol. X, p. 8. ‡ Nat. Hist. of Ceylon, p. 296.

§ Journl., Bomb. Nat. Hist. Soc., Vol. XI, p. 546.

¶ Cat. Rept., Brit. Burm., p. 64.

|| Ind. Snakes, p. 173.

** The Rept. of Sind, p. 56.

†† Journl., Asiatic Soc. of Bengal, Vol. XXXIX, p. 374.

VIPERA LEBETINA—The Levantine Viper.

Identification—The sublinguals touching 4 or 5 infralabials; the subcaudals divided; and the absence of the large lateral spots on the sides so typical of the last when occurring together, will suffice to identify this from the rest of the group.

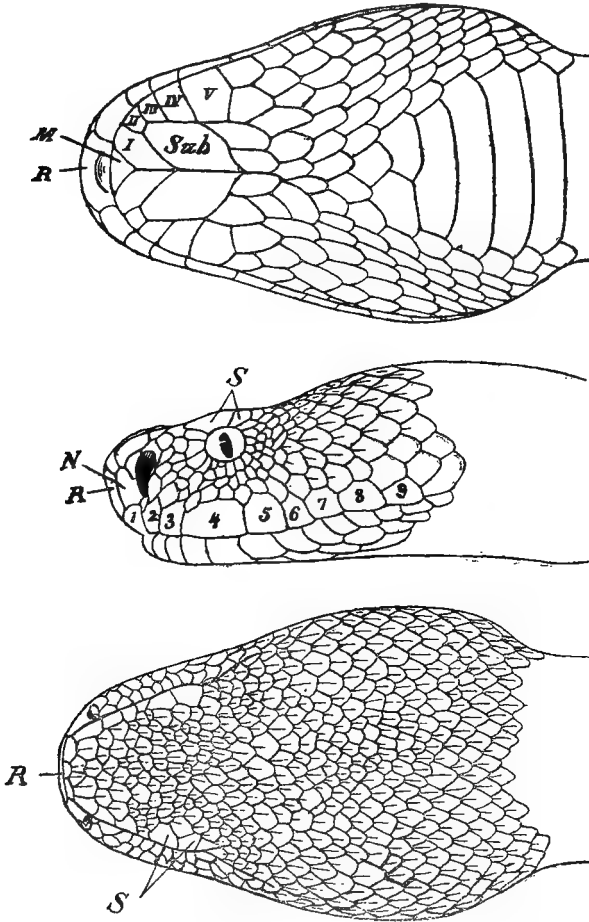


FIG. 40.—*Vipera lebetina* (nat. size).

Supplementary characters.—*Supraocular* well developed or broken up into small shields. *Nasal* touches the rostral and the 1st supralabial. *Eye*.—Diameter about equals its distance to the nostril, about half its distance from the labial margin; 2 or 3 rows of scales between it and the supralabials. *4th supralabial* the largest of the series. *Sublinguals* touch 4 or 5 infralabials and 2 scales behind. *Infralabials*.—5 large normally, the 5th touching 2 scales

behind. *Scales* in middle of body 23 to 27. *Ventrals* not ridged laterally. *Subcaudals* divided.

Distribution.—An inhabitant of Northern Africa and South-Eastern Europe, it extends through Asia Minor eastwards so as to include Baluchistan and Kashmir on the fringe of its distribution.

Poison.—Nothing known.

Dimensions.—Grows to 5 feet.

Colour.—Grey or pale brown above, with a dorsal series of large brown spots, often edged with blackish which may be confluent into an undulous band, or with small dark spots or cross-bars, small dark lateral spots, and vertical bars; a large V-shaped marking on the upper surface of the head, and a V-shaped one on the occiput, may be present; a dark streak behind the eye to the angle of the mouth; and usually a dark blotch or bar below the eye; whitish beneath, powdered with grey-brown, with or without dark brown spots; end of tail yellow. All the markings sometimes very indistinct (Boulenger).

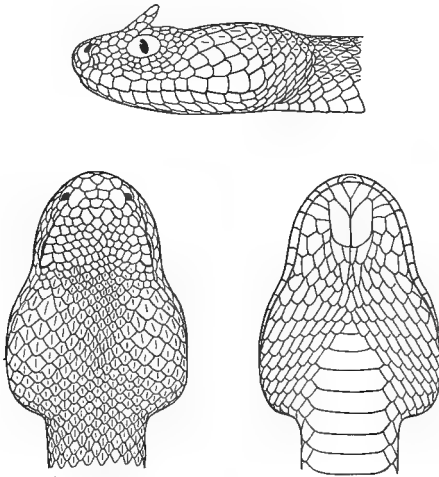


FIG. 41.—*Pseudocerastes persicus* (nat. size).

PSEUDOCERASTES PERSICUS.—The Perso-Baluch horned viper.

Identification.—Most easily recognised by the so-called “horn” above the eye, and the scales in midbody numbering 23 to 25.

Supplementary characters.—*Supraoculars*.—Three subequal, elongated, obliquely erect shields, which form the horn above the eye.

Nasal not touching the rostral or the 1st labial. *Eye*. Diameter rather less than distance to nostril, and distinctly less than distance to labial margin; 3 to 4 rows of scales between it and the supralabials. *Supralabials* 13, the 5th and 6th largest. *Infralabials* 4, the 4th largest, and in contact with two scales behind *Sublinguals*. One pair. *Costals* in 23 to 25 rows in midbody. *Ventrals* not ridged. *Subcaudals* divided.

Distribution.—Persia and Baluchistan.

Poison.—Nothing known.

Dimensions.—Grows to about 3 feet.

Colour.—Greyish brown with six series of large, ill-defined, blackish spots which alternate with those in the adjacent rows. The two median are often more or less confluent to form short transverse bars. An ill-defined blackish broad streak from the eye through the gape, and another less distinct shorter band from the lore to the lip. Belly whitish with grey spots.

Captain Jolly, I.M.S., tells me it is common about Kacha Thana in Baluchistan.

PSEUDOCERASTES BICORNIS.—Smith's Viper.

The description of this new viper is based on an examination of the head and forebody of the only specimen known (now preserved in the museum of the Bombay Natural History Society), and notes on the complete snake in life supplied me by Major O. A. Smith. The specimen, a female, was encountered at Khajuri Kach, above Gwaleri kolal in the Gomal Pass, Waziristan. Major Smith tells me he would not have noticed it had not its loud hiss attracted his attention from a distance of about 4 yards.

Lepidosis. *Rostral*.—More than twice as broad as high, in contact with 8 small scales. *Supranasal*. An enlarged shield placed above the nostrils but not actually bordering it. *Supraoculars* not or hardly enlarged, but two scales are somewhat elongate, and free. These are now depressed, but Major Smith says in life they were erect, and formed the so-called "horns." They are separated from the ocular ring by a row of small scales. No other enlarged or modified shields above. *Nasals*.—Two, the anterior and larger is not in contact with the rostral, and forms about three-fourths of the circumference of the nostrils; the posterior,

small, completes the circumference of the nostrils. *Supralabials*—Many, the 5th largest, and in contact with 2 scales behind. *Sublinguals*.—One large pair, in contact with two scales on each side behind, and with 4 infralabials. *Costals*.—Two heads-lengths behind the head 24, midbody 21, keels present in the anterior three-fourths of each scale where they dilate, and end before the apex; no oblique rows. Ultimate row but slightly larger than those immediately above it.

Dimensions.—It measured $24\frac{1}{4}$ inches, the tail accounting for 3 inches.

Colour.—Dusky khaki with a series of broad short transverse bars on each side of the spine. These are alternately dusky sienna, and dusky bluish grey. Outside these bars are other bars, and spots of dusky bluish, and a fine speckling of blackish. Head uniform. Tail blackish at tip. Belly white. The iris was a dull yellow ochre.

The snake appears to have harmonised very strikingly with its surroundings, for the soil around was of the same hue as the ground colour, and many stones were scattered about tinged with brown and bluish of almost the same shades as the markings on the back.

AZEMIOPS FEÆ—Fea's Viper.

Identification.—(1) scales in midbody 17 (see fig. 7). (2) 6 supralabials of which the 3rd only touches the eye. These two points when co-existing will serve to differentiate this from every other Indian snake.

Supplementary characters.—*Frontal* unusually broad, about 3 times the breadth of each supraocular. *Nasal* touches 1st and 2nd infralabials only. *Loreal* present. This is the only poisonous snake with large shields on the head in which this shield occurs. *Præoculars* 3. A very unusual feature. (Except the pit-vipers I know of only one other snake where these shields

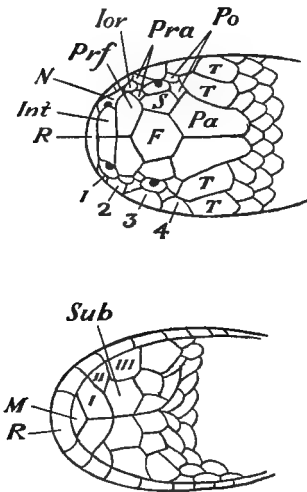


FIG. 42—Azemiops feæ (after Boulenger).

are 3, viz., *Lytorhynchus paradoxus*.) *Temporals* 2. The upper touching one supralabial only, viz., the 4th. Eye with vertical pupil. *Supralabials* 6, the 3rd only touching the eye. *Sublinguals*.—One pair only each in contact with 2 scales behind. *Infralabials* 3 only.

Distribution.—One specimen only known discovered by Signor Fea in the Kachin Hills of Upper Burmah.

Poison.—Nothing known.

Dimensions.—2 feet.

Colour.—Boulenger* says:—"Lower parts olive-grey with some small lighter spots; chin and throat variegated with yellow." He further remarks it is strikingly like a harmless colubrine in external appearance.

* Fauna of Brit. Ind., *Reptilia* and *Batrachia*, p. 419.

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Snake Bite and Snake Poisoning.

In reading the reports of snake casualties, which appear from time to time in various publications, I have been forcibly and repeatedly struck with the very incomplete way in which many of these cases are recorded, and also by the fact that in many cases the diagnosis of snake-poisoning (*ophitoxæmia*) appears to have been quite unjustified. Frequently one or two of the most obvious symptoms in a case are mentioned—not necessarily symptoms of *ophitoxæmia* at all—to the exclusion of many others which though less obvious are perhaps of greater importance in establishing a diagnosis. It appears to me that the term “snake-bite” is often used as synonymous with “snake-poisoning,” and the mere fact that a man has been bitten by a snake, or is reported as having been bitten by a snake, has been the only justification for considering and recording the case as one of snake-poisoning. Many cases appear to be recorded as snake-poisoning which should have been returned wound punctured, or wound lacerated.

Now in cases of snake-bite, whether the wounds are inflicted by a harmless or a poisonous species, a certain train of symptoms follow which are the direct result of fright, and kindred emotions, pain, etc. Some of these are so serious that they end fatally, but whether fatal or not a great many of these cases are wrongly diagnosed, the symptoms due to fright being misinterpreted as the result of snake-poison.

Complicating Effects of Fright.

The gravity of symptoms due to fright does not appear to me to be sufficiently recognised, though there is no doubt in my mind that fatal cases from this cause are abundant, especially among the timid natives of this country.

To take examples, Fayrer records the case of a man who was bitten by a slow loris (*Nycticebus tardigradus*), a perfectly harmless little creature of the order Primates. Natives believe that the bite of this animal is fatal and this man sharing the conviction of his race, became alarmed, and within five minutes was in a senseless state necessitating 5 or 6 hours of vigorous stimulating measures to restore him. Mr. M. H. Oakes has written to me of a fatality from the bite of a “bis cobra,” one of the monitor lizards (probably

Varanus bengalensis). The subject was a woman of 50 who was bitten on the toe, and she died in $1\frac{1}{2}$ hours. I believe there is not the slightest doubt that this lizard is completely innocuous. Mr. E. E. Green remarks that year by year in Ceylon the Registrar General's annual mortality report returns one or more cases of death from the bite of the brahminy lizard (*Mabuia carinata*), a little skink which is perfectly harmless, though the natives think otherwise.

A man came under my care in Rangoon in an unconscious state having been bitten by a harmless water snake (*Tropidonotus piscator*), and was 17 hours unconscious in spite of vigorous stimulating remedies. Dr. Willey reported the death of a woman in Ceylon from the bite of a common wolf-snake (*Lycodon aulicus*), a perfectly harmless species, and Dr. Ewart met with serious symptoms after the bite of the same snake, all of which were due to fright.

The same species was responsible for another instructive case reported by me in the Bombay Natural History Journal (Vol. XX, page 521).

A cooly woman in Chanda (C. P.), aged 22, was bitten by a snake in two places on the middle of the middle right finger. Twenty-five minutes later she was brought to a dispensary, where the subordinate in charge believing the snake which had been killed and accompanied her, was a krait, applied a ligature, freely incised the wounds, rubbed in crystals of permanganate of potash, and injected two doses of antivenene 30 c.c. each. She complained of thirst, burning pain in the hand and arm which later became numb and vomited twice. Half an hour later, she was comatose with respirations 30 per minute, and a weak pulse of 120, which later became imperceptible, and swallowing was reported as impossible. After lying unconscious for $5\frac{1}{4}$ hours, with brief intervals of consciousness, she revived, asked for water, drank, and then slept soundly to awake next day quite well, except for burning pain and numbness in the hand and arm.

Fortunately the snake was killed, and our correspondent had it placed in a bottle, and sent it to the Honorary Secretary of the Bombay Natural History Society when it proved on examination to be the harmless Wolf snake (*Lycodon aulicus*). The notes of the case were supplied by the Hospital Assistant who attended the case.

The symptoms—vomiting, collapse with weak pulse, and incapability to swallow (not a genuine paralysis)—are all to be attributed to an attack of syncope, in the main due to fright, but probably aggravated by the pain occasioned by the surgical wounds, and the burning of the permanganate.

Had the snake not been killed, this casualty like so many others would probably have been reported as “another case of snake-poisoning cured by antivenene or permanganate of potash.”

Now it appears to me that quite a large number of cases are reported each year as snake poisoning which have never shown a symptom of toxæmia, but which are comparable to the cases quoted above, the gravity of the symptoms being wrongly interpreted as due to the action of snake venom.

I think the conditions to be met with in the two states, *i.e.*, fright and colubrine poisoning require emphasizing, especially as they are in almost every detail strikingly different, and as a result call for completely different methods of treatment.

To begin with fright operating through the nervous system mainly affects the heart. The symptoms may vary from a transient pallor and giddiness, to syncope of so profound a nature that unless combative measures are speedily employed the condition may pass insensibly on to death.

Now if we take the cobra as the type of a colubrine snake the toxæmia produced by its bite exerts its main force upon the nervous system, and principally operates upon the respiratory centre, the heart remaining unaffected. The constitutional effects seen in the two cases are as follows :—

Fright.

- (1) Onset of weakness often sudden.
- (2) Involuntary prostration; often the patient falls in a faint and is brought in this state to hospital.
- (3) Complete or semi-unconsciousness.
- (4) Syncope—
 - (a) Pallid face,

Cobra-poisoning.

- (1) Onset of weakness very gradual.
- (2) Recumbency voluntary after some time owing to gradual loss of power of legs. The patient often walks to hospital by himself or with help.
- (3) Consciousness not impaired.
- (4) Heart not affected—
 - (a) Face natural at first, livid later on,

Fright.

- (b) Cold clammy skin,
 (c) Feeble or imperceptible pulse.

- (5) Breathing shallow, sighing and weak, and unduly frequent (30 a minute or more).
 (6) No paralyses.

Cobra-poisoning.

- (b) Skin warm,
 (c) Pulse of normal force and regularity.

- (5) Breathing gradually becomes more and more laboured, and quickened; gasping towards close.
 (6) Paralyses. Gradual weakness of legs mounting upwards to trunk and head. The head droops. The eyelids droop. Swallowing becomes difficult, the lower lip falls away from the teeth, and saliva dribbles from the mouth. Articulation too becomes difficult.
 (7) Death from cardiac depression. (7) Death from respiratory depression.

The symptoms of fright often very speedily declare themselves, far more speedily than is ever the case in snake poisoning. In some reported cases we read that the patient is seen or is brought to hospital in a senseless or nearly senseless condition, it may be a few minutes after the bite, and as one reads the record it seems that this unconscious state has been interpreted as the outcome of absorption of venom, and remedial measures have been at once taken on this supposition.

The incomplete and unsatisfactory fashion in which many cases of snake-bite are reported, makes one feel that much valuable information relating to the clinical manifestations of the various venoms is being lost to science each year, and with regard to many of these ophitoxins we know absolutely nothing. I would propose that every case of snake-bite should be returned on a prescribed form similar to those now in use for recording cases of cancer and enteric fever. If all cases were so returned a greater uniformity and value in the records would be forthcoming, one would be able to judge the constancy of the signs both local and general which accompany the various forms of poisoning, any differences in the clinical manifestations occasioned by the various venoms would

become apparent, one would be able to discriminate between genuine and spurious cases of ophitoxæmia, and possibly form some opinion as to the number of fatalities in India, reported as snake bite, which are due to poisonous bites as compared with non-poisonous. I would propose a form somewhat as follows :—

Casualty return of snake-bite.

Station.
Sex.
Age.
Date and hour of bite.
Hour of admission
Part bitten.....
Species of snake.....
Result.....	If fatal, method of death, syncope or asphyxiaTime elapsed since bite
Symptoms—Local—	
(a) Pain
(b) Swelling
(c) Sanious oozing
(d) Appearance of tissues when cut into
(e) Characters due to mechanical causes
General—	
(a) Consciousness
(b) Respiration
(c) Syncope, Pallor—
Warmth and activity of skin.....
Pulse.....
Vomiting
Onset of weakness
(d) Paralyzes. Onset of weakness
Sequence
Drooping head.....
Drooping eyelids.....
Articulation

Phonation
Deglutition
Salivation
(e) Hæmorrhages
(f) Other symptoms
Treatment.	

In addition to this I would suggest that in every case where a snake is produced it should be sent to the Parel Laboratory, the Bombay Natural History Society or the Indian museum, for competent and confirmatory identification, no matter whether believed to be harmless or poisonous.

From the foregoing remarks it is obvious that the symptoms of snake-poisoning must be thoroughly understood before rational treatment can be carried out. First of all it is most necessary to determine whether a case is one of snake bite or snake poisoning. The symptoms due to fright have been sufficiently detailed in the comparison of this condition with that of cobra-poisoning.

Another train of symptoms is likely to be met with in snake bitten subjects of a hysterical type which may complicate the case for the diagnostician. I have on more than one occasion witnessed such. It is not uncommon for a reputed snake bitten subject to be brought to hospital in an apparently unconscious state. Such acts as vacantly staring with open eyes and twitching or tightly closing the eyes, or partially closing them so that the patient may see what is going on around him, without appearing to do so to the anxious relatives, rolling the eyeballs, puffing out the cheeks, twitching the lips, gnashing the teeth, and lying prostrate with rigid limbs may deceive those who do not realise these are exaggerated acts, not paralytic ones.

These symptoms are of great importance in diagnosis, and treatment, and should always influence the inductions to be drawn in a case of recovery from supposed snake bite.

In this connection it is most important that the following facts should be realised :—

- (1) *That it is quite possible to be bitten by a poisonous snake without being poisoned.*

- (2) *That in the case of so fatal a snake as the cobra, it is quite possible to be poisoned, but to receive a sublethal dose.* (Lamb says that 30 per cent. of cobra poisoned subjects would not die for this reason.)
- (3) *It is possible for a person to die from the bite of a harmless snake, death being brought about by the depressing influence of fear or anxiety on the heart. Such cases have been already cited.*

Snake Poisons.

I propose first to state briefly the main effects of snake poisoning, explain the mode of action, and where possible illustrate my remarks by quoting well reported cases in medical literature.

Before doing so perhaps I should say that in the present recognised system of classification, the snakes of the world are divided into 9 families. All the known poisonous species belong to one or other of two of these families, *viz.*, the *Colubridæ*, and the *Viperidæ*. Many Colubrines are harmless, but all Vipers are poisonous, and among our Indian poisonous snakes we have several representatives of both families.

Now snake venoms of the Colubrine class differ but slightly among themselves in their composition, and main effects upon animals, and the same remark applies to the Viperine class. The symptoms evoked by the Colubrine class taken collectively differ considerably from those produced by the Viperine.

Colubrine poisons act chiefly on the central nervous system (cord and brain) and cause death by paralysing the respiratory centre in the brain. Their effects upon the blood are slight compared with the Viperine class, so that hæmorrhages are not usual, and when present are not severe.

Viperine poisons have no paralysing effect upon the nervous system, except on the vaso motor centre, but a very marked effect on the heart and blood, death being usually brought about by paralysis of the vaso motor centre, exhaustion from profuse and persistent bleeding, or from septicæmia (a blood poisoning due to germs).

THE COBRA.

TOXINS OF COBRA VENOM.*

Chemical formula $C_{34} H_{20} O_{52}$ (Faust).

- (1) Toxins operating on nerve cells.—
 - (a) A depressor that paralyses the respiratory centre. (Brunton and Fayrer.)
 - (b) A depressor to the vasomotor centre. (Lamb.)
 - (c) A depressor that paralyses centres in the bulb. (A. J. Wall.)
 - (d) A depressor that paralyses the ends of nerves including the phrenic nerves. (Brunton and Fayrer.)
 - (e) A destructive agent to cells generally in the spine and cord. (Lamb and Hunter.)
- (2) A direct stimulant to cardiac muscle. (Brunton and Fayrer, Fraser and Elliot.)
- (3) A vaso constrictor. (Brunton and Fayrer.)
- (4) Toxins affecting the constitution of the blood.—
 - (a) An antifibrin ferment that reduces clotting of blood. (Brunton and Fayrer.)
 - (b) "Hæmolysin" destructive to red blood cells. (Hilson, Ragotzi.)
 - (c) "Leucolysin" destructive to white blood cells. (Flexner and Noguchi.)
 - (d) An antibactericidal principle that destroys the normal activity that blood exhibits towards the invasion of germs into the body. (Flexner and Noguchi.)
- (5) Toxins destructive to other cell elements.—
 - (a) "Hæmorrhagin" destructive to the lining membrane of arterioles. (Flexner.)
 - (b) Agents destructive to cells at the seat of injury.

* Because many of these elements have received special names according to their specific action it must not be inferred—in this or in other venoms—that each is a separate toxic entity. On the contrary it is probable that the element in cobra venom that operates on the respiratory centre (neurotoxin) is also the destructive agent to the red-blood cells, in which case the name "hæmolysin" should be suppressed. Possibly other elements have a dual or even a triple action.

ANALYSIS OF THE ACTION OF COBRA TOXINS.

- (1) (a) The "neurotoxin" paralysing the respiratory centre is the predominant agent in causing death. It is assisted by 1 (*d*), and slightly by 4 (*b*).
 - (b) The effects of the "toxin" that depresses the vaso-motor centre, are neutralised by the combined antagonistic action of 2 and 3. The result is that after breathing has ceased, artificial respiration is capable of prolonging life for many hours.
 - (c) This "toxin" operating on several centres in the bulb evokes symptoms exactly similar to those seen in bulbar palsy where lips, tongue, throat and voice are paralysed.
 - (d) Assists 1 (*a*) in the production of asphyxia.
 - (e) The toxin "amyotrophin" destroys nerve cells generally, but its action is not seen in the human being, death being brought about by the more speedy action of 1 (*a*). When 1 (*a*) is insufficient to cause death, the dose of "amyotrophin" is too small to produce death, or even to seriously affect the bitten subject.
- (2) The cardiotoxic element by reinforcing the heart muscle helps to preserve the blood pressure, and helps to counteract the effect of 1 (*b*).
- (3) The vaso constrictor by reducing the calibre of the blood vessels raises blood pressure, and acts indirectly in reinforcing the heart. With 2 it completely counteracts the effect of 1 (*b*).
- (4) (a) The effect of the antifibrin ferment is seen in the reduction of clotting power in the blood, and this in conjunction with 5 (*a*) accounts for the hæmorrhages that may occur.
 - (b) "Hæmolysin" by destroying red blood cells contributes to the asphyxia produced by 1 (*a*) aided by 1 (*d*).

- (c) "Leucolysin" by destroying white blood cells reduces the normal power of the system to resist microbic invasion of all sorts.
- (d) This aids the action of 4 (c) if indeed it is a separate entity.
- (5) (a) "Hæmorrhagin" by damaging the lining membrane of the arterioles allows leakage of the vessel contents. It in conjunction with 4 (a) favours the occurrence of hæmorrhages.
- (b) The local destruction of cells accounts for sloughing of the wounds, and the formation of an ulcer when the dead tissues have separated.

Compared with the venom of other snakes, cobra venom ranks third in virulence among our Indian Snakes. The sea snake *Enhydrina valakadya* stands first and the krait, *Bungarus cœruleus*, a good second. Lamb's estimates based on injections into rabbits show that cobra venom is one-seventh as virulent as the former, and one-fourth to one-fifth the latter. Rogers experimenting on pigeons puts cobra venom at one-tenth the former, and half the latter.

Cunningham computed the capacity of the glands at ten lethal doses for man, but Lamb shows that under exceptional circumstances a single cobra may contain twenty lethal doses in its glands.

It seems to me that in Colubrine poisons two toxic elements are associated in the murderous assault upon the nervous system, the one precipitate by nature, the other dilatory. This appears to be the interpretation of the facts disclosed to us by those who have worked experimentally in the field of ophitoxicology. It would seem that the more hasty associate (neurotoxin) has no destructive action upon nerve cells (?). How else can one explain the completeness, and rapidity with which a cobra bitten subject recovers when the dose is sublethal? In waning the symptoms in such cases disappear about, if not quite, as rapidly as they appear when waxing. Lamb and Hunter state that in monkeys that succumbed to an injection of cobra venom before two hours had elapsed, the nerve cells showed no degenerative changes. If death was delayed

beyond this time degenerative changes were produced. When once degenerative changes have occurred can recovery be so complete and rapid? It is inconceivable to my mind that nerve cells that show marked degeneration such as is recorded by Lamb and Hunter in monkeys that died from cobra, and *cæruleus* poison can regain their normal structure and function in a few hours. To allow that this is possible is to concede to nerve cells structurally injured by snake poison, a power of regeneration never seen in nerve cells structurally damaged by any other morbid processes, and greatly in excess of that shown by any other body cells that have suffered structurally, which under the most favourable circumstances require three or four days to regain their normal tone and functions.

The fact however that a degeneration of nerve cells does undoubtedly occur after lethal doses of cobra and *cæruleus* poisoning appears to me to be the result of the other member of the partnership (for which I propose the name "amyotrophin"). It has been shown by Lamb and Hunter that in the chronic form of poisoning from *Bungarus fasciatus*, very extensive degeneration of the nerve cells occurs in the brain and cord, and that a peculiar form of fatal illness is provoked which is not seen after any other form of snake poisoning which has yet been investigated. It appears to me that cobra and *cæruleus* venoms contain both the toxic principles "neurotoxin" and "amyotrophin," but that the "neurotoxin" is the predominant associate producing death very often before the more lazy, and in these poisons weaker associate "amyotrophin" has had time to make itself felt. It is probable too that the "amyotrophin" present in a sub-lethal dose is too small in quantity to work much, if any destruction, on the nervous elements, so that when the effects of the "neurotoxin" wane, recovery is complete and rapid.

In the case of the banded krait (*B. fasciatus*) the position seems reversed, and "amyotrophin" is the predominant partner. Hence, although early symptoms attributable to "neurotoxin" are evoked, the bitten subject may recover from these entirely, only to acquire a new train of symptoms later on, the result of "amyotrophin," which, though more dilatory in its attack, may prove more inexorable and cause death.

COBRA TOXÆMIA.

Symptoms of Cobra Poisoning.

In a subject poisoned by a cobra, which we may take as the type of Colubrine toxæmia, the earliest constitutional symptom is a feeling of intoxication, but this frequently passes unnoticed in an unobservant subject. Later the patient feels his weakness (paralysis) insidiously creeping upon him, till, uncertain of maintaining the upright posture, he voluntarily reclines. His paralysis begins in his legs, mounts to the trunk, and finally affects the head which droops. Synchronising with this drooping of the head, a drooping of the eyelids may be noticed, and simultaneously the muscles of the lips, the tongue and throat become gradually paralysed. As a result the lower lip falls away from the teeth, allowing the saliva to dribble from the mouth, speech becomes increasingly difficult, till the subject, unable any longer to control his lingual and labial muscles, attempts by signs to communicate to those around him, often striving with his fingers to remove the viscid saliva that clings to his mouth. Breathing soon becomes embarrassed, later laboured, and finally impossible. Distress is written on the countenance, which becomes increasingly livid from defective aeration of the blood. Swallowing similarly becomes difficult, and later impossible, so that fluids taken into the mouth are apt to regurgitate through the nose. Nausea and vomiting are frequent symptoms. A convulsion often heralds the cessation of respiration, but the heart goes on beating for a minute or two longer. Consciousness is retained till the end. There appears to be no special sequence in the development of these paralyses. Those affecting the muscles of the lips, tongue, voice, throat develop synchronously, and evoke a train of symptoms exactly comparable to the organic nerve disease "bulbar palsy," as first pointed out by that excellent observer A. J. Wall. Such are the effects produced by the paralysing influence of the poison on the cord and brain, which may cause death in from 1½ to 6 hours usually. (Nicholson says after 12, or even 24 hours).

Symptoms arising from the action of another toxin, viz., hæmorrhagin on the blood may be present, but a discussion of

these will come more appropriately when dealing with Viperine poisoning (*see* page 103).

Local effects are always more or less in evidence, but these again being much more pronounced in Viperine poisoning are better considered under that head (*see* page 103).

The action of cobra venom can be easily remembered from the word COBRA. CO stands for COOrd, and BRA for BRAin, showing that it is the central nervous system that is mainly affected. Again, COBR stand for Clotting Of Blood Reduced, and A gives us the mode of death in the word Asphyxia from paralysis of the respiratory centre.

Illustrative Cases.

No. 1.

Cobra bite. Toxæmia. Death in 65 minutes. Autopsy.

Reported by Dr. Hilson (*Indian Medical Gazette*, October 1873):—

“ On a night in June, at about half-past 12 o'clock, Dabee, a Hindu punkah coolie, was bitten on the shoulder by a cobra, whilst sleeping. On inspecting the wound, there were found over the prominence of the right deltoid muscle, and about three-quarters of an inch apart, two large drops of a clear serous-like fluid tinged with blood, which had apparently oozed from two small punctures, so minute that they could not be perceived by the naked eye. A burning pain was complained of in the neighbourhood of the bite, which rapidly increased in intensity, and extended so as to affect a circular portion of the integument of the size of an ordinary saucer; and, judging from the description given of it, it was very similar in character to that produced by the sting of a scorpion.

“ At 12-45 A.M., or about a quarter of an hour after being bitten, he complained of a pain in his shoulder shooting towards his throat and chest, and said he was beginning to feel intoxicated; but there was nothing in his appearance at this time to indicate that he was in any way under the influence of the poison. On the contrary, he was quite calm and collected and answered all questions intelligently, at the same time that he was fully alive to the danger of his

condition. The pupils were not dilated, and they contracted when exposed to the light of a candle; his pulse was normal, and there was no embarrassment of the respiration. About five minutes after he began to lose control over the muscles of his legs, and staggered when left unsupported. At about 1 o'clock, the paralysis of the legs having increased, the lower jaw began to fall, and frothy and viscid saliva to ooze from the mouth. He also spoke indistinctly like a man under the influence of liquor. At 1-10 A. M. he began to moan, and shake his head frequently from side to side. The pulse was now somewhat accelerated, but was beating regularly. The respirations were also increased in frequency. He was unable to answer questions, but appeared to be quite conscious. His arms did not seem to be paralysed. At 1-15 A. M., twenty-five minims of liquor ammoniæ were rapidly injected under the skin of the forearm; but as this produced no results, the basilic vein was laid bare, and twenty-five minims injected into it.

“The operation caused no amelioration in the symptoms, and the condition was evidently becoming critical. He continued to moan and shake his head from side to side, as if trying to get rid of viscid mucus in his throat. The respirations were laboured, but not stertorous. The external jugular vein of the left side was next exposed, and twenty-five minims of the liquor ammonia injected into it; but without producing any good effect. The breathing gradually became slower, and finally ceased at 1-44 A. M., while the heart continued to beat for about one minute longer. No convulsions preceded death, which took place in one hour and five minutes after the infliction of the bite.”

Post-mortem examination at 6 A. M., or five hours after death.

Rigor mortis well marked; countenance placid. Nothing abnormal could be noticed externally, except a slight tumefaction of the bitten shoulder. The apertures formed by the fangs of the snake could not be seen with the naked eye, but on removing the skin extensive ecchymosis of the cellular tissue was disclosed around the bitten part. The blood was everywhere fluid and of a peculiar claret-like colour.

The large thoracic and abdominal veins were gorged, and all the cavities of the heart were distended with the fluid blood.

Both lungs were much congested, and on making a section, blood flowed freely from them.

The liver, spleen, and kidneys were of a deeper colour than usual, but otherwise they were healthy.

The membranes of the brain were much congested, but only a small quantity of serum was found either external to that organ or in its ventricles. The brain substance was nowhere softened or diseased, but a section displayed numerous *puncta cruenta*.

The *post-mortem* appearances, in short, were identical with those seen in the lower animals after cobra bite, except that the blood did not coagulate on exposure to the air. On examining this fluid with a microscope, magnifying 500 diameters, I was unable to observe any of the peculiar cell formations which are said by Professor Halford to be discernible in it after death from snake-bite. The white cells were not increased in numbers, while the red corpuscles were, to a great extent, broken up and had coalesced, so far as to form bright red amorphous masses. Many of them, however, had undergone no change.

No. 2.

Cobra bite. Severe toxæmia. Recovery.

Reported by Dr. Vincent Richards and quoted by A. J. Wall-
(Indian Snake Poisons, p. 45.)

A man named Bamon Das, aged forty years, was bitten by a snake on the shoulder about 3 o'clock in the morning. From his description it was probably the snake termed by the natives of Bengal the "Teutuliah Karis" (a spectacled cobra) about four feet long. He had complained, after the bite, of feeling intoxicated, had vomited, and could neither stand nor speak though he had continued to be perfectly conscious. At 10 A.M., when Dr. Richards saw him, he was being supported in the sitting posture by two men. Near the posterior border of the deltoid of the left arm were two rather indistinct fang-marks at some considerable distance from each other; one fang-mark, however, more resembled a scratch than a puncture. The arm was painful, hot,

and swollen, measuring eleven inches in circumference, whereas the other arm at a similar part measured only nine and a half. On cutting through the punctures the track of the fang was clearly visible though the staining of the areolar tissue was very slight indeed. He had no power whatever over the eye-lids, which had dropped, leaving only the lower parts of the pupils visible. The pupils were perfectly natural and the irides responded. When asked to identify people he pushed his head back so as to bring the person into the line of vision. He could, in fact, see perfectly well. The hearing was not affected. There was profuse salivation, the saliva streaming down from the corner of his mouth. The lower lip had dropped. He could speak but very indistinctly, so indistinctly that his friends had to ask him to repeat what he said. The intonation was peculiarly nasal, much resembling that of persons who have lost part of their palate. The lips were not used in his endeavour to pronounce his name, consequently the labial "b" was omitted, and he answered with a very indistinct and nasal "Aon Das." On attempting to swallow some water it was returned through the nostrils. He was unable to clear his throat, which caused him some distress. He felt some difficulty in breathing, though the respiration was but slightly embarrassed. The superficial temporal and frontal veins were very distinct and tortuous, being gorged with blood. He could not walk himself, but if supported walked with an unsteady gait, though he had perfect control over his upper extremities. Still felt intoxicated and his body hot. Pulse 96, full and strong. Temperature 100.5 F. Occasional retching.

11-30 A.M.—Slightly better; still feels intoxicated; temperature 101.1 F.

1 P.M.—Temperature 101.5 F.

2-30 P.M.—Arm a little more swollen—now measures eleven inches and a half; has passed a large quantity of urine.

Feels a little sick, and the veins about the face are still rather gorged. Now speaks distinctly, and can swallow. Suffering rather severely from the pain in the arm.

From this time the arm got gradually better, and the man completely recovered.

[*Remarks.*—In this case we have an example of severe cobra-poisoning, well described, in which the nerve symptoms were fully developed and yet, when they had passed away, the man was at once in a state of thorough and complete recovery.—F. W.]

No. 3.

Cobra bite. Toxæmia. Recovery due to antivenene injections.

Reported by Captain Lamb, I.M.S., in the "Bombay Natural History Journal" (Vol. XIV, p. 233). The "officer" referred to was Captain Lamb himself, and the note is on this account specially valuable.

An officer of the laboratory, while assisting in extracting the poison from a full-sized cobra, put his fingers where he had no business to, that is, in the neighbourhood of the snake's mouth. In a moment the animal had buried one of its fangs in the point of the right thumb. The thumb was at once withdrawn, but not before the total amount of poison in the gland had been injected. The symptoms, both objective and subjective, &c., which followed, were carefully noted as they occurred. Locally there was much pain at the site of the injection. Swelling of the parts soon began and gradually became well marked. A bloody serum oozed out from the puncture and continued to do so for 24 hours.

Fortunately for the experiment no fresh serum was available, and we had to inject two bottles of a serum which had been the property of the Society and which was at least four years old. Just the week previous to the accident I had tested this serum with cobra venom on rats and had found that it had little or no neutralising power. The patient then went on with his work. About three hours after the bite he began to get lethargic and lazy, did not wish to work and preferred to lie down. This was soon followed by sickness and violent vomiting. Then he noticed that his legs were weak, he was unable to move about and had perforce to adopt the prone position. It appeared then that the

serum had had little or no effect and that the case was hopeless. Just at this time, however, some fresh serum arrived. Ten cubic centimeters were at once injected and the symptoms watched. In about an half an hour the paresis of the legs showed signs of improvement. A short time later our patient was able to walk away and drive to the club. Locally, the pain and swelling continued for some time. A small slough formed. This, on separating, left a deep hole which gradually healed up. A depressed scar is now the only sign of the accident remaining.

No. 4.

Cobra bite. Severe toxæmia. Recovery without treatment.

Quoted by Calmette (*Venoms, Venomous animals. &c.*, p. 370).

A snake-charmer, Kingilien by name, aged 25, was bitten in the first phalanx of the right forefinger, when taking hold of a cobra in the courtyard of the Pondicherry Hospital. Refusing an injection of antivenomous serum, the man ran off as fast as he could go, after having a simple ligature applied to his wrist. Scarcely had he reached his dwelling, when he fell into a deep coma, in which condition he was carried to Cottacoupom, to the abode of one Souraire Kramani, a kind of sorcerer, who administered to him a certain medicament in a betel leaf. After having vomited a large quantity of bile he was taken home. At this time, according to the summary investigation that we caused to be made, the patient was unable to utter a single word; he could only open his mouth with difficulty, and his eyelids remained closed. Kingilien, who had partially regained consciousness, seemed to be suffering from continuous attacks of vertigo; his head, if pushed to one side, drooped, and the man was incapable of voluntary movement. Respiration was fairly easy, swallowing painful. The entire hand was greatly swollen; poultices of leaves were applied to it, after a few incisions had been made with a knife in the back of the hand, in order to reduce the congestion. The arm was rubbed from above downwards with the very bitter leaves of the Vembou, or mango-tree and prayers were recited. This is all the information that I have been able to obtain with reference to this man, who, after a prolonged convalescence, is said to have recovered.

No. 5.

Cobra bite. Some local effects. No toxæmia.

Reported by Dr. Nicholson (Indian Snakes, p. 160).

Two pariahs, who used to bring snakes, got drunk one Sunday and were bitten whilst playing with the snakes they were keeping to bring me on the morrow. They came to me in great fright. One had two lacerated fang marks on a finger, his hand also being swollen; the other was slightly scratched on the leg. The former had fastened a string round the finger above the wound; the latter had done nothing, the scratch being trifling. The seriously wounded man wanted medicine, as the wounds were inflicted about a quarter of an hour before. I did not see much use in interference, and as the man had a good quantity of arrack inside of him I contented him by means of a draught of water coloured pink with dentifrice lotion, and they soon took their departure without any constitutional symptoms appearing. The man's hand was swollen when I saw him next day. The cobra by which these men had been bitten, and which they brought with them, was in perfect condition.

In these cases the cobras had evidently bitten without injecting poison; I have no doubt that this happens frequently and that many of the authentic recoveries ascribed to antidotes are really due to the want of malice on a part of the snake. Had I been an antidote enthusiast, I might have made some nice cases of cure out of these accidents.

THE HAMADRYAD.

THE TOXINS OF HAMADRYAD VENOM.

- (1) Toxins operating on nerve cells—
 - (a) A depressor that finally paralyses the respiratory centre. (Rogers.)
 - (b) A depressor that paralyses the ends of the phrenic nerves. (Rogers.)
- (2) A cardiotoxic agent stimulating heart muscle. (Lamb.)

- (3) A vaso constrictor that increases blood pressure, and further stimulates the heart. (Lamb).
- (4) Agents affecting the constitution of the blood—
- (a) An anti-clotting ferment. (Lamb.)
 - (b) Hæmolysin, that destroys red blood cells but feeble in action. (Rogers.)
- (5) Haemorrhagin? A principle damaging the lining membrane of blood vessels is probably present.

ANALYSIS OF THE ACTION OF HAMADRYAD TOXINS.

These are sufficiently detailed under the subject of Cobra venom.

Lamb by experiment on rabbits found hamadryad venom as virulent as cobra venom. Rogers by experiment on pigeons found the virulence rather less than cobra venom. Rogers estimated with some doubt that about ten lethal doses (for man?) could be discharged at one bite.

Being a much larger snake than the cobra, the mortality from its bite is almost certainly much higher than in that species.

HAMADRYAD TOXÆMIA.

Although there appear to be no records of hamadryad poisoning in the human subject in which a fully detailed account of the symptoms are given, we may infer a great deal from the researches of Rogers on the lower animals.

The points to be noted are that the venom acts almost exactly like cobra venom, producing death by paralysing the respiratory centre in the brain, which action is further augmented by a paralysis of the terminations of the phrenic nerves (the nerves that contract the diaphragm, one of the most important muscles of respiration). Its effects on the blood are but slight. From these considerations, we may expect to find a bitten subject present almost the same picture as in the case of the cobra, but bloody discharges are still less likely to be met with. Local signs will be present in some marked degree (*see* page 103).

Theobald (Cat. Rept. Brit. Burma, 1868, p. 61) saw a snake charmer bitten by one in Burmah die within a few minutes. Evans (Bombay, Nat. Hist. Journl., Vol. XIV, p. 413) mentions a case of a foolhardy Burman, believing himself snake poison proof, teasing one belonging to a Shan snake charmer, he was bitten in the hand, and soon afterwards died. The same observer also records another instance of a Burman being bitten by one in the base of the index finger, with the result that he died shortly afterwards. He also furnishes another case in which this time the victim was a bullock, which was bitten by a hamadryad, which the bullock cart passed over. The animal died soon afterwards.

Raby Noble (Bombay. Nat. Hist. Journl., Vol. XV, p. 358) mentions one 10 feet 1 inch in length (identified by Mr. Phipson) making an unprovoked assault on a cooly woman in Assam, seizing her by the leg, and maintaining its hold for at least 8 minutes, when it was beaten off. She was treated by a "Doctor Babu" (treatment not specified), but succumbed in about 20 minutes. The symptoms were local pain and swelling, vomiting, laboured breathing and prostration. Theobald (Cat. Rept. Brit. Burma, 1868, p. 61) records, on the information of a Burman, an elephant being bitten on the trunk by a hamadryad whilst browsing on some foliage, with the result that death ensued in about three hours.

It is interesting to note that Dr. Nicholson reported a case where a Burman snake-catcher was bitten by a ten-foot specimen in good condition. He chewed some vegetable pulp and applied it to the wound, and "was none the worse for the bite."

THE COMMON KRAIT (*BUNGARUS CÆRULEUS*).

THE TOXINS OF CÆRULEUS VENOM.

These are in the main identical with those enumerated under cobra venom.

Lamb by experiment on rabbits estimated the virulence of the venom as four to five times greater than that of cobra venom. Rogers operating on pigeons fixes the virulence as twice that of cobra venom. With some doubt the latter estimated that an adult is capable of injecting three lethal doses (for man?) at a bite.

CÆRULEUS TOXÆMIA.

The action of the venom as tested by experiment by both Lamb and Rogers is found to be almost identical with cobra poison. The respiratory centre, and the ends of the phrenic nerves are paralysed, and death supervenes from asphyxia. On the blood it has no action in reducing coagulability, but the red blood cells are destroyed as in other Colubrine poisons. The local effects are sometimes marked; on the other hand these have been so trivial in some reported cases that none could be discovered. Pain at first absent has sometimes later become a prominent symptom.

From the above one would expect to find the subject of Krait poisoning, suffering in almost the same manner as in cobra poisoning. One peculiarity, however, is very frequently reported, *i.e.*, violent abdominal pain.

The fact that Elliot found submucous hæmorrhagic spots in the stomach and intestine of all the monkeys he *post-mortemed* who died from experiment with this venom, taken in conjunction with the abdominal pain so frequently noted in the human subject strongly suggests internal hæmorrhage. As shown when dealing with *Echis* toxæmia internal hæmorrhages are by no means uncommon, and it may be that "hæmorrhagin" causes internal bleeding in this toxæmia in the human subject. In case No. 3 indeed definite hæmorrhages are reported, though the toxæmia appeared slight

Illustrative Cases.

No. 1.

Bite from krait over 3 feet long. Toxæmia. Death in 12 hours.

Reported by Assistant Surgeon Jadul Kristo Sen. (*Indian Medical Gazette*, February, 1874.)

Thacoorprasad, Hindoo, male, age about 60 years, was bitten by a snake (krait) on the left index finger, at about 9 o'clock on the night of the 15th July, while he was sleeping in a room in the Bulrampore Maharajah's cooty.

He was admitted into hospital at 5 o'clock next morning, with the following symptoms:—Giddiness; drowsiness; incoherence of speech; difficulty of breathing, and a choking sensation in the throat. Pulse 98; temperature normal; conjunctivæ congested, pupils dilated, but acted on by light. Had had no stools since he was bitten, but passed urine several times. The left hand was livid, swollen, and painful, and its motion was much impaired, but not completely lost; could not walk or sit up unsupported.

On washing off the paste of native medicines with which the finger was covered, two fang-marks, about $\frac{1}{3}$ of an inch apart from each other, were observed on the dorsal aspect of the finger, about half an inch from its root.

There being no potent remedy known to meet the exigency of a case of this nature, Dr. Halford's method was adopted. Immediately after admission, liquor ammoniæ (mxxx diluted with mxx of water), was injected in the left basilic vein, and a similar dose with camphor water was given by the mouth: no effects.

6. A.M.—Parotids swollen; complained of severe shooting pain in the left thigh; vomited once; the vomiting consisted of tenacious mucus of a greenish tinge. Liquor ammoniæ mxxx undiluted was injected into the right basilic vein: no effects.

6-30. A.M.—Distressing nausea; vomited three times; voice became very low; breathing very much oppressed. Became very restless; complained of smarting pain in the left hand. Liquor ammoniæ mxxx, diluted with an equal quantity of water, was injected again into the left basilic vein, through the former puncture: no effects.

7. A.M.—Could not swallow medicines; could not speak; eyes drooped; constantly putting the right hand into the mouth. Spasmodic twitchings commenced in the muscles of the legs. Pupils acted on by light. Pulse fair. Ordered, Ammon. carb. gr. x., rum oz. i., as an enema, every half an hour.

7-15 A.M.—Vomited once; no stool and urine. At 7-14 Dr. Heffernan saw the patient. He injected liquor ammoniæ mxxx,

diluted with mxxx of water into the left saphenous vein : no effects.

8 A.M.—Rattling noise in the throat ; respiration difficult ; passed urine in his clothes. Liquor ammoniaë, of the same dilution, was injected into the right saphenous vein : no effects.

8-30 A.M.—Breathing slow and noisy. Head turned on the left side ; viscid saliva dribbling from the mouth ; pulse fair ; extremities cold. Injection was repeated into the left saphenous vein : no effects.

9 A.M.—Died in convulsions, in the presence of Dr. Heffernan, about 12 hours after the infliction of the bite.

Post-mortem was not allowed.

The snake which had bitten the man was caught on the spot, and brought to the dispensary alive. It was a vigorous krait upwards of three feet in length.

No. 2.

Bite from krait, one and-a-half feet long. Toxæmia. Death in $5\frac{1}{2}$ hours.

I am indebted to Colonel F. W. Dawson for the following :— A keeper in the Trivandrum Museum was bitten on the right index finger by a small krait, one and-a-half feet long, at about 1-30. P.M., 13th August 1907. The bite felt like a pinprick, there was no bleeding, and indeed no mark whatever of a puncture. He went home, having declined all persuasions to go to hospital, and apparently stayed in his house till about 3 P.M., when he began to feel a burning pain in the bitten finger. He walked to a Hakim's house without any difficulty, and soon after arriving suffered intense pain in the abdomen. At 5-30 his neck became rigid so that he could not turn his head, and his body became rigid so that he could not stoop. He was unable to talk. His respirations became laborious and coma set in. Frothy matter, and a quantity of phlegm-like mucous passed with great difficulty from the mouth and nostrils. Towards the climax he had two

convulsive seizures, and he died apparently from suffocation at about 7 P.M. the same day. It was observed that the heart pulsed some time after breathing had ceased. Further, Colonel Dawson says: "There have been several cases of death from bites of the krait here lately, in all of which the prominent symptoms were burning pain of the bitten part, rigidity of the neck, and pain in the abdomen." He was informed by his headkeeper that a neighbour's boy of 6 or 7 years of age had awaked one morning recently with an intense pain in the abdomen. He was treated in hospital for stomach-ache, and sent home. On removing the mat on which the child had slept a krait was discovered. A train of symptoms very similar to those experienced by the keeper who died followed, and the child died. No mark of a puncture could be found on the body.

No. 3.

I am indebted to Captain Leonard Forsyth, I.M.S., for the following notes of a case.

Two bites from a *Ceruleus*,* 3 feet long. Slight Toxæmia
Death, from other causes (?) in 36 hours.

History.—At eleven o'clock on the night of the 27th April 1911, a male, aged 48, a paniwala, well nourished and well developed, was bitten in his own compound. He stated that he walked on something which at first he mistook for a frog, this bit him twice rapidly.

He was at once attended to by a native hakim whose knowledge of treatment confined itself to "snake stone" tom-toms, the application of mud to his abdomen and eyes. The case was not reported to me until eleven o'clock in the morning, 12 hours having elapsed.

Onset of Symptoms.—Ascertained from his friends the natives. Giddiness and headache, came on about one hour after the actual bite. He complained of some abdominal pain and weakness in his

* The very detailed description given of the snake which was killed leaves no doubt as to the identification.

legs. When seen by me at 11 o'clock on 28th April 1911, the following symptoms and signs were present :—

He lay in the dorsal decubitus in a condition of semicoma, his pulse was full and bounding, regular in frequency and 100 per min. Respiration was hurried and embarrassed by secretion in his bronchioles 30 per minute.

His pupils were very dilated, did not react to light but the conjunctival reflex was still present. Both pupils were equal. His conjunctivæ were injected and the seat of ecchymotic hæmorrhage. There was slight bleeding from the mucous membrane of his nose and bronchi—at least, on cough his saliva was stained by red blood. His reflexes were normal (knee jerk, plantar, etc.). He had been bitten in the foot but there was absolutely no trace or abrasions as evidence of the bite. There was no swelling of the lymphatics of the limb or in any other part of the body.

I advised him to go to the regimental hospital at once, but this was very stubbornly refused. Accordingly I gave him an injection of antivenene at once; no local treatment of the bite was resorted to as it was considered too late. To keep his heart going, although it showed no signs of failure he was given hypodermically 1/100 grain of digitalin and kept covered up as his surface temperature was then lowered. I gave him also an injection of permanganate of potash (1 grain to the ounce) 4 oz. subcutaneously. At 1 o'clock same day, two hours later I saw him again. His pupils were now much smaller though still dilated, he opened his eyes himself and rested his head on his hand on the pillow. He had recovered from his condition of semicoma to a sort of dazed condition. I was told that he had sat up and taken "kunjji" and asked for water. His pulse was then much slower and respirations were only twenty to the minute.

I did not see him again since I was discouraged by the native treatment that prevailed whenever I went away. They removed the warm blankets about 4 o'clock, and covered him up with wet clothes. My hospital assistant saw him at 6 o'clock and reported that his temperature had gone up to 102. Later in the night about 10 o'clock the hospital assistant reported it to be 103.

He was not seen again by the Hospital Assistant or myself and death was reported at 10 o'clock next day, 36 hours after the bite.

(*Remarks.*—There seems little doubt that the man was poisoned, but he appears to have recovered from the toxæmia. Twenty-six hours after the bite he was able to swallow. It is impossible to say what caused death, but it is possible that the unscientific native treatment was directly responsible for this.—F.W.)

No. 4.

Communicated to me by Lieut.-Colonel Dimmock, I.M.S.

Bite from *Cæruleus* about 2 feet long. No toxæmia. Symptoms of fright. Recovery.

A Hindu male, aged 35, was bitten on the dorsum of the right foot at 11 P.M. on the 29th November 1907, by a small krait, "about two feet long," identified as such at the Parel Laboratory. At the Railway Hospital, Bombay, two punctures half an inch apart, at the seat of the reported bite were slightly incised, and permanganate of potash applied. He was transferred to the Jamsetjee Jejeebhoy Hospital, where the punctures were freely incised and permanganate crystals rubbed in. On admission he was reported as "suffering from fright, pretended to be insensible but is quite conscious." "In the night his pulse became slow and feeble, and respirations shallow and hurried. Next morning he was quite well and went home at noon." Internally he was treated with ammonia, and hypodermically with strychnia.

(*Remarks.*—No symptoms occurred other than those directly referable to fright. Ammonia and strychnia have both been proved powerless agents in snake-bite, though, of course, they are powerful restoratives in combating fright—F. W.)

THE BANDED KRAIT (*BUNGARUS FASCIATUS*).

THE TOXINS OF FASCIATUS VENOM.

(1) Toxins operating on nerve cells.

(a) A depressor that paralyses the respiratory centre.
(A. J. Wall.)

- (b) A depressor that paralyses the vasomotor centre. (Rogers.)
 - (c) A depressor that paralyses centres in the bulb. (A. J. Wall.)
 - (d) A depressor that paralyses the ends of the phrenic nerves: (Rogers.)
 - (e) A toxin destructive to nerve cells generally in brain and cord. (Lamb and Hunter.)
- (2) Toxins affecting the constitution of the blood.
- (a) A fibrin ferment that clots blood. (Lamb.)
 - (b) An antifibrin ferment reducing the clotting power of blood. (Lamb.)
 - (c) "Hæmolysin." A substance destructive to red blood cells. (Rogers.)

ANALYSIS OF THE ACTION OF FASCIATUS TOXINS.

- (1) (a) The "neurotoxin" paralysing the respiratory centre is the predominant agent in causing early death. It is assisted by 1 (d) and slightly by 2 (b) in bringing about asphyxia.
- (b) The toxin paralysing the vasomotor centre, reduces blood pressure, and so weakens the heart. When (1) (a) fails to cause death this toxin also may fail to bring about a fatal issue which unhappy result falls to the lot of 1 (e). When breathing ceases, from the effects of 1 (a) if artificial respiration is carried out, life is but slightly prolonged, and death is then due to cardiac failure.
- (c) The toxin operating on the centres in the bulb, by its action imitates the condition known as bulbar palsy where lips, tongue, throat, and voice are paralysed.
- (d) Assists 1 (a) in producing asphyxia.
- (e) Produces a general muscular weakness, and atrophy such as is not seen in the toxæmia of any other Indian snake and is the direct cause of death from exhaustion when 1 (a) and 1 (b) fail to achieve dissolution.

- (2) (a) The effects of the clotting ferment are only produced by large doses of venom. The dose capable of injection by any banded krait would not be large enough to produce clotting in the human subject.
- (b) This effect is only seen in doses of moderate concentration.
- (c) The amount of "hæmolysin" is relatively small in this venom. Slight augmentation of the respiratory embarrassment set up by 1 (a) probably occurs.

Lamb by experiment on rabbits found the virulence of the venom nine to ten times less than that of Cobra venom. Rogers by experiment on pigeons fixed the virulence at about one fourteenth that of Cobra venom. From this we are justified in concluding that fatalities in the human subject are unusual.

This inference receives actual support from the Burmese who are an observant race, and remarkably well informed concerning their animals and trees. This snake is very common in the Burmese Province, and though there is conflicting evidence on the subject of fatalities from its bite, many Burmese vigorously protest that it is a poisonous snake. In Assam too it is a very common snake, and it is remarkable that we have no records of casualties under the circumstances, although it is a notably lethargic and peaceful species.

FASCIATUS TOXÆMIA.

As already stated, this venom, like that of other Colubrines, contain two toxic elements that may produce death by their action on nerve cells in the brain and cord, *viz.*, "neurotoxin" and "amyotrophin."

It differs from that of other Colubrines, in that the "neurotoxin" is less concentrated, so that the "amyotrophin" is the predominant partner in the association. The result is that two very different forms of toxæmiæ are seen from the effects of the same dose of poison. In that produced by "neurotoxin" the symptoms show themselves in a couple of hours or so; they are evoked by the

same toxic principle that occurs in Cobra and *caeruleus* poisoning, and the symptoms are identical. Similarly death is produced in from a few hours to a couple of days from paralysis of the respiratory centre. As pointed out by Rogers, the respiratory embarrassment as in the case of the Cobra and common krait (*caeruleus*) is augmented by a paralysis of the terminations of the phrenic nerves.

In Cobra and *caeruleus* poisoning no heart weakness is noticed, in spite of the depression of the vasomotor centre that synchronises with the action on the respiratory centre. The reason for this is that both these poisons contain an element that directly stimulates the heart muscle, and another that does so indirectly by contracting the arterioles. *Fasciatus* venom seems to lack these principles, and hence a tendency to faintness is observed directly referable to the poison, apart from other influences, such as fright and pain.

In *fasciatus* poisoning, the symptoms evoked by "neurotoxin" may be very severe, and yet, as in cobra poisoning may decline till recovery appears complete. Subsequently, however, even where symptoms referable to "neurotoxin" have not been manifested, a new train of symptoms may appear. These seem to be due to the other and more lazy partner in the toxic association, *viz.*, "amyotrophin".

This toxin produces a degeneration in the nerve cells of the brain and cord and produces a clinical picture almost identical to that seen in the last stages of the spinal disease amyotrophic lateral sclerosis. The result is that in from 2 to 6 days, after the bite, the bitten subject develops rapid general paralysis, great general depression, loss of appetite, marked and rapid muscular weakness and emaciation, with a reduction in the urine. Purulent discharges from the eyes, nose, or rectum occur later and the victim dies of exhaustion in from 6 to 12 days. (A. J. Wall).

Rogers attributed death in these chronic cases to paralysis of the vasomotor centre, but Lamb and Hunter (*The Lancet*, September 23rd, 1905) have shown that this is not the cause of death.

The local signs in this form of poisoning are according to A. J. Wall exactly those seen in cobra poisoning, but less intense.

Lamb says they are much less obvious, and, in fact, sometimes little or nothing is to be observed at the site of the bite.

Fayrer records a case of a woman bitten in the foot at Tavoy. She suffered from tingling and swelling locally but exhibited no constitutional effects. It certainly seems from the local condition that some poison had been injected into the wounds.

THE DABOIA OR RUSSELL'S VIPER (*VIPERA RUSSELLI*).

TOXINS OF DABOIA VENOM.

- (1) Toxins operating on nerve cells—
 - (a) A depressor paralysing the vasomotor centre. (Rogers.)
 - (b) A depressor to nerve cells generally.
- (2) Agents affecting the constitution of the blood—
 - (a) A fibrin ferment clotting the blood. (Lamb.)
 - (b) An antifibrin ferment reducing the clotting power of blood. (Cunningham, Lamb.)
 - (c) "Hæmolysin" destructive to red blood cells. (Cunningham, Lamb.)
 - (d) "Leucolysin" destructive to white blood cells. (Cunningham.)
- (3) "Hæmorrhagin" destructive to the lining membrane of blood vessels. (Cunningham.)
- (4) A depressor to cardiac muscle (Lamb.)
- (5) A vaso constrictor. (Rogers.)
- (6) "Cytolysins" destructive to (a) liver, kidney and testis cells. (Flexner and Noguchi.)
and (b) to tissue cells at the site of the wounds.

ANALYSIS OF THE ACTION OF DABOIA TOXINS.

- (1) (a) The depression of the vasomotor centre is seen in the reduction of blood pressure, and cardiac weakness culminating in early death.
- (b) The depression to nerve cells generally, explains the great depression of spirits and reduced vitality in

Daboia poisoning. It is not sufficient to produce paralysis.

(2) (a) In large doses blood clots firmly within the vessels, and convulsions ending in death occur almost immediately. The Daboia cannot inject sufficient venom into the human subject to bring about this effect, which is only seen in relatively small animals injected with very large doses.

(b) In moderate doses a contrary effect is produced in the blood, and this, in conjunction with (3) accounts for the hæmorrhages which so frequently occur.

(c) "Hæmolysin" is potent in this venom and still further lowers the general vitality induced by 1 (b). It also has some effect in embarrassing the respiration.

(d) Reduces the normal resistance to microbic invasion.

(3) "Hæmorrhagin" by damaging the lining membrane of the arterioles favours leakage of the vessel contents. In conjunction with 2 (b) hæmorrhages are frequently the result.

(4) In conjunction with 1 (a) this causes heart failure.

(5) A vaso constrictor has been demonstrated by Rogers. By reducing the calibre of the blood vessels, blood pressure would be raised, and the heart reinforced. Any tendency towards such action appears to be nullified by a more potent element operating on the vasomotor centre, *viz.*, 1 (a).

(6) (b) Causes sloughing locally with the formation of an ulcer.

The venom is three to five times less potent than cobra venom according to Lamb.

Its action upon the blood is as follows :—

A toxic body (hæmolysin) destroys the red blood cells whose function it is to carry oxygen to the various tissues. As a result all vital processes are lowered. Another toxic principle (antifibrin ferment) very profoundly alters the consistency of the blood, and reduces its clotting powers. The action of this latter principle is exactly that of citric, phosphoric, oxalic and other acids, and is probably like them due to a precipitation of the calcium salts in

the blood. This in itself does not account for the transudation that occurs; but the fact that the lining membrane of the walls of the blood vessels themselves are damaged by another toxic element (hæmorrhagin) renders them more permeable. The effects of these two toxins is seen in the great tendency to hæmorrhages which are characteristic of the Viperine class of poisons. These hæmorrhages may be visible or invisible, and the whole case reminds one forcibly of scurvy, or purpura, diseases mainly characterised by similar blood changes. It frequently happens that the fang punctures continue to bleed or discharge bloody serum, or having stopped, bleeding recommences some hours or even days later. There may be bloody discharges from any mucous orifice. Invisible hæmorrhages* in the abdomen may cause pain, tenderness, and vomiting recalling to the physician's mind the identical state of affairs one sees in Hænoch's purpura. Similarly there may be extravasations into joints, or other serous cavities which may become painful, and swollen reminding one of another clinical condition, *viz.*, arthritic purpura. Hæmorrhages are apt to occur under the skin producing port wine discolourations in the form of spots or patches of various sizes, or they may occur in muscles, and other tissues giving rise to painful and tender swellings.

Death due to cardiac failure induced by toxins (1) (a), and (4) or later on to exhaustion may occur in from 1 to 14 days, or even longer.

DABOIA TOXÆMIA.

Daboia poisoning may be taken as the type of Viperine toxæmiæ.

Experimentally in animals three forms of toxæmiæ are observed.

It was observed by A. J. Wall, Fayrer, and others experimenting on animals that when the dose of poison was large, rapid and violent convulsions were induced resulting in death from asphyxia in a few seconds or minutes. Lamb was the first to correctly interpret these phenomena. He found that daboia venom contains a ferment that coagulates blood. This ferment is only operative

* These are specially well exemplified in the cases of *Echis* poisoning referred to later.

in highly concentrated doses of poison. It causes clotting of blood inside the blood vessels, and to this is attributable the convulsive seizure, and death from asphyxia. He further showed that the dose necessary is so large that it is very unlikely that this mode of death would ever be seen in the human subject.

The other two forms of toxæmiæ, one acute, the other sub-acute or chronic, are to be met with in the human subject. In the *acute* form there is general depression, in which the vital functions connected with both the heart and respiration are profoundly affected. The pulse becomes rapid and weak, and breathing rapid and irregular. General weakness of the muscular system is seen, and the mental activities may be reduced to the degree of unconsciousness. Nausea and vomiting are frequent, and the pupils become dilated, and insensitive to light. The surface temperature is reduced, and the skin is cold and often bedewed with sweat. There are no paralyses such as we see in Colubrine toxæmiæ. Concurrently with these effects on the nervous system, others dependent on the altered state of the blood are most likely to be exhibited. Bleedings from various mucous orifices, beneath the skin, or invisible hæmorrhages into serous cavities. The watery state of the blood may occasion œdema in dependent parts, or in organs, especially the lungs. Death from cardiac or respiratory failure, may terminate this toxæmia, or these symptoms may decline, and the patient appear as if about to recover. A repetition of the above may occur, or recovery may pass on to the third form of toxæmia.

The *sub-acute* or *chronic* poisoning appears to be connected with the local state of the wounds, which from the intensely virulent nature of the poison are very apt to be seen in a sloughing condition, favouring the development of septic germs. In the course of a few days added to the general depression, emaciation sets in and anæmia which will depend in degree largely upon the extent of the hæmorrhages. The enfeebled state of the system can oppose little resistance to the effects of invasion of any germs into the local wounds, and various forms of blood-poisoning, including tetanus, may result. Diarrhœa may occur, albumen appears in the urine and the patient ultimately dies of exhaustion from

hæmorrhages, or from blood-poisoning the effects of any invading germs that have gained entry into the local wounds. This state of affairs, however, need not necessarily prove fatal.

LOCAL SIGNS OF DABOIA POISONING.*

The local effects in daboia poisoning are usually very severe. Extravasations of blood are likely to occur in the neighbourhood of the punctures, the various tissue cells are destroyed by the virulence of the poison, and a slough forms which when separated leaves a deep ragged ulcer.

(1) *Pain*.—Where venom has been injected pain is an almost certain symptom. It is burning or stinging in character, often extremely acute and it comes on immediately. It is possible that the stings of certain other creatures such as scorpions, spiders, hornets, etc., might be as severe, and as rapidly produced; but if pain is experienced only to the degree normally met with in ordinary wounds from mechanical agency, it is highly probable that poison has not been introduced. The pain due to the poison may be masked by that produced by surgical interference, that evoked by local remedies of a caustic nature such as acids, and permanganate of potash, and also that occasioned by ligatures which is usually very distressing.

(2) *Swelling*.—Snake venom is an extremely powerful local irritant, and as such causes swelling in the injured part almost at once, similar to that seen after the bite of a mosquito. Swelling to an equal degree, and as rapidly manifested might result from insect or scorpion stings. If however no trace of swelling accompanies the wound, there is good reason to consider no poison has gained entry, and the longer the interval since the bite the greater the justification for assuming a non-venomised wound.

(3) *Bleeding*.—One of the chief effects of snake venom, whether Colubrine or Viperine in quality, is its power of reducing the coagulability of the blood and this fact affords very valuable information as to whether or not venom has been introduced into a wound, since when it has gained entry a constant oozing of thin bloody serum results which often continues for many hours.

* These signs are seen in most cases of Viperine poisoning, and to a less degree in Colubrine poisonings of every kind.

In a case recorded by Lamb and Hanna this continued for 24 hours. If lacerations or punctures are seen sealed up with blood within a few minutes of the casualty, as in the case of ordinary wounds, there is very strong justification for believing that no poison has been injected.

(4) *Tissue changes.*—Should the presence of any of the above local conditions call for local operative measures, the condition of the tissues as revealed by incision will furnish confirmatory testimony of the entrance of snake poison which, in the opinion of my namesake A. J. Wall, is absolutely characteristic. He says the areolar tissue becomes purple in colour and infiltrated with coagulated purple blood-like fluid. This fades gradually to a pinkish colour, and this again to normal conditions as the site of the poisoned wounds are receded from. These changes are extremely rapidly produced having been seen by this authority within 30 seconds of the entrance of the poison.

To the surgeon this sign is invaluable. Its presence proclaims the envenomed nature of the wound, and dictates a course of action completely different from that necessary in its absence.

(5) *Discolouration.*—A greenish or bluish tinge is frequently observed in the skin in the immediate neighbourhood of the punctures within a few minutes of the bite when venom has been introduced. Sometimes, indeed, the skin is purplish from severe subcutaneous bleeding.

(6) *Sloughing.*—The tissue cells in the vicinity of the wounds frequently die as a result of the powerful action of the poison. The result is the formation of a slough varying in extent to the dose of the poison injected. This dead matter under normal constitutional conditions offers an ideal pabulum for putrefactive germs to flourish. But in snake poisoning among other things it has been shown by Ewing, that the natural germicidal properties of blood are rendered inert, so that with the entry of germs there is a grave danger of another form of blood poisoning to be set up, and death may be the result of an intoxication from such germs. When the slough separates a deep ulcer is left which takes some time to heal.

Before quitting the subject of local signs I wish to make a few remarks on the characters of wounds resulting from snake-bite due to mechanical causes alone. There is a popular belief that the pattern left by a snake's teeth in the act of biting can furnish a clue to the poisonous or non-poisonous character of the offender. Fayrer has done much to foster this belief by his illustrations of the dentition marks of certain snakes, and in the remarks on this subject in I. A. F. M. 1248 given to Military Hospitals with directions for the treatment, etc., for snake-bite, these views are reiterated. Without denying that it may sometimes be possible to guess at the nature of the snake, I am very decidedly of opinion that, in the generality of cases of snake-bite, it is quite impossible for even an expert to say from the pattern of the punctures whether the snake that occasioned them was a harmless or poisonous variety. I might even go further, and say it is impossible to say with any proximation to certainty whether the wounds were inflicted by a snake at all. Vincent Richards says *apropos* this subject: "Not the slightest reliance is to be placed in the appearance of the scratches or punctures, though very much stress has been laid upon them as a means of diagnosing the bite of a venomous snake." A. J. Wall similarly remarks: "Now the mark of the teeth is no guide, or next to none, because a Cobra may not leave a single mark visible to the naked eye: and on the other hand fanged harmless snakes, like *Lycodon* and *Dipsas* may leave punctures in the skin that might easily be mistaken for the wounds caused by the fangs of venomous snakes." I have several times been bitten by harmless snakes, including those referred to by A. J. Wall, that have long fang like teeth situated like those of poisonous snakes, and in all cases the wounds have been lacerated, not punctured. Generally speaking a snake cannot make its jaws meet tooth to tooth on the flesh, its mouth being too small to grasp the limb or other part, but it fastens itself obliquely, and the teeth slip off and tear the skin.

Illustrative Cases.

No. 1.

Daboia-bite. Toxæmia. Death after about 23½ hours.

Reported by Dr. Spaar (*Spolia Zeylanica*, May 1910).

At midnight on April 6th, 1910, I was hastily summoned to see the late Mr. MacIntyre, Postmaster of Trincomalee, who had been bitten by a Polonga. On arrival at his residence, thirty to forty minutes after the accident, I found him seated erect on a chair on his verandah. He was bathed in a cold, clammy sweat, and complained of feeling sick, and was vomiting continually. The ejected matter consisted of a few grains of boiled rice and water and bile-stained fluid, and later on of glairy mucus. He had been attended to, within five or ten minutes of the accident by a constable, who applied to the wound a black "snake stone" such as I have seen in the possession of "snake charmers." Internally a remedy, prepared by dissolving part of a light green stone in water, had been administered with the object of producing vomiting. * * *

Three hemp ligatures were applied by his wife round the injured limb: one just above the ankle, another round the knee, and the other round the lower part of the thigh. The wound is said to have bled freely, staining all the bed linen. Careful examination, after cleansing of the limb, revealed a single, black, pin-point puncture on the inner side of the right heel, about an inch above the sole. There was then no bleeding, and but very slight pain complained of. The tissues around had a faint bluish tint, and the limb was swollen from the knee downwards. The ligatures, I found, were not tightly applied. The patient complained of great weakness, and there was much restlessness, violent retching, and inability to sleep.

I incised the wound freely, and injected into it a saturated solution of permanganate of potash. A series of punctures were also made all round, and the same solution injected hypodermically into the tissues. Powdered crystals were then rubbed in, and the wound packed with the same. The limb was postured, and compresses also of the solution applied and frequently renewed.

Four fluid ounces of whisky and half an ounce of sal volatile were administered internally at once, and a full dose of strychnine and ether injected hypodermically into the arms an hour later. The subsequent treatment consisted of a mixture of carbonate of ammonium, citrate of caffein, strychnine and digitalis, and hypodermic injections of adrenalin and strychnine. The treatment

adopted was that described by Dr. J. W. Watson Stephens, and in his hands proved very successful in Siam. The vomiting ceased after the first dose of whisky had been administered. I was not certain as to whether the vomiting and cold sweats were due to the snake poison or to the emetic administered by the constable, but it was evident later that these were effects of the former. The poison, therefore, had undoubtedly entered the general circulation before I first saw the patient. At dawn the patient was not so restless, but complained of great thirst and hunger. The bowels had acted once and were relaxed, the skin was warm, the tongue dry, the expression anxious, and the eyelids had now a very heavy appearance, and he was unable to open them wide.

The elevators of the lids exhibited parietic symptoms. The pupils were contracted, fixed, and equal. Pulse was quick, 115 per minute, and moderately full. Finding that the ligatures were rather lax, I proceeded to remove them, following the procedure recommended by Prentiss Wilson in the "Arch. of Internal Medicine," June, 1908, by intermittingly relaxing the ligature nearest to the heart, letting it become looser and looser until it was entirely removed, and the other ligatures removed in the same manner, at the same time watching the effect on the patient. At midday vomiting commenced again, but was not persistent. The tissues all round the wound were slightly tumefied and inflamed. Bleeding took place every now and again, especially if the patient exerted himself. A noteworthy feature of the blood was that it was thick, dark in colour, and did not coagulate. Restlessness was more marked. Weakness, depression, and exhaustion and pain in the small of the back were complained of, but there were no cramps, no paralysis of the limbs, and no convulsions. The skin again began to break out in cold, clammy sweat. The abdomen was distended and tympanitic, the upper part exhibiting a board-like hardness. Eructations were frequent, but did not appear to relieve the patient. He complained of suffocating pains, as if both sides of his chest were being compressed. There was great oppression. Respiration was hurried and laboured, and the pulse was becoming weak and more rapid—125 per minute. Sight was rather dimmed, but recognition of objects and persons was possible. Sinapisms were applied to the

feet and over the prae-cordial region, and saline infusions injected per rectum, and the patient seemed to rally somewhat, the pulse falling to 118 per minute. At this stage, however, his case was taken over by a native "snake physician of known repute," and English treatment given up, but the case was watched by me with interest to the end.

Drops were instilled into the eyes by the "vedarala," and this appeared rather to aggravate the dimness of sight. Internal remedies were also administered, but with the withdrawal of stimulants there was a steady rise in the pulse, till at 5 p.m. it registered 132 beats per minute, and was soft and feeble. Respiration also became more hurried and difficult.

At 10 p.m. the pulse rose to 142 per minute, and slight signs of lividity were noticed about the face. The native physicians were now making preparations against the twenty-fourth hour, which is stated to be a critical time with cases of snake bite. At about 11 p.m. dried bile from chickens was insufflated into the nostrils, which made the patient feel very short of breath. Within a couple of minutes he called out to his wife to hurry quickly up to him, and taking leave of her dropped back on his pillow and expired instantly. Consciousness and power of speech were retained to the very last. Death appeared to have been due to asphyxia and heart failure, and I am firmly convinced that free stimulation from the very onset is strongly indicated in cases of snake bite, if only to prevent the extreme exhaustion which marks these cases.

The external appearances noticed eight hours after death were lividity of the face, which was almost black. The lower portion of the face was swollen. Livid patches were also seen on the neck, chest, and lower extremities. The palmar aspect of the fingers was black in colour, and the nails were of a deep purple hue. A blood-stained fluid was issuing from the mouth and nostrils. The pupils were widely dilated, and the eyeballs congested. Post-mortem rigidity had disappeared, and decomposition was setting in early.

(*Remarks.*—In this case it is impossible to say to what extent the early vomiting, and tendency to collapse were due to the venom, as these very symptoms might have been entirely produced

by the emetic given (probably sulphate of copper). By dawn these symptoms had subsided, so that the recurrence of collapse at midday may be certainly ascribed to the action of the venom alone. The embarrassment of breathing was a secondary result of the failing heart, and the consequent starvation of the respiratory centre in the brain, not to any direct action on the centre itself.—F. W.)

No. 2.

Reported by Dr. Nicholson (Indian Snakes, p. 146).

Bite from *daboia** about 2½ feet long. Toxæmia. Death in 27 hours.

A case of death from its bite occurred while I was in Burma in the person of a strong gunner of the battery stationed at Thyetmyo..... The reptile turned and bit him on the finger. The snake held on for a short time and it was with some little difficulty the man shook it off. The man came at once to hospital, being advised by one of his comrades to do so, when on the way he became very weak. The Apothecary saw the patient on his arrival at hospital. It is supposed that a lapse of 20 minutes must have occurred from the time he received the bite until he reached the hospital and nothing had been done meanwhile in the way of remedies. The Apothecary immediately scarified the wounded finger freely, made the patient suck the wound and administered ammonia. For twelve hours no prominent symptoms appeared beyond swelling of the arm, restlessness and slight feverishness. Next morning he was found in a state of collapse, soon became unconscious and died 27 hours after the bite.

(Death appears to have been due to cardiac failure.—F. W.)

THE SAW-SCALED VIPER (*ECHIS CARINATA*).THE TOXINS OF *ECHIS* VENOM.

(1) Toxins operating on nerve cells.—

(a) A depressor acting on and paralysing the vasomotor centre? (Fraser and Gunn?)

* The snake was killed, and identified as a Russell's viper.

- (b) A depressor to nerve cells generally (Lamb and Martin), but insufficient to cause paralysis other than that of the vasomotor centre.
- (2) Agents affecting the constitution of the blood.
- (a) An anti-clotting ferment. (Fraser and Gunn.)
- (b) 'Hæmolysin,' destructive to red blood cells. (Fraser and Gunn.)
- (3) "Hæmorrhagin," damaging the lining membrane of blood vessels, is probably present.
- (4) A depressor to cardiac muscle. (Fraser and Gunn.)

Lamb estimates the virulence of the venom as from three to five times less than that of cobra venom. Fatalities in the human subject are much more frequent than used to be supposed. Probably about 20 per cent. of bitten subjects receive a lethal dose in a single bite, but this is largely a matter of conjecture.

ANALYSIS OF THE ACTION OF ECHIS TOXINS.

- (1) (a) A depressor to the vasomotor centre is probably present to explain the reduced blood pressure, and cardiac weakness noted by Fraser and Gunn. The same phenomena are seen in *Daboia* poisoning which Rogers demonstrated were due to a paralysis of the vasomotor centre, early death from heart failure ensuing.
- (b) This toxic element accounts for the great depression of vitality seen in *Echis* poisoning. It is insufficient to cause paralysis.
- (2) (a) The anti-clotting ferment accounts for the defective clotting capabilities produced in blood, and in conjunction with (3) for the hæmorrhages which are so frequently seen.
- (b) "Hæmolysin" in this venom is relatively potent, and hence destruction of red blood cells is a prominent feature. This produces some respiratory embarrassment, and contributes to the depression of vitality produced by 1 (b).

- (3) "Hæmorrhagin" is relatively more potent in this than in any other of our Indian snake venoms. In conjunction with 2 (a) it causes profuse bleedings.

ECHIS TOXÆMIA.

The poison of the saw scaled viper (*Echis carinata*) has no direct effect upon the central nervous system, except upon the vasomotor centre (as shown by Rogers). Consequently paralysis are conspicuously absent, and the chief constitutional symptoms observed are indicative of cardiac weakness. In addition the constitution of the blood is profoundly altered and the blood vessels have their lining membrane damaged with the result that hæmorrhages almost always occur.

The local symptoms are usually very severe. (See page 103).

In *Echis* toxæmia the heart labours under great difficulties. Like Colubrine venoms this powerfully depresses the vasomotor centre in the brain, producing a fall in blood pressure, and a weakening of the heart's pulsations in consequence. Fraser and Gunn have also demonstrated a direct weakening effect that this venom exerts on the cardiac muscle itself. By a destruction of the red blood cells an impoverished quality of the blood is supplied to the heart's muscle, which suffers again on this account. Further, the activity of the heart is lowered proportionately to the degree to which hæmorrhages occur. Over and above all these influences are the emotional ones, due to anxiety, fright, and pain. It is not surprising therefore that death is due to heart failure. Any attendant tendency to asphyxia that may present itself is brought about indirectly by the cardiac weakness supplying insufficient blood to the respiratory centre, and not by any direct influence on the respiratory centre, in the brain, or on the terminations of the phrenic nerves.

Illustrative Cases.

No. 1.

Bite from *Echis*, 12 inches long. Death in 27 hours. Autopsy.

Reported by Captain C. H. Reinhold, I.M.S., (*Indian Medical Gazette*, November 1910).

At Hangu, on 13th July, at 7 a. m., dhooly bearer R., age about 40, while removing a dhooly from a tent, was bitten by a snake on the outer side of the forearm, 3 inches above the wrist.

He at once went to the Hospital Assistant and told him what had happened. The Hospital Assistant with commendable promptitude applied a ligature immediately above the site of the tooth marks, from which oozed two minute drops of blood, he then incised across the tooth marks and removed semi-circular flaps of skin to the size of an eight-anna piece, induced free bleeding and rubbed in crystals of permanganate of potash.

By this time the snake had been killed by some sepoy, and the Hospital Assistant went to see it; recognising it as a poisonous one he applied a further ligature round the fleshy part of the forearm of the man.

Since the hospital at Hangu is only a camp one, the patient was removed in a cart to the civil dispensary, and here at 9 a. m., rubber ligatures, above and below the elbow, were substituted for the cloth bandages, and potassium permanganate re-applied.

No antivenine being available, it was not used.

The wound in the arm continued to ooze all day, but the patient complained of severe pain in the arm, which was attributed to the ligatures; however, he managed to get some sleep.

At 5 p. m. there was considerable swelling of the arm, and severe pain complained of: as the general condition of the man remained satisfactory, it was decided to remove the ligatures.

At 7 p. m., the patient passed a diarrhœic motion in bed, but got up later to pass water and clean himself. There was no blood in the motion or urine, and active bleeding had ceased from the wound in the arm, the dressing being merely stained.

At 10 p. m. the patient complained of pain in the abdomen and was given aromatic spirits of ammonia and cinnamon water.

At mid-night the pain in the abdomen was worse; patient described it as a burning sensation. There was no vomiting.

At 2 a. m. the patient passed a diarrhœic motion (no blood) going out, with assistance, to the latrine, 20 yards away, for the

purpose. Patient had no sleep during the night, and was restless, complaining continually of the abdominal pain.

July 14th, 7 A.M., the wound was dressed, there was no fresh bleeding, the patient was quite conscious though the pulse was imperceptible, at the wrist. It was not noticed that he was blanched or cold. He complained of thirst and drank *sherbet*.

There was no sign of any paralyses.

About an hour before death he became very restless and ceased to recognise his surroundings.

He died at 10 A.M. 27 hours after the accident. I saw the case first an hour after death; rigor mortis had not yet set in. There was some swelling of the left arm, and blisters above and below the elbow where the rubber ligatures had been applied.

A *post-mortem* examination was made at 6 P.M., 8 hours after death.

Rigor mortis was well-established.

The wound in the arm was circular and about the size of an eight-anna piece, it had penetrated well into the connective tissue, but was not deeper. There was no sanious discharge from the wound, though the blood stains on the dressing were watery.

Lungs.—Emphysematous and anæmic, old pleuritic adhesions on the left side.

Heart.—Left ventricle strongly contracted and empty, right ventricle engorged with blood.

The blood was quite fluid and notably light coloured; there was no trace of clotting.

Abdomen.—No peritonitis or petechial hæmorrhages; the coils of intestine were distended with gas, and the omentum was anæmic.

The bladder was strongly contracted and the urine not blood stained.

Liver.—Normal, anæmic.

Kidneys.—Normal, anæmic—the capsule stripped easily.

Spleen.—Normal, small.

An enormous retro-peritoneal hæmorrhage distended the left side of the abdominal cavity extending from the diaphragm to the brim of the pelvis, but not crossing the middle line. The blood forming the hæmorrhage was dark, and had formed a curiously tough stringy clot, which was not easily broken up. It was impossible to discover what vessel was the source of bleeding; the arterial system generally was not atheromatous and the vessels of the kidney did not show any gross degenerative changes.

I satisfied myself that the hæmorrhage had no connection with the spleen or kidney (enquiries as to whether he had fallen or sustained any injury subsequent to the snake-bite produced no evidence of trauma).

I examined the snake which bit this man and identified it as an *Echis carinata*, 12 inches long; this has subsequently been kindly confirmed for me by the Bombay Natural History Society.

No. 2.

Bite from *Echis*, about 2 feet long. Toxæmia. Death 9 days later.

Reported by me in the Bombay Natural History Journal (Vol. XX, page 522).

A Mr. Neale was bitten below the inner bone of the right ankle on the night of the 12th of May by a snake which he saw and described as being about two feet long. He was wearing socks at the time. He came indoors, applied a ligature above the ankle, and unable to persuade his servants to cut open the site of the injury had to do so himself, making two superficial incisions with a razor, and then he applied crystals of permanganate of potash. From the notes I conclude that the incisions and the application of the salt were not what a Surgeon would consider at all satisfactorily performed.

He passed a restless night, and in the morning sent for a local snake charmer, who grasped the tissues as well as he could two or three times with his teeth, and sucked with the idea of forcibly extracting the poison. Being in great pain, he sent for an

Assistant Surgeon, 11 miles distant, who arrived in the afternoon of the 13th instant. He found two fine punctures half an inch apart at the seat of the injury, one being very slight, the other was still bleeding and the foot was enormously swollen and discoloured greenish-blue. Mr. Neale was in great agony.

On the 14th instant the patient developed bleeding from the gums. He appears to have remained in much the same state till the 18th instant, when he was carried to Tankari, and admitted into hospital at 4 A.M. on the 19th. His wounds had healed, and the foot had completely subsided to normal proportions, but there was swelling of the right calf and thigh. Later, pain in the right groin shooting into the abdomen was experienced, and the abdomen became distended, painful and tender. There were blood stained patches and spots in the skin of the arms and chest, and a large extravasation formed over the right buttock, and bled freely externally. His gums were still bleeding. He had no fever; but his respiration was hurried, and his pulse weak and frequent. An enema relieved the abdominal symptoms, the stool being very dark (probably from admixture with blood).

On the 20th he had very severe pain in the right hip, which became swollen, and he could not bear to have it touched or moved. He was decidedly weak as shown by his pulse and at 2 A.M. had a fainting fit. He then complained of burning pain all over the body, and still had special pain in the right calf and thigh, but his abdominal pain was less and the swelling reduced. He vomited three times during the day. Bleeding from the buttock had stopped and the bleeding from the gums was slight. He had another fainting fit at 6 P.M. but rallied again. At 10-30 that night the Civil Surgeon arrived from Broach and found the patient quite conscious, free from any nervous disturbance, but very exhausted. The breathing was distressed, and there was some congestion of the lungs. The pulse was thin and weak. The extremities were cold and livid. The gums were blue, spongy and bleeding, and expectoration blood stained. There were extravasations of blood beneath the skin of various sizes on the face, chin, neck, chest and back, and a very large one over the right buttock, and another on the inner side of the left (right?) thigh. The right foot was swollen to

twice the size of the left, the tissues round the bitten part were loughing, and there was cellulitis of the foot and ankle.

The abdomen was tender and swollen. He saw a tarry stool that had been recently voided. In spite of every endeavour to save the patient, he continued to grow weaker and died from heart failure at 2 A.M. on the 21st May.

(*Remarks.*—The symptoms detailed above are all due to diminished coagulability of the blood, such as we know is induced by the poisons of both vipers and colubrine snakes. The absence of any nervous phenomena negatives the idea that the culprit was a colubrine species, whilst the severity of the symptoms arising from the altered state of the blood, which we know, are specially pronounced in viperine toxæmiæ, strengthens the assumption that it was a viper that inflicted the injury. The casualty occurring near Broach clearly points to the offender being either the “Phoorsa” (*Echis carinata*), or Russell’s viper (*Vipera russelli*), but we cannot be certain which.—F. W.)

No. 3.

Reported by me in the Bombay Natural History Journal (Vol. XIX, p. 266).

Bite from *Echis*, 15 inches long. Toxæmia. Death 7 days later.

Thanks to letters from Colonel Russell, R.A.M.C., and Mr. C. A. Owen, I am able to put on record an instructive case of *Echis toxæmia* which ended fatally.

The bitten subject was a muscular male European, aged 47, total abstainer and non-smoker, and in excellent health. He was bitten at 10 A.M. on the 15th August 1908 at Rawal Pindi, wounds being inflicted on fingers and back of the right hand and the back of the left hand. He went “at once” to the station hospital where the wounds were “freely incised” and crystals of permanganate of potash then rubbed in. Antivenene was then injected subcutaneously. He had no symptoms that day up till 5 P.M., when he left hospital at his own request.

On the 16th at 6 P.M. his wounds began to bleed spontaneously, and he discharged blood in his urine and by the bowel.

He was re-admitted into the station hospital where his pulse, respiration and temperature were found to be normal. His tongue however was swollen and discoloured, and his right arm too up to the shoulder. He passed blood in his urine, and also from the bowel. He was given internally calcium chloride, adrenalin chloride and ergot.

On the 19th he had severe vomiting, necessitating feeding and medication by the bowel, but his bleedings had reduced, and his general state was reported quite good. He complained only of pain in his hands.

He continued to improve, and the bleeding diminished until the 21st (the 7th day after the bites), no blood appearing then in the stools. At 4 P.M. that day however he suddenly collapsed, became delirious and then comatose. He was given strychnia and other stimulants, and transfusion of salt solution was performed. Under this treatment he rallied temporarily, but a recurrence of the collapse at 10-30 P.M. culminated in death.

The case was thus a very typical one of viperine toxæmia. There were no symptoms at any time referable to the nervous system, all the action of the poison being exerted upon the blood. The reduction in the coagulability of this fluid was responsible for the visible hæmorrhages, and there is no doubt that the swollen and discoloured condition of the tongue, and the tissues in the right arm was due to subcutaneous hæmorrhages. The actual cause of death was obviously heart failure, due no doubt to the drain upon the system from continued and persistent bleedings. One could not expect beneficial results from antivenene in this case, which was a wholly unsuitable one for the exhibition of this remedy. The serum prepared at Kasauli is only antitoxic to the venoms of the cobra and the daboia.

I have examined the snake that caused this fatality, and have the skull in my collection.

THE COMMON HIMALAYAN VIPER (*ANCISTRODON
HIMALAYANUS*).

THE TOXINS OF HIMALAYANUS VENOM.

No experimental work has been done with this venom.

HIMALAYANUS TOXÆMIA.

Illustrative Cases.

No. 1.

Bite. Severe local effects. Recovery without treatment.

My brother Colonel E. W. Wall was bitten by this snake in Kashmir in September 1912 in a locality so remote that no attempt at treatment could be made. The paharis, who call the snake "pohur", claimed to be quite familiar with the toxic effects, as many subjects they said are bitten annually, and they get well in a couple of days or so. Their predictions were confirmed in this case. The seat of injury was above the boot. And almost immediately my brother felt lancinating burning pain in the punctures. On removing his boot he noticed a sort of blood blister. The foot and leg up to the groin rapidly swelled, the pain continuing, but there was no bloody or serous oozing from the punctures after a few minutes, and no hæmorrhages from mucous surfaces or the wound subsequently. The boot could not be put on for a couple of days, but the swelling then subsided, and the tissues in the vicinity of the wound were much discoloured for some time.

This is an extremely valuable record for the purposes of this book.

No. 2.

Major Frost, I.M.S., has favoured me with the following case:—

Bite from adult *Himalayanus*, 18 inches long—Toxæmia—Recovery.

On Sunday, the 14th May 1911, at about 5 P.M., Rifleman Himantia Thapa of the 2/4 Goorkhas was bitten on the right fore-finger by a Himalayan viper *Ancistrodon himalayanus* (identified as such by the Secretary, Bombay Natural History Society), about 18 inches long.

Himantia thought but little of the bite and went off to take his evening meal, however his hand rapidly became very painful, and much swollen up to the wrist, and he came to Hospital for treatment at 7 P.M., two hours after the infliction of the bite. A ligature was applied round the wrist by me, and crucial incisions were

made deeply into the fang wounds, and potassium permanganate crystals were rubbed in generously. Afterwards the wound was bathed freely and for a long time (over an hour) with warm water, massage and pressure being applied downwards and outwards with a view to expelling from the wounds as much snake poison as possible. It appears to me that it would be well worth while to adopt this procedure without the application of potassium permanganate, if none were at hand, in the hope of reducing the quantity of snake venom in the tissues from a lethal to a non-lethal dose.

Being in camp I had no antivenene to administer. The patient made a good and rapid recovery. The day following the bite his forearm was much swollen up to the elbow, and this swelling persisted for four days, disappearing on the fifth. He suffered a good deal of pain in the armpit for two days, but I could not detect any hard or swollen glands there.

The wounds in the finger remained free throughout from septic trouble and inflammation.

A wet antiseptic dressing was employed with the object of permitting free oozing of blood and serum, possibly containing some snake venom, from the wounds.

For forty-eight hours after the bite temperature was sub-normal, (lowest 96·8) subsequently normal.

I have a few other records of casualties occasioned by this species, none of which proved fatal. Lt.-Col. Fenton mentions four casualties in the B. N. H. S. journal (Vol. XIX, p. 1002), three of which were treated with ligature, incision and permanganate of potash, and the fourth with vinegar. Pain and swelling were the main symptoms, and these passed off in two or three days, and all the cases recovered. I have no record of a fatality, and the natives everywhere agree in saying that bitten subjects recover. It would be a mistake however to argue from these cases, and native reports that treatment is unnecessary. It is highly probable that there are subjects to whom the poison of our least venomous snakes may prove fatal.

THE LARGE SPOTTED VIPER (*LACHESIS MONTICOLA*).

THE TOXINS OF MONTICOLA VENOM.

No experimental work has been done with this poison.

MONTICOLA TOXÆMIA.

Illustrative Case.

Reported by me (*Indian Medical Gazette*, November 1907.)

Bite from snake 1 foot 9 inches long. Poisoning. Recovery.

On the 23rd of August at Shillong (Khasi Hills, Assam, 4,900 feet) I arrived home at 6 P.M. to find my snakeman awaiting me with the report that he had been bitten in the finger whilst trying to effect the capture of a viper. He produced the snake which proved to be a pit viper (*Lachesis monticola*) common in these hills.

The injury had been sustained about 4 P.M.

I accompanied him to the Civil Hospital walking. On examination I found the wound had been inflicted on the dorsal aspect of the second phalanx of his right middle finger. The wound had been cauterised in a very superficial and perfunctory sort of manner with nitrate of silver, and a single string ligature applied above the wrist by a native practitioner. The patient complained of much pain which he said was increasing in the hand, and I have little doubt was due mainly to the ligature. The whole limb was much swollen, and the swelling extended slightly to the subcutaneous tissues beneath the axilla. The hand was most swollen, partly doubtless due to the ligature for it was cold.

I removed the ligature, made four parallel incisions to the bone, rubbed in crystals of permanganate of potash, and dressed the part.

The patient walked to my house, a mile distant, where I told him to sleep in case of developments. He passed a fair night, and said he slept all right, and he seemed fairly easy in the morning, but his swelling had increased. I sent him home and told him to keep quiet.

At 12-25 inclement weather having brought me home unexpectedly I found him sitting on my doorstep, his clothes saturated with blood, and his finger bleeding copiously. He had been there fifteen minutes. He must have lost at least a pint and a half of blood where he sat. The bleeding he said came on suddenly whilst he was asleep. I controlled the bleeding as best I could with

improvised tourniquets, and had him taken to the Civil Hospital where I packed the wound, applied a tight bandage and gave him a hypodermic injection of Ergotin 1/200 grain, and morphia sulphate $\frac{1}{4}$ grain. I ordered him adrenalin chloride grs. x every hour, and calcium chloride grs. xv with Ext. Ergotæ Liq. every second hour. After 7 P.M. calcium chloride alone was given in fifteen grain doses every two hours whilst awake, and a generous supply of milk. He remained in hospital, and for his subsequent history I am indebted to Major D. R. Green, I.M.S., Civil Surgeon.

On admission, 24th August.—Pulse 65 weak. Respiration and temperature normal. Evening—Pulse better, stronger, respiration normal, temperature 99 F.

August 25th.—Pulse stronger, having nearly regained a normal force. Respiration and temperature normal. Passed a good night. Wound left undressed, the finger tip being warm and sensitive. No further hæmorrhage from the finger, nor from any mucous surfaces. The urine contained no blood nor albumen. The bowels acted, and the dejecta were normal. Calcium chloride was continued as before, and he had a generous supply of milk.

August 26th.—The wound was dressed. On removing the plugging, some oozing re-commenced, but soon ceased when re-bandaged. No constitutional symptoms of any sort. Treatment as before.

August 27th.—Left the hospital at his own request. On the following day some slight oozing from the wound recurred, but soon stopped. I have seen him since on several occasions, and he has had no further ill consequences.

The toxic effects were typically viperine. He never showed the least constitutional disturbance, and no nervous manifestations. His companion reported to me that he shivered immediately after his accident, but this I feel little doubt was nothing more than an emotional manifestation, for it was transient, and part of a fit of sobbing which his first alarm evoked, and there was no repetition of a similar nature. The blood was evidently profoundly altered in quality for that which was shed upon my verandah steps showed no trace of coagulation an hour or so afterwards.

The drugs administered internally appear to have had the desired effect of restoring coagulability, for no further hæmorrhage occurred, though there was some tendency to a recurrence after their suspension.

On another occasion in Shillong, I met a cooly who had been bitten by one. There was a slight wound on his ankle from which blood was oozing, and the surrounding parts were slightly swelled. He had placed a single cord rather tightly above the wound, refused my proffered surgical attention, and showed no apprehension, telling me the snake was a harmless one (*i.e.*, not deadly). A few days later I saw him and he said he had suffered very little in consequence. The wound was healed and he told me he had applied ginger to it after infliction. The Revd. C. Leigh (*The Field* 1st January 1910) says: "A case has been reported to me of an old woman who died in consequence of being bitten by a viper of this species. But this can hardly be taken as a test; for what proved fatal to a weak and aged woman might have comparatively slight effect in the case of strong healthy man."

THE GREEN PIT VIPER (*LACHESIS GRAMINEUS*).

THE TOXINS OF GRAMINEUS VENOM.

This venom has not been investigated experimentally.

GRAMINEUS TOXÆMIA.

Illustrative Case.

The Revd. J. H. Lord has sent me the following particulars of a case :

Dual-bite. Severe local effects. Recovery.

A cooly at Sai, in the Konkan when cutting brushwood, was bitten in two places on the left side of the head. He killed and brought the snake with him. About half an hour after the injury, the left side of the head was very swollen, so much so indeed that the eye on that side was almost closed. He complained of pain of feeling that the ground came up very close to his eyes. Both wounds were incised and permanganate rubbed in. From one incision serum escaped, and from the other blood. Ammonia,

chloric ether and brandy were given internally. On the following day his head was much more swollen, and both eyes closed. The local symptoms which it will be seen were very severe gradually subsided day by day, the pain included. He suffered no further ill-effects.

THE ANAMALLY VIPER (*LACHESIS ANAMALLENSIS*).

THE TOXINS OF ANAMALLENSIS VENOM.

- (1) Toxins operating on nerve cells.—
 - (a) A depressor paralysing the vasomotor centre. (Rogers).
 - (b) A depressor to nerve cells generally. (Rogers).
- (2) Agents affecting the constitution of the blood.—
 - (a) A fibrin ferment clotting the blood. (Rogers).
 - (b) An antifibrin ferment reducing the clotting power of blood. (Rogers).
 - (c) “Hæmolysin” destructive to red blood cells. (Rogers).
- (3) “Hæmorrhagin” destructive to the lining membrane of arterioles. (Rogers).

ANALYSIS OF ANAMALLENSIS TOXINS.

Identical with that of Daboia. The clotting ferment is less potent than that of Daboia, but the hæmorrhagin is more powerful, hence hæmorrhages are likely to be more profuse. Rogers is the only investigator who has experimented with this venom.

ANAMALLENSIS TOXÆMIA.

There is no well reported case.

Jerdon (Journal, Asiatic Society of Bengal, Vol. XXII, p. 525), has known several cases of bite, but none proved fatal. Mr. Henderson has informed me by letter how he was once bitten by one in the finger. The snake was half grown. He sucked the wound, and cauterised it at once, and “suffered very little discomfort.” For some time afterwards he experienced a sense of weight in this arm when it was held down. Ferguson (Journal, Bombay Nat. Hist. Soc., Vol X, p. 9) relates how Baron Von Rosenberg was bitten, and walked 10 miles before pain asserted itself. He then found the

member, so swollen he had to cut the boot off. After a night of pain and fever, a cupful of blood and matter came away, and it was several days before he could wear anything but a slipper. A year later the place swelled up again, became painful, and discharged matter. Ferguson also mentions having met a hillman with a withered right arm which he (the native) attributed to a bite from this reptile.

Uncertainty of the effects of Snake-bite.

The effects of snake-bite are most uncertain as will be seen from a few examples quoted below.

Snake literature is simply full of parallel examples. The evidence is so conflicting that those who study the question sooner or later abandon the attempt to make inductions from snake-bite records. They are obviously too illusory. The only satisfactory knowledge we have acquired, and are likely still further to acquire, is derived from direct experiment in animals where a known dose of venom is injected by the agency of a hypodermic syringe. The evidence from such experiments carefully conducted is incontrovertible.

There is one consolation to be derived from records such as those I give, *viz.*, one need never lose hope in a case of snake-bite, or even snake-poisoning. Many serious cases undoubtedly recover without any treatment.

ILLUSTRATIVE CASES.

<p>(1) Elliot¹ records the following: "I myself saw a large powerful Daboia (3 feet 8 inches long) strike fairly at a dog, hold it, shake it, and only let go, when the dog had fled yelping several yards dragging the snake along the ground. The part bitten was soft, and fleshy, the bite was apparently a fair one, the glands of the snake, when dissected, though emptier than usual, both proved to contain poison. From one gland alone I obtained more poison</p>	<p>(1) Elliot¹ again records : "Eight days later the same animal was fairly struck by a vicious Daboia (3 feet 4 inches long), the bite being almost instantaneous in its shortness, and this time the victim died in less than three hours."</p>
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¹ Trans., South Ind. Branch Brit. Med. Asscn. 1895, pages 7 & 36.

than another *Daboia* emitted through a leaf in a vigorous bite; add to all this, that there was a well marked subcutaneous extravasation around the bite, and the case seems perfect. Though it became rather ill, did not die."

¹Surgeon-Major Browning, I.M.S., records the following:—

(2) "A healthy cobra bit a dog in two places with no result."

(3) ²Nicholson records a snake catcher who was bitten by a hamadryad "in perfect condition" and about 10 feet long. He suffered no ill consequences.

(4) ⁴Russell records the following:—

"I was told at Vizagapatam, of an old invalid who happened to be present at an exhibition of snakes, and observing, among other feats, the snakeman thrust a large *Cobra de Capello* into his bosom, he asserted he could himself do the same, swearing at the same time, that no snake could live an hour in his country, nor would the most venomous do any mischief to an Irishman. It was in vain that the spectators remonstrated and warned him; for resolutely putting the animal between his shirt and his skin, but ignorant of the acquired art of handling it, he was bitten severely in the breast. The consequence was excruciating pain, some alarming symptoms of poison, and a local ulcer, which was a month in healing.

(2) "Another bite from the same cobra on the same animal resulted in death."

(3) ³Theobald mentions a snake charmer, who was bitten by a hamadryad, who died in a few minutes.

(4) ⁵Fayrer records the death of a man in half an hour from cobra poisoning, and another case where a woman died two hours after being bitten by a cobra."

¹ Trans., South Ind. Branch Brit. Med. Asscn, 1895, page 7.

² Ind. Snakes, p. 148.

³ Cat. Rept. Brit. Birma, p. 61.

⁴ Ind. Serpents, p. 88.

⁵ Thanatoph, pp. 42 & 43.

(5) ¹Fayrer records the following:—

A krait, about 30 inches long, was bitten by a krait 48 inches long without ill-effect.

²Another krait, about 20 inches long, was bitten by a krait, 42 inches long, with a similar result.

(6) ⁴Fayrer also records that a large cobra was bitten in the body by a Daboia and showed no ill-effect.

(5) ³Fayrer mentions a small krait bitten near the tail by a large one at 1 p.m; it was dead the next morning.

(6) ⁵Fayrer again reports a full grown cobra being bitten in the body by a Daboia. It died two days later.

¹ Thanatoph Ser. 23 Expt. 8.

² Loc. cit. Ser. 24 Expt. 14.

³ Loc. cit. Ser. 21 Expt. 17.

⁴ Loc. cit. Ser. 8 Expt. 28.

⁵ Loc. cit. Ser. 8 Expt. 29.

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Treatment of Snake Poisoning.

In Clifford Allbutt's last edition of medicine the article on snake poisons may be assumed to be the standard one, written as it is by two such distinguished workers in ophitoxicology as Martin and Lamb. It is disappointing to see that these two authorities confine their remarks on the treatment of snake bite other than by antivenene to a few lines, and that they state that beyond antivenene nothing can be done in cases of snake poisoning but keeping the patient quiet and warm, applying a ligature, excising the wounded parts, and introducing strong permanganate of potash in solution. I think there are other means than those mentioned at our disposal from which one may expect good results.

The measures to be adopted in combating the effects of snake poison may be considered under three headings. (1) *Preventive* which aims at preventing the absorption of any venom that may have been discharged into the wounds, or reducing the dose absorbed. (2) *Antidotal* which aims at introducing into the blood an agent that will neutralise, and render inert any venom that has been absorbed. (3) *Symptomatic* which aims at undoing the evil effects wrought by the absorption of venom into the system.

(1) PREVENTIVE TREATMENT.

Under this heading I include all those measures undertaken with the object of preventing the absorption of the venom injected into the fang punctures, or at any rate reducing the dose of the venom absorbed to a minimum. These measures may be (A) *Medicinal* and (B) *Mechanical*.

(A) *Medicinal*.—Various agents are known to neutralise snake venom when mixed with it in a vessel, a chemical decomposition arising which robs the venom of its poisonous qualities. Such are permanganate of potash, chloride of gold, silver nitrate, the chloride and hypochloride of lime, etc. When Fayrer (in 1869) first discovered that permanganate of potash possessed this property, there was theoretically every reason to suppose that it would prove antidotal when introduced within the tissues holding the snake venom, but experiment proved otherwise. What is true of permanganate is probably equally true of the other bodies enumerated. Snake venom

when once introduced into living tissues is locked up in them so tenaciously that it can only with great difficulty, and then only imperfectly be brought into chemical relationship with neutralising agents of this class.

Colonel Bannerman has convincingly shown from a large series of experiments on living animals that permanganate is for all practical purposes of little if any avail. In these experiments, some of which I witnessed, the tissues were cut into after the injection of venom by a hypodermic syringe specially fitted on to the fang of a Russell's viper, thoroughly opened up and permanganate was then (within a second or two) rubbed into the incised wounds. The circumstances were obviously far more favourable than could possibly ever obtain in a bitten subject in medical practice, yet the salt rarely reprieved the death sentence.

(B) *Mechanical*.—The measures which have been suggested, and practiced with a view to mechanically preventing the venom being absorbed into the general circulation are (a) *Ligature*, (b) *Excision*, and (c) *Amputation*.

In a paper read by me at the Bombay Medical Congress in 1909 I attempted to show the dubitable value of all these measures, my opinion being based on the experiments of Fayrer, and reported in his *Thanatophidia of India*. I repeat these considerations here.

LIGATURE.

I think I am fairly if not completely accurate when I say that in every text book treating of the subject of snake poisoning, ligature is recommended as one of the most essential practices to be adopted. This being so it appears to me that the lessons to be learnt from the magnificent experimental work carried out by the late Sir Joseph Fayrer have gone completely unnoticed. I will quote some of the experiments conducted by that authority,¹ and then ask you if ligature as usually practiced is entitled to rank as the valuable measure which has unanimously been conceded to it.

In experiment 4 of the 15th series a ligature well soaped to make the knot run easy was tied "with the greatest amount of tension that a man's hand could exert" round a fowl's thigh before a bite

was inflicted by a cobra below the ligature. This however did not prevent the absorption of the poison. Symptoms appeared in 23 minutes and the fowl died in 43 minutes from typical cobra poisoning.

In experiment 2 of series 15 a dog's forearm was bitten by a cobra. Within 5 seconds a soaped ligature was "tightened as firmly as a man's strength could draw it," and immediately strong carbolic acid was rubbed into the wound and then a red hot iron applied. The dog died in 11 minutes (not 21 as stated by Fayrer).

In experiment 14 of series 16 a dog's forearm was bitten by a cobra. A ligature "was tied as tightly as it could be drawn" within 2 seconds of the bite. Carbolic acid was applied locally and 10 drops of carbolic acid in an ounce of water given internally. Death took place in 51 minutes.

In experiment 13 of series 16 a dog's forearm was bitten by a cobra. A ligature was immediately applied "as tightly as two persons could pull it," and the parts disorganised with the actual cautery. Yet the dog died in 35 minutes.

It must be noted here that in five other experiments on dogs in which no treatment was carried out at all, the average duration of life after the bite of a cobra was less than 36 minutes, so that the ligature in three cases quoted above could not even be claimed to have postponed the fatal issue which took place in 11, 51, and 35 minutes respectively or an average of 32 minutes.

Now it must be borne in mind that in these experiments the assistants were ready and everything was to hand in anticipation of the coming events. The utmost expedition therefore was enforced, and yet the procedure proved a signal failure. It is manifestly impossible for any surgeon to apply a ligature in practice with anything approaching the despatch which characterised Fayrer's efforts. Further if the circulation cannot be controlled by a ligature on a dog's leg where the muscular tissues are comparatively moderate, and we are told in one case that the united strength of two men was insufficient to accomplish this, how vastly more difficult will its arrest be in the much larger muscular development of the human arm or leg?

Fayrer himself says "it is almost physically impossible with the power of one pair of hands to so tighten a cord round a dog's leg as thoroughly to strangulate the limb." Wall (A. J.) too says: "an ordinary cord or string, or bandage, is nearly useless compared with the India rubber band. I have known fatal absorption take place when a string has been applied so tightly as actually to cut the flesh, and apparently strangulate the limb completely, causing acute suffering, evidently from the cord not accommodating itself accurately to the form of the member, and thus leaving a small channel for the circulation." Wall thus indicates the faultiness which, if there is any virtue in ligature at all, lay in Fayrer's technique. My remarks upon ligature above are only intended to apply to the application of a cord or inelastic band which, as far as my limited experience goes, is the method usually adopted in attempting to arrest the circulation in cases of snake-poisoning. In all the cases I have seen treated, or questioned bitten subjects upon, this form of ligature had been used. Mine may be a unique experience, still it is a fact, and as long as our text books continue to advocate ligature without specifying what is intended by this term, and refrain to warn the operator of what is known to have proved useless in this procedure so long will futile ligature be practised.

Now Wall (A. J.) tested the use of Esmarch's bandage with very marked results, says: "the India-rubber band is nearly painless, and properly applied is an absolute safeguard against further absorption." As far as I can ascertain the elastic band was but twice applied by Wall, once in the human subject, and once experimentally in a dog, and in both cases recovery was complete. I do not think these two cases sufficient test of the method, and I am inclined to think therefore that Wall was too sanguine in his opinion.

My reasons for doubting the value of ligature, as applied even on the lines advocated by Wall are based upon experiments by Fayrer with regard to excision and amputation.

EXCISION.

In experiment 14 of series 15, a fowl was bitten in the thigh by a cobra. The part was immediately excised but the bird died in 21 minutes.

In experiment 11 of series 16, a fowl was bitten in the thigh by a cobra. The part was "immediately" excised (within 2 seconds). A ligature applied before the bite was relaxed just before excision. Death took place in 64 minutes.

In experiment 3 of series 16, a dog was bitten by a cobra in a fold of skin in the groin. The wound was entirely excised "at once", but death took place in 60 minutes.

In experiment 13 of series 15, a dog was bitten by a cobra in a fold of skin in the groin. The part was "immediately" excised (within 2 seconds). The animal succumbed in 2 hours and 35 minutes.

It is to be observed that in 7 experiments in the cobra-bitten fowls, all bitten in the thigh, the average duration of life was rather less than two minutes (116 seconds), so that it is clear that excision performed under very exceptional circumstances—so exceptional indeed that they could never be repeated in general practice—merely prolonged life, did not save it. With reference to the experiments on dogs too it must be remarked that the skin of the groin was held up with forceps, so that the injury sustained could only have implicated the skin, and too that such a proceeding offered every facility for complete removal of the poisoned tissues, yet in spite of this death was not averted.

AMPUTATION.

In experiment 6 of series 16 a cat was bitten in the tail by a cobra. Amputation was performed in 20 seconds, but sufficient poison had been absorbed in this brief interval to give rise to profound toxæmia, the breathing became hurried, and the dejecta sanguineous.

In another experiment of Fayrer's a dog was bitten in the tail by a full sized cobra, but in spite of the tail being amputated within a few seconds the dog died. In a third case reported by Russell (*Indian serpents*, p. 73) a cobra was made to bite a dog twice (to expend some of its venom?) after which it was made to bite a pigeon in the thigh. The limb was amputated one minute later, but the bird died.

From these experiments one thing is certain. In the case of small animals at any rate, a lethal dose can be absorbed into the

system in a few seconds. Whether this rate of absorption is maintained can only be a matter of conjecture, but one cannot find a reason to see why the initial absorption should be less rapid than that which follows. If as seems probable the same rate of absorption is sustained then the outlook for bitten subjects who have had a large supralethal, dose injected into the wounds seems hopeless after the lapse of a few minutes. As we cannot speak positively on this point such measures as excision and amputation seem justifiable in the human subject on the chance that a supralethal dose may thereby be converted into a sublethal one.

(2) ANTIDOTAL TREATMENT.

There is only one remedial agent in snake poisoning that can claim attention as an antidote, *viz.*, antivenene. This as prepared in India is only curative against the toxæmiæ of the cobra and Russell's viper.

It is an antitoxin derived from the blood of horses that have been subjected to progressively increasing doses of these two poisons. These animals in time acquire a tolerance for these poisons considerably in excess of the normal lethal dose for horses. It is possible of course to prepare antitoxins that would be operative against the poisons of any snake, but in India a difficulty arises in collecting sufficient venom from other species to confer upon horses the degree of immunity required.

Calmette, who (synchronously with Fraser) discovered the antidotal properties of blood serum withdrawn from an immunised animal, claims (*Venoms, venomous animals, etc.*, published in 1908) that his antivenene prepared mainly from cobra venom is curative against the poisons of many species of snakes. This claim is refuted, however, (1) by Lamb, and others of high repute by direct experiment on animals, (2) it is in direct opposition to the laws that govern the action of antitoxic sera (see * footnote), and (3) it is

* Behring and Warnicke established the law that the serum of an animal artificially protected against any particular poison is capable of transmitting the immunity so derived to another animal, by the introduction of the first animal's serum into the second animal's blood. Antitoxins of all kinds are specific in their action, that is, are antidotal only against the particular poison which was used in their production. Lamb by direct experiment demonstrated that antivenene in no way differed from other antitoxins in this respect, however close the affinities between the poisons of two different species of snake appeared.

not upheld from the records given by Calmette in support of his claim, many of which I have attempted to show are open to every doubt from a variety of causes (*Indian Medical Gazette*, August 1909).

Antivenene must be injected into the tissues or blood of the bitten subject, preferably into the blood stream. Its success depends upon— (1) the freshness of the preparation employed; (2) the time that has elapsed since the casualty; (3) the method adopted; and (4) the dose employed. The Kasauli preparation is claimed to retain its virtues for one year, losing in this interval from 5 to 10 per cent. of its power. It probably still further attenuates, but retains a fair degree of virtue for much longer than a year. The shorter the interval between the bite and the injection of the antidote, the more favorable the chances of recovery. The results obtained from direct injection into a vein are more rapid and more pronounced than those derived from injection into the tissues, and this treatment may be the means of saving life even after a considerable degree of paralysis has become manifest. The intravenous method should obviously be left to the Surgeon alone. The dose recommended by Lamb for intravenous injection in 1904 was at least 350 cubic centimetres. Lamb and Martin more recently fix the initial dose at 100 c. c. for the polyvalent serum now prepared at Kasauli, which I was informed by Colonel (now Sir David) Semple has the same antitoxic value against cobra venom, volume for volume, that the older antivenene possessed. A second or third dose may be injected if the symptoms are not subdued. It is to be noted that children and small persons require a larger dose than adults.

(3) SYMPTOMATIC TREATMENT.

The third line of treatment aims at combating the effects wrought by snake venom after absorption into the system. Scores of reputed remedies of animal, vegetable, and mineral origin have received the attention of various experimentalists in this field. Many have been suggested by a knowledge of the action of agents used in Western medicine, many others owing to reputed virtues ascribed to them by the natives of the East. None of these however when submitted to a thorough test in the lower animals have

been found capable of averting death where a lethal dose has been injected. In considering this method of treatment attention must first be directed to the nature of the effects of snake venom on the system as already outlined in Part II. These are in the main two-fold. In the one case that of colubrine poisoning the danger is from paralysis of the respiratory centre in the brain, in the other that of viperine poisoning, the chief danger lies in cardiac weakness and tendency to hæmorrhages. The question naturally arises can we undo the effects produced in either case? In the first case the answer is probably "No." In the second emphatically "Yes." It was thought at one time that the depressing influences of colubrine poisons on the respiratory centre might be overcome by the exhibition of drugs that stimulated that centre, such as ammonia, strychnia, or alcohol, but all have been proved useless.

AMMONIA.

Jussieu appears to have been the first to recommend this drug as an antidote. Later in 1777, Monsieur Sage wrote a pamphlet in its praise, but when put to the test by Fontana in Europe it was condemned by him as useless, if not positively hurtful. Later on towards the close of the last century, Dr. Halford of Melbourne revived an interest in the drug, and firmly believed in its efficacy against Australian snake poisons. It was put to the test by Fayrer and Vincent Richards in the lower animals, being given internally, and by injection into the tissues and veins, but no benefit resulted. It was tried in the human subject in cobra poisoning by Dr. Hilson in the case of his quoted by me on page 81, and by Assistant Surgeon Jadul Kristo Sen in a case of krait poisoning quoted by me on page 90, and has been given in many other cases. When recovery took place this was wrongfully ascribed to the virtues of ammonia.

STRYCHNIA.

This powerful nervous stimulant was again vaunted as a specific some 18 to 20 years ago, in spite of experiments conducted by Fayrer in the seventies which served to show that it was worse than useless, being positively harmful. In three experiments death appeared to be actually due to the "remedy" before the poison against which it was used could claim its victim! Cunningham and

Elliot both independently arrived at the same conclusion in the early nineties of last century. Elliot in two valuable papers read before the Madras Medical Association in 1894 and 1895 convincingly showed by experiment on animals, and by administration in snake poisoned subjects that it is useless. He says "I can confidently state that, from beginning to end, I never saw one atom of benefit derived from the administration of strychnine." He went still further, and described its action as "vicious" in ophitoxæmia, and later published a pamphlet entitled "The dangers of strychnine in snake-bite."

ALCOHOL.

Scarcely any case of ophitoxæmia escapes a generous dose of this drug, though it certainly does no good, often on the contrary positive harm. Fayer tried it in the form of brandy in dogs, but no success attended its use. That it is actually harmful nobody understanding its effects on the system can deny. Vincent Richards says very sagely "Over-stimulation in a case of snake-poisoning can only expedite the absorption of the poison, which it should be our aim to prevent being taken up into the general circulation." He speaks again even more emphatically in the following words: "In my opinion every person who resorts to it should be punishable for malpractice." The dangers of alcohol in these cases are—(1) though at first it may stimulate the heart where there is a tendency to syncope, this effect is evanescent, and then replaced by a depression which may still further cripple its flagging efforts; (2) by dilating the blood vessels, and temporarily increasing the vigour of the heart, it operates in two ways—favourable to the production or aggravation of any bleeding tendencies, and by stimulating the blood current augments the rapidity of absorption of venom from the seat of injury; (3) alcohol induces a marked general fall in the opsonic index (*i. e.*, one's capability for resisting disease). This action would prove most detrimental in cases like *fusciatus* and *Echis* poisoning where the bitten subjects often live many days.

It is obvious from the above considerations that drugs that stimulate the centres in the brain are useless if not actually baneful. All the three dealt with above, which are those perhaps most frequently

given, are probably harmful and the most harmful of the three is undoubtedly alcohol.

All these remedies would be suitable in stimulating a weakened heart, not the result of ophitoxæmia, such as one sees from emotional causes in non-venomous snake, and other animal bites. Where one cannot positively exclude the possibility of the injuries having been inflicted by a poisonous species these remedies should certainly be withheld. It is better to do nothing than damage a patient's chances of life by meddling medication.

In considering the second question "can we influence the effects produced in the blood by snake poison" the answer is emphatically "Yes." There are at least three drugs known to us now that act upon the blood vessels and the blood too, in the very direction required. These stand out above all others, and should prove of the greatest use in reducing or actually controlling hæmorrhages. These are calcium, adrenalin, and pituitrin.

CALCIUM.

In discussing the effects of snake poison in Part II, p. 100, reference was made to the reduction of the clotting power induced. Now calcium not only increases the coagulability of the blood in any subject, but will, if given in suitable quantities, actually restore clotting power lost under the influence of such acids as phosphoric and citric, which act (like snake venom?) by decalcifying the blood. Further it probably restores calcium to the vessel walls, and renders them less permeable. We may assume, until disproved by experiment, that calcium will operate similarly on blood and blood vessels decalcified by snake venom. By experiments on tadpoles Professor Ivor Bang of the Royal Veterinary School, Copenhagen, and Ernst Overton have demonstrated that Calcium actually abates the toxicity of Cobra Venom. Solutions of the Chloride in water (1 in 200) required 100 times as much venom to produce the same effects. Lime water was found to be still stronger than the Chloride, but magnasium, and soda-water were found to be weaker in action. In a few reported cases in the human subject, it has been used with dubious benefit, but I venture to think that its full virtues have never yet been put to the test. It

has always, as far as I am aware, been given internally, and it has been pointed out through experiment in human beings that calcium so taken has little or no effect on the coagulability of the blood, being only slightly, and very slowly absorbed in the stomach. On the other hand if it is injected under the skin the effect on the clotting power is very remarkable, and very rapid. Further, in the cases known to me where it has been employed, the dose of the chloride has been about 10 or 15 grains. Leonard Williams says that it may be given in one drachm doses three times daily without fear of ill-effects. These considerations may very materially enhance our opinion of the value of the drug if pushed in viperine toxæmiæ.

ADRENALIN (HEMISINE OR EPININE).

This, the active principle of a gland situated near the kidney, has been shown to have a very potent action in reducing or actually arresting hæmorrhages, and has been employed in a few cases of viper poisoning. It very powerfully constricts the small blood vessels, which we have seen are structurally damaged by snake venom, so allowing transudation of their contents. This constricting influence by rendering the walls denser should overcome to some extent the damage wrought by the venom. Hitherto it has not controlled the bleeding in all cases in which it has been used, but like calcium, it may be that it has not been sufficiently pushed. Its effects are rather evanescent, but even so a hæmorrhage from a leaking vessel may be sealed in a few minutes, especially in conjunction with the action of calcium just referred to. More important still in cases of ophitoxæmia is its action upon the heart. It reinforces that organ in two ways. It exerts a direct stimulating effect on the heart muscle, and further stimulates it indirectly by increasing blood pressure. It will be seen from these remarks that it is the ideal remedial agent in cases of viperine poisoning, acting in almost every particular in an antagonistic manner. In colubrine poisonings where the heart is weakened, notably in the banded krait (*fasciatus*) and hamadryad it is for the same reasons an ideal remedy.

PITUITRIN (PITUITARY EXTRACT, ETC).

Another and even more potent remedy than the last is pituitrin, the active principle of a small gland at the base of the brain. It

acts like adrenalin by constricting the blood vessels, but is much more powerful, and its effects longer sustained. It has a similar but more forcible action on the heart, and is an ideal stimulant in snake poisoning and its accompanying hæmorrhages. I am not aware of its trial in practice, but it seems to me to offer great possibilities, especially in conjunction with calcium chloride.

In being confronted with a reputed case of snake-bite, even if a poisonous snake is produced one's first duty is to ascertain whether the case is one of snake-bite, or snake poisoning. One's decision should, I think, be based primarily upon the local signs as already discussed in Part II. p. 103.

I have now seen twelve cases of reputed snake-bite in this country, one of which was without doubt snake poisoning, the viper being produced. I have always been guided by external signs, have in consequence never administered antivenene, and only once permanganate of potash, and every case recovered. Had I been an enthusiast for the ammonia, permanganate, or any other form of treatment, a series of cases such as these would have appeared very convincing testimony of the efficacy of such to many people.

(4) LOCAL TREATMENT.

A wide excision of the bitten part is perhaps the best practice and the application of permanganate of potash crystals. One must do something in cases where life is seriously jeopardised, and disappointing as these measures have proved by experiment, there is always the possibility of reducing a supralethal dose to a sublethal one. All wounds require special attention to render them aseptic as already shown under *Daboia* (page 143).

(5) SPECIAL TREATMENT IN THE CASES OF THE COMMONER SNAKES.

TREATMENT OF COBRA POISONING.

The intravenous injection of 100 c. c. of antivenene as speedily as possible after paralytic symptoms have become manifest is imperative. If the symptoms after fifteen minutes exhibit a "crescendo" rather than a "diminuendo" tendency, this should be repeated, and, if necessary, a third or fourth injection given.

It is *known* to be a specific to this form of toxæmia, and hence should be pushed as long as the symptoms continue to increase. If the patient is seen in the last stages of toxæmia, and the breathing stops, artificial respiration should be employed, and continued for hours. Experiments conducted by the Indian Snake Poison Commission in 1873 showed that animals could be kept alive many hours after breathing had ceased, and in one case a dog's death was postponed by this means for thirty-seven hours and fifty minutes. So long as the circulation can be kept going, any antivenene injected may, probably will, fulfil its purpose.

Owing to antivenene, and the wonderful effect of artificial respiration in this toxæmia, we are better able to control, and successfully treat the effects of a supralethal dose of cobra poison than that of any other of our fatal Indian snakes.

Cases have been reported, it is true, where antivenene failed to avert death. One explanation is probably that the dose of the antidote was not sufficient. Another that it had not retained its virtue. A third that suggests itself is that the case may have been complicated with syncope (though this is unusual), and suitable measures to combat this were neglected.

It would be quite possible for a man after the toxic effects of cobra venom had been completely nullified by antivenene, to die from an attendant syncope due to fright, etc., if this condition were left unnoticed and untreated, death in such a case would almost certainly be wrongly attributed to cobra poisoning, and discredit thrown upon an active antivenene that had faithfully fulfilled its mission. Treat syncope as detailed (p. 146) and the wounds as under *Daboia* (p. 143).

TREATMENT OF HAMADRYAD POISONING.

No suitable antivenene* is available for this poisoning, and as we have seen it is as potent as cobra venom, and that a much larger dose is likely to be discharged by so large a snake, the prospects of recovery are not good. Attention to the general state as laid down under syncope (p. 146) offers the best hopes of benefiting the patient. A free excision of the wounds and treatment with permanganate may reduce the dose injected to something sublethal. Later treat wounds as shown under *Daboia* (p. 143).

* Lamb tested the efficacy of antivenene against this poison and found it useless.

TREATMENT OF COMMON KRAIT (*CÆRULEUS*) POISONING.

In no case of ophitoxæmia brought about by Indian Snakes are we so powerless to ward off the inevitable consequence of a lethal dose as we are in krait poisoning; we have no suitable antivenene* at our command for one thing, and hence artificial respiration is futile. There is little doubt this latter measure would postpone death, but it would not avert it. As in cobra poison the paralysis of the vasomotor centre is nullified for a time by toxins that increase the vigour of the heart's action, one operating on the heart muscle itself, another (or the same) acting indirectly by constricting the arterioles. Sooner or later, however, the vasomotor centre becomes paralysed, and artificial respiration would be no longer of any avail.

Being a colubrine snake the respiratory centre is destined to become paralysed, if the dose is lethal, and no human aid yet revealed can prevent this.

One cannot stand idly by, however, and watch the progress of events. We cannot know that a lethal dose has been absorbed, however grave the symptoms may be, and, forlorn as the case may appear, we must act for the best hoping that the dose is sublethal. It may even be possible to convert a lethal into a sublethal dose in some cases by prompt attention to the wounds. Extensive excision and treatment with permanganate is therefore more than justifiable. Treat wounds later as under *Daboia* (p. 143). The heart for the reasons given above, though not overcome by the poison, may suffer through the effects of fright and pain, and one should be on the look out for syncope, and treat it as stated under that heading (p. 146).

Where abdominal pain is complained of, on the assumption that it may be the result of internal hæmorrhage, calcium, and adrenalin, or pituitrin should be tried, as already advocated in viperine toxæmiæ (pages 138 and 139).

TREATMENT OF BANDED KRAIT (*FASCIATUS*) POISONING.

As previously shown the potency of this venom is greatly inferior to that of the cobra, and its nearer relative *cæruleus*, so that the outlook for a poisoned subject is correspondingly more favorable if not actually good.

* Lamb has tested antivenene (the product of pure cobra venom) against this poisoning and found it inert.

We have seen there are two distinct types of toxæmiæ produced by this snake. Being a colubrine species paralysis of the respiratory centre is to be feared, and early death will be attributable, to this action, to undo which nothing short of a specific antivenene* will avail. As there is no suitable antivenene prepared the case should be treated on general principles in the hope that the respiratory centre will escape complete paralysis. The toxic elements in cobra, and *cæruleus* venom that stimulate the heart are absent in this venom so that syncope is likely to occur (see treatment of syncope, p. 146). In the chronic form of poisoning we can do nothing, but maintain the patient's strength, keep him warm, and remove depressing influences of every kind. Treat wounds as under Daboia (p. 143.)

TREATMENT OF DABOIA POISONING.

The polyvalent antivenene prepared at Kasauli is a specific against this toxæmia, and if administered as already indicated under the treatment of cobra poisoning all cases should recover from the acute stage. In this toxæmia, however, there is great nervous depression, which, with the weakening effects of hæmorrhages, if not controlled produce a great tendency to syncope. This syncope should receive every attention or death may occur from this cause in spite of a sufficient dose of an active antivenene. Pituitrin, adrenalin, and calcium are to be relied upon to control the hæmorrhages visible and invisible (See pages 138 and 139). Extensive excision and the application of permanganate may reduce a lethal to a sublethal dose and should be practised.

In the *subacute* or *chronic* condition it seems dubious whether the symptoms are directly attributable to the venom, and it is quite likely that they may be simply the result of infective germs. At any rate it is imperative that the local wounds should receive the most careful attention. After incision and permanganate the wounds should be aseptically dressed. It would be even wiser to concede the great probability of infective germs being present, and treat the wounds antiseptically, and when sloughing of the parts has actually occurred this is all the more imperative. The best treatment probably one can adopt is to inject peroxide of hydrogen

* Lamb tested the efficacy of antivenene as now prepared against this poisoning and found it useless.

(two to five volumes) into the wounds freely. Enzymol by liquifying and digesting the dead tissues will help to rapidly clear up the unhealthy state, and promote antiseptis. It should be used with equal parts of water. A dressing of cyanide gauze should cover up the wounds, and the member be kept still on a splint. Bier's cups afford another excellent method of treatment. If fever continues a vaccine prepared from the patient's own flora should be injected. The general strength of the patient should be kept up with nourishing soups and a generous diet without alcohol. Should tetanus, or any other disease occur attributable to any specific germ the anti-toxin or other suitable serum should be injected. The layman will probably do best by immersing the wounds where practicable for an hour or more in perchloride of mercury solution (1 in 2000), or permanganate solution of a clear bright crimson hue (1 in 5000). After this the wounds should be dressed with lint, and bandaged, and the immersion repeated each day.

TREATMENT OF ECHIS POISONING.

Here no suitable antivenene * is available, and our efforts must be directed towards a control of the hæmorrhages. Bleeding is so characteristic and so profuse in this poisoning that it will be wise to anticipate it, and exhibit such drugs as calcium, pituitrin, or adrenalin as soon as possible (See remarks on pages 138 and 139). Syncope is very likely to be present in some degree, and this too should be anticipated, and every care taken to prevent or control it as laid down under that heading (p. 146). Being so small a snake the amount of venom injected is likely to be correspondingly small, and local treatment in the form of excision, and treatment with permanganate may in many cases reduce a lethal to a sublethal dose. Subsequently treat the wounds as laid down under *Daboia* (p. 143.)

TREATMENT OF OTHER VIPERINE POISONINGS.

Hæmorrhages are specially likely to occur in every form of viperine poisoning, and they are best anticipated, and treated by calcium, pituitrin and adrenalin as already advocated (pages 138 and 139).

* Lamb tested the efficacy of antivenene against *Echis* venom and found it inoperative.

Attention must always be directed to any attendant syncope (see treatment of syncope, page 146), which is always likely to be met with in viperine toxæmia. Treat wounds as under Daboia (p. 143.)

No antivenene has been prepared against these venoms, and that prepared from cobra venom has been shown by Lamb to be inoperative against at least one of these vipers, *viz.* *Lachesis gramineus*.

(6) Treatment to be adopted by Non-professional People.

To recapitulate, in being confronted with a case of supposed snake-bite, the first thing to decide is whether it is a case of snake-poisoning. In this decision be guided *entirely* by the local conditions, the pain, the swelling, and discharge of bloody serum from the wounds (p. 103). Usually the pain is so instantaneous, severe, stinging and persistent in character that this alone will proclaim the injection of venom. If moderate and transient, its existence may be dismissed in the absence of swelling and bleeding. The aching pain of a ligature will mask the usual characters of pain due to poison. Swelling in a poisoned wound comes on in a few seconds, or minutes, and increases progressively for hours. If a ligature has been applied this symptom may be masked, the whole limits below the ligature becoming tumefied. Absence of swelling is sufficient justification to believe poison has not gained entry. If the punctures are not sealed up in a few minutes, but continue to discharge blood or bloody serum there can be no doubt as to the injection of venom. Any *one* of these signs points to poisoning.

Next if a diagnosis of snake-bite is made, and snake-poisoning negatived, keep the patient under close observation, seek to allay any apprehension he may feel, and reassure him as to his fate with every confidence. Take special note of his surface temperature at frequent intervals, so that any impending faintness may be discovered early, and the measures laid down on pages 146 to 149 resorted to. It should however be born in mind that if antivenene is used it should only be injected subcutaneously by the layman and *not* intravenously. Above all give no alcohol.

If one's decision is one of snake-poisoning, whether dubious or certain, do not waste time with ligatures, but incise the poisoned

tissues freely and apply permanganate crystals moistened with water, remembering that the larger the amount of surface exposed to the action of the salt the larger will be the amount of poison neutralised. Make your incisions therefore a series of parallel slices, and cut in the length of the limb to avoid wounding large blood vessels as far as possible. Let the wounds bleed freely. Put the patient to bed, and keep him warm, and treat any tendency to faintness as laid down on pages 146 to 149. Avoid alcohol. Dress the wounds aseptically. See Daboia, p. 143. These are general rules to be observed by ordinary non-professional people.

The special treatment for cases where a poisonous snake accompanies the bitten subject has been already discussed.

Where it is obvious that a poisonous wound has been inflicted, but the culprit escaped destruction, it is justifiable, I think, to inject antivenene, as the cobra and daboia are such common snakes. In watching the progress of a case if paralytic symptoms begin to appear, the injection of antivenene subcutaneously is all the more justifiable, if not actually imperative. If no paralytic symptoms come on before six hours, there is considerable probability that the culprit was not a colubrine snake.

Bloody discharges in the absence of paralytic symptoms make it more and more probable as time elapses that the culprit was a viperine snake, and after six hours if measures to prevent bleeding have not been up till then commenced, these should be no longer deferred, see remarks on calcium (p. 138), adrenalin (p. 139), pituitrin (p. 139), and antivenene should be injected on the chance that the snake was a Daboia.

(3) Syncope and its Treatment.

Reference has been frequently made in the foregoing pages to the effects of pain, and the emotions upon the heart. Among the latter we may include the shock sustained by a timid individual in merely seeing a snake. A bite from so repulsive an object will certainly accentuate that shock as will also the sight of blood and the pain from the wounds it may inflict. Later the knowledge that many snakes are venomous, and even deadly leads to a natural anxiety, which may be intensified to actual fear of the possible consequences.

These depressing influences on the heart are likely to be operative in any case of snake-bite, but in cases of snake poisoning, over and above these non-toxic influences are others directly attributable to the snake venom. Such are (1) general depression of the whole nervous system, (2) the depression of the vasomotor centre, and (3) in viperine poisoning especially, the depressing effects of hæmorrhages. In either case whether a subject has or has not been poisoned syncope is very likely to be met with, and the condition and its treatment demands attention in these pages.

Explanation of the symptoms.—The symptoms have already been sufficiently detailed in contrast to the effects of cobra poisoning (page 71), but with an explanation of the symptoms the “raison d’être” for the treatment called for will become obvious.

Shock produces a paralysis of the vasomotor nerves (nerves that regulate the calibre of blood vessels, and so control blood pressure), especially those of the splanchnic system (the visceral blood vessels in the abdomen). By this paralysis the abdominal vessels dilate, and are then capable of accommodating the whole body content of blood! All other parts of the body suffer in consequence, and blood pressure generally is reduced, but the ill-effects of this vicarious anæmia and reduced blood pressure are first seen on the vital centres in the brain for the heart, and respiration. These being underfed strike work. The patient so stricken is unable to stand, turns giddy and falls. The heart beats become weak, and unduly frequent and the breathing also shallow and frequent. The loss of blood in the skin is seen by the pallor of the face, and coldness of the body surface.

Treatment.—We can assist the restoration of blood to these centres in the following ways:—(1) by general stimulation, (2) determining blood to the starved centres, (3) by invigorating the heart’s muscle, (4) by increasing the general blood pressure, and (5) by removing contributory influences to the depression.

(1) *General Stimulation.*—Suitable food is a valuable restorative. Alcohol we have seen is contraindicated where any chance of snake poisoning is present. Hot nourishment in the form of soups.

milk, tea, and black coffee are most comforting and beneficial. To obtain the best results small quantities—a table spoonful or two—every ten minutes or quarter of an hour should be given if swallowing is possible.

(2) *Determining blood to the starved centres.*—The position of a fainting person is most important. If the head is kept lower than the body, and the legs flexed at right angles to the trunk, blood by gravity alone will pass into the brain, and supply the needful stimulus to the centres. One may even more or less invert the patient with this object in view. Again if the limbs are tightly bandaged from below upwards, their contained blood small, though it may be, will be expressed, and conserved for the heart to act upon, and drive into the brain. Further a tight broad abdominal binder such as we apply after confinement will greatly assist in this direction.

(3) *Direct stimulation to the heart muscle.*—The following drugs will act in this direction and are best administered by hypodermic injection. Pituitary extract one cubic centimetre (18 minims) every four hours intramuscularly, adrenalin chloride in ten minim doses, or digitalin $1/130$ of a grain. Half these doses every two hours may prove more satisfactory.

(4) *Restoration of general blood pressure.*—The drugs pituitary extract, adrenalin chloride, and ergot, by constricting the blood vessels increase blood pressure and indirectly reinforce the heart. Ergot may be given intramuscularly in the form of ergone, or citrate of ergotine in $\frac{1}{100}$ to $\frac{1}{50}$ grain doses, or as tyramine in $\frac{1}{3}$ grain doses. Saline injections of various sorts also act powerfully, though less speedily in restoring the lost blood pressure. The best method for the layman is by the rectum, an enema syringe being introduced into the rectum, and salt solution allowed to gravitate into the bowel. It should not be pumped in. The salt solution should be warm (100 F.), and of the strength of one and a half teaspoonfuls to the pint. One pint is allowed to flow in from a vessel attached to the tube, and the vessel held not higher than one foot above the level of the patient's anus. Other methods of saline infusion open to the physician are subcutaneous injections, intravenous injections, and proctolysis.

(5) *Removal of removable contributing causes of cardiac depression.*— Cold has a most depressing influence on the heart, and will certainly intensify any tendency to shock caused by other influences. It is most important therefore to promote the body warmth in every way. We can do this by rubbing the limbs alternately, and the body with mustard, and ginger, keeping the parts not being so rubbed at the moment well covered with blankets, until hot water bottles can be got ready, when eight or a dozen should be applied around the patient. Pain again is a powerful agent in the production of shock, and is removable. The judicious administration hypodermically of sulphate of morphine $\frac{1}{6}$ to $\frac{1}{4}$ of a grain, with sulphate of atropine $\frac{1}{100}$ of a grain is likely to assist materially in relieving shock from this cause.

(6) If the heart actually ceases work, artificial respiration should be tried, and also the forcible dilatation of the anus with the forefingers.

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