The theoretical section takes up about one third of the book. This includes modern concepts of complete ionization, acid-base relationships, etc., which are made an integral part of the later discussions in the experimental sections. Especially noteworthy are the up-to-date sections on hydrolysis and amphoterism, the author's treatment of "ammonium hydroxide" as ammonia, and the inclusion of a separate chapter dealing with the concept of activity. To this reviewer, the entire theoretical section, although admirable in its scope, was somewhat lacking in general clarity and readability. There might be mentioned, as contributing to this, rather poor arrangement of the topics discussed and occasional misplaced emphasis. Thus more space is devoted to the nomenclature of Werner compounds than is given to an explanation of their structure.

In the experimental section which follows, 100 pages are devoted to the 24 common cations. General discussion of the chemistry of the several metals and preliminary experiments on the reactions of their ions precede the actual group analysis. The latter follows customary procedure, employing sodium hydrogen sulfide to separate the copper and tin subgroups, and making use of only the most essential organic reagents. A good discussion of the analysis of each cation group follows the description of the procedure. It is unfortunate that a complete summary of the several cation groups is to be found nowhere in the section on cations, but only in the Introduction, and that here the sulfides of Group II are described as "sparingly soluble in water," while those of Group III are said to be "almost insoluble in water."

To the analysis of 24 anions the author devotes 72 pages. The selection is somewhat arbitrary, including for example chlorate, bromate, iodate and perchlorate, but excluding silicate, arsenite, acetate and oxalate. The procedure followed is that of Sneed and Duschak[1] adapted for semimicro use by the author and A. Lerner. It has the advantage of dividing the anions into five mutually exclusive groups, which are separated in order by means of successive precipitations of the calcium, barium, cadmium and silver salts under proper conditions. Without first-hand experience of the method, this reviewer can not form an adequate opinion of its merits.

The Appendix of 21 pages contains several valuable features, including a good review of mathematical operations used in qualitative analysis, and an exhaustive but uncritical list of reference books in qualitative analysis and inorganic chemistry.

This first edition of Professor Heisig's book is marred by numerous small errors and omissions. Misspellings, especially of proper names, are frequent; it is to be hoped that these will be corrected subsequently.

WENDELL H. TAYLOR

DISEASES OF DOMESTIC ANIMALS

The Infectious Diseases of Domestic Animals. By WILLIAM ARTHUR HAGAN, D.V.M., D.Sc., professor of bacteriology and dean of the faculty, New York State Veterinary College, Cornell University. 665 pp. 145 ill. Ithaca, N. Y.: Comstock Publishing Co., 1943. Price, $6.00.

THIS is a well-integrated and entirely adequate account of the host of infectious diseases to which domesticated mammals and birds are subject, of the specific microorganisms involved and of available methods of diagnosis and control. The introductory section of the book is a consideration of the general aspects of infection and disease production by microorganisms, and of the nature and development of the immune response, with a brief review of allergic conditions and of iso-antibodies. Discussion of groups of microorganisms is arranged under the following section headings: Pathogenic Bacteria, Bacteria-like Pathogenic Organisms of Uncertain Classification, *i.e.*, Spirochetes, Rickettsiae and Pleuropneumonia Group, Pathogenic Fungi, Pathogenic Protozoa and Viruses. For each of these groups or, where justified, for individual organisms, consideration follows the general pattern: morphology, reactions in culture, natural habitat, pathogenicity and types of disease in susceptible hosts, diagnostic and control methods, immune response, and, where appropriate, relation to disease of man. These divisions of the subject matter are clearly marked by subtitles in bold face type, giving ready access to any part of the material. To each chapter and to many chapter subdivisions short lists of well-chosen references are appended. This is not a textbook of bacteriology in the usual sense. Instead, its purpose is much broader, and it presents a well-balanced treatment of the important aspects of infectious diseases of lower animals.

HERBERT L. RATCLIFFE

SPECIAL ARTICLES

OBSERVATIONS CONCERNING THE ETIOLOGY OF PRIMARY ATYPICAL PNEUMONIA

THE clinical syndrome currently known as primary atypical pneumonia may be caused occasionally by viruses of the psittacosis group[1,2,3,4,5] or by Rick-

[1] *Jour. Chem. Ed.*, 8: 1177–86 and 1386–95, 1931.

[1] M. D. Eaton, M. D. Beck and H. E. Pearson, *Jour. Exper. Med.*, 73: 641, 1941.

ettsiae,[6] but in the great majority of cases it is apparent that some other agent is responsible. Within recent months a number of investigators have reported attempts to isolate and identify this agent, which is presumably a virus.[7,8,9,10,11]

Eleven patients severely ill with primary atypical pneumonia were selected for the present study. Sputum, throat washings and blood from nine of these cases were inoculated to animals and birds by several routes, and serial passages were carried out. Specimens of lung and spleen were utilized in a similar manner from the remaining two cases, both of whom died of other causes while the pulmonary infection was present. No gross or microscopic evidence of disease was observed in any of the following species, either after primary inoculation, or after serial blind passage: ferrets, Swiss mice, deer mice, Syrian hamsters, rabbits, white rats (normal, x-rayed, adrenalectomized, splenectomized), cotton rats, rice birds, pigeons.

However, in recently weaned guinea pigs 10 strains of a readily transmissible agent have been isolated from 7 of the 11 patients, as indicated in Table 1.

TABLE 1

Case	Age and sex	Source of strains	Day of disease isolated
J. A.	M 22	Sputum	5
		Blood	4
B. W.	F 22	Throat washings	4
		Sputum	11
D. K.	M 55	Lung	9
		Spleen	9
E. V.	F 24	Sputum	10
B. R.	F 26	Sputum	12
E. L.	F 55	Lung	13
L. F.	M 24	Throat washings	10

Pulmonary lesions appeared in the animals on first passage with two strains, on second passage with two strains, on third passage with five strains and on fourth passage with the remaining strain. The agent produces a disseminated bronchopneumonia which becomes manifest grossly in from 12 to 20 days and shows complete resolution after 45 to 60 days. Successful transmission has been accomplished only by the intranasal route of inoculation. The infective titer is low, usually 10^{-2}, although in one instance a titer of 10^{-4} has been observed.

The various strains have now been carried through from 3 to 16 passages, employing a total of 387 guinea pigs, with no demonstrable alteration in character of the pulmonary disease, and no increase in virulence of the agent. In 90 control guinea pigs the intranasal inoculation of suspensions of normal guinea pig lung, with 3 to 5 subsequent passages, has invariably given negative results.

The agent will pass through a Berkefeld V candle but is retained by filters of smaller porosity. It is unstable and deteriorates rapidly in saline suspensions at both room and ice-box temperatures, but may be protected by broth or normal serum. Potency is maintained for at least 6 months in the frozen state.

Pathologically the pneumonia in the guinea pig is characterized by thickening of the alveolar septa due to congestion and infiltration with mononuclear cells, scanty collections of monocytes in the alveoli and lymphocytic cuffing about the blood vessels and bronchi. Bronchitis is slight or absent. Inclusion bodies, elementary bodies and Rickettsia-like organisms have not been observed. Routine cultures of the lungs on blood agar under CO_2, and at intervals on other media to reveal the presence of anaerobes or organisms of the pleuropneumonia group, at no time have revealed bacteria that could be considered responsible for the disease.

Identity of the various guinea pig strains has been demonstrated by cross-protection tests, recovered animals showing immunity to reinfection with both homologous and heterologous strains. However, pooled convalescent guinea pig sera and sera from immunized rabbits have failed uniformly to neutralize the agent by any of several techniques employed. Neutralization tests with acute and convalescent sera from cases of atypical pneumonia have likewise been unsuccessful. Complement fixation tests with patients' sera proved to be impractical, since we encountered nonspecific fixation with normal tissue antigens.[12]

Attempts to cultivate the agent in the developing hen's egg and in various types of tissue cultures have thus far given negative results.

Although we have been unable to produce pulmonary lesions in the cotton rat by the inoculation of human material, a bronchopneumonia has been regularly produced in this animal following intranasal inoculation of all strains of the guinea pig agent. The incubation period in the cotton rat aver-

[2] K. F. Meyer, *Medicine*, 21: 175, 1942.
[3] J. M. Stickney and F. R. Heilman, *Proc. Staff Meet. Mayo Clin.*, 17: 369, 1942.
[4] J. E. Smadel, *Jour. Clin. Invest.*, 22: 57, 1943.
[5] C. B. Favour, *Am. Jour. Med. Sci.*, 205: 162, 1943.
[6] R. E. Dyer, N. H. Topping and I. A. Bengtson, *Pub. Health Rep.*, 55: 1945, 1940.
[7] J. A. Baker, SCIENCE, 96: 475, 1942.
[8] M. D. Eaton, G. Meiklejohn, W. Van Herick and J. C. Talbot, SCIENCE, 96: 518, 1942.
[9] F. G. Blake, M. E. Howard and H. Tatlock, *Yale Jour. Biol. and Med.*, 15: 139, 1942.
[10] J. H. Dingle *et al.*, *War Med.*, 3: 223, 1943.
[11] F. L. Horsfall, Jr., E. C. Curnen, G. S. Mirick, L. Thomas and J. E. Ziegler, Jr., SCIENCE, 97: 289, 1943.
[12] L. Thomas, E. C. Curnen, G. S. Mirick, J. E. Ziegler, Jr., and F. L. Horsfall, Jr., *Proc. Soc. Exper. Biol. and Med.*, 52: 121, 1943.

ages one week. Pulmonary involvement is often extensive, but the animals rarely die. On section the lungs of infected cotton rats show an alveolar exudate rich in polymorphonuclear neutrophiles. Bronchitis is frequently observed. The picture suggests a bronchopneumonia caused by bacteria, but cultures have regularly failed to reveal significant microorganisms. Moreover, the agent is transmissible after filtration through a Berkefeld V candle, as in the guinea pig.

Selected cotton rat strains have been carried through 12 to 19 passages, employing a total of 334 animals. In 47 control cotton rats no pulmonary lesions have been observed, following the intranasal inoculation and subsequent passage of normal lung suspensions.

The cotton rat strains have been shown to be antigenically similar to one another by cross-protection tests. Furthermore, recovered cotton rats are immune to reinoculation with homologous and heterologous strains of the guinea pig agent, thus demonstrating that the agents in the cotton rat are identical with the original guinea pig strains.

Neutralization and complement fixation tests with the cotton rat strains, employing sera from patients, convalescent rats, convalescent guinea pigs and immunized rabbits, have given inconclusive results.

However, both guinea pigs and cotton rats repeatedly injected by the intranasal route with human material, including sputum, throat washings, lung and spleen, develop a partial or complete immunity to infection with passage strains of the agent from either the guinea pig or the cotton rat.

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LOSS OF PROPRIOCEPTIVE REFLEXES FOLLOWING RETROGRADE DEGENERATION[1]

ACHESON, Lee and Morrison[2] have reported a decrease in spontaneous respiratory activity in the cut phrenic nerve during the period of retrograde degeneration. To check the nature of this deficiency and to define it in terms of spinal reflexes a study was made of a series of cats in which the sciatic nerve had been cut. Stimuli were delivered as single shocks to the peripheral nerves or to the dorsal roots, and recordings were made from the dorsal and ventral roots with a cathode ray oscillograph.

FIG. 1. Potentials recorded from 7th lumbar ventral root in a cat which had had the right sciatic nerve cut 12 days previously. The stimulus was in each case a single condensor discharge to the dorsal root. (*a*) Left side. (*b*) Right side. Time intervals, 1 millisecond.

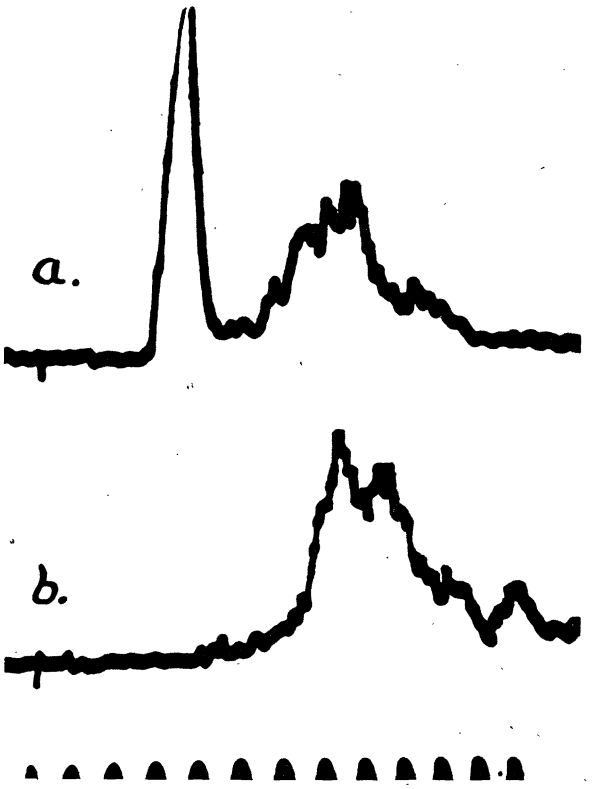

FIG. 2. Potentials recorded from seventh ventral root of same preparation in response to stimulation of the central ends of the cut sciatic nerves. The conduction distances on the two sides were approximately equal. (*a*) Left side. (*b*) Right side. Time intervals, 1 millisecond.

[1] From the Department of Neurology, College of Physicians and Surgeons, Columbia University. Aided by a grant from the National Foundation for Infantile Paralysis.

[2] G. H. Acheson, E. S. Lee and R. S. Morrison, *Jour. Neurophysiol.*, 5: 269, 1942.