type U. S. P. H. & M. H. S., 9467, from Woodman, Montana.

Opinion 79.—Case of Lamarck's (1801a) Système des Animaux sans Vertèbres: "Rigidly construed," Lamarck's (1801a) Système des Animaux sans Vertèbres is not to be accepted as designation of type species.

Opinion 80.—Suspension of Rules in the Case of *Holothuria* and *Physalia:* The Echinoderm genus *Holothuria* Linn., 1767, restr. Bruguière, 1791, type *H. termula* 1767 = *H. tubulosa* 1790, and the *Siphonophorae* genus *Physalia* Lamarck, 1801, type *P. pelagica* 1801 = *Holothuria physalis* 1758, are hereby placed in the official list of generic names.

Opinion 81.—The Genotype of *Cimex, Acanthia, Clinocoris* and *Klinophilos:* On basis of the premises before the commission, the common bedbug of Europe, *Cimex lectularius,* is the genotype for *Cimex* 1758, *Acanthia* 1775, *Clinocoris* 1829, and *Klinophilos* 1899 (*Clinophilus* 1903), and its proper technical designation under the Rules is *Cimex lectularius. Cimex* Linn., 1758, type *C. lectularius* is hereby placed in the official list of generic names.

NOTICE TO ZOOLOGISTS OF CERTAIN GENERIC NAMES TO BE INSERTED IN THE OFFICIAL LIST

THE following generic names (with genotype in parentheses) have been submitted to the International Commission on Zoological Nomenclature for inclusion in the official list of generic names.

The secretary will delay final announcement of the votes on these names until November 1 in order to give to any zoologists, who may desire, the opportunity to express their opinions.

Protozoa: *Balantidium* Clap. and Lachm., 1858b, 247 (*entozoon*); *Endamoeba* Leidy, 1879a, 300 (*blattae*); *Giardia* Kunstler, 1882, 349 (*agilis*); *Trichomonas* Ehrenb., 1838a, 331 (*vaginalis*); *Trypanosoma* Gruby, 1843a, 1134 (*sanguinis*).

Cephalopoda: *Argonauta* L., 1758a, 708 (*argo*); *Octopus* Lam., 1799, Prodromus, 18 (*vulgaris*); *Sepia* L., 1758a, 568 (*officinalis*).

Gasteropoda: *Acteon* Montf., 1810, 314 (*tornatilis*); *Ampullaria* Lam., 1799, Prodromus, 76 (*urceus*); *Buccinum* L., 1758a, 734 (*undatum*); *Bulimimus* Ehrenb., 1831 (*labrosus*); *Bulla* L., 1758a, 725 (*ampulla*); *Calyptraea* Lam., 1799, Prodromus, 78 (*chinensis*); *Columbella* Lam., 1799, Prodromus, 70 (*mercatoria*); *Helix* L., 1758a, 768 (*pomatia*); *Limax* L., 1758a, 652 (*maximus*); *Littorina* Feruss., 1821, Tabl. Syst., XXXIV (*littorea*); *Natica* Scop., 1777, 392 (*canrena*); *Physa* Drap., 1801, 31 (*fontinalis*); *Planorbis* Müller, 1774, 152 (*cornea*); *Succinea* Drap., 1801, 32 (*putris*); *Tethys* L., 1758a, 653 (*leporina*); *Vitrina* Drap., 1801, 33 (*pellucida*).

Lamellibranchiata: *Anodonta* Lam., 1799, 87 (*cygneus*); *Cyprina* Lam., 1818, 556 (*islandicus*); *Dreissena* Van Bened., 1835, 25 (*polymorpha*); *Mactra* L., 1767, 1125 (*stultorum*); *Margaritana* Schum., 1817, 137 (*margaritifera*); *Mya* L., 1758a, 670 (*truncata*); *Mytilus* L., 1758a, 704 (*edulus*); *Ostrea* L., 1758a, 696 (*edulis*); *Sphaerium* Scop., 1777, 397 (*cornea*); *Tellina* L., 1758a, 674 (*virgata*); *Teredo* L., 1758a, 651 (*navalis*); *Venus* L., 1758a, 684 (*mercenaria*).

Polyplacophora: *Chiton* L., 1758a, 667 (*tuberculatus*).

Scaphopoda: *Dentalium* L., 1758a, 785 (*elephantinum*).

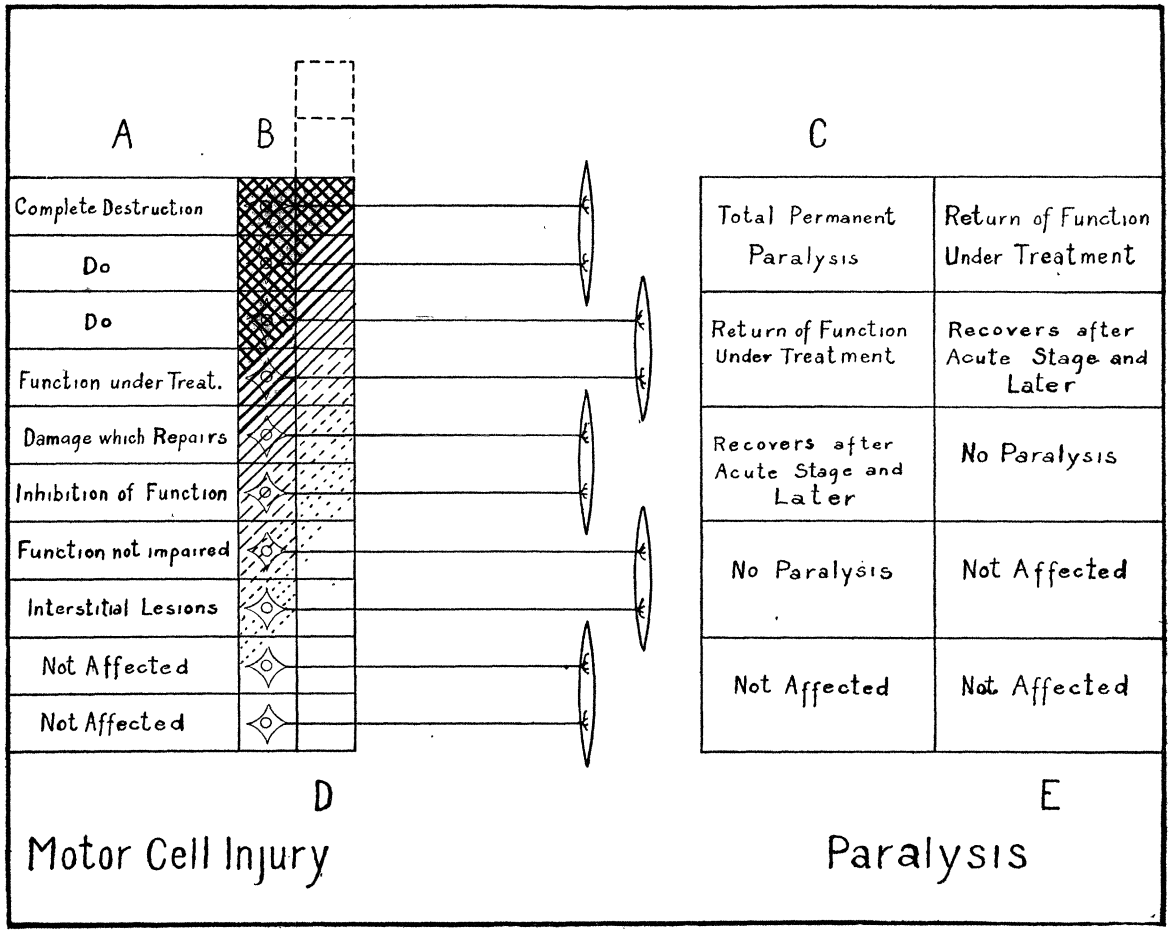
Tunicata: *Ascidia* L., 1767, 1087 (*mentula*); *Botryllus* Gaert., 1774, 35 (*schlosseri*); *Clavelina* Savig., 1816, 174 (*lepadiformis*); *Diazona* Savig., 1816, 35 (*violacea*); *Distaplia* de Valle, 1881, 14 (*magnilarva*); *Molgula* Forbes, 1848, 36 (*oculata*).

C. WARDELL STILES,
Secretary to the International Commission on Zoological Nomenclature
HYGIENIC LABORATORY,
U. S. PUBLIC HEALTH SERVICE

SPECIAL ARTICLES

THE RELATION OF PLURISEGMENTAL INNERVATION TO RECOVERY IN INFANTILE PARALYSIS

THE earlier conception of the pathology of infantile paralysis attributed the paralytic features of the disease to a primary degeneration or atrophy of the anterior horn cells. This conception has been enlarged to include the several other changes in the central nervous system which appear in the acute or paralytic stage of the disease, but damage to the anterior horn cells still holds its place as being directly responsible for the paralytic manifestations. The initial change in the motor cell itself in this disease is atrophy of the intracellular network of the neurofibrillae. The cell body swells and becomes more globular. With this swelling a disintegration of the Nissl granules occurs and often extends throughout the whole cell. In many instances the nucleus retains its normal appearance for a remarkably long time. Generally, it also retains its position in the middle of the cell. The nucleus may even remain after the peripheral part of the cell body is degenerated and dissolved. When a severer change occurs in the cell body, the nucleus is converted into a deeply staining irregular structure. Complete karyolysis takes place at times. Occasionally vacuoles may be seen in the protoplasm. Finally round cells invade and ingest the ganglion cells till only a clump of round cells with greatly increased protoplasm remains to mark the site of the ganglion cell. The nerve filaments of the gray matter are faintly stained and of irregular contour. Sometimes they present small swollen nod-



ules, or they may be degenerated so that only fragments may be recognized.

Although the muscular areas involved in the paralytic process have been more or less closely associated with the lesions in corresponding segments of the spinal cord, this can not be said to have been worked out in sufficient detail to permit an accurate correlation between the varying degrees of cell injury and the several grades of severity which are observed in the paralyses which result. Undoubtedly such a relation exists. A correct conception of this relation is highly desirable, especially from the standpoint of treatment of the paralyses.

During the past two and one half years, I have inoculated a number of monkeys (*Macaccus rhesus*) with a strain of the virus of infantile paralysis which was obtained on October 29, 1921, from the spinal cord of a fatal case of the disease. This virus has produced the experimental disease in monkeys in a milder form than is usually seen. For the most part the animals have recovered, but always with more or less paralysis. Hence it has been possible to observe the progress of the paralytic manifestations over varying periods of time. In respect to disability, atrophy, contractures and improvement, the events in the course of the experimental disease correspond to those in human beings. No attempt has been made to treat the individual paralyses, but the fact that these lame monkeys restrict their activities to a great extent is probably a factor in the improvement which takes place. In this respect, the animals may be compared with a patient following a régime of rest.[1]

The progress of these animals as to recovery from paralysis may be divided as follows:

Improvement in paralysis at termination of acute stage.
Improvement after six to eight weeks.
A gradual improvement later.
No improvement.

The improvement which occurs at the termination of the acute stage of the disease is often considerable and abrupt. These circumstances suggest that it is to be associated with the removal of factors which have merely inhibited the function of the motor cells, such as edema. The improvement which takes place after six or eight weeks or longer may also be exten-

[1] Jones, Robert, and Lovett, Robert W.: "Orthopedic Surgery," William Wood and Company, 1923.

sive as well as quite sudden. This we are inclined to associate with motor cells which have suffered actual injury and have undergone repair.[2] The gradual improvement which sometimes occurs seems more properly to be associated with the taking up of function by other motor cells.

Upon the unisegmental theory of innervation, the varying degrees of recovery are ascribed largely to the stimulation of remaining active fibers, but this does not entirely account for the improvement which takes place, especially that which comes late in the disease. The doctrine of plurisegmental innervation of individual muscle fibers which has recently been emphasized by Cattell and Stiles[3] seems to offer a more adequate explanation of the phenomena of recovery and to stimulate the hope that function may be restored in muscles which under the older conception are sometimes considered beyond repair. The manner in which plurisegmental innervation may operate in recovery is presented schematically in the accompanying diagram. The different degrees of motor cell involvement are given under A and illustrated graphically under B, with the corresponding paralysis shown under C. This schematic drawing shows both nerve cells of the first muscle fiber completely destroyed, which produces a total and permanent paralysis of this fiber. One nerve cell of the next muscle fiber is completely destroyed but its fellow is brought into play under reeducational treatment. The next cell represents the lesion which is associated with a paralysis which recovers sometime after the acute stage of the disease, while the sixth cell is one whose function is inhibited only while the acute process exists in the central nervous system tissue.

There is still a question whether the motor cell injury in infantile paralysis is produced by the direct action of the virus or toxins produced by it, or whether the injury is secondary to the interstitial lesions. There is considerable evidence that the latter is the case—that the nerve cell undergoes changes resulting from disturbances in its nutrition which are due to the interstitial lesions.

When it is recalled that the damage to the motor cells is accomplished in a very short time, one can not but be impressed by the effects which would undoubtedly follow even a slight reduction in the extent or duration of these interstitial lesions. Among the possibilities in this connection are the reduction of edema, the promotion of the circulation of the perivascular fluid, the destruction of even a small portion of the virus present by specific means, or the neutralization of irritating substances acting locally. Recent important advances in the physiology of the central nervous system[4] have opened new avenues of approach to this question.[5] It does not seem too much to hope that means can be found which will change the picture in the central nervous system to that represented in D, thus modifying the problem of the orthopedist to that shown in E.

W. LLOYD AYCOCK

FROM DEPT. OF PREVENTIVE MEDICINE AND HYGIENE, HARVARD MED. SCHOOL, BOSTON, AND THE RESEARCH LAB., VERMONT STATE BOARD OF HEALTH, BURLINGTON

[2] Buzzard, E. F., and Greenfield, J. G.: "Pathology of the Nervous System," 1923.

[3] Cattell, McKeen, and Stiles, P. G.: SCIENCE, 1924, N. S., LIX, 383.

[4] Weed, L. H.: *Jour. Med. Research*, 1914, N. S., XXVI, 93; Weed, L. H., and Hughson, Walter: *Am. Jour. Physiol.*, 1921, LVIII, 53; Weed, L. H., and McKibben, Paul S.: *Am. Jour. Physiol.*, 1919, XLVIII, 512.

[5] Aycock, W. L., and Amoss, H. L.: *Johns Hopkins Hospital Bull.*, 1923, XXXIV, 361.

THE AMERICAN CHEMICAL SOCIETY

SECTION OF HISTORY OF CHEMISTRY

F. B. Daines, *chairman*
Lyman C. Newell, *secretary*

Lewis C. Beck, M.D., a pioneer in the food and drug adulteration movement of America: L. F. KEBLER. Dr. Beck received both a literary and a medical training. He was identified with several institutions of learning, among them the Albany Medical College and Rutgers College of New Jersey. He taught materia medica, pharmacy, botany and other natural sciences. These all gave him a good background to write his book entitled "Adulterations of Various Substances Used in Medicine and the Arts," printed in 1846. In 1848 an appropriation of $1,000 was made by Congress to make certain chemical analyses. Dr. Beck was selected to do this work, and his two reports made in 1848–9 constitute the first records of the Department of Agriculture dealing with the great problem of food and drug adulteration.

A sketch of agricultural chemistry in America from 1663–1863: C. A. BROWNE. The history of agricultural chemistry in America is briefly reviewed from the time when Governor John Winthrop, of Connecticut, presented his report before the Royal Society upon the "Description, culture and use of maize," down to the time of the establishment of the U. S. Department of Agriculture in 1862. Rev. John Clayton's report on the "Observables of Virginia," in 1688, contains the first reference to the chemistry of American soils. The interest of Presidents John Adams and Thomas Jefferson in the practical applications of chemistry to agriculture is mentioned. Allusion is made to the early investigations of John Taylor, Gerard Troost, C. V. Sheppard, Edmund Ruffin, S. L. Dava, J. P. Norton, Evan Pugh, J. W. Draper, Ebenezer Emmons, Eben L. Horsford, Benjamin Silliman, Jr., C. A. Goessman, S. W. Johnson and other chemists and to their publications upon agricultural chemistry. A great impetus was given to agricultural chemistry in America in the decade 1840–1850 as a result of the publication of Liebig's "Chemistry in its Application to Agriculture,"