Physiological MATERIA MEDICA

Containing
All that is known of the physiological action of our remedies

Together with their characteristic
INDICATIONS and PHARMACOLOGY

Third Edition

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INDEX.

CLASSIFICATION.

All medicines have for their *starting point* or *center of action* the *nervous centers*, either *animal* or *organic*. Those that have their center of action in the *animal* (cerebro-spinal) nervous system, are the *true* remedies for *acute* and *sub-acute* diseases; and those that have their *center of action* in the *organic* (ganglionic) nervous system, are the true remedies for *sub-acute* and *chronic* diseases. The cerebro-spinants can often be alternated advantageously with the organic remedies.

ANIMAL GROUP.

Cerebro-Spinants.

For Acute and Sub-Acute Diseases.

Aconite.
Æsculus hip.
Ambra grisea.
Ammonium carb.
Amyl nitritum.
Antimonium cr.
Antimonium tart.
Apocynum can.
Arnica montana.
Arum triphyllum.
Asafœtida.
Asparagus offic.
Acidum benz.
Acidum carbol.
Acidum hydroc.
Acidum salicyl.
Baptisia.
Belladonna.
Bryonia alba.
Cactus grand.
Camphora.
Cannabis sativa.
Cantharides.
Capsicum.
Caulophyllum.
Causticum.
Chamomilla.
Chelidonium.
Chloroformum.
Cimicifuga.
Cina.
Cinchona.
Cinnamomum.
Cocculus Indicus.
Coffea cruda.
Colchicum.
Collinsonia Can.
Colocynthis.
Conium mac.
Copaiba.
Corallium rub.
Crocus sativus.
Crotalus hor.
Cuprum.
Digitalis pur.
Dioscorea vil.
Duboisia.
Dulcamara.
Ether.
Eucalyptus glob.
Eupatorium per.
Eupatorium pur.
Euphrasia.
Gelsemium sem.
Glonoine.
Hamamelis.
Helleborus niger.
Homatropin.
Hyoscyamus nig.
Hypericum per.
Ignatia amara.
Ipecacuanha.
Iris versicolor.
Jaborandi.
Kali bromidum.
Lachesis.
Lilium tigrinum.
Mezereum.
Millefolium.
Moschus.
Nux moschata.
Nux vomica.
Opium.
Ostrya Virginica.
Phytolacca dec.
Plantago major.
Platina.
Plumbum met.
Pulsatilla nigr.
Rhus tox.
Robinia pseudac.
Sabina.
Sambucus nigra.
Sanguinaria Can.
Scutellaria.
Secale cornutum.
Senecio aureus.
Senega.
Spigelia.
Stannum.
Sticta pulmo.
Stramonium.
Tabacum.
Tanacetum vulg.
Tarantula Cuben.
Tarantula Hispa.
Terebinthina.
Teucrium m. v.
Thlaspi bur. pas.
Trillium pend.
Uranium.
Urtica urens.
Ustilago madis.
Valeriana.
Veratrum album.
Veratrum viride.
Zincum val.
Zincum metal.

ORGANIC GROUP.

Ganglionics.

For Sub-Acute and Chronic Diseases.

Aloes.
Apis mellifica.
Argentum nit.
Arsenicum alb.
Aurum.
Acidum fluor.
Acidum muriat
Acidum nit.
Acidum phos.
Acidum sulph.
Baryta carbonica.
Borax.
Calcarea carb.
Carbo animalis.
Carbo vegetabilis.
Chimaphila.
Croton tiglium.
Euonymus.
Ferrum.
Filix mas.
Graphites.
Gummi guttæ.
Helonias dioica.
Hepar sulphur.
Hydrastis Can.
Iodine.
Kali bichrom.
Kali bromidum.
Kali carbonicum.
Kali chloricum.
Kali hydriodicum.
Kreosotum.
Leptandra Virg.
Lycopodium.
Magnesia.
Magnesia carb.
Magnesia mur.
Manganum.
Mercurius.
Mercurius jod.
Natrum mur.
Petroleum.
Phosphorus.
Podophyl. pelt.
Polyporus offic.
Psorinum.
Rheum.
Rumex crispus.
Sarsaparilla.
Secale cor.
Sepia.
Silicea.
Spongia.
Stillingia sylvat.
Sulphur.
Thuja occident.

Classification According to Tissues.

Cerebral Group.

Aconitum.
Amyl nitritum.
Arsenicum.
Acidum carbol.
Belladonna.
Cannabis sativa.
Chloroformum.
Cinchona.
Coffea.
Crotalus.
Cuprum.
Digitalis.
Ether.
Gelsemium.
Glonoine.
Helleborus.
Hyoscyamus.
Kali brom.
Lachesis.
Opium.
Plumbum.
Pulsatilla.
Rhus tox.
Sanguinaria.
Scutellaria.
Silicea.
Stannum.
Stramonium.
Tabacum.
Tarantula.
Veratrum viride.

Spinal Cord (Motor Group).

Aconitum.
Alumina.
Amyl nitritum.
Antimonium tart.
Argentum.
Arnica.
Arsenicum.
Asafœtida.
Acidum carbol.
Acidum hydroic.
Acidum phos.
Acidum salicyl.
Baptisia.
Belladonna.
Camphor.
Cantharides.
Causticum.
Chloroformum.
Cimicifuga.
Cina.
Cinchona.
Cocculus.
Colchicum.
Colocynthis.
Conium.
Copaiba.
Crocus.
Cuprum.
Digitalis.
Dioscorea.
Dulcamara.
Ether.
Eucalyptus.
Eupatorium per.
Eupatorium pur.
Gelsemium.
Helleborus.
Hyoscyamus.
Ignatia.
Ipecacuanha.
Kali brom.
Kreosotum.
Lachesis.
Manganum.
Mercury.
Mezereum.
Moschus.
Nux moschata.
Phosphorus.
Phytolacca.
Plumbum.
Polyporus.
Psorinum.
Rhus tox.
Secale.
Spigelia.
Stannum.
Stramonium.
Tabacum.
Tanacetum.
Tarantula.
Terebinthina.
Uranium.
Ustilago.
Valeriana.
Veratrum album.
Veratrum viride.

Spinal Cord (Sensory Group).

Aconitum.
Ambra grisea.
Antimonium tart.
Argentum.
Arsenicum.
Asafœtida.
Acidum hydroc.
Acidum phos.
Acidum salicyl.
Baptisia.
Belladonna.
Camphor.
Capsicum.
Caulophyllum.
Chamomilla.
Chloroform.
Cimicifuga.
Cinchona.
Coffea.
Colchicum.
Colocynthis.
Crocus.
Dioscorea.
Ether.
Eupatorium per.
Eupatorium pur.

Ignatia.
Kali brom.
Mercury.
Mezereum.
Moschus.
Natrum mur.
Nux moschata.
Opium.
Phosphorus.
Platina.
Plumbum.
Pulsatilla.
Rhus tox.
Sanguinaria.
Scutellaria.
Secale.
Senecio.
Spigelia.
Tarantula.
Terebinthina.
Valeriana.
Veratrum viride.

Skin.

Aconitum.
Aloes.
Antimonium cr.
Antimonium tart.
Apis mellifica.
Apocynum can.
Argentum.
Arnica.
Arsenicum.
Aurum.
Acidum carbol.
Acidum fluor.
Acidum mur.
Acidum nit.
Acidum phos.
Acidum sulph.
Belladonna.
Borax.
Calcarea.
Cantharides.
Carbo animal.
Cinchona.
Colchicum.
Copaiba.
Crotalus.
Croton tiglium.
Dulcamara.
Ether.
Eucalyptus.
Eupatorium per.
Graphites.
Hepar sulphur.
Iodine.
Ipecacuanha.
Iris.
Kali bichrom.
Kali brom.
Kali hyd.
Kreosotum.
Lachesis.
Lycopodium.
Manganum.
Mercury.
Mezereum.
Natrum mur.
Nux vomica.
Opium.
Petroleum.
Phytolacca.
Plantago.
Pulsatilla.
Rhus tox.
Rumex.
Sambucus.
Sarsaparilla.
Secale.
Sepia.
Silicea.
Stillingia.
Stramonium.
Sulphur.
Tabacum.
Terebinthina.
Thuja.
Urtica urens.
Ustilago.
Veratrum viride.

Mucous Membranes.

Aconitum.
Alumina.
Ammonium carb.
Antimonium cr.
Antimonium tart.
Apis mellifica.
Apocynum can.
Argentum.
Arsenicum.
Arum triphyllum.
Asafœtida.
Acidum carbol.
Acidum fluor.
Acidum mur.
Acidum nit.
Acidum salicyl.
Baptisia.
Belladonna.
Borax.
Bryonia.
Cannabis sativa.
Cantharides.
Capsicum.
Carbo veg.
Causticum.
Colocynthis.
Copaiba.
Croton tiglium.
Dulcamara.
Eucalyptus.
Euphrasia.
Ferrum.
Hepar sulphur.
Hydrastis.
Iodine.
Ipecacuanha.
Kali bichrom.
Kali brom.
Kali carb.
Kali hyd.
Kreosotum.
Lycopodium.
Magnesia carb.
Magnesia mur.
Manganum.
Mercury.
Mezereum.
Natrum mur.
Nux moschata.
Nux vomica.
Opium.
Phytolacca.
Pulsatilla.
Rhus tox.
Rumex.
Sabina.
Sambucus.
Sanguinaria.
Senega.
Silicea.
Stillingia.
Stramonium.
Sulphur.
Tabacum.
Teucrium.
Thuja.
Trillium.
Urtica urens.
Veratrum album.
Veratrum viride.

Serous Membranes.

Aconitum.
Apis mellifica.
Apocynum cann.
Arnica.
Arsenicum.
Acidum salicyl.
Bryonia.
Cantharides.
Colchicum.
Colocynth.
Dulcamara.
Helleborus.
Hypericum.
Iodine.
Jaborandi.
Kali carb.
Kali chlor.
Kali hyd.
Mercury.
Mezereum.
Phytolacca.
Plumbum.
Pulsatilla.
Rhus tox.
Senega.
Sulphur.
Uranium.

Fibrous Tissues.

Aconitum.
Argentum.
Acidum benz.
Bryonia.
Colchicum.
Hamamelis.
Kali bichrom.
Kali hyd.
Manganum.
Mercury.
Phytolacca.
Plumbum.
Rhus tox.
Sabina.
Senega.
Silicea.
Spigelia.
Stillingia.
Sulphur.

Osseous Group.

Aurum.
Acidum fluor.
Acidum phos.
Calcarea.
Ferrum.
Hepar sulphur.
Manganum.
Mercury.
Phosphorus.
Plantago.
Silicea.

Lymphatics.

Arsenicum.
Arum triphyllum.
Aurum.
Acidum fluor.
Acidum nit.
Acidum sulph.
Baptisia.
Baryta carb.
Belladonna.
Calcarea.
Carbo animal.
Carbo veg.
Chimaphila.
Graphites.
Helonias.
Hepar sulphuris.

Hydrastis.
Iodine.
Iris.
Kali hyd.
Kreosotum.
Lachesis.
Lycopodium.
Mercury.
Mezereum.
Natrum mur.
Petroleum.
Phytolacca.
Psorinum.
Rhus tox.
Rumex.
Sarsaparilla.
Silicea.
Spongia.
Stillingia.
Sulphur.
Ustilago.

Salivary Glands.

Apis mellifica.
Argentum.
Arum triphyllum.
Acidum mur.
Acidum nit.
Acidum sulph.
Belladonna.
Cantharides.
Digitalis.
Helleborus.
Helonias.
Iodine.
Iris.
Jaborandi.
Kali brom.
Kali chlor.
Kali hyd.
Lachesis.
Mercury.
Natrum mur.
Podophyllum.
Rhus tox.
Sanguinaria.

Pancreas.

Arsenicum.
Ether.
Helleborus.
Helonias.
Iodine.
Iris.
Mercury.

Spleen.

Arsenicum.
Cinchona.
Eucalyptus.
Ferrum.
Natrum mur.
Ostrya.
Phosphorus.

Liver.

Aloes.
Arsenicum.
Aurum.
Acidum nit.
Chamomilla.
Chelidonium.
Cinchona.
Cocculus.
Colchicum.
Crotalus.
Croton tig.
Cuprum.
Digitalis.
Dioscorea.
Ether.
Eupatorium per.
Helleborus.
Helonias.
Hepar sulphuris.
Iodine.
Iris.
Jaborandi.
Kali bichrom.
Kali brom.
Lachesis.
Leptandra.
Lycopodium.
Manganum.
Mercury.
Natrum mur.
Ostrya.
Phosphorus.
Podophyllum.
Polyporus.
Rheum.
Sanguinaria.
Sepia.
Sulphur.
Veratrum viride.

Kidneys.

Amyl nitritum.
Apis mellifica.
Arsenicum.
Asparagus.
Acidum benz.
Acidum salicyl.
Belladonna.
Camphor.
Cantharides.
Chimaphila.
Cina.
Cinchona.
Coffea.
Colchicum.
Crotalus.
Cuprum.
Digitalis.
Dulcamara.
Eucalyptus.
Eupatorium pur.
Ferrum.
Gelsemium.
Gummi guttæ.
Helleborus.
Helonias.
Hyoscyamus.
Ignatia.
Iodine.
Jaborandi.
Kali bichrom.
Kali brom.
Kali chlor.
Kali hyd.
Kreosotum.
Magnesia carb.
Magnesia mur.
Mercury.
Mezereum.
Opium.
Phosphorus.
Phytolacca.
Plumbum.
Pulsatilla.
Sabina.
Sarsaparilla.
Senecio.
Sepia.
Tanacetum.
Terebinthina.
Thuja.
Uranium.
Valeriana.

Bladder.

Conium.
Nux vomica.
Plantago.
Secale.
Thuja

Ovaries.

Apis mellifica.
Argentum.
Asafœtida.
Aurum.
Belladonna.
Cantharides.
Cimicifuga.
Cinchona.
Cocculus.
Coffea.
Conium.
Ether.
Gelsemium.
Graphites.
Hamamelis.
Ignatia.
Iodine.
Kali brom.
Kali carb.
Kreosotum
Lachesis.
Lilium.
Magnesia carb.
Magnesia mur.
Mercury.
Moschus.
Natrum mur.
Nux moschata.
Nux vomica.
Opium.
Phosphorus.
Platina.
Pulsatilla.
Secale.
Sepia.
Spongia.
Stramonium.
Tanacetum.
Thuja.
Ustilago.
Valeriana.

Uterus.

Belladonna.
Borax.
Cantharides.
Caulophyllum.
Cimicifuga.
Cocculus.
Coffea.
Crocus.
Digitalis.
Ether.
Gelsemium.
Graphites.
Hepar sulphuris.
Iodine.
Jaborandi.
Kali brom.
Kreosotum.
Lilium.
Mezereum.
Opium.
Pulsatilla.
Sabina.

PHYSIOLOGICAL

MATERIA MEDICA.

ACONITUM NAPELLUS.

Wolf's-bane.

Habitat, Central Europe. Tincture of the fresh plant, Class I.; of Root, Class III.

Antidotes.—Vegetable acids, Wine, Bell., Coff., Verat.

Through the cerebro-spinal nervous system, Aconite has nine special centers of action:

I. HEART. *Inhibitory Paralysis; Blood-Pressure Lessened.*
II. CIRCULATION. *Vaso-Motor Paralysis.*
III. TEMPERATURE. *Depressed, with Diaphoresis.*
IV. CEREBRO-SPINAL NERVOUS SYSTEM. *Paralysis.*
V. MUCOUS MEMBRANES. *Sthenic Inflammation.*
VI. STOMACH. *Emesis; Congestion; Neuralgia.*
VII. LUNGS. *Centric Vagi Paralysis; Congestion; Inflammation.*
VIII. TENDONS AND FIBROUS TISSUES. *Rheumatoid Inflammation.*
IX. SEROUS MEMBRANES. *Plastic Inflammation.*

Heart and Arterial Capillary Blood-Vessels.—Toxic doses of Aconite paralyze directly the nervous ganglia of the heart, which are its rhythmically discharging centers, producing at first a reduction of the number of heart-pulsations, and then an increased action; with evident loss of muscular power; finally, irregular systolic movements, with very long intervening pauses, ending in diastolic paralysis. Each individual pulsation is lessened until death, when the muscle of the heart will not respond to galvanic irritation, its contractility being destroyed.

Dr. Sydney Ringer says: "Aconite certainly affects either the muscular substance or the contained ganglia of the heart; on this point all observers are agreed; for Aconitia affects the heart after section of the pneumogastric, or the administration of Atropia, which paralyzes this nerve; and it affects the extirpated heart in the same way as it affects this organ in situ. It is maintained, however, that it acts also through the pneumogastric; Boehm and Wartmann believing that it paralyzes the termination of this nerve; Achscharumow, that it first stimulates the inhibitory center of the pneumogastric, and so slows the heart, and then the pneumogastric becomes exhausted and at last paralyzed, and then the heart beats quickly and irregularly."

"The action of Aconite upon the circulation is very decided. According to Dr. Achscharumow, in the frog a moderate toxic dose of Aconitia produces at first a reduction in the number of the heart's pulsations, then an increase of rapidity of its action, with very evident loss of power, and finally irregular systolic movements, with very long intervening pauses, ending in diastolic arrest. Drs. Rudolf Boehm and L. Wartmann have confirmed these observations.

"In animals, the exhibition of Aconite in sufficient doses yields similar results. In the dog and cat, there is a steady sinking of the arterial pressure; in the rabbit, this fall is preceded by a brief rise. The rate of the heart's pulsation also undergoes reduction; and there is finally diastolic arrest in these and other mammals.

"The method by which the Aconite influences the heart is not certainly settled. According to the experiments both of Boehm and Wartmann, it produces a gradual paralysis of the peripheral vagi; a constant increase of the intensity of a galvanic stimulation of the pneumogastric nerves being required to influence the heart as the poisoning deepens, until finally the vagi entirely refuse to transmit any inhibitory impulse.

"In a single experiment, Achscharumow found, that, after section of the vagi in the early stage of Aconite-poisoning, there was an immediate rise, both in the number of the cardiac pulsations and in the arterial pressure. From these data he argues that the slowing of the pulse during the early stage of Aconite-poisoning, is due to stimulation of the inhibitory centers in the medulla oblongata. Boehm and Wartmann repudiate this conclusion; because, according to their experience, the phenomena of Aconite-poisoning occur in the usual manner, after section of the vagi, or in the atropized animals. It is evident that there is no necessary contradiction in the asserted facts of these observers, as it is possible that the slow

ing of the pulse may be due to two immediate causes, one having its seat in the medulla oblongata, the other in the heart. Although the explanation cannot be considered proven, it is probably correct; as Lewin agrees with Achscharumow that there is a primary rise of the pulse when Aconitia is given after section of the vagi, but states that this rise is of very brief duration, and is soon followed by the usual reduction. It is very certain that Aconitia also influences directly the heart, or its contained ganglia, for Achscharumow has found that it acts upon the frog's heart removed from the body, and Liegeois and Hottot have observed the ordinary cardiac phenomena of Aconite-poisoning, produced by the alkaloid placed directly upon the viscus. Boehm and Wartmann have also noted that in Aconite-poisoning the force of the individual beat is lessened. After death the cardiac muscle fails entirely to respond to galvanic irritation, its contractility being lost."—*Dr. H. C. Wood, Materia Medica.*

"Aconite is a protoplasmic poison, and destroys the functions of all nitrogenous tissue; first of the central nervous system, next of the nerves, and last of the muscles; but it has an especial affinity for the sensory apparatus, paralyzing first the sensory perceptive center, and, through this central nervous system, is a powerful depressant of the motor nerves and muscles. Aconite affects all the structures of the heart; first its ganglia, next its nerves, and last its muscular substance."—*Ringer.*

Upon the arterial capillary vessels, through the vaso-motor nerves and heart, we have paralysis, with its attendant congestion, and all the symptoms of inflammation, in all the tissues that have capillary vessels. Dr. Wood does not believe Aconitia acts on the vaso-motor nerves, but that it destroys the conducting power either of the afferent nerves or of the cord, so that in the animal under its influence no impulse can be transmitted from the periphery to the vaso-motor centers in the medulla; but Bartholow says it is a direct sedative to the vaso-motor nervous system.

Cerebro-Spinal Nervous System.—The action of Aconite upon the animal nervous system, is not fully settled. Liegeois and Hottot believe that it first paralyzes the perceptive centers, above the spinal cord, and afterward the terminations, and lastly, the trunks of the sensory nerves.

Boehm and Wartmann conclude that Aconite first paralyzes the sensory and then the motor part of the cord.

Achscharumow concludes that it paralyzes both the trunks and terminations of the cerebro-spinal motor nerves, but leaves the muscles unaffected.

Dr. G. Hunter Mackenzie says: "Aconite's action on the nervous system consists in first irritating, and secondly, paralyzing, the peripheral sensory nerves, and posterior roots of the spinal nerves. Increases the irritability of the peripheral motor nerves, and of the motor columns of the cord. Does not induce muscular paralysis, but, on the contrary, increases the irritability of the voluntary muscles, inducing convulsions, mainly augmenting the irritability of the anterior column of the cord, and the motor nerves and muscles."

"It may be considered settled that Aconite has no *decided influence upon the motor nerves.* At the same time the poison would seem to have some such influence, because, when brought in contact with an exposed nerve, Aconitia rapidly destroys its functional activity, and, after death in the Aconitized frog, the motor nerves lose their irritability more rapidly than normal. Further, it should be noted, that, when in frogs the convulsions are very severe, the motor nerves seem temporarily to lose their functional power from exhaustion.

"A very complete and beautiful investigation of the action of Aconitia upon the spinal cord has been made by Dr. Liegeois and M. Hottot. According to these observers, in Aconite-poisoning loss of sensibility occurs in the frog's legs simultaneously with, or even before, the disturbances of respiration, and long before the power of voluntary motion is lost; and even when the reflex activity is intact. This sensory paralysis, according to the experiments of the French investigators just quoted, first appears in the hind legs of a frog poisoned with Aconitia, and has not its primary seat either in the peripheral nerves or in the spinal cord; for it was found that tying the aorta close to its abdominal bifurcation, so as to prevent access of the blood—i. e., of the poison—to the posterior nerves, did not affect the development of the anæsthesia; further, that closing the artery nearer its origin in such a way as to shut off the circulation to the cord and spinal nerves, but to allow the passage of the blood to the cerebrum, did not cause sensory paralysis to come on more slowly than normal in poisoning by Aconite.

"Of course, it is possible for the peripheral ends of the sensory nerves to be paralyzed either at the same time that the perceptive center is, or afterward; and, of course, the center being paralyzed, it becomes very difficult to determine whether the periphery is or is not affected. Liegeois and Hottot assert that this paralysis of the center occurs before any serious implication of the peripheric nerves, because, after Aconitic anæsthesia had been pro-

duced, Strychnia was able to produce tetanus; afterward, however, the extreme peripheric nerves became affected, so that irritation of the skin in the doubly poisoned frog would not provoke convulsions, even at a time when irritation of the trunk of a nerve would produce general reflex motor disturbance. At last galvanization of the nerve-trunk itself failed to induce response. From these facts, Liegeois and Hottot deduce—very logically, I think—the conclusion that Aconite induces anæsthesia by paralyzing, first, the perceptive centers; secondly, the peripheral extremities of the nerves; thirdly, the nerve-trunks themselves. The observers alluded to also confirmed this conclusion by other experiments than those already noticed. They found, that, although Aconitia applied directly to a nerve-trunk paralyzes its sensibility, yet when the veins of a frog's leg are tied and the alkaloid injected into the artery and allowed to penetrate the tissues of the leg, the skin loses its sensibility long before the nerve is affected.

"In regard to motion, Liegeois and Hottot found, that, in a certain stage of Aconitia-poisoning, the frog lies with his limbs extended, relaxed, and perfectly paralyzed, and yet is capable of executing vigorous voluntary movements, and evinces nearly normal reflex activity. They attribute this condition of apparent but not real motor paralysis, to loss of sensibility from paralysis of the perceptive center; as the unpoisoned frog evinces the same phenomena after division of all the posterior spinal roots. After a time the reflex activity is also lost, the power of voluntary movement remaining. Liegeois and Hottot believe that this loss of reflex activity is spinal; but, in their experiments upon the conjoint action of Aconite and Strychnia, it was found that at a certain stage, when no amount of irritation of a nerve would induce convulsions, a slight direct irritation of the cord would cause violent Strychnic spasms. This would seem to show that at least the earliest abolition of the reflex activity was due to paralysis of the afferent nerve-fibers.

"In some particulars the researches of Liegeois and Hottot have been confirmed by the later studies of Dr. George Hunter Mackenzie. The persistence of voluntary movement, after abolishing reflex actions, which was first noted by Boehm and Wartmann, and afterward by Liegeois and Hottot, as well as by Mackenzie, proves that, at a certain stage of the poisoning, while the motor pathway from the brain along the anterior columns and the afferent nerves is open, either the sensory nerves or the receptive centers of the cord are paralyzed. The experiments of Liegeois and Hottot upon the joint action of Aconitia and Strychnia are also ac-

cordant with those of Mackenzie; for that observer found, that, when a nerve was protected from the poison by tying its supplying artery, irritation of it caused reflex actions when the remainder of the frog's periphery was insensible; also that there is a stage of poisoning in which irritation of the extreme peripheral nerves fails to induce reflex movements, although such movements are called out by irritation of the sensory nerve-trunk; later, irritation of the trunk was powerless, while irritation of the posterior columns of the cord still produced wide-spread movements. It must, therefore, be considered proven that Aconite *paralyzes the sensory nerves, commencing at their peripheral endings,* and that the loss of reflex activity is due, at least in great part, to such cause.

"The supposed action of Aconitia upon a higher perceptive center is at present very doubtful. S. Ringer and R. Murrell (*Journal of Physiology* i., Nos. 4 and 5,) deny the accuracy of the delicate experiments of Liegeois and Hottot. They affirm reflex action is never maintained after abolition of sensation, and that Aconitia diffuses itself through the cellular tissue of the frog so rapidly as to throw doubt upon any experiments in which the progress of the power is supposed to be arrested by tying an artery. Curiously enough, Drs. Ringer and Murrell, while doubting the experiments of Liegeois and Hottot, accept the conclusions founded upon these asserted erroneous experiments, seemingly because they themselves have found that Aconitia causes abolition of reflex action more rapidly in brainless than in normal frogs. It is evident, that, if this asserted fact were true, it would in no way prove the conclusion of Liegeois and Hottot. Further, the experiments on brainless frogs were only three in number and it is perfectly possible that the rapid reflex palsy was simply the result of batrachian idiosyncrasies. The only safe conclusion on the evidence, is that the evidence does not warrant any conclusion.

"For reasons already given, the deductions are, however, now warranted by their premises. All the phenomena, except the final loss of voluntary power, are explainable by the action of the drug upon the sensitive nerves. The final extinction of voluntary movement must be due to an action upon the motor tract of the spinal cord, as the peripheral motor apparatus is not distinctly affected. Experiments by Mackenzie on frogs, have yielded apparently contrary results to those of Boehm and Wartmann as to the effect of removal of the influence of Setschenow's center upon the cord in Aconitized frogs. The difference probably depends upon difference in the doses employed. Boehm and Wartmann distinctly state, that, when minute doses of Aconitia are employed,

there is a primary period of excitement of the spinal centers. Mackenzie has found, that the convulsions which are so severe in frogs after small quantities of Aconite, are chiefly of spinal origin, but that the peripheral motor apparatus shares the stimulation with the spinal motor tract. M. Guilland also affirms this primary stimulant spinal action. If it exists at all in mammals, it is in them completely masked. The convulsions seen in Aconite-poisoning in some mammals are cerebral, not spinal; as I have experimentally determined that they do not occur in those portions of the body separated by spinal section from cerebral influence.

"As Boehm and Wartmann found that the reflex activity was lost more rapidly than the power of voluntary movement, and that no increase of reflex activity occurs in the Aconitized frog when the cord is cut so as to release it from the influence of Setschenow's reflex inhibitory centers, they draw the conclusion that the Aconitia first depresses the reflex activity of the sensitive spinal centers, and afterward that of the motor spinal centers, until the cord is completely paralyzed."—*Dr. H. C. Wood.*

Aconite is a protoplasmic poison, and destroys the functions of all nitrogenous tissue; first, of the central nervous system; next, of the nerves; and, last, of the muscles; paralyzing first the sensory perceptive center; and, through this central nervous system, it is a powerful depressant of the motor nerves and muscles. Aconite affects all the structures of the heart; first its ganglia, next its nerves, and lastly its muscular substance.

Upon the brain, we have intense hyperæmia, from paralysis of the capillary blood-vessels; as shown by the swollen face, blue lips, violent pain in the head; partial insensibility, delirium, mania, despair, whizzing noises in the ears, loss of sight, vertigo, anxiety, restlessness, etc.

Vagi.—Upon the respiratory center, Aconite is a direct depressant and paralyzant. "Aconite acts very directly upon the respiratory centers; and this effect is not produced by any stimulation of the inhibitory fibers of the vagus, and so arresting the action of the discharging center, but by the effects upon the center itself, in the medulla. The respiration, when lethal doses are taken, becomes slower and deeper, until the respiratory center is completely exhausted. We have then violent dyspnœa, inflammation and death.

"The action of Aconite upon the respiration is very decided. In mammals, the respirations, under the influence of the drug, are slow, with a prolonged expiration following immediately upon

the inspiration. After the expiration there is a long pause. The whole breathing cycle resembles very much that occurring after section of the vagi; and, like the alteration in breathing after this section, seems to be due, at least in part, to paralysis of sensory or afferent fibers. The known influence of Aconite upon the peripheral afferent nerves in general, suggests that the poison disturbs repiration by paralyzing the peripheral afferent fibers of the vagi. The evidence upon this point is somewhat contradictory, Mackenzie affirming, that, in the Aconitized animal, section of the vagi produces no effect on the respiration, while Boehm and Wartmann affirm that Aconite produces its usual effect after division of the nerves. It is plain, that, even if the Aconite does paralyze the peripheral afferent vagi, it must also act upon the respiratory centers, since arrest of respiration could not be caused by afferent palsy. As the arrest occurs in the frog before the motor nerves are affected by the poison, Liegeois and Hottot believe that the disturbance is centric; and I think there can be no doubt that Aconite is a depressant and paralyzant of the respiratory centers." —*H. C. Wood.*

Serous Membranes.—Aconite affects especially the capillaries of serous membranes, producing paralysis and intense congestion, through a direct impression upon the retinæ of the cerebro-spinal and vaso-motor nerves supplied to them. Inflammation of serous membranes does not generally go on to ulceration, sloughing and gangrene; but the fluid that is thrown out in the second stage takes on what is called "adhesive inflammation." The fluid effused undergoes such an organizing process as to glue the opposing surfaces of the serous membranes together. As soon as the second stage, that of effusion, takes place in serous membranes, the usefulness of Aconite ceases, and Bryonia, Arnica, Sulphur, or some other remedy must be chosen; but, up to the stage of effusion, Aconite is the remedy *par excellence*. The inflammation caused by Aconite is rheumatic in character; and most examples of inflammation in serous membranes are rheumatic in character; and this explains why Aconite is so useful in these inflammations.

Tendons and Fibrous Tissues.—Aconite has a special and specific action on the joints, producing inflammation of a rheumatic character; and no remedy will be so often called for in acute inflammation of the joints, especially if used locally as well as internally, prepared as an ointment, using from two to ten grains of Aconitia to the drachm of Cosmoline.

Mucous Membrane.—In cases of poisoning with Aconite, inflammation of nose, mouth, and fauces, is a striking effect of the drug. Salivation and vomiting are often well marked. This is accompanied with violent pains in the stomach, though rarely does it produce colic and diarrhœa; but constipation is most frequent and marked. Results of autopsies: The lips, and mucous membrane of the mouth and throat, are found to be destitute of epithelium, the mouth and fauces being intensely congested; the hyperæmia often extending to the stomach and small intestines, with here and there small patches of inflammation; and, in a few cases, gangrene has been found. The liver, spleen, and kidneys are all engorged, and the bladder strongly contracted.

Temperature.—Aconite first produces chilliness, more especially down the spine and sides, soon followed by general and constant burning heat, with elevation of the temperature; this is followed by copious perspiration, from an increased flow of blood to the skin. In fatal cases of poisoning with Aconite, the temperature has been found to fall three degrees; and, in acute inflammations with high temperature, no remedy will reduce the temperature with such certainty and rapidity.

"Fothergill attributes the effect of Aconite and other cardiac depressants, on inflammation, to their influence on the vascular system. It has been shown that the vascular system is always in a state of semi-contraction, and that, by paralyzing the vasomotor nerves, it is possible to double its capacity. Aconite dilates the arterioles, and greatly increases the capacity of the vascular system, and by this means drains blood away from the inflammation; in fact, this drug 'bleeds the patient into his own vessels.' As the vessels leading to an inflamed organ are already paralyzed, Aconite does not augment the supply of blood to it; but, in medicinal doses, it stimulates these dilated arterioles, and causes them to return to their normal state of semi-contraction, thus curing symptoms similar to those which it causes in toxic doses.

"Aconite slows and weakens the heart; hence, the circulation becomes less rapid, with corresponding decrease in its chemical changes; this diminished oxidation involving, of course, diminished production of heat." The heat is also diminished by radiation and evaporation from copious perspiration. Aconite often fails to produce perspiration in children; but, at the same time, the temperature is quickly reduced, by the action of this drug, in all synochal inflammations.