June 16, 1881.

THE PRESIDENT in the Chair.

The Presents received were laid on the table, and thanks ordered for them.

Professor William Edward Ayrton, Mr. Henry Walter Bates, Dr. John Syer Bristowe, Mr. William Henry Mahoney Christie, Professor Herbert McLeod, Mr. John Arthur Phillips, Mr. William Henry Preece, Rev. Henry William Watson, and Dr. Charles R. Alder Wright were admitted into the Society.

The following Papers were read:—

I. "On the Differences in the Physiological Effects produced by the Poisons of certain species of Indian Venomous Snakes." By A. J. WALL, M.D. (Lond.), Surgeon H.M. Indian Army. Communicated by Sir JOSEPH FAYRER, M.D., K.C.S.I., F.R.S. Received May 2, 1881.

Hitherto no clear distinction has been recognised to exist between the actions of the poisons of the various species of venomous reptiles. It will be the object of this paper to examine closely the symptoms produced by these poisons in order to detect any differences that may be present, and to see if the poisonous agents can be classified according to their physiological effects. As poisonous reptiles admit of a simple anatomical classification into colubrine and viperine, one member of each group will be taken. It will be convenient to begin with the cobra, which is one of the most virulent and best known of colubrine venomous snakes.

I.—*The Physiological Effects of the Poison of the Cobra* (Naja Tripudians).

In order to exhibit the effect of this venom, it will be necessary to detail the results that follow when animals of different classes have been poisoned by this snake.

Experiment I.

At 10.46 A.M., a pariah dog was bitten in the thigh by a cobra.
11.14 A.M. Very lame in the bitten limb.

11.27 A.M. Affected by the poison. Staggers when he attempts to walk. Chewing movement of the jaw and lips.
11.28 A.M. Salivation.
11.30 A.M. Pupils somewhat small.
11.31 A.M. Attempting to vomit.
11.35 A.M. Respiration becoming slower.
11.37 A.M. Respirations 16 per minute.
11.38 A.M. Copious salivation.
11.39 A.M. Respirations fallen to 12 per minute.
11.42 A.M. Tongue hanging out of the mouth.
11.43 to 11.46. Convulsions.
11.47½ A.M. Respiration completely ceased. Pupils dilating.
11.49 A.M. Heart stopped. Dead.

Experiment II.

A fine large cock had 2 mgrms. of dried cobra poison in solution injected into its leg.
3.13 P.M. Injection.
3.20 P.M. Respirations 30 per minute.
3.38 P.M. Head drooping as if the neck were too weak to support it; but from time to time the head is raised with a jerk.
3.41 P.M. Respirations 25; can barely stand.
3.48 P.M. Respiratory movement very slight.
3.50 P.M. Cannot stand.
3.55 P.M. Respirations 19; pupils somewhat contracted.
4.3 P.M. Respirations 16; movement exaggerated.
4.5 P.M. Comb has become of a dusky purple colour; slight convulsive movements of the body.
4.9 P.M. Respirations 9.
4.17 P.M. Convulsions.
4.20 to 4.22 P.M. Convulsions continuing, but gradually becoming less violent.
4·25 P.M. Pupils widely dilated. Dead.

Experiment III.

A medium-sized frog (*Rana tigrina*) had 5 cgrms. of dried cobra poison dissolved in water, injected into its dorsal sac:—
12.42 P.M. Injection.
1.23 P.M. Struggling violently to escape.
1.40 P.M. Becoming paralysed.
1.53 P.M. Dead.

In these three experiments the effects of cobra poison on mammals, birds, and amphibia, are well shown. But as the symptoms in man, owing to the differences in his nervous system, are peculiar, it is requisite to give an outline of the results of cobra poisoning in his

case. The one selected is condensed from Dr. Hilson's account ("Indian Medical Gazette," October, 1873).

A punkah coolie was bitten by a cobra on the right shoulder about half-past 12 o'clock at night. He immediately felt an acute burning pain on the spot, which increased in severity. A quarter of an hour afterwards he said he was beginning to feel intoxicated, but seemed quite rational, and answered questions intelligently. The pupils were natural, the pulse normal, and the respiration was easy. He next began to lose power in his legs, and staggered. Half an hour after he was bitten his lower jaw began to fall, and frothy and viscid saliva to run from his mouth, and he spoke indistinctly, like a man under the influence of liquor, and the paralysis of the legs increased. Forty minutes after the bite he began to moan and shake his head from side to side, and the pulse and respiration were somewhat accelerated.

He was unable to answer questions, but appeared to be quite conscious, and his arms were not paralysed. The breathing then became slower and slower, and finally ceased about one hour and ten minutes after the bite, the heart beating for about one minute longer.

From these cases, and from the evidence given by other experiments, we can draw up a summary of the chief facts to be noticed during cobra poisoning. The first manifestation of cobra poison having been injected beneath the skin is a sensation of pain in the bitten part. The evidence of pain occurring in animals is very clear; the animal turns and licks the spot, and if it is the leg that is wounded it either limps on that leg or, what is more usual, draws it up so as to ease it.

This action has been termed paralysis of the bitten leg, due to the local contact of the poison with the muscles. Now, though it can be proved that the local effect of cobra poison on muscle is to weaken it, yet after the bite of a cobra a very small extent indeed of muscle comes in contact with the poison—very often none at all; and if the limb were really paralysed it would hang uselessly down, dragging upon the ground, instead of being drawn up.

This pain is accompanied by, or rather is dependent on, a very characteristic local condition, that is worthy of careful attention. If the body of a man or animal killed by snake-bite be examined, there may be scarcely a sign to mark the spot where the snake bit—a scratch or puncture may apparently be the extent of the injury. If an incision be made through the skin and carried through the punctures, very little change will be found in the true skin. It may be somewhat more injected with blood than normal, and the punctures will be found to be intensely so just at their edges, and a small quantity of blood may be effused there. But the areolar tissue lying beneath the true skin is the site of the chief changes. It will frequently be found to be of a purple colour, and to be infiltrated with a large quantity of

coagulable purple blood-like fluid. In addition the whole of the neighbouring vessels are intensely injected. This injection gradually lessens as the site of the poisoned part is receded from, so that a bright scarlet ring surrounds the purple area, and this, in its turn, fades into the normal colour of the neighbouring tissue. At the margin also the purple blood-like fluid is replaced by a pinkish serum, which may often be traced up in the tissues surrounding the vessels that convey the poison to the system. In one case in which the victim was bitten on the hand, I traced this effusion around the veins as high as the elbow. The local appearances differ considerably in different cases, varying from those excessive ones just described to a mere hyperæmia. It has been asserted that these changes are merely the result of hæmorrhage from the divided vessels. But this will not account for the pain or for the intense injection of the surrounding parts, or for the fact that should the bite not prove fatal the site of it nearly always suppurates. The real explanation is that cobra poison is an intense irritant and produces acute inflammation. Thus, if a small quantity be placed in the eye, the most severe inflammation of the conjunctiva follows. The existence of this local inflammation is of great value. It takes place with startling rapidity, one minute or less producing marked hyperæmia. This local hyperæmia is, therefore, the first indication that we can obtain that snake poison has really entered the system. As life itself depends upon the rapidity with which snake poisoning can be recognised, it will be seen that this is a matter of the greatest practical importance.

An interval now occurs before any fresh symptom is noticed, but the length of it varies greatly in different cases. In dogs bitten by cobras the average of four experiments gave 18·2 minutes as the length of this interval.

It is not possible to determine with exactitude the average length of this period in man, but in the case given it was fifteen minutes, in another instance reported by the same observer it was an hour and fifteen minutes; and an eye-witness, who evidently described a case with great accuracy as to details, stated four hours passed before any change was noticed. The evidence available on the subject makes it probable that an interval of an hour is the average of this period in man.

This pause, it can be proved, depends on two separate factors. The one factor is the time required for the absorption of the poison; for it is lessened by the poison being injected simultaneously into several different sites. The other is clearly dependent on some secondary change produced by the poison for which time is necessary; for if the part into which the poison has been injected be excised before the occurrence of a single constitutional symptom, yet, nevertheless, the animal may die apparently as rapidly as if no interference had been

attempted; showing that the mere presence of the poison in the blood, even in sufficient quantity to kill, is not capable of producing directly a physiological effect. On the other hand, to prevent grave misconception, it should be stated that it is quite possible to save life by excision of the bitten part, if it be done sufficiently quickly to prevent any considerable absorption.

The symptoms once developed follow one another with great rapidity. In man a feeling of intoxication appears to be the first constitutional effect of the poison. It is very generally complained of, but not universally, as it would require some intelligence on the victim's part to mention it. It is not possible to get evidence of a purely subjective condition in animals.

In man the next symptom is loss of power in the legs. There is first staggering, then inability to support the body, and finally there is complete incapacity to move the lower limbs. At the same time there is scarcely any loss of power in the arms, which may remain completely under the influence of the will. The exact nature of the action of cobra poison on the nervous system is, however, a very difficult subject. Sir Joseph Fayrer and Dr. Brunton, in their valuable series of papers on the subject, maintain that though the greater part of the nervous system is affected, yet the terminations of the motor nerves suffer especially, and in a very marked manner. They base their reasoning on the results produced by experiments in which the excitability of two nerves of the same animal is tested, one of which has been subjected to the action of the poison, and the other has been kept from the contact with the poisoned blood by the limb to which it is distributed being ligatured, the nerve, however, being left intact. These experiments I have repeated with, however, some differences in the arrangements, with the result that though the poisoned limb lost its excitability to a very much greater extent than the non-poisoned limb, yet the spinal cord, as long as it was capable of stimulation at all, could convey stimulation to the poisoned and unpoisoned nerve, but that the excitability of the cord was exceedingly quickly lost. These results would imply that the terminations of the motor nerves only suffered, *pari passû*, with the cord itself, and that there is no special elective affinity for the endings of the nerves. Nor are the results of the experiments of Sir Joseph Fayrer and Dr. Brunton incompatible with this view. For when one thigh of the subject of the experiment was ligatured, and the other was poisoned, when the cord was excited by a current, the stimulus had to be transmitted to the non-poisoned leg through the trunk of the nerve which was unaffected; whereas on the other leg it had to overcome the resistance induced by the paralysing poison. There is no need to suppose a special effect of the poison on the ends of the motor nerves; the different lengths of the trunks affected would account for a con-

siderable difference. To this we have also to add the paralysing effect on muscle, which, though not so great as on the nerve, is yet not unimportant, and would tell on the same side. Moreover, it does not follow that because a nerve to which poison has had access conveys electrical *stimuli* in a very imperfect manner, or not at all, therefore the effect of that poison has been to paralyse the nerve. It is unfortunate that the only test we have of the vitality of a nerve is its power of causing contraction in a muscle when irritated by electricity or mechanically.

It would be going too far to say, therefore, that because a nerve did not transmit such rude *stimuli* it was dead; and when an animal loses the power of withdrawing a member that is being painfully stimulated, the break in the power of conducting impressions or *stimuli* may be in any part of the nervous chain involved, or may be distributed equally throughout. A poison also that produces death by totally different means than paralysis may yet cause in the nerves a complete deadness to *stimuli*.

Experiment IV.

The right thigh of a frog (*Rana tigrina*) was ligatured, so as to completely prevent circulation through the limb, the nerve being included in the ligature. Two cgrms. of strychnia in solution were then injected into its dorsal sac.

12.53 P.M. Injection.
12.56 P.M. Tetanus.
1.10 P.M. Reflex action ceased.
1.23 P.M. Muscles of right leg infinitely sensitive.
1.25 P.M. Muscles of left (poisoned) leg contract with 0·75 volt.
1.39 P.M. Right (unpoisoned) sciatic nerve infinitely sensitive, causes muscular contraction with less than ·0001 volt.

Now strychnia certainly does not kill by paralysis, and yet the difference between the poisoned and the non-poisoned sides in regard to their nerves was more marked than was recorded in similar experiments made with cobra poison. In another frog this difference was very pronounced before the strychnia had ceased to produce tetanus, o that it occurs long before exhaustion has taken place. The complete interference with the vital functions produced by tetanus is the real cause of death, and the deadening of the nerves is simply the result of the excessive nervous discharges that have taken place through them. Thus, though the trunk or extremity of a nerve may be found paralysed, it does not follow that it is the direct action of a poison that may be present, or that it was the paralysis that caused death.

In cobra poisoning also it is possible to get very distinct evidence of sensation long after the nervous centres of organic life have been so

completely destroyed by poison, as to render the animal dependent on artificial respiration for life.

In this direction another point must be taken into account. It has already been stated that one of the most characteristic features of cobra poisoning in the human subject is paralysis of the legs. The patient is unable to walk or to stand, though his arms have not as yet experienced any loss of power. Now, it would be difficult to suppose that this was due to the terminations of the motor nerves of the legs becoming paralysed, while those of the arms remain unaffected. It is much more probable that the spinal cord is becoming paralysed, one of the first effects of which would be that it would lose the power of maintaining the tone and necessary contraction in the many complex groups of muscles on which the upright posture is dependent. But in cobra poisoning in dogs paralysis of the hind limbs without the fore is rarely seen. In the vast majority of cases power is lost simultaneously in all four members. In those few cases in which it has been noticed that the hind legs have suffered first, the animal has been bitten on the hind leg, which would always cause a certain amount of lameness and difficulty in walking with the hind quarters, due to the local effect of the poison. But even with this source of fallacy it holds good that in men suffering from cobra poisoning paraplegia is a most constant symptom, whereas it is very exceptional in dogs. The reason of this great dissimilarity is to be found in the different functions of the inferior portion of the spinal cord in the two cases. In man the lower portion of the cord is, to a great extent, a distinct nervous centre. The ganglia there not only are the centres on which the lower limbs rely for their nerve power to maintain the upright posture, but the sensation of contact with the ground of one foot in walking is translated in these lower centres into a motor stimulus to excite movement in the other leg, and paralysis affecting these centres would at once destroy the process. But in dogs the mechanism is very different. They move the foreleg of one side with the hindleg of the other. It is necessary, then, that the centres governing the movements in these limbs should be coupled—so to speak—together. The stimulus that moves the foreleg of one side has to excite simultaneous movement in the hind leg of the other. Therefore the posterior extremity of the cord has in the dog merely to transmit the motor impulse from the forepart, whereas in man it has to translate sensations into stimuli to excite movement, and this in man is the first faculty destroyed by cobra poison. It is, therefore, probable that the earliest injury inflicted on the nervous system by cobra poison is a paralysis of the centres in the lower part of the spinal cord.

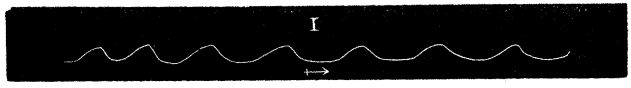
The next symptoms of cobra poisoning are very characteristic. The patient loses power of speech, of swallowing, of moving the lips, the

tongue becomes motionless and hangs out of the mouth, and the saliva which is secreted in large quantities runs down the face, the patient being equally unable to swallow it or to eject it. It is singular that the striking resemblance of these symptoms to the disease known as glosso-laryngeal paralysis has not been previously noticed. Now, the preponderance of opinion attributes this latter disease to lesion of certain tracts in the medulla oblongata. It cannot, therefore, be thought anything but reasonable to connect both diseases with paralysis of those centres in the medulla oblongata which are so closely associated together, and which are in connexion with the roots of the vagus, the spinal accessory, and the hypoglossal nerves, and the lower nucleus of the facial. But the resemblance does not end here. In both diseases the respiration becomes feebler and feebler, and the victim at last dies suffocated. In other words the lesion in the one case, and the paralysing poison in the other, have invaded the respiratory nucleus so near to the centres they have already destroyed, and have thus rendered the respiratory act difficult, and at last impossible. Lastly, after all the lower centres have been completely paralysed, the one by which connexion is made with the second, fifth, and seventh nerves still acts, and the eye is closed when touched, and even when approached, after the animal is dependent on artificial respiration for life. For these reasons it seems natural to conclude that the principal action of cobra poison on the nervous system consists of an extinction of function, extending from below upwards, of the various nerve-centres constituting the cerebro-spinal system; but in addition to this, there is a special and rapid action on the respiratory and allied nuclei, and it is to this special action that death is to be attributed in most cases of cobra poisoning. In very rapid cases of poisoning, when a very large quantity of poison has entered the circulation at once, instead of the gradual extinction of function of the cerebro-spinal centres, the poison appears to act almost immediately by stopping the action of the respiratory centre. There is, of course, no time then to watch the gradual extension of the influence of the poison on the nervous system. In these cases, the slight stimulation of the centres which almost always precedes the paralysis, instead of being represented by slight irregular contractions of the muscles, is exaggerated into violent clonic convulsions, but they are almost instantly followed by complete paralysis.

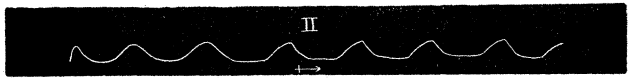
The action of the cobra poison on the respiration is of the deepest importance in the light it throws on the special action of the poison, and its relation to other poisons.

The first change that is noticed in the breathing of an animal after the introduction of cobra poison, is a decided quickening and deepening of the respiratory movements. Sir Joseph Fayrer and Dr. Brunton have shown that this effect is no longer to be perceived after section of both vagi. The inhibitory effect of section of the vagi may be too power-

No. 1.—Tracings of Respiratory Movements of a Fowl under gradual Cobra Poisoning.

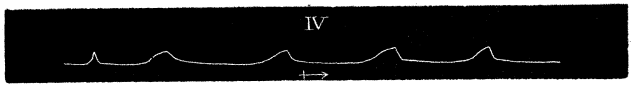


Normal respiration.

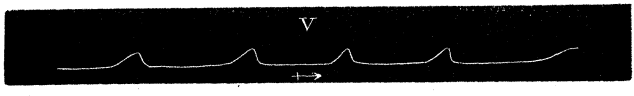


Slightly exaggerated breathing.

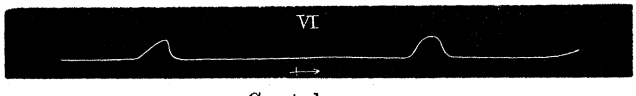
Commencing slowing.



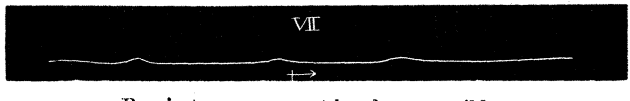
Slowing.



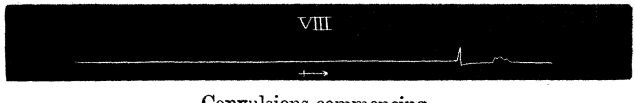
Slowing greater. Sudden and abrupt inspiration and expiration.



Great slowness.

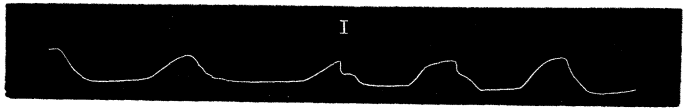


Respiratory movement barely perceptible.

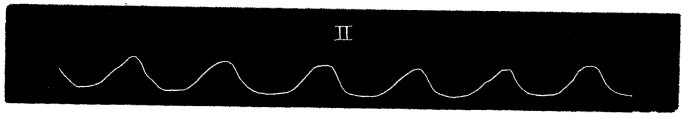


Convulsions commencing.

No. 2.—Respiratory Tracings from a Fowl that died very rapidly from a large dose of Cobra Poison. The tracings are nearly continuous.

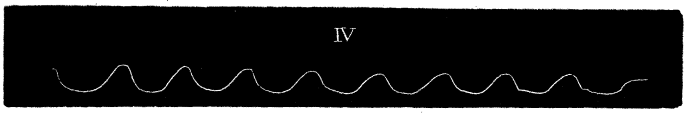


Slow and deep but natural respiration.

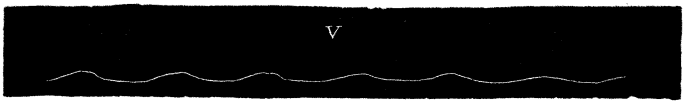


Quickened.

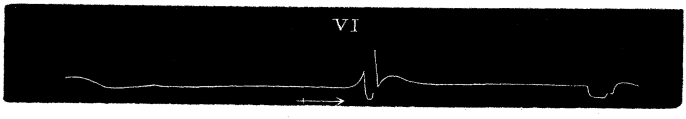
Still more quickened.



Slightly lessened excursus.



Excursus very slight.



Respiration ceased—Convulsions.

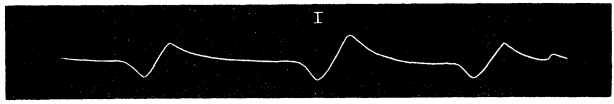
ful to allow of the accelerating action of the poison being perceived. But this quickening, however produced, is merely temporary, and is followed by retardation. The simplest form in which to see the effect of cobra poisoning on respiration is afforded by the common fowl. The stethometric chart, marked No. 1, gives in a concise manner the effects of cobra poisoning when the action is very gradual.

The main points to be noticed are the slight quickening first perceived, and the increase of the excursus. These are followed by rapidly increasing retardation with a certain amount of lessening of the excursus, though the excursus is less affected than the frequency. It is also to be noticed that inspiration becomes sudden and abrupt, and is immediately followed by an expiration equally sudden. The movement that remains is, therefore, peculiarly unfavourable for respiration. In the end the respiratory movement is entirely abolished, and after a pause the convulsions of asphyxia terminate life. Chart No. 2 presents some contrasts of interest. It gives nearly the whole course of the respiratory movements of a fowl from the injection of the poison till death, in a case in which a large amount of cobra poison was given, causing death very rapidly. From it will be seen how very much more pronounced the acceleration is when a large quantity of poison is given, and that when the stage of acceleration is passed the excursus is lessened quite as rapidly as the frequency. The respirations before the administration of the poison bear to the respirations at their greatest degree of acceleration in the tracing IV a ratio of 4 to 7. Chart No. 3 is a series of tracings from a large pariah dog, the acceleration followed by retardation, and the accompanying diminution of excursus are well shown, but the chief points of interest are in the tracings of the occurrence of convulsions; they begin by regular contractions of the inspiratory muscles, in the period of their greatest violence they lose all respiratory character, and then gradually fade away in gentler and gentler attempts at inspiration. The cat is an animal that shows a peculiar power of resisting cobra poison, presenting a marked contrast to the dog. Chart No. 4 is from a cat in whom this resisting power was well shown. After the retardation of respiration is accomplished, it will be noticed that an occasional deep respiration occurred; it is as if the animal, aware of its lessening breathing power, made conscious efforts to assist respiration. A similar feature appears to have been frequently noticed in the human subject under like circumstances. The series of tracings in Chart No. 5 shows graphically the instantly destructive character of cobra poison on the respiratory function. They were taken from a dog on whom the following observations were made.

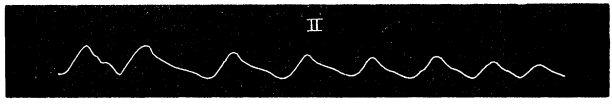
Experiment V.

A powerful pariah dog had 1 cub. centim. of fresh cobra poison

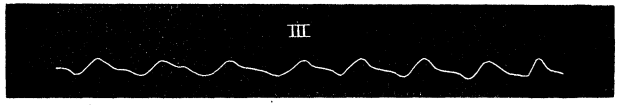
No. 3.—Tracings of Respiratory Movements of a Dog with Cobra Poisoning.



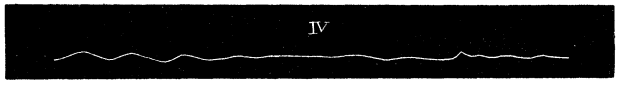
Normal but deep respiration.



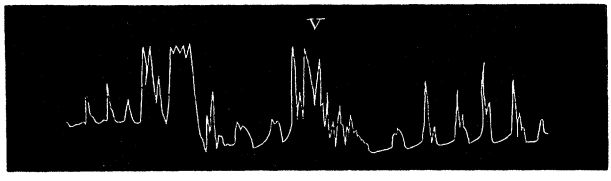
Quickened.



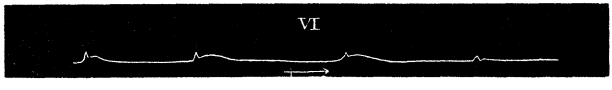
Lessened excursus.



Movement greatly lessened.



Convulsions.



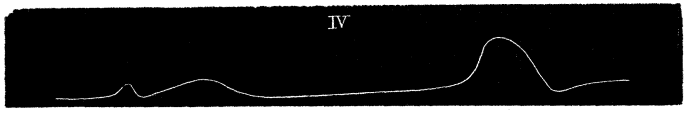
Termination of convulsions.

No. 4.—Tracings of the Respiratory Movements of a Cat under the Influence of Cobra Poison.

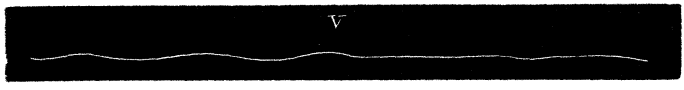
Normal respiration.

Quickened by cobra poison.

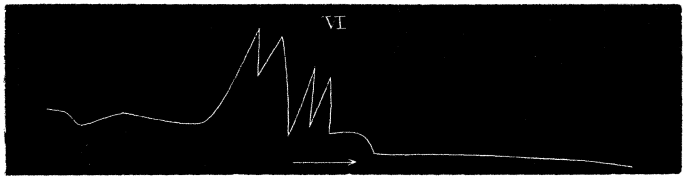
Commencing slowing.



Occasional deep inspiration.



Respiration barely perceptible.



Convulsions.

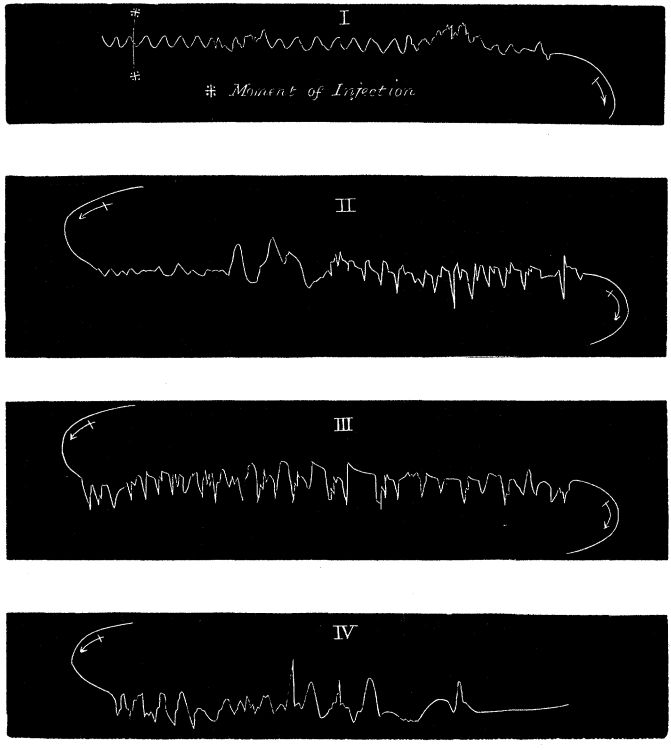
injected into its saphena vein. No change was noticed for thirty seconds, at the end of that time normal respiration abruptly stopped, its place being taken by violent and irregular contractions of the respiratory muscles. The heart's action continued strong, but increased greatly in frequency. Very soon all movement ceased, with the exception of that of the heart, which continued acting for about ten seconds longer. The whole time from injection to death was under 100 seconds.

Chart No. 5 is a continuous tracing of the respiration of this animal from the administration of the poison till death. It will be seen that the normal respiratory rhythm is suddenly displaced by violent alternate upheavings and depressions of the chest walls, and that then all movement ceases suddenly. But it will be noticed that the convulsions, though of course irregular, have a distinct respiratory character. When cobra poison enters the blood rapidly and in large quantity, its first action is to stimulate the respiratory centre so as to cause more irregular respiratory movements, and then one might almost say simultaneously it paralyses it completely, the heart acting for a short time longer. This, however, is not the usual course of events. A much more common method is the one depicted in the stethometric charts, Nos. 1, 2, and 3, where a fair amount of cobra poison is gradually absorbed, as from the bite of a cobra, and where there is in consequence gentle primary acceleration of respiration, with gradual lessening of its rapidity and excursus, and death with convulsions. In still more gradual cases, the primary acceleration is very faint, and the diminution of the respiratory function very gradual, and there is often an attempt—half conscious—to fight against the poison by occasional deep inspirations; at last respiration is arrested, and generally without convulsions, the heart stops after a short interval. This appears to be the usual course in man.

On the circulation, cobra poison cannot be said to exercise a very great influence. The heart nearly always acts for some little time after respiration has ceased, and if the place of normal respiration be supplied by artificial means, the heart will continue acting for very many hours. On the temperature, also, no decided effect can be determined. There may be a slight rise or even fall, but in several cases, watched carefully for the purpose, no change was observed. Nor is there any noticeable effect on the special senses, the poisoned animal appears to hear and see perfectly. The pupil of the eye is also unaffected, and answers to light. If life be preserved by artificial respiration, the pupil remains somewhat contracted. I once noticed the pupil remain contracted after death from cobra poisoning, in which long continued artificial respiration had been resorted to.

On secretion, generally, cobra poison has great influence; nearly all secreting tissues are stimulated by it. The lachrymal glands act

No. 5.—Continuous Tracing of Respiratory Movements of Dog from the moment of Intravenous Injection of Cobra Poison till Death.



freely, and salivation is a most marked and constant symptom. It is rarely absent either in men or in dogs. Saliva often runs in streams from the mouth. The whole of the mucous tract is also in an active state of secretion. After the stomach has been thoroughly emptied by vomiting, the animal will often bring up repeatedly large quantities of mucus, and mucous discharges are also frequently evacuated from the rectum. The respiratory mucous membrane is also similarly affected. Little can be known of its action on the liver or kidneys, the time of observation being so short; in the more chronic cases the kidneys often act freely, but sometimes there is a diminution of urine.

There is but little evidence of the effect of cobra poisoning on the blood. The blood is clearly the carrier of the poison to the system, and it is necessary for it to be some short time present in the blood

before a physiological effect is produced, except in those rare cases in which an overwhelming quantity of poison is injected into a vein directly. In man, after cobra poisoning, the blood is nearly always found incoagulable, though in animals, especially dogs, the blood generally coagulates as usual. That there is no great change in the blood is evident from the fact that, when an animal has survived the nerve symptoms produced by cobra poison, it is found to be quite well and to suffer no further inconvenience from blood poisoning or other causes.

Experiment VI.

A large pariah dog had 5 mgrms. of dried cobra poison dissolved in water injected into its hind leg.

12.12. P.M. Injection.

4 P.M. Vomited.

7.30 P.M. Salivated. Looks depressed.

10.15 P.M. Still depressed; salivation slight; respirations 14.

12.5 A.M. Still salivated; very depressed.

8 A.M. Very weak, hardly able to walk; all the legs equally affected.

8.25 A.M. Respirations 12; pupils somewhat dilated, but contract to light. Site of injection very red, hot, and swollen.

11.30 A.M. Respirations 15; frothy salivation.

12 P.M. Rectal temperature, 39° C.

12.38 P.M. Pulse about 120, extremely irregular; chewing movement of jaw and lips; salivation considerable.

2.21 P.M. Better, can walk, salivation ceased; ate sparingly.

7.30 P.M. Recovering fast. Urinated—no albumen in the urine.

8 A.M. Seems quite well. Purulent discharge from the site of the injection; respirations 20, pulse 120.

The dog was kept under observation but remained quite well. The normal pulse rate was 90 and respirations 28.

Here, though the most severe nerve symptoms were present, when they passed off the animal was quite well, and suffered no further inconvenience. The same also occurs in the human subject. Dr. Vincent Richards relates a case of cobra poisoning, from his own observation, in which a man lost completely all power over his legs, was unable to speak, to move the lips, or to swallow, and where there was profuse salivation, and yet after a few hours complete recovery ensued, the man by the next day being well. The microscope also gives no evidence of structural change in the blood. In cobra poisoning, also, albumen in the urine is unknown. In animals that have suffered most severely from nerve symptoms in which I have tested the urine, albumen has not been present in a single case either fatal or non-fatal. When, however, artificial respiration has been performed for some

time after apparent death, it is not unusual for blood to be present in the urine; but if the kidneys are examined in these cases the wonder will be, not that blood was found in the urine, but that any urine was secreted at all, so great is the renal congestion. This circumstance can, therefore, hardly be taken as evidence of blood change, and coupled with the fact of the rapid recovery that ensues in cobra poisoning after the nerve symptoms have passed off, leads us to the conclusion that all cases of death from cobra poisoning have their origin in the direct action of the poison on the nerve-centres.

II.—*The Physiological Effects of the Poison of the* Daboia Russellii.

The *Daboia Russellii*, or Russell's viper, is selected as being an extremely deadly member of the viperine family, and is the best known of the Indian vipers. The following experiments show the course of the symptoms produced by this snake.

Experiment VII.

A somewhat small pariah dog had a pulse of 88 per minute, respirations 30, rectal temperature 39° C.

3.24 P.M. Bitten by a *Daboia Russellii* in the thigh.

3.28 P.M. General muscular spasm of the most violent character, all parts of the body taking part. The animal fell down and rolled about in convulsions, even the muscles of the eyeballs being affected —jerking the eyes about in the strangest way.

3.30 P.M. Convulsions ceased, complete paralysis of both hindlegs, tries to stand, but can only rest on his forelegs.

3.31½ P.M. Contractions of muscles of eyeball continue; respirations very shallow, 68; pupils somewhat dilated.

3.37 P.M. Muscles of eyeball at rest; seems utterly prostrate and unable to move; respirations 40, chiefly abdominal.

3.40 P.M. Respirations 56, shallow, occasionally a deep sigh.

3.44 P.M. Pupils somewhat dilated but contract to light.

3.47 P.M. Respirations 67, about every tenth one is very deep.

3.53 P.M. Pulse 156.

3.56 P.M. Moaning. Temperature 39°·5 C.

4.1 P.M. Respirations 32; sighing and moaning, lying down paralysed, sanious discharge from the rectum, pupils widely dilated.

4.9 P.M. Respirations 36.

4.12 P.M. Respiration reduced to a quick inspiratory spasm, followed by relaxation.

4.15 P.M. Respirations 16.

4.17 P.M. Dead. Temperature 39°·4 C.

Experiment VIII.

A small quantity of fresh daboia poison (about 0·1 cub. centim.) was injected into the thigh of a pariah dog.

12.20 P.M. Injection.

12.25 P.M. Very lame in the leg in which the injection was made.

12.52 P.M. Very drowsy, unsteady in walking.

12.54 P.M. Pupils widely dilated; iris only just visible.

12.56 P.M. Moaning.

1 P.M. Panting; raised himself to a standing posture, but his legs gave way under him, and he fell to the ground.

1.5 P.M. Violent respiratory movements; respirations 28.

1.6 P.M. Still able to move his head freely.

1.15 P.M. Tries occasionally to rise.

1.17 P.M. Moaning.

1.20 P.M. Moderately loud screams.

1.25 P.M. Convulsions.

1.34 P.M. Dead.

Experiment IX.

A pariah dog had 5 mgrms. of dried daboia poison injected subcutaneously, dissolved in 1 cub. centim. of water.

12.56 P.M. Injection.

1.54 P.M. Quite well. Respirations 44.

7 P.M. Ate freely.

6 A.M. Looks a good deal depressed. Respirations 48 a minute.

7.20 A.M. Pupils contracted, panting; respirations 60 a minute.

8.15 A.M. Panting excessively.

8.30 A.M. Passed a sanious discharge per rectum.

8.45 A.M. Respirations 80, excessive dyspnœa, blood oozing from mouth.

9.30 A.M. Dead.

After death the fatal dyspnœa was found to be dependent on œdema of the lungs.

Experiment X.

A small pariah dog was bitten in the thigh by a small *Daboia Russellii*.

12.34 P.M. Bitten.

12.35 P.M. Slightly panting.

12.35½ P.M. Fell over suddenly in violent convulsions, the hind-legs being especially strongly convulsed, pupils contracted.

12.36 P.M. Slight attempt at respiration.

12·39 P.M. Dead.

From these experiments it will be seen that there is a great

diversity in the symptoms between different cases of daboia poisoning. The animal may fall down at once in convulsions and expire as in Experiment X, or it may never have any nerve symptoms at all, but die many hours after from a remote affection as in Experiment IX.

The following experiments throw light on the causes of these differences.

Experiment XI.

A pariah pup had about 0·1 cub. centim. of fresh daboia poison injected subcutaneously into the shoulder.

12.52 P.M. Injection.
1 P.M. Vomiting.
1.4 P.M. Can stand but cannot walk.
1.8 P.M. Cannot stand; moaning.
1.11 P.M. Slight sanious discharge from the rectum.
1.15 P.M. Short rapid respiration with an occasional deep inspiration.
1.30 P.M. Respiration very slight.
2.6 P.M. Dead.

Experiment XII.

A pariah pup of the same litter as the one in the last experiment, and as nearly as possible of the same size, had 0·3 cub. centim. of fresh daboia poison injected into its shoulder subcutaneously.

1.31 P.M. Injection.
1.35 P.M. Fell over in convulsions.
1.37 P.M. Unable to stand.
1.45 P.M. Lying down, is quite paralysed, but occasionally groans.
2.1 P.M. Totally unconscious.
2.16 P.M. Respiration failing.
2.29 P.M. Dead.

In these two dogs, which were of the same age and size, the daboia poison was injected into exactly the corresponding part of each, and subcutaneously; but, in the latter experiment, three times as much poison was injected as in the former case. This was the only difference in the two experiments, and the result was that the animal which had the larger quantity of poison suffered in four minutes from violent convulsions, and from that time was quite paralysed; whilst the other only gradually became paralysed, and had no sign of convulsions at all. The poison in both cases was from the same viper, and extracted at the same time. In the same way it can be proved that an equal quantity of poison injected into two animals of different sizes will kill the small one almost instantly in convulsions, but will only cause death after a long interval, and without violent symptoms, in the larger one. Daboia poison, therefore, commences its constitutional

action by causing convulsions, though a certain proportion of animals will escape them through an insufficient quantity of poison having been injected. It should be stated here, that the preliminary effects of the poison differ only in degree from those of cobra poisoning. The local pain appears to be peculiarly acute, and the accompanying inflammation exceedingly severe; and, from the amount of blood-stained serum effused, might almost be said to have a "specific" character.

The contrast between the effects of the two poisons comes out singularly clearly when birds are selected as the subject of experiment.

Experiment XIII.

1 cgrm. of dried daboia poison was dissolved in 1 cub. centim. of distilled water, and the solution was injected with great care just beneath the skin of the leg of a fowl.

3.10 P.M. Injection.
3.11 P.M. Slightly lame in the leg in which the injection was made.
3.12 P.M. Violent convulsions.
3.12¾ P.M. Dead.

Experiment XIV.

1 cgrm. of dried cobra poison dissolved in 1 cub. centim. of distilled water was injected just beneath the skin of a fowl.

3.30 P.M. Injection.
3.34 P.M. Drowsy.
3.36 P.M. Beak resting on the ground; unable to stand; eyelids closed, pupils contracted.
3.37 P.M. Unable to lift its head from the ground.
3.40 P.M. Convulsions.
3.42 P.M. Dead.

In these two experiments the greatest care was taken to ensure the conditions being exactly the same; yet, with daboia poison, the bird died in two and three-quarter minutes with only the occurrence of convulsions; while, with cobra poison, it died after twelve minutes, and went through the regular course of paralysis. The convulsions, therefore, are not dependent upon the injection of a large quantity of poison directly into a vein. The next point to be determined is whether the convulsions are due to asphyxia, like those that often occur after the cessation of respiration from paralysis in cobra poisoning.

Experiment XV.

A fowl had its trachea opened, and a tube in connexion with a bellows for artificial respiration inserted into it. 2 cgrms. of dried

daboia poison in solution were then injected subcutaneously into its leg. Immediately on the injection being completed, artificial respiration was commenced.

2.23 P.M. Injection.

2.23¼ P.M. Artificial respiration commenced.

2.26 P.M. Convulsions, in which the bird died.

Here, though ample means were employed to keep the blood oxygenated, the bird died at once in convulsions. The convulsions of asphyxia that occur after the paralysis of cobra poisoning are removed by artificial respiration. It is clear, therefore, that the convulsions of daboia poisoning are *primary*, and in no way due to defective aëration of the blood.

The convulsions are exceedingly violent in character, the whole of the voluntary muscular system being affected, even to the muscles of the eyeball. Sometimes, but rarely, the convulsion is tonic, the muscles clasping, as it were, the frame of the victim. The convulsions may be due either to the direct action of the poison on the muscles, or on the muscular terminations of the nerves, or on the central nervous ganglia. The following experiment determines this.

Experiment XVI.

A fowl was placed under the influence of chloroform, and its right crural nerve divided; the left crural nerve was then isolated, and a ligature placed round all the other structures so as completely to obstruct the circulation, but the nerve was left uninjured. The moment the bird became conscious, a small quantity of solution of daboia poison was subcutaneously injected into the tissues at the back of the neck. Almost directly after, convulsions occurred, but, in these convulsions, the muscles supplied by the right or divided nerve took no part, remaining perfectly flaccid, while the muscles of the left leg were violently tetanized. Here the muscles of the right leg which were freely supplied with the poisoned blood, but the nerve to which was divided, escaped the convulsions; while those of the left leg, which had no contact with the poisoned blood, but whose nerve was still in communication with the central nervous system, took part in them. It is evident, therefore, that the convulsions depend upon the direct irritation of the central nervous system by the poison, and in no way on the contact of the poison with the muscles or with the terminations of the nerves.

These *primary* convulsions occur in from one to ten minutes after the infliction of the bite, and they may have any degree of severity, from those producing almost instant death to merely a few muscular twitchings. The course of the symptoms after the convulsions also varies. Respiration may never be thoroughly re-established, the

animal dying very soon, or respiration may recommence, but complete general paralysis is found to have supervened, and the animal dies of consequent asphyxia. It may happen that, for a short time, the victim recovers completely from the convulsions, walking about for a few minutes as if nothing had occurred, but the fatal paralysis is only delayed for a short time.

It appears as if daboia poison acted somewhat differently in different classes of the animal kingdom. Birds are most easily affected, and next to them come the Lacertilia.

Experiment XVII.

About 0·4 cub. centim. of fresh daboia was injected beneath the skin of the upper surface of the tail of a large specimen of *Calotes versicolor*.

The respirations were 54 per minute.

3.49 P.M. Injection.
3.51 P.M. Convulsions.
3.52 P.M. Quite paralysed.
3.53 P.M. Respirations 12 per minute.
3.56 P.M. Respirations 4 per minute.
3.59 P.M. Dead.

Mammals also are very easily affected by the convulsion-producing property of the poison, whereas the Amphibia only have symptoms of gradual paralysis.

Experiment XVIII.

A medium sized frog (*Rana tigrina*) had 1 cgrm. of dried daboia poison in solution injected into its dorsal sac.

1.12 P.M. Injection.
4.30 P.M. Violent respiratory efforts.
4.39 P.M. Fast becoming paralysed.
4.53 P.M. Completely paralysed.
5.11 P.M. Dead.

Though birds are so sensitive to the agent in the poison that produces these nervous discharges, that it is difficult to poison them at all without the occurrence of primary convulsions—1 mgrm. often producing them—yet by heating daboia poison in solution to 100° C. it loses completely the power of causing this symptom even in them.

Experiment XIX.

5 cgrms. of dried daboia poison in solution were heated for a short time to 100° C. The solution was then injected subcutaneously into the leg of a fowl.

12.27 P.M. Injection.
12.34 P.M. Seems drowsy; eyes closed.

12.45 P.M. Breathing rapid.
12.47 P.M. Respiration failing, comb becoming purple.
12.49 P.M. Lying down, head drooping.
12.50 P.M. Fallen over on its side; respiration fainter.
12.53 P.M. Gasping.
12.56 P.M. Convulsions.
12.58 P.M. Convulsions lessening.
12.59 P.M. Dead.

Here, though the amount of poison injected was more than fifteen times that required to cause almost instant death from convulsions, simple exposure to a heat of 100° C. completely altered the symptoms. They were those of gradual paralysis which caused the respirations to cease, and the convulsions which occurred at the end of the paralysis were simply the expression of carbonic acid poisoning. They were secondary, and no longer primary. Even with the abolition of primary convulsions, however, the symptoms differed materially from those of cobra poisoning.

Should primary convulsions not occur the history is that of advancing paralysis. The respirations and pulse become greatly accelerated, and there is gradual loss of power in all the limbs; vomiting may occur. Sanious discharges issue from the rectum and other parts. The pupils are usually widely dilated, and the respirations become less and less, and may cease with or without convulsions.

But there is a third form of death from daboia poisoning, quite unlike anything that is seen in cobra poisoning. It occurs in those cases in which but a very small quantity of poison indeed has been injected. The animal has very few nervous symptoms, very likely none at all; but on the second day he appears ill, refuses food, has diarrhœa, his urine contains albumen, and he may linger on in this state for days, dying exhausted, or some acute complication may supervene as in Experiment IX, causing death rapidly. In that case it was an œdematous condition of the lung that proved fatal, whereas in a case recorded in the "Indian Medical Gazette" (June 1, 1872), it was a hæmorrhagic condition of the system generally. The snake was evidently from its description the *Daboia Russellii*, or its congener the *Echis carinata*.

A Mahomedan, forty years of age, was bitten by a snake on the finger. The bite was soon after incised, and stimulants given. The hand and arm became much swollen, and on the same day he passed blood by the rectum, with his urine, and he also vomited blood. The next day he was sick, and was still passing blood from all the channels. In this state he remained eight days, losing blood constantly, and dying in consequence exhausted on the ninth day.

In this case no symptoms occurred for many hours, but when they did supervene they increased in severity till the man died from

hæmorrhage. There was no paralysis or any other special symptom. The case presents in every detail a complete contrast to Dr. Richard's case of cobra poisoning, where for some hours the patient was in the greatest danger from nerve symptoms, and yet the next day made a full and complete recovery.

This chronic form of daboia poisoning occurs whenever only a small quantity of daboia poison is injected.

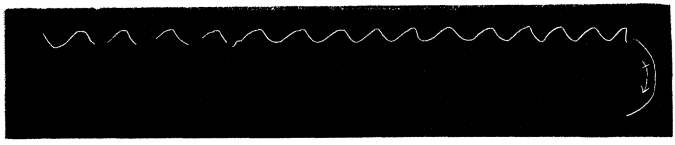
Experiment XX.

A cat had about 5 mgrms. of daboia poison in solution injected into its leg. No symptom was noticed for about twelve hours, when it became ill, refused food, had diarrhœa, and remained in this state till the fourth day, when it died.

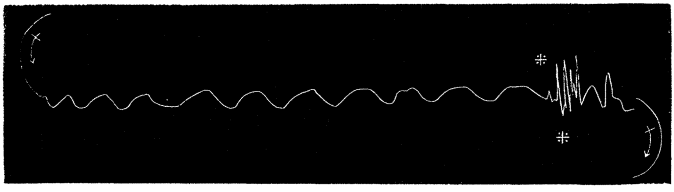
In regard to the paralysis caused by daboia poison, there a few points to be observed. It certainly supervenes earlier than in cobra poisoning, but it does not extinguish the respiratory function nearly so soon. Paraplegia is occasionally noticed in dogs, especialiy if the hind legs have been much convulsed. It is probably due to exhaustion of the conducting elements of the cord by the violence of the convulsion-producing discharges. Care, however, should be taken not to confound it with inability to use the hind quarters occasioned by the pain of the local injection, which appears to be peculiarly severe. Again, the paralysis of the lips, tongue, larynx, and pharynx, which is so marked a feature in cobra poisoning, is absent in daboia poisoning. The tongue, instead of being pendulous, is retracted, and the larynx, so far from being paralysed, gives utterance to loud screams, often as long as life lasts, as in Experiment VIII. It is as if the poison exerted its paralysing influence on the main motor tract, and had not that marked affinity for the respiratory and allied centres that cobra poison has; a conclusion borne out by the way in which it commences its action, and by the time it takes to extinguish the respiratory function.

The course of the respiration in daboia poisoning is of necessity as varied as there are modes of death from the poison. Chart No. 6, from a fowl, gives a typical example of respiration when the primary convulsions are fatal. It contains nearly all the chest movements from the moment of injection till death. The first sixteen respirations are normal, the excursus is then slightly increased, and a slight retardation is to be observed; then without further warning, the most violent convulsions took place, and with them life was extinguished. It affords corroborative evidence of the fact, that asphyxia has nothing to do with the causation of the convulsions, as respiration is perfectly well performed up to the moment of their occurrence. Chart No. 7 is from a fowl that was poisoned by daboia poison, which had been heated in solution to 100° C., and so deprived of its power of causing

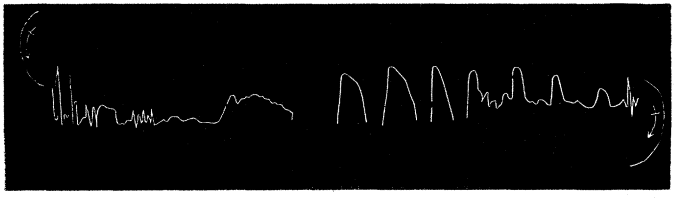
No. 6.—Tracing of Respiratory Movements in Fowl under the Influence of Daboia Poison.



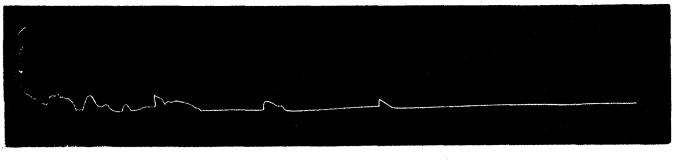
Respiration after the injection of the poison, but still normal.



Slightly exaggerated breathing, suddenly interrupted by convulsions.



Convulsions.

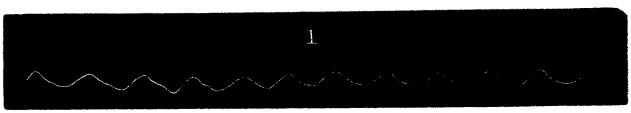


End of convulsions.

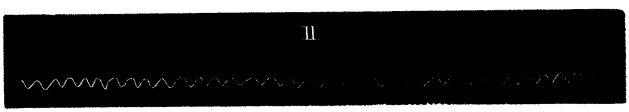
convulsions. The acceleration of the respiratory movements is most remarkable, as is also the way in which the excursus is exaggerated and maintained; it is only at the very end of life that the respiratory movement is diminished.

Chart No. 8 is from a dog, to which sufficient poison was not given to cause convulsions. The very much greater acceleration of the

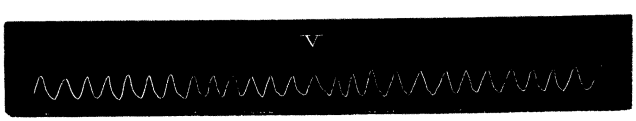
No. 7.—Tracing of Respiratory Movement in a Fowl under the Influence of Daboia Poison that had been heated to 100° C.



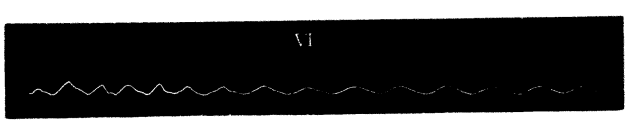
Normal.



Great acceleration.



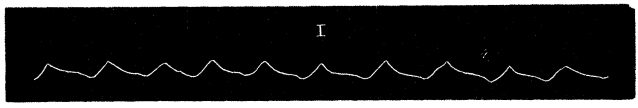
Great acceleration with increased excursus.



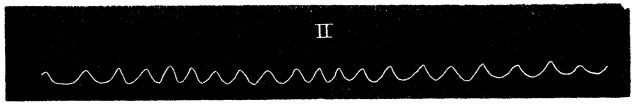
Lessened excursus.

respiration than can be caused by cobra poisoning is seen, the respirations in I and III being as two to five. But in IV, a singular condition is to be noticed, that is exceedingly common in daboia poisoning. It is a peculiarly deep inspiration, followed immediately by expiration. It occurs generally once in every ten or fifteen respirations, and at a

No. 8.—Tracings of Respiratory Movements of Dog under Daboia Poisoning.

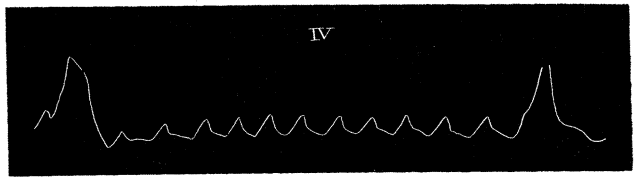


Normal respiration.

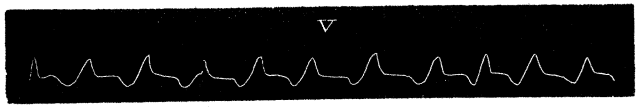


Acceleration.

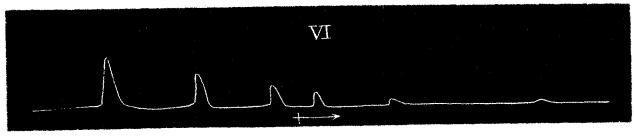
Respiration still quicker.



Irregular respiration.



Respiration slowing.



End of respiratory movements.

time when the animal is often profoundly unconscious. It is quite characteristic of daboia poisoning, though it is not always present, sometimes the whole body taking part in the movement. After the primary convulsions have occurred, it is exceedingly rare for death to be preceded by the convulsions of asphyxia, the respiratory function appearing gradually to fade away, as in the tracing VI.

In its influence on the temperature and circulation, daboia poison does not differ materially from cobra poison. But while cobra poison has no effect on the pupil of the eye, wide dilatation is always, or nearly always, to be seen during some stage of daboia poisoning. The iris is sometimes barely visible during the primary convulsions. Over secretion, daboia poison has also some power, though less than cobra poison, and the mucous discharges are nearly always largely mixed with blood. But salivation, the constant accompaniment of cobra poisoning, is almost unknown in daboia poisoning. Out of a large number of experiments, I have only seen it once, and in this case it was by no means marked, and might easily have been overlooked, unless special attention had been paid to its occurrence.

In daboia poisoning there is a good deal of evidence to be considered as to the effect of the poison on the blood. It is almost universally found after death, that the blood has been rendered uncoagulable, the only exceptions being when the animal has died almost instantly in convulsions, or in those cases where the animal has struggled for a long time against the poison, when the coagulation will be found to be imperfect, as if an attempt towards restoring the normal condition of the blood had been made. Even in rapid cases of daboia poisoning, we have proof that the relation of the blood to the tissues is altered. In Experiment VII a sanious discharge occurred thirty-seven minutes after the infliction of the bite. So grave are these changes in the blood, that they are by far the most frequent causes of death in daboia poisoning (Experiments IX and XX). The hæmorrhages chiefly take place from the rectum and kidneys, but I have seen them from the mouth, and even the skin.

In cobra poisoning albuminuria is unknown, but in every case of daboia poisoning in which symptoms were present, and in which six hours elapsed before death, I have detected albumen in the urine. It is generally not in large quantity, but it is quite unmistakeable.

Experiment XXI.

A solution of about 5 mgrms. of daboia poison was injected into a dog.

4 P.M. Injection.

8 A.M. No symptom.

4 P.M. 5 mgrms. more of daboia poison injected.

9.30 P.M. Affected; respirations 50; pupils widely dilated.

6 A.M. Depressed and ill.
3 P.M. Passed some albuminous urine.
9 P.M. Diarrhœa.
12 P.M. Urine albuminous; still very ill.

As it was clear that the animal would suffer the usual prolonged course of blood-poisoning, more poison was injected to shorten life.

1.30 P.M. Injection.
3.17 P.M. Cannot stand.
3.20 P.M. Respiration failing.
3.29 P.M. Dead.

There is evidence, therefore, that daboia venom is a most severe blood poison, producing death when not administered in sufficient quantities to cause serious nerve symptoms, and that it is even possible for death to occur early from this cause alone, nerve symptoms not having supervened.

Conclusions.—The regular course of the symptoms of cobra poisoning is slowly advancing general paralysis, coming on after a well-marked interval without symptoms, with special paralysis of the lips, tongue, larynx, and pharynx, and complete destruction of the respiratory function. Death is often attended by convulsions, which depend on asphyxia.

Daboia poison commences its action by inducing violent general convulsions, which are often at once fatal, or may be followed immediately by paralysis and death, or may also be for a short time recovered from, paralysis and death following later. These convulsions do not depend on asphyxia, and they may be absent if only a small quantity of poison has been injected. The paralysis that succeeds is general, and lasts a considerable time before respiration is extinguished, and there is no special paralysis of the lips, tongue, larynx, and pharynx.

Cobra poison very quickly destroys respiration; after slight acceleration the respiration becomes slower, and the excursus is lessened.

Daboia poison at first quickens the respiration very much more than cobra poison does, and the lessening of the excursus, and the retardation of the respiratory movements do not occur so soon.

Daboia poison invariably kills birds and reptiles at once in convulsions; cobra poisoning only after paralysis.

The effect of cobra poison on the pupil is so small as to be a matter of doubt; daboia poison causes wide dilatation of the pupil. Salivation is a constant symptom in cobra poisoning; it is exceedingly rare in daboia poisoning.

The effect of cobra poison on the blood is not great, sanious discharges are rare, albuminuria has not been seen, and should the patient not die from the paralysis, recovery is perfect and complete, no symptom being left in a few hours.

In daboia poisoning, sanious discharges are the rule; albuminuria is always found, should the victim live any time, and after the nerve symptoms pass over, the subject has to go through a period of blood poisoning little, if at all, less dangerous than the primary symptoms, from which he may die as late as the end of the second week.

Lastly, the physiological properties of daboia poison undergo great change by its being heated to 100° C. in solution, whereas cobra poison remains unaltered.

II. "On Pendent Drops." By A. M. Worthington, M.A. Communicated by Professor B. Stewart, F.R.S. Received May 16, 1881.

[Plate 7.]

About two years ago I was led to examine the forms of pendent drops of liquid by a method of great simplicity, which seems capable of being used with considerable accuracy for determining the value of the surface tension.

Previous observers, so far as I am aware, have observed only the weight of drops which fall, and, making this the basis of calculation, have endeavoured to find the influence on the size of such drops, of the rate of influx of liquid, shape of terminal, as in the case of Dr. F. Guthrie,* or to ascertain the value of the surface tension of the liquid as in the case of Professor Quincke† and M. Dupré.‡

Under no circumstances, however, is the weight of the drop which falls exactly the weight of the volume which it is necessary to know in order to ascertain the value of the surface tension, though under certain circumstances it approximates thereto. Hence we find that Prof. Quincke rejects this method of finding the tension, or recommends it only where other methods fail;§ albeit all his results obtained by this method are vitiated by an assumption to which I shall have occasion to draw attention. The principle of the method which I will now describe is simply to project a magnified image of a drop pendent from a cylindrical tube on to a screen, and there to trace its outline at any required stage of its development.

A vertical cylindrical glass tube, A, whose lower end is ground truly flat and with a sharp edge, communicates by means of a bulb or wider tube, B, and a piece of india-rubber or lead tubing, C, with an air-tight syringe, D, some 10 or 12 feet away. For the usual syringe piston a cup of mercury, E, is substituted, which can be gradually

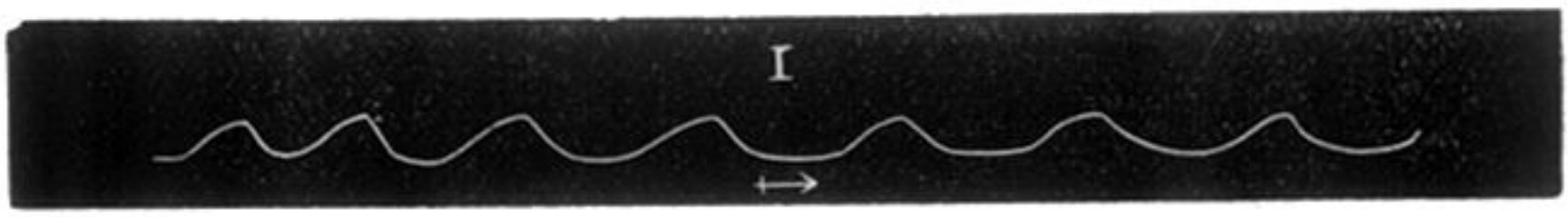
* "On Drops." By Dr. F. Guthrie. "Proc. Roy. Soc.," vol. 13, p. 444.

† "Poggendorff's Annalen," vol. cxxxv, p. 621. "Phil. Mag.," 1869.

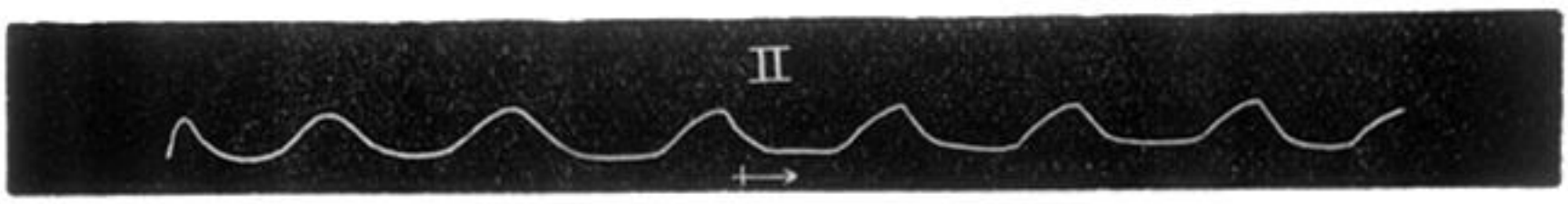
‡ "Théorie Mécanique de la Chaleur," p. 332.

§ *Loc. cit.*, p. 637, § 12.

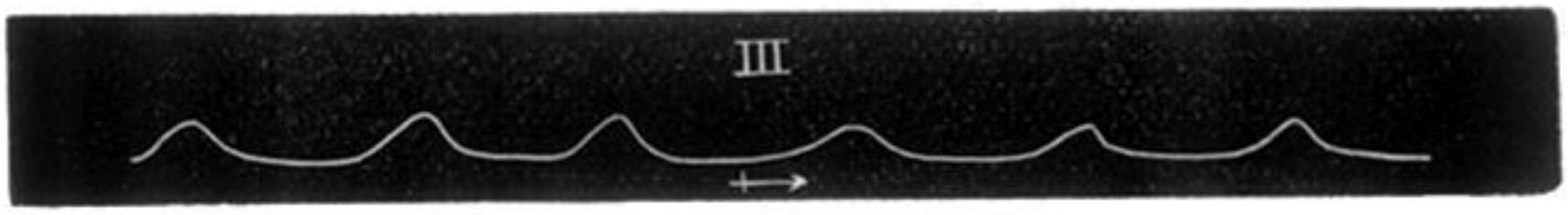
No. 1.—Tracings of Respiratory Movements of a Fowl under gradual Cobra Poisoning.



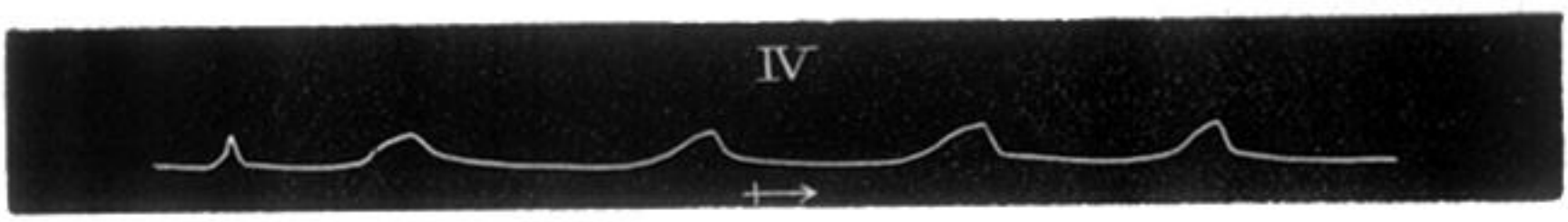
Normal respiration.



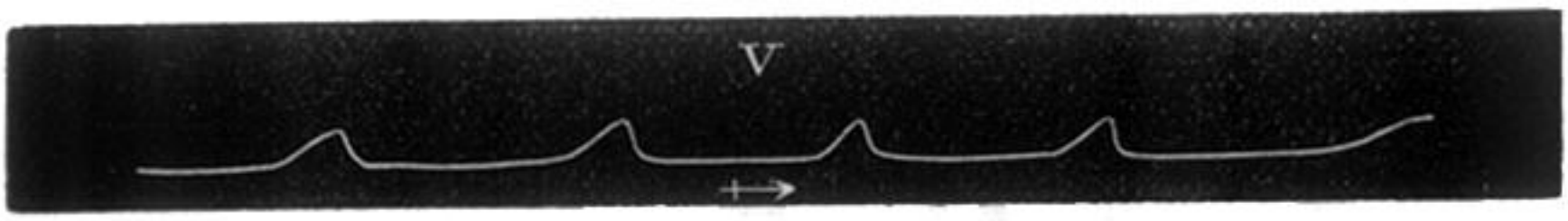
Slightly exaggerated breathing.



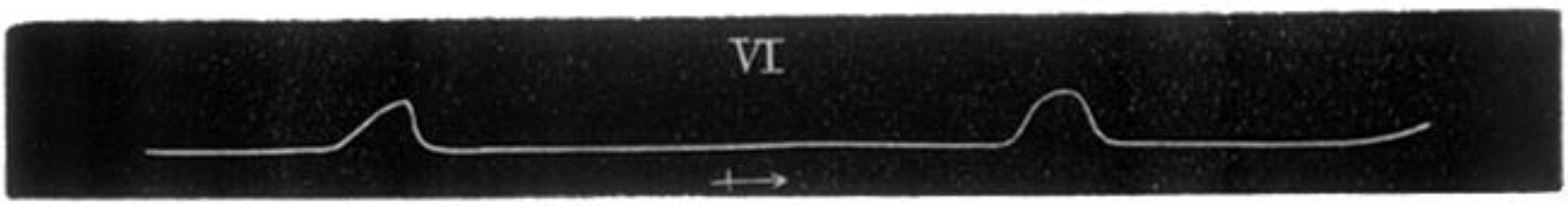
Commencing slowing.



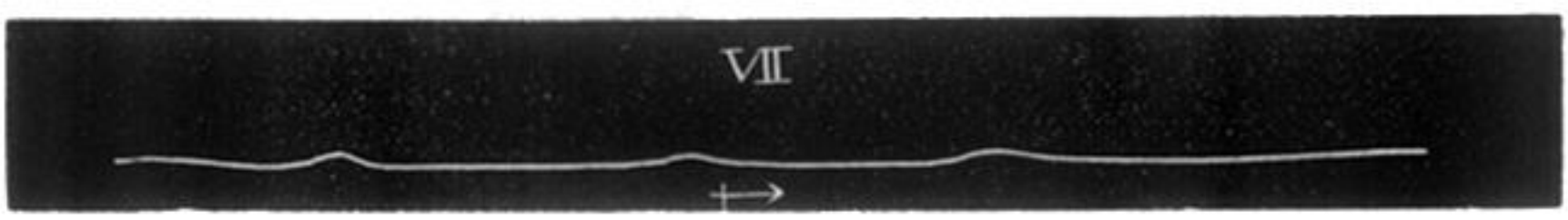
Slowing.



Slowing greater. Sudden and abrupt inspiration and expiration.



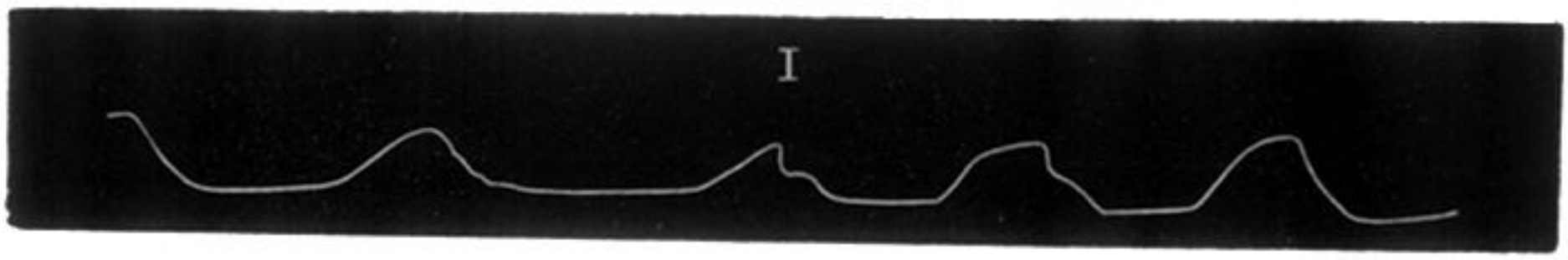
Great slowness.



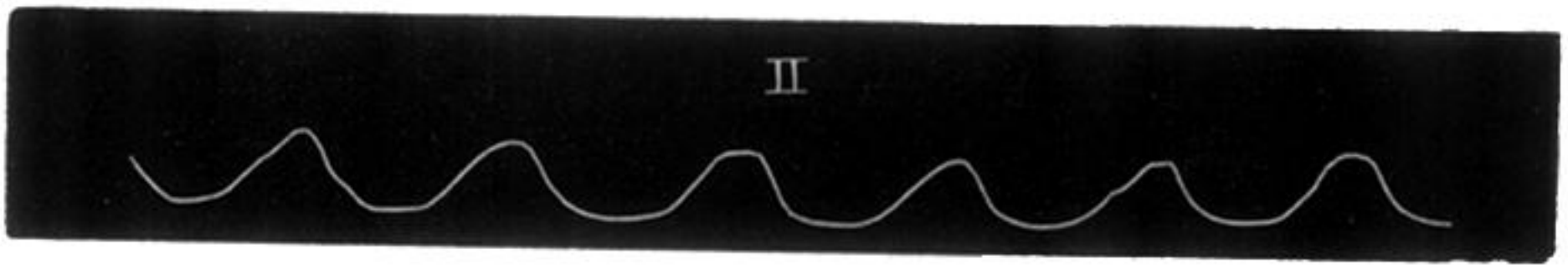
Respiratory movement barely perceptible.

Convulsions commencing.

No. 2.—Respiratory Tracings from a Fowl that died very rapidly from a large dose of Cobra Poison. The tracings are nearly continuous.

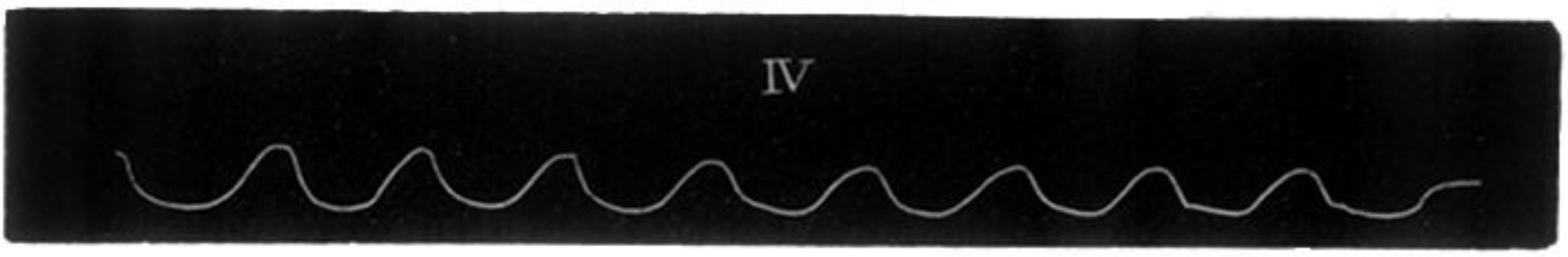


Slow and deep but natural respiration.



Quickened.

Still more quickened.

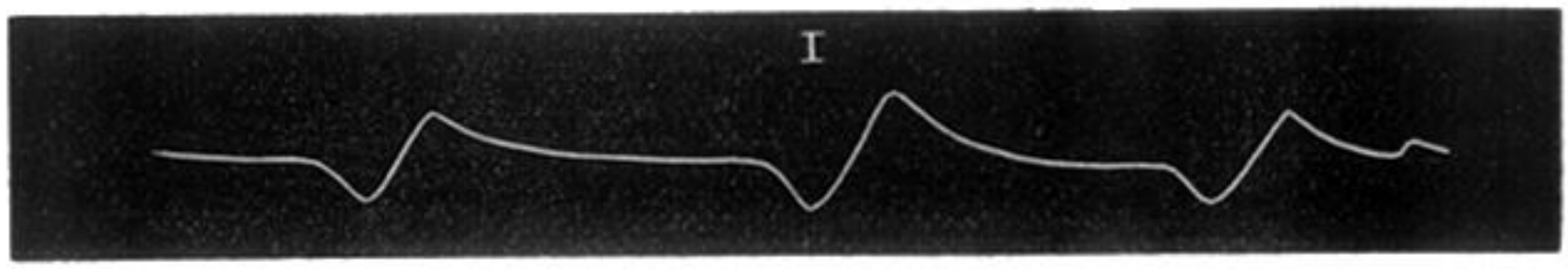


Slightly lessened excursus.

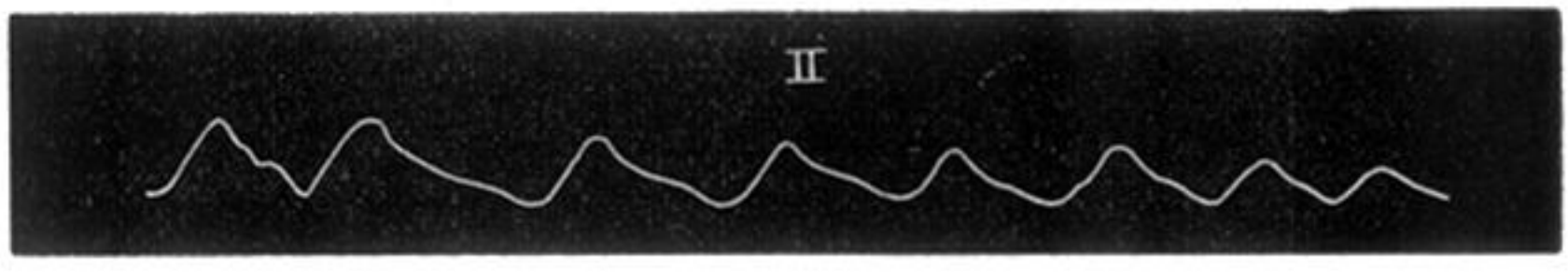
Excursus very slight.

Respiration ceased—Convulsions.

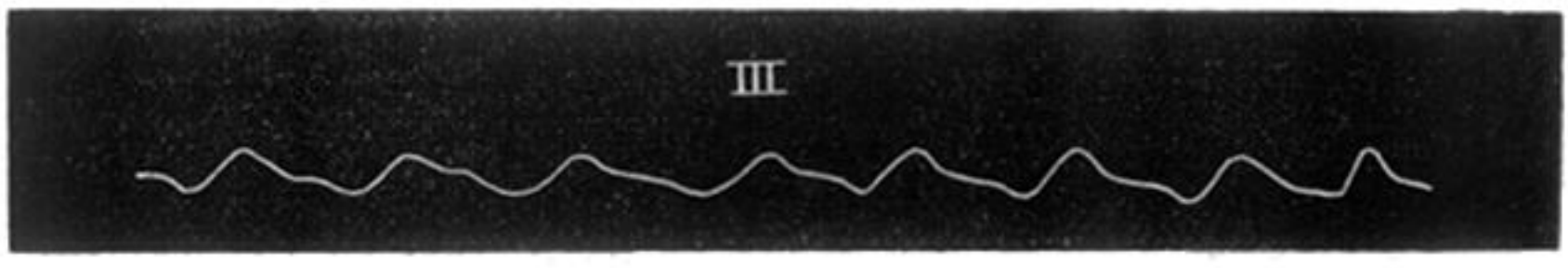
No. 3.—Tracings of Respiratory Movements of a Dog with Cobra Poisoning.



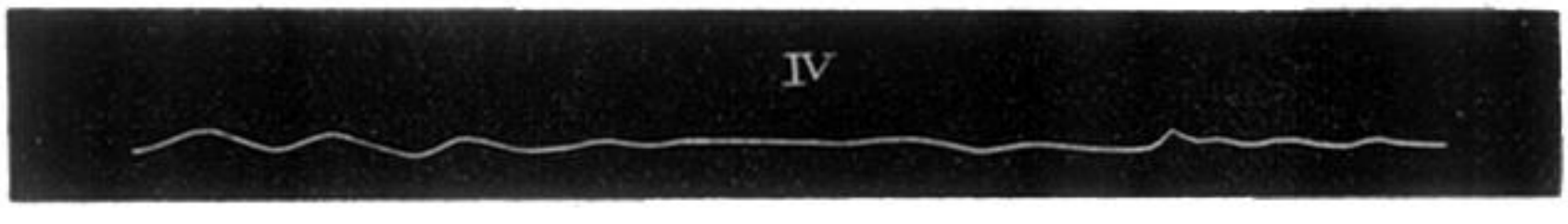
Normal but deep respiration.



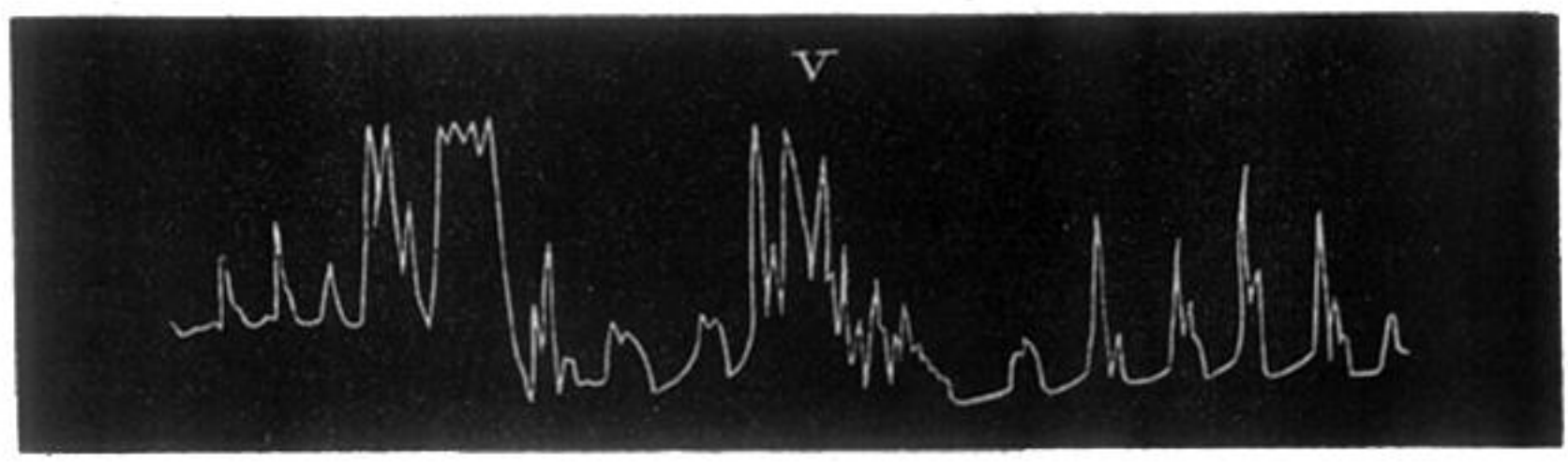
Quickened.



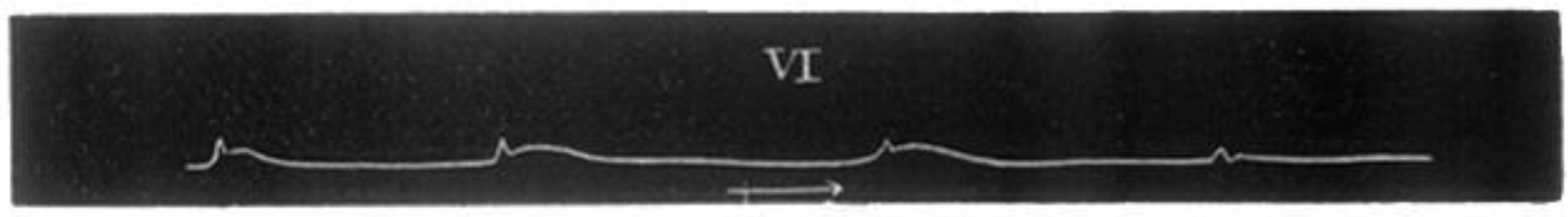
Lessened excursus.



Movement greatly lessened.

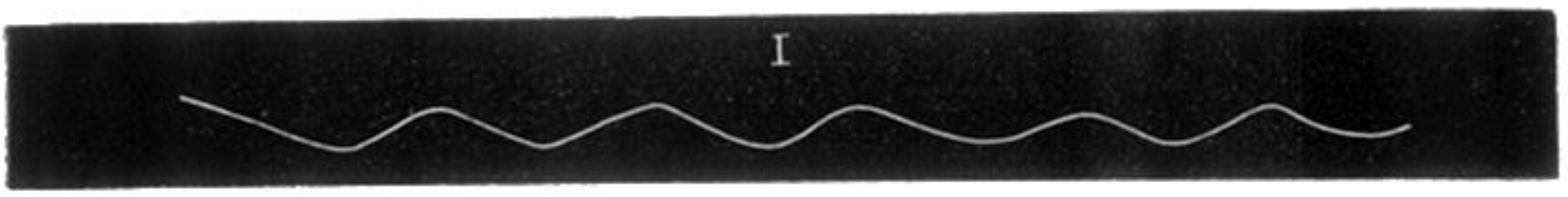


Convulsions.

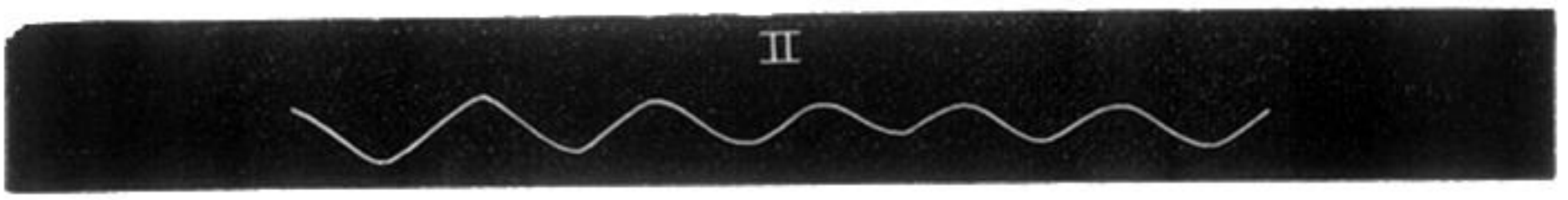


Termination of convulsions.

No. 4.—Tracings of the Respiratory Movements of a Cat under the Influence of Cobra Poison.

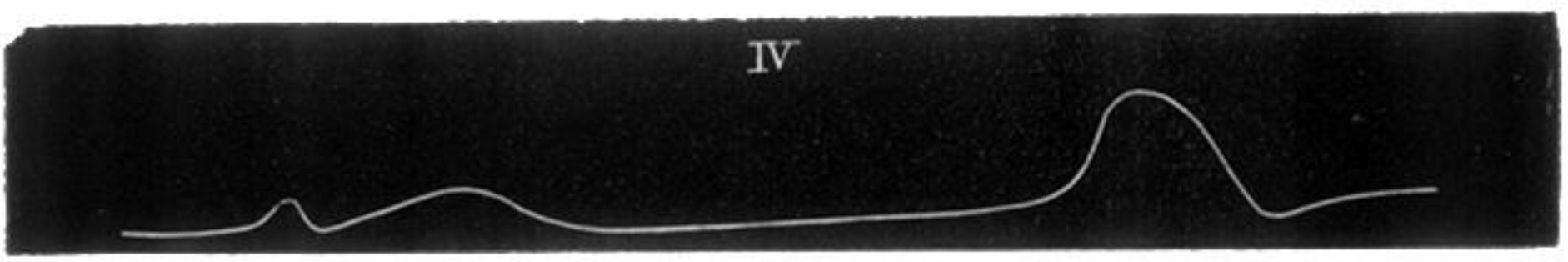


Normal respiration.

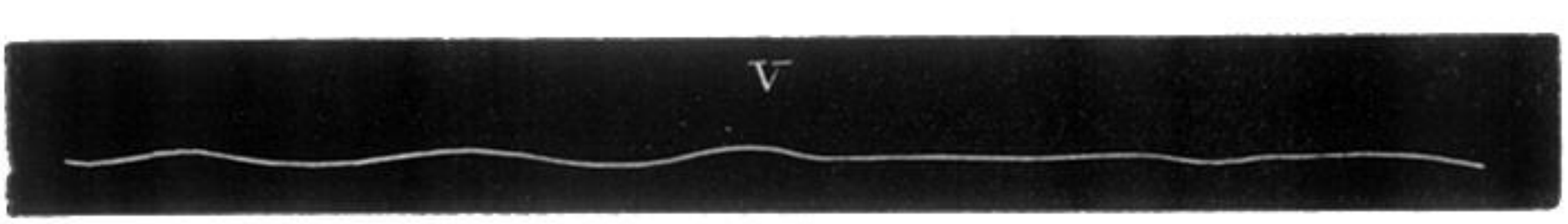


Quickened by cobra poison.

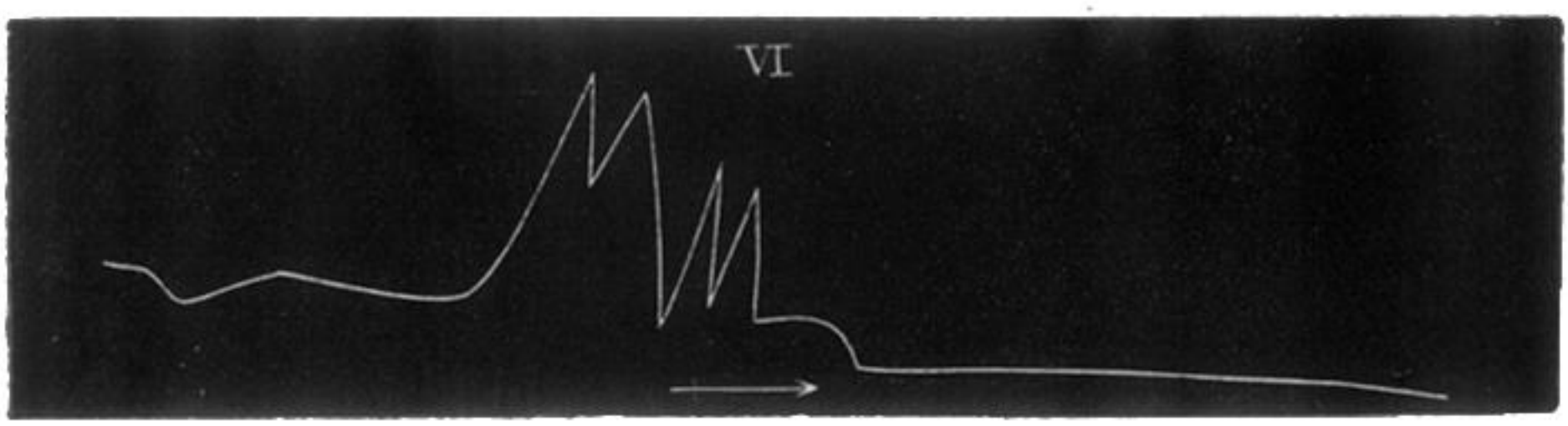
Commencing slowing.



Occasional deep inspiration.

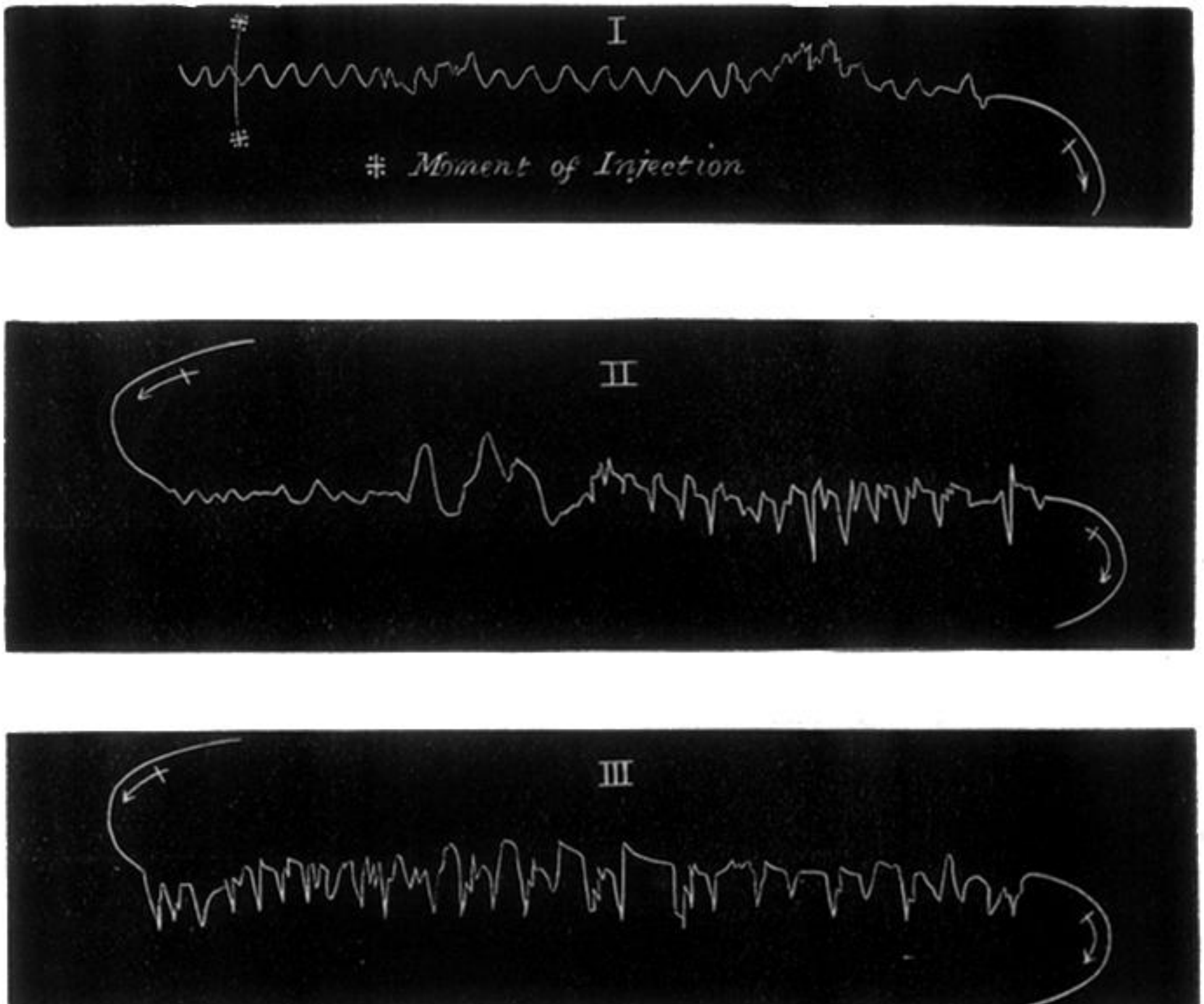


Respiration barely perceptible.

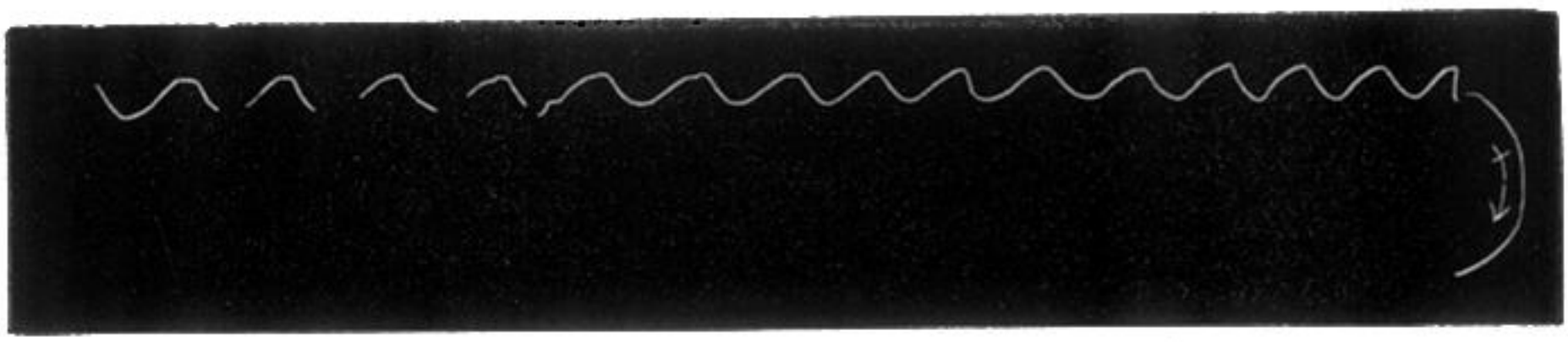


Convulsions.

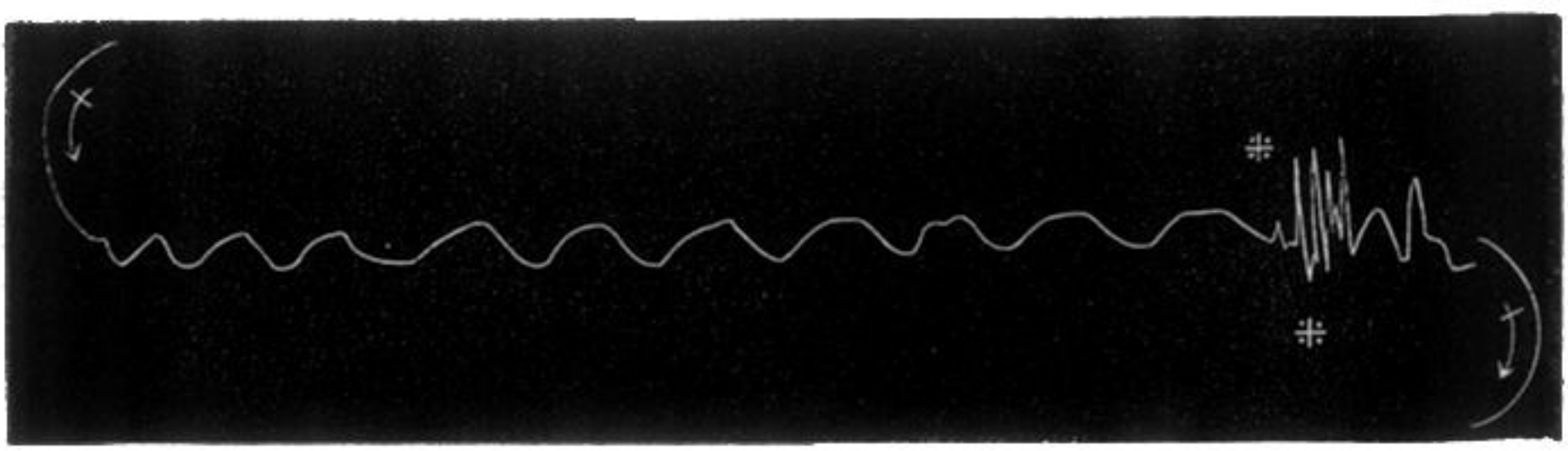
No. 5.—Continuous Tracing of Respiratory Movements of Dog from the moment of Intravenous Injection of Cobra Poison till Death.



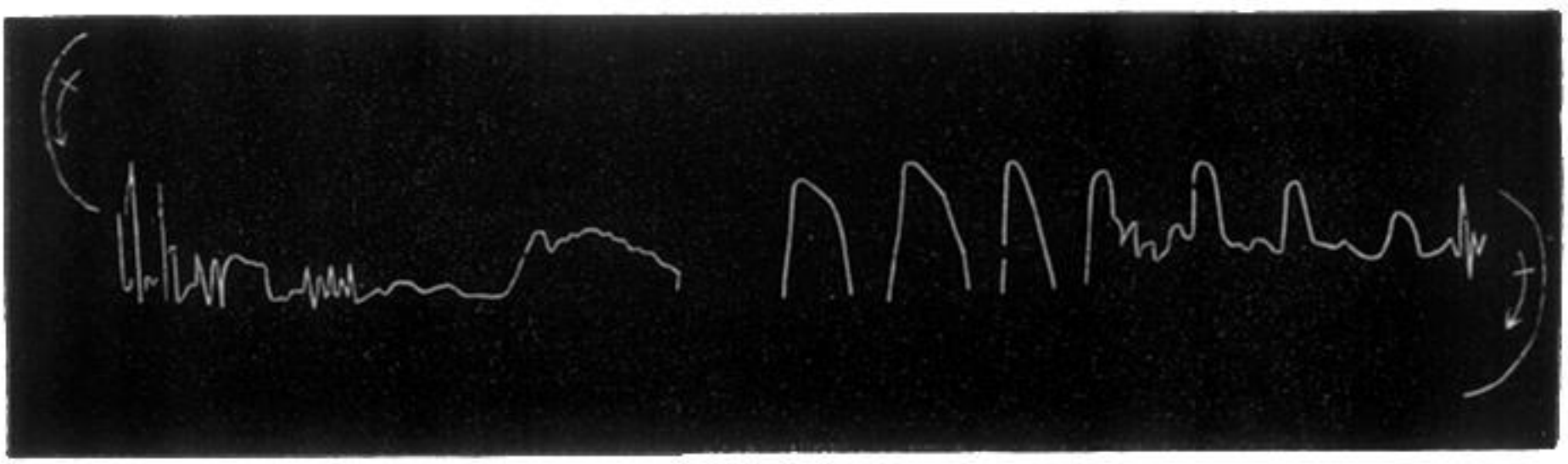
No. 6.—Tracing of Respiratory Movements in Fowl under the Influence of Daboia Poison.



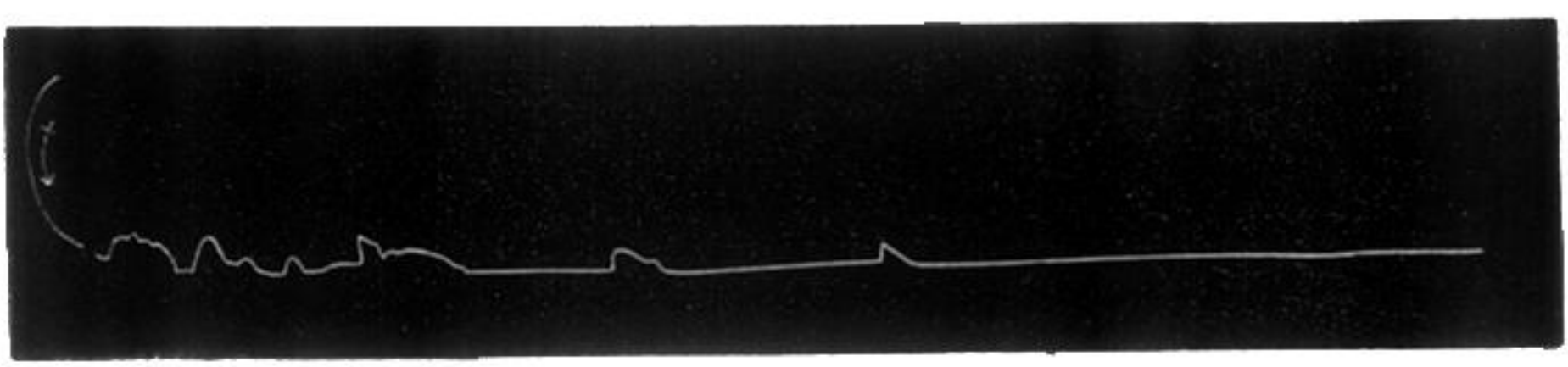
Respiration after the injection of the poison, but still normal.



Slightly exaggerated breathing, suddenly interrupted by convulsions.

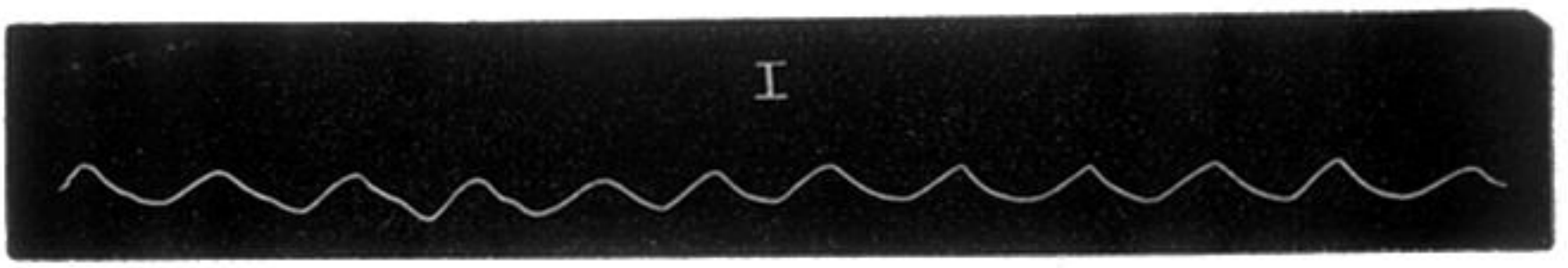


Convulsions.

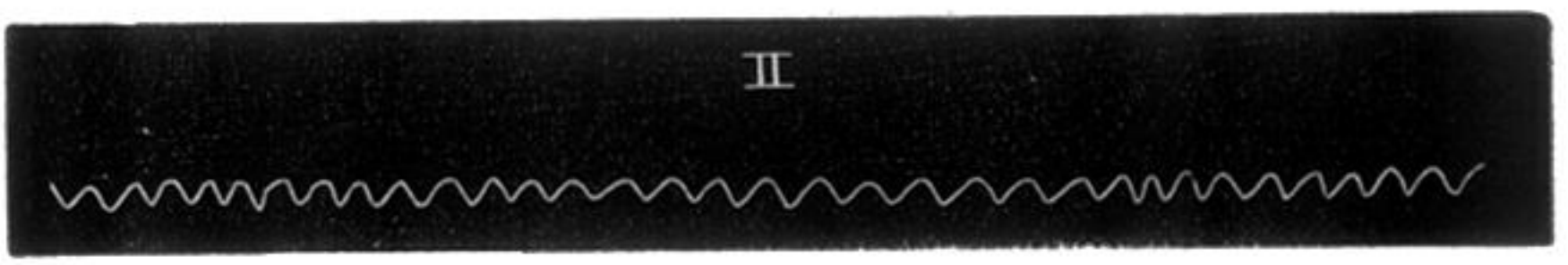


End of convulsions.

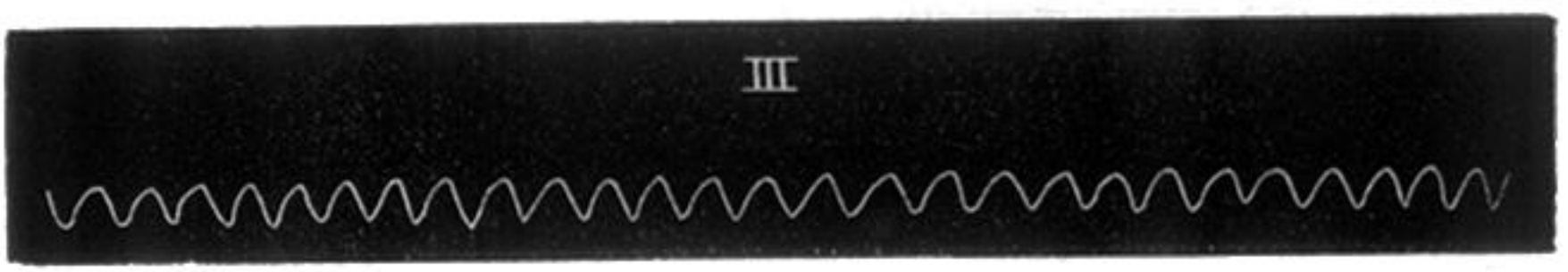
No. 7.—Tracing of Respiratory Movement in a Fowl under the Influence of Daboia Poison that had been heated to 100° C.

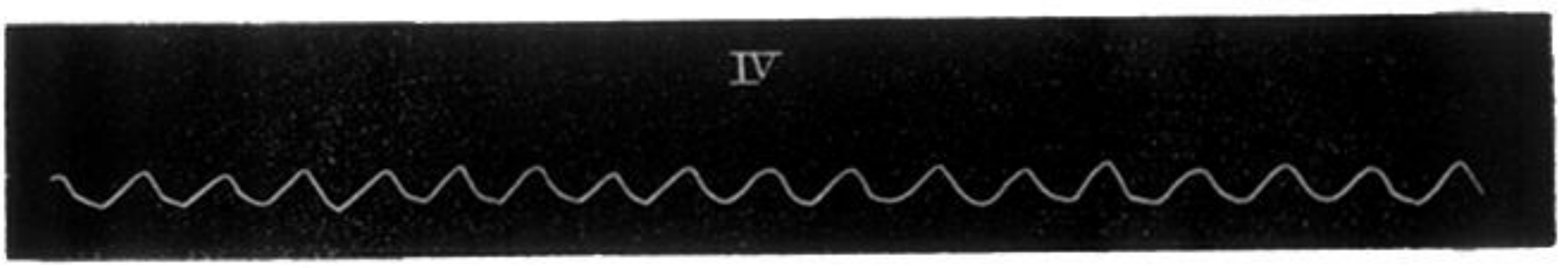


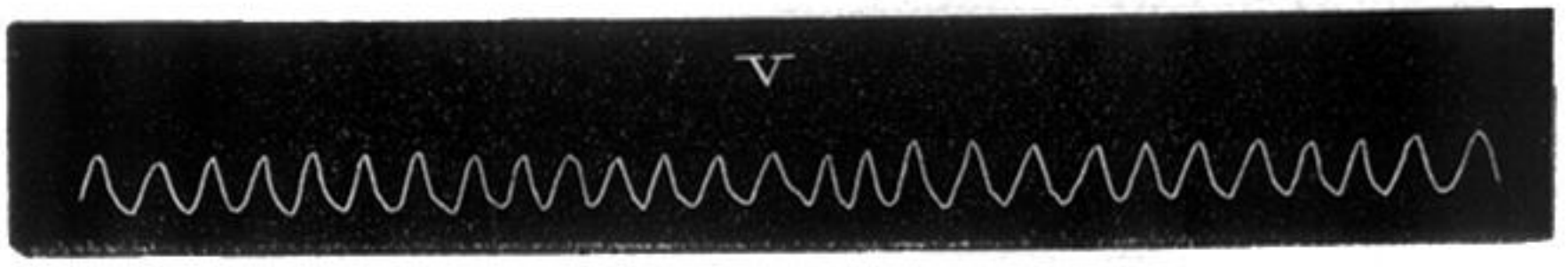
Normal.



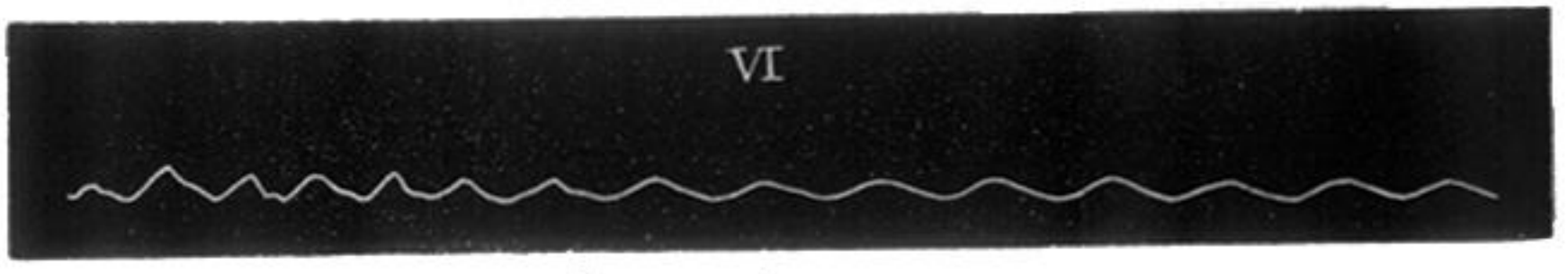
Great acceleration.





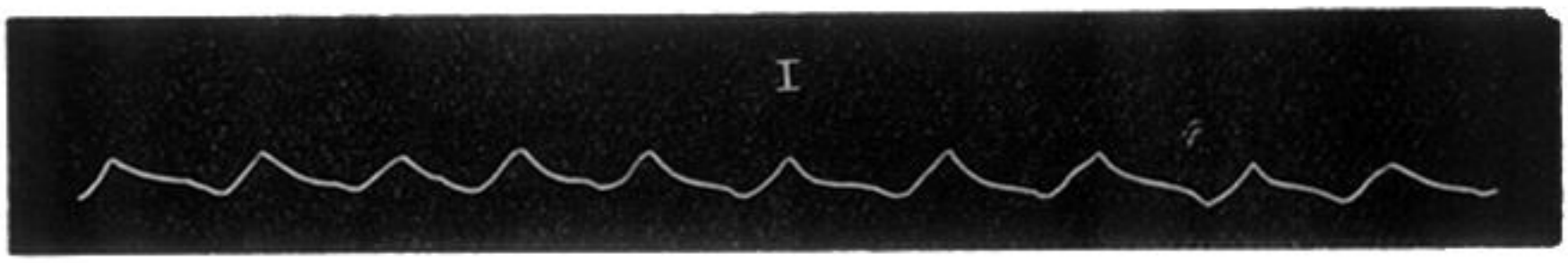


Great acceleration with increased excursus.

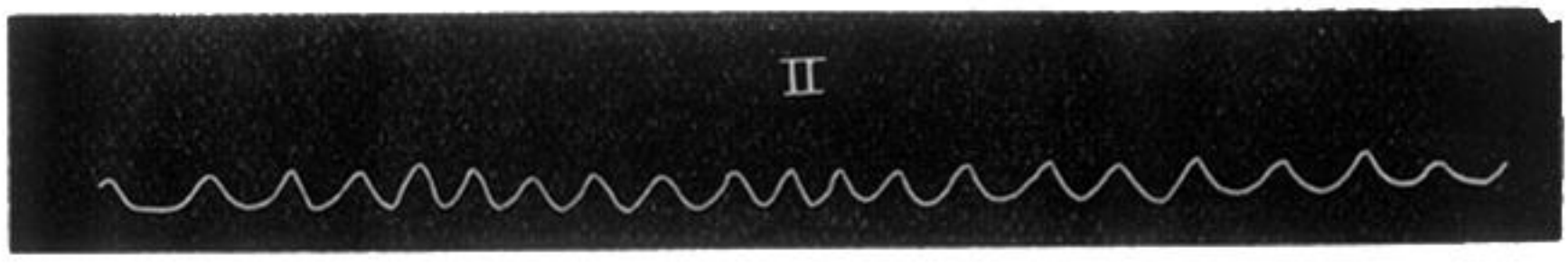


Lessened excursus.

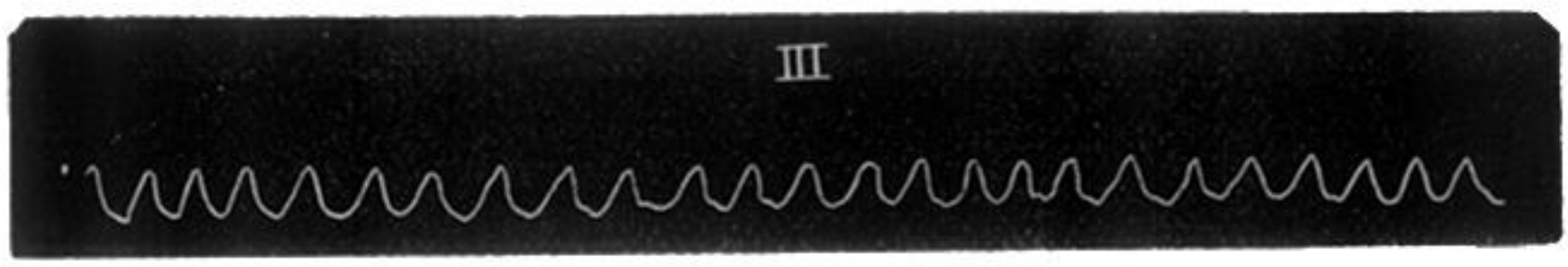
No. 8.—Tracings of Respiratory Movements of Dog under Daboia Poisoning.



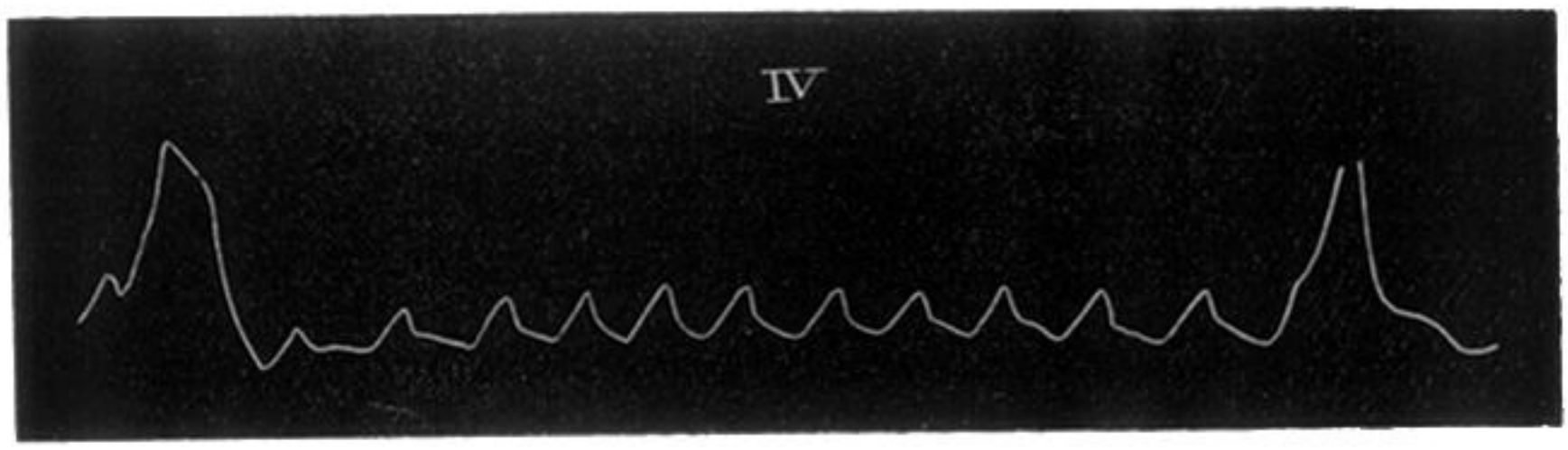
Normal respiration.



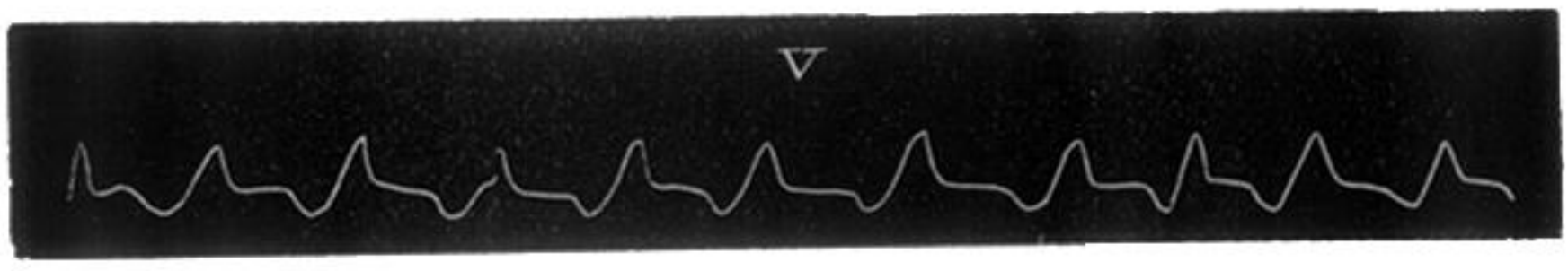
Acceleration.



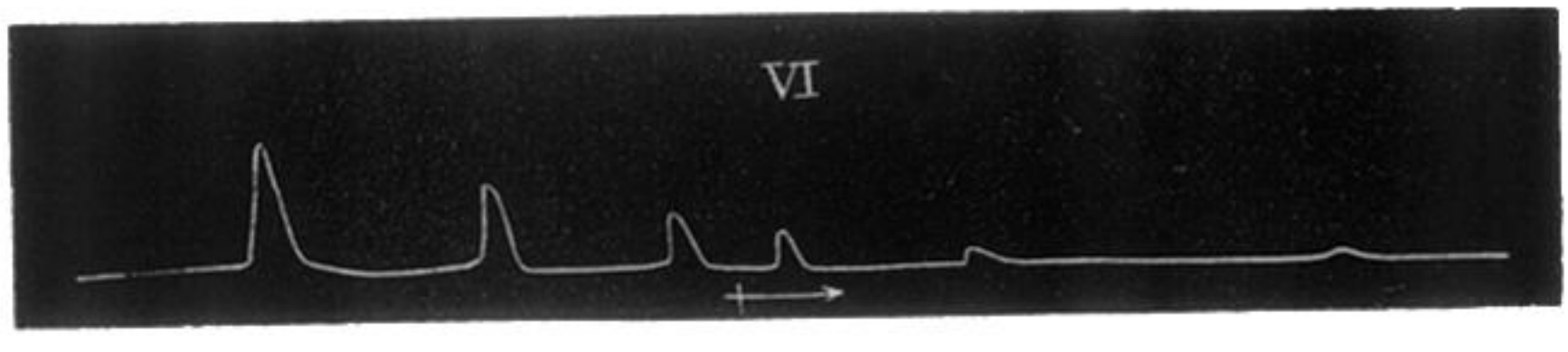
Respiration still quicker.



Irregular respiration.



Respiration slowing.



End of respiratory movements.