

**Coalition For Informed Choice**

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*“For a successful technology, reality must take precedence over public relations, for Nature cannot be fooled” ...Richard P. Feynman*

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CFIC's book scanning project in association with Steve Solomon of [www.soilandhealth.org](http://www.soilandhealth.org) is intended to salvage the great works of the pioneers of Natural Hygiene in public health and clinical practice. —Gary Krasner, Dec. 2004

This book is posted in full in the Health Library of [www.soilandhealth.org](http://www.soilandhealth.org). Steve Solomon wrote the following introductory comments about the book:

Rational Bacteriology, by J. R. Verner, C. W. Weiant, R. J. Watkins.  
Self Published by the authors, 1953.

*This book is like a double barreled elephant gun. On one hand it prepares chiropractic students to pass their state medical licensing board examinations. To pass this gauntlet of orthodoxy the student must walk a tightrope, parroting the official line and viewpoints about bacterial diseases. So reading the first part of this book fully explains the official medical model. On the other hand the book simultaneously offers an alternative view of bacteria and the diseases they are reputed to cause, a viewpoint that does not support vaccination or most other forms of attacking bacteria whilst seeking to cure a disease that the bacteria did not cause. Perhaps more than any other book in our collection, Rational Bacteriology expounds the best alternative to the conventional theory of infectious disease—a viewpoint that was begun by Béchamp and refined later by J. Tissot and others.*

*While some minor assertions in the book are out of date (as science learned a few things in microbiology since 1953), the theoretical framework is as sound as it was when Bechamp was alive. Thanks are owed to Gary Krasner, founder and director of Coalition For Informed Choice in New York City, for supplying the scans of this book.*

*The Book downloads as a pdf file of 1.55 MB*

**This file is formatted for printing to paper. CFIC makes available a pdf file designed for optimum reading from the computer screen.**

**The following excerpts from the book describes microbiologist J. Tissot:**

The authors write in the Preface to the Second Edition:

*Excerpt:*

*“Section II is entirely new. In large part it is an exposition of the researches of the late Professor J. Tissot, of Paris, whose monumental work (as yet untranslated into English), product of a lifetime of vast scholarship and ingenious laboratory investigation, marks the fitting culmination of that great era in the history of French microbiology which began nearly a century ago with Béchamp and a few of his contemporaries. We believe firmly that it is impossible to exaggerate the importance of these researches, and hence make no apology for the amount of space devoted to them.”*

And from Part II on page 92:

*Excerpt:*

*“The late Dr. J. Tissot was Honorary Professor of General Physiology, National Museum of Natural History, Paris, France. His death occurred in June, 1950. He was a scientist in the fullest sense of the word. For several years he occupied himself with a series of cancer studies in collaboration with medical colleagues. He did not allow popular dogma to influence his interpretation of findings; he simply investigated, goaded on with the eternal question: Why? Preliminary findings were published in 1926, when he had gathered enough new material to warrant a book. For another ten years he continued his researches on the constitution of animal and vegetable organisms. During this period he made discoveries closely paralleling those of Béchamp. Further extensive research over the next twenty years confirmed Béchamp’s findings and elaborated upon them with technics more refined than any available to Béchamp.”*

*“These revolutionary findings of Tissot have been published in French under the title *Constitution des Organismes Animaux et Végétaux, Causes des Maladies Qui les Atteignent*. They are published in three separate volumes, each with an accompanying catalog of photomicrographs which clearly illustrate each point. Since the last volume went to press in 1946, it is definitely not outdated. As the work has not yet been published in the English language, it is necessary that we present an extensive analysis of it in order to show the need for a revision of bacteriology. We proceed, therefore, with the salient points of this vast generalization.”*

# **RATIONAL BACTERIOLOGY**

**J. R. Verner C. W. Weiant R. J. Watkins**

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J. R. Werner, C. W. Weiant, & R. J. Watkins  
Manufactured in the United States  
of America by H. Wolff, New York  
Second Edition, Revised and Enlarged

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## PREFACE TO SECOND EDITION

Aside from the fact that the first edition of this work has long been out of print, there are compelling reasons why a new, enlarged, and thoroughly revised edition should be brought out at this time.

In the past fifteen years drugless therapy has made enormous strides. The number of practitioners has doubled; the number of their adherents has tripled. Chiropractic in particular may be said to have come of age scientifically. Its educational institutions are now fully commensurate with its status as a mature and autonomous profession. One development, however, is deplorable.

In the absence of any organized body of material dealing critically with the germ theory of disease and the public health practices based thereon, it has become necessary to fall back upon medical textbooks in this field, if only to prepare students for state examining boards. Too often the instruction has been taken over by a new generation of younger teachers, generally well trained in the basic sciences at the university, but quite unfamiliar with the classic protests of an earlier generation against orthodoxy, as well as ignorant of the brilliant achievements of such modern representatives of unfettered investigation as Tissot and Speransky. Small wonder that many of these teachers have been inclined to make broad concessions to medical orthodoxy, concessions which need never have been made. If chiropractic is to maintain its traditional place in the vanguard of the fight for health freedom and rational therapy, the gap must be filled, and filled quickly. Hence the urgency felt by the authors in publishing a second edition of RATIONAL BACTERIOLOGY.

In the new edition, Section I has been completely rewritten to conform to the newer classification of micro-organisms, as well as to expand the material to the extent necessary to meet present day basic science board requirements. Section II is entirely new. In large part it is an exposition of the researches of the late Professor J. Tissot, of Paris, whose monumental work (as yet untranslated into English), product of a lifetime of vast scholarship and ingenious laboratory investigation, marks the fitting culmination of that great era in the history of French microbiology which began nearly a century ago with Béchamp and a few of his contemporaries. We believe firmly that it is impossible to exaggerate the importance of these researches, and hence make no apology for the amount of space devoted to them. Section III, dealing with the applications of bacteriological knowledge to the problems of disease, is the least altered portion of the book, although here too certain revisions and some new material will be found.

In view of the vastness of the material involved in our subject, the authors are well aware that much which might have been included has been omitted. We are likewise aware that the organization of material falls considerably short of perfection. However, in view of the urgency already pointed out, we feel that it would be unwise to delay publication in the vain hope of producing eventually a book with which we could feel completely satisfied. If the ideas herein advanced stimulate even a few readers to appropriate action and lead others to look more deeply into the sources and pursue the quest a bit further, our purpose will have been served.

J. ROBINSON VERNER, C. W. WEIANT , and R. J. WATKINS

## **PREFACE TO FIRST EDITION**

This Outline is written with two objects in mind. It aims, first of all, to give to the student and the drugless practitioner those basic facts and principles of bacteriology which underlie the hygiene of the communicable diseases and sanitation, which create an appreciation of the true role of bacteria in disease, and which make possible the interpretation of diagnostic laboratory reports. Incidentally, this is the knowledge usually required to pass a state board examination in the subject.

The book has, however, a second and more important object, namely, that of making public some of the outstanding results of medical and bacteriological research of the past few years which undermine the whole germ theory of disease causation and the practices of serum and vaccine therapy or prophylaxis based thereon. It is hoped by the authors that this material, all of which will be found carefully authenticated, may speedily become of service not only to professional groups, but to all laymen, especially parents and educators, who are interested in having the truth prevail.

New York C. W. Weiant  
May 2, 1933 J. R. Verner

## I: Formal Bacteriology



## 1 BACTERIA IN GENERAL

### *The discovery of bacteria*

In the latter part of the 17th century there lived in Delft, Holland, a lens-grinder named Anton van Leeuwenhoek. With a microscope of his own construction he examined specimens of water, excreta, scrapings from the teeth, and various other material in which he found a multitude of tiny moving forms which he called “animalcules.” A letter which he wrote to the Royal Society of London in 1683 describing his discovery, as well as the drawings he submitted, leaves no doubt that he was the first human being to see bacteria.

### *Development of bacteriology*

It was nearly a century before the scientific world paid much attention to Leeuwenhoek’s discovery. Müller, of Denmark, in 1786, contributed something to the knowledge of bacterial structure, and Ehrenberg, in 1838, published a work pointing out the fundamental differences between the larger forms. Succeeding investigators gathered more and more information concerning bacterial physiology and growth.

Modern bacteriology is said to have begun with the work of Pasteur during the latter half of the 19th century. Pasteur is generally credited with having demonstrated the relation of bacteria to fermentation. The credit for this discovery actually belongs, however, to Béchamp, whose writings were plagiarized by Pasteur. E. Douglas Hume, in her book *Béchamp or Pasteur?* has shown that Pasteur, far from being the genius and the idealistic benefactor of humanity he is commonly pictured, was in reality a careless and slipshod experimenter in many instances, that he was incapable of drawing correct inferences from his results, that he repeatedly appropriated the work of Béchamp as his own, and that it was only his clever courting of publicity and mastery of showmanship that enabled him to override the more modest, patient, and untiring Béchamp, the true scientist.

Bullock, in his *History of Bacteriology*, is critical of Béchamp for “talking too much.” Actually Béchamp was presenting basic ideas in biology, bacteriology, and pathology! Bullock did not examine the early work of Béchamp—the material which is really controversial.

Since the real relationships of Pasteur and Béchamp to bacteriology are not generally known, the matter is better left unmentioned on state board examinations. Tissot’s opinions will be dealt with later in this book. For more facts of the case refer to Hume’s book just mentioned.

Great impetus was given to the development of bacteriology by the German contemporary of Pasteur, Robert Koch, who introduced solid culture media, perfected the technic of obtaining bacteria in pure cultures, and established beyond question a relationship between bacteria and disease.

### *Nature of bacteria*

Bacteria are generally defined as unicellular plants, as distinguished from unicellular animals or protozoa. Many biologists, however, realizing that the distinction between plant and animal ceases to have much meaning in life-forms as primitive as these, place them in an indeterminate class by themselves. They lack the chlorophyll characteristic of plants, yet, like plants, are often capable of depending upon purely inorganic matter for food. Thus they are often considered the simplest

forms of fungi. On the other hand, they tend, like many of the lower animal forms, to utilize copper in the synthesis of pigments (instead of the magnesium of vegetable pigment) and, unlike most plants, many of them possess independent movement. Furthermore, their cell membrane contains no cellulose.

#### *Size and shape*

Bacteria are so small that they have to be magnified from 600 to 1000 times before they can be studied. Even then many varieties appear as mere points, and there is reason to believe that bacteria too small to be detected by any microscope exist. The unit in which the measurements of bacteria are given is the micro-millimeter or micron. This is equal to 1/1000 of a millimeter (about 1/25, 000 of an inch). It is generally designated in scientific writings by the Greek symbol  $\mu$ .

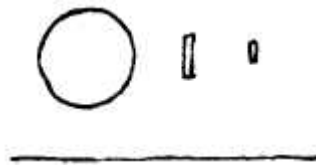


FIG. 1 Comparative size of human red blood corpuscle, typhoid bacillus, and influenza bacillus. (Line represents 1/25, 000 inch. )

Bacteria can be recognized as belonging roughly to three different groups classified according to shape:

- (1) rod-shaped forms or bacilli
- (2) spherical forms or cocci
- (3) spiral forms or spirilla

The cocci are further classed as diplococci, when they occur in pairs, streptococci, when they occur in chains, and staphylococci, when they occur in masses resembling bunches of grapes.



FIG. 2 Bacillus, coccus, and spirillum

For many years this grouping constituted the chief basis of classification, but the modern discovery that these forms are by no means fixed and the growing complexity of bacteriology have led to the adoption of a much more intricate scheme of classification.

#### *Structure*

Many bacteria (according to some, all bacteria) are surrounded by a capsule of carbohydrate nature. The cell membrane is not strongly differentiated. It is generally stated that bacteria have no nucleus. Some investigators, however, report the formation of a nucleus just prior to cell division, and practically all agree that nuclear material (chromatin) is present as scattered granules throughout the cell. Particles of nutritive and waste matter can also be detected in the protoplasm. The former include masses of fat, glycogen, and protein. Probably the most plausible view is that presented by the late George W. Crile, noted surgeon and biophysicist. Since the

bacteria are generally acid with relation to their immediate environment, just as the cellular nuclei are acid with relation to their cytoplasm, Crile suggests that the bacteria are naked nuclei or naked nuclear fragments with their immediate environment acting as the cytoplasm. This view coincides remarkably well with the views of Béchamp and Tissot as discussed later in this book.

### *Motility*

Some bacteria are equipped with delicate projecting filaments or flagella by means of which they are able to propel themselves through a fluid. The flagella are variable in number and may be terminal (attached only at one or both ends of a bacillus) or distributed around the entire cell. Motility varies with the environment. The organism may travel a distance 2000 times its own length in an hour. Flagella are not found on cocci. The criterion of motility has given rise to the following classification:

- A. Gymnobacteria—those without flagella
- B. Trichobacteria—(flagellates), those with flagella
  - 1. Monotrichous—bacteria with a single terminal flagellum
  - 2. Lophotrichous—bacteria with a single tuft of two or more flagella
  - 3. Amphitrichous—bacteria with bipolar tufts of flagella
  - 4. Peritrichous—bacteria with flagella around their entire periphery

### *Conditions favoring bacterial growth*

Bacteria in general thrive best in a warm, moist environment in which food is available. Very few can resist drying, heat above 100 degrees C., or direct sunlight. There are wide individual differences which will be noted in succeeding chapters.

### *Spore-formation*

In the life-cycle of some bacilli there develops within each rod a tiny oval body known as a spore. The spore is analogous to the seed of a plant. It is highly resistant and may exist for a long period during which unfavorable conditions prevent the growth and reproduction of the organism. In fact, such conditions are sometimes a necessary stimulus to spore-formation.

### *Mode of reproduction*

The usual mode of reproduction of bacteria is that of simple cell division or fission. Under favorable conditions this takes place about every twenty minutes. Variations from this typical process have been observed, however. For example, a budding, such as occurs in yeasts, or a Y-like splitting, may take place.



FIG. 3 Fission—Budding—Y-Formation

### *Growth of bacteria*

Bacteria may reproduce by binary fission at an average maximum rate of every twenty minutes but only for a short time. Cultures traverse the following periods of growth: (1) Lag phase of little multiplication while bacteria are becoming accustomed to the new medium; (2) Logarithmic period when multiplication is at maximum rate and the growth curve has become a straight line; (3) Stationary period when bacteria are dying as fast as they are being formed; (4) Death phase, usually fifteen to twenty-four hours, when bacteria are dying faster than they are being formed. This last phase is extremely interesting because it is here that involution forms gradually outnumber the “typical” forms. Since this involution and variation is not generally understood, it is not emphasized by orthodox bacteriologists. Its importance becomes quite apparent in the later sections of this book. In order to stress “monomorphism” and “fixed” species the laboratory cultures are quite frequently re-planted on fresh media and kept under rigid environmental conditions.

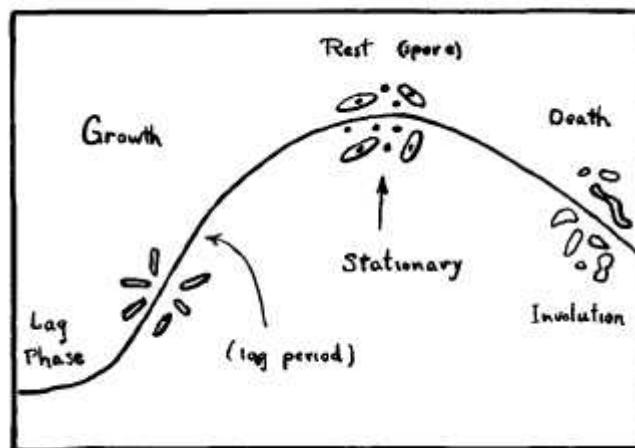


FIG. 4 Growth and death of bacteria

### *Other characteristics*

There are bacteria which require oxygen; these are known as aërobes. Others, strangely enough, require the absence of oxygen and are called anaërobes. Still others are quite indifferent in -this matter; these are called facultative anaërobes. Some bacteria, like those found regularly in the intestines, get along well only at the expense of another organism or host and are termed parasitic, while others, like those in the soil which decompose the bodies of dead animals, are quite able to shift for themselves and are termed saprophytic. Bacteria which produce highly colored pigments are called chromogenic; those which become phosphorescent and give off light, photogenic; and those which generate gas, aërogenic.

### *Biological importance of bacteria*

Of bacteria George A. Dorsey wrote: “Their daily bread is a few simple minerals. Without bacteria, air, land, and ocean to-day would be lifeless. They were the primordial chemists, finding food in a foodless world, drawing their energy and their nutrition from lifeless compounds. . . . Of living organisms, bacteria are presumably the lowest, simplest, and most primitive. Sulphur bacteria obtain their energy by the oxidation of sulphuretted hydrogen to sulphuric acid; with that energy

they fix nitrogen of the air and synthesize carbon compounds. We may speak of their energy as a bioelectric current; their growth as electro-synthesis. They deal direct with inorganic matter. They are a link in organic evolution. Whatever life is, they had it. They made more complex bodies possible.”

Of the origin of these remarkable organisms nothing is known. Bacteria have been found everywhere from beds of coal to the interior of meteorites. They may have brought life from interstellar space to this planet.

#### *Economic importance of bacteria*

It should not be forgotten that relatively few bacteria are regarded as pathogenic (disease-producing). Many are of the greatest economic importance. The nitrogen-fixing bacteria of the soil, for instance, make fertilizers unnecessary. Attaching themselves to the roots of certain plants, they take nitrogen from the atmosphere and combine it with other elements to form nutritive compounds which the plant can absorb through its roots. Tanning, cheese-making, the curing of tobacco, the fermentation of sauerkraut, and vinegar-making are essentially bacterial processes, while the great industry of food preservation would be an absurdity, if bacteria did not exist.

“Given a well-roasted mouse or two, baked straw, leaves, and other plant remains, a few lumps of granite, limestone, and apatite—all sterilized—and access to filtered air and boiled water, how would the plant fare? These things contain all that a plant needs, but the plant is unable to make use of ingredients, in such undigested forms. Plant and animal remains are in nature made available to plants through the agency of micro-organisms. Micro-organisms also take part in making minerals available to plant roots.” (Nicol: *Microbes by the Million*) Without bacteria in action there would be no more complex life forms. Where there is life there are germs. (Recent experiments at Notre Dame with “aseptic” animals are explained under the section on Tissot and do not alter the statements on the absolute necessity of bacteria in living processes. )

#### *Bacterial variation (Quotations are extracts from Jordan: General Bacteriology)*

Although bacteria are classified with extreme precision into orders, families, genera, species, types, and subtypes, there is definitely variation of morphology and functional characteristics. “Some of the earlier bacteriologists believe in the almost unlimited variability of bacteria.” This variation is entitled Pleomorphism. The opposing viewpoint, monomorphism, is Pasteur’s dictum that each species is rigidly fixed and invariable. Oligomorphism is a compromise term meaning few forms.

“Bacteria, like the higher forms of life, exist as fixed and stable organisms and there is no more likelihood of the transformation of the typhoid bacillus into the cholera vibrio than of the lion into the elephant or the oak tree into a pine. Although not unquestioned, as will be seen, this is still the dominant view in bacteriology. The concept of bacterial specificity has had a pragmatic triumph in the fields of epidemiology and public health.

“At the same time it must be admitted that the phenomena of variability in bacteria present certain special and peculiar problems. Sudden changes in bacterial cultures do occur; often these changes apparently overstep boundaries of ‘species’ and ‘genera’ formerly thought impassable.

“Knobbed, swollen, spindle-shaped, spiral, rod-shaped, and spherical cells have all been found in cultures previously descended from a single ‘typical’ cell of a familiar bacterial species such as the typhoid bacillus. The various morphological

types represent stages in a more or less complex life cycle. Growth in a particular stage may become stabilized so that the cells representing that stage go on reproducing themselves, as may happen in a 'standard' culture medium. It is not fair to regard the cells of this arrested stage as any more characteristic of the species than the cells of any other phase of development."

Yes, indeed! But the description of any species given in standard texts and required by state board examiners is always the "typical," arrested stage with almost no reference to any "atypical" forms. The latter present peculiar problems which are let alone by orthodox bacteriologists as being "too hot to handle," and that is what we must do before board examiners. The enigmas find their solution later on with Béchamp and Tissot.

"Connected with the variability of bacteria is their remarkable plasticity or adaptability to diverse conditions of life. By a series of inoculations or transfers it is possible to so alter bacteria that qualities originally present are sometimes accentuated, sometimes abolished. Theobald Smith has made the important suggestion that bacteria of great pathogenic power should be looked upon as incompletely adapted parasites that have not yet succeeded in establishing an equilibrium between themselves and their host. It may be urged that the ability of cell fragments to regenerate is no new thing in biology, and that it is quite as plausible to regard minute filterable forms of cocci or tubercle bacilli as portions of fragmented cells as to look on them as representing a significant filterable phase in their life history."

## **Glossary**

**acquired variations** usually brought about as a result of adaptation-temporary.

**adaptations** variations due to environmental changes (as in a patient).

**artificial selection** fixed characteristics brought about by rigid laboratory conditions.

**drug fast** resistant to the action of drugs. For example, gonococci are now generally resistant to the sulfonamides and are becoming quite resistant to the current antibiotics.

**hereditary variations** the permanent changes usually due to mutations.

**microbial dissociations** variations resulting in involution forms, gradual pleomorphism within a colony. Common in "aging" colonies.

**monomorphism** the concept of many separate, rigidly fixed bacterial species.

**mutations** sudden variants (sports) arising without reference to known environmental changes. Usually permanent.

**pleomorphism** the concept of many variations of one or a few bacterial species.

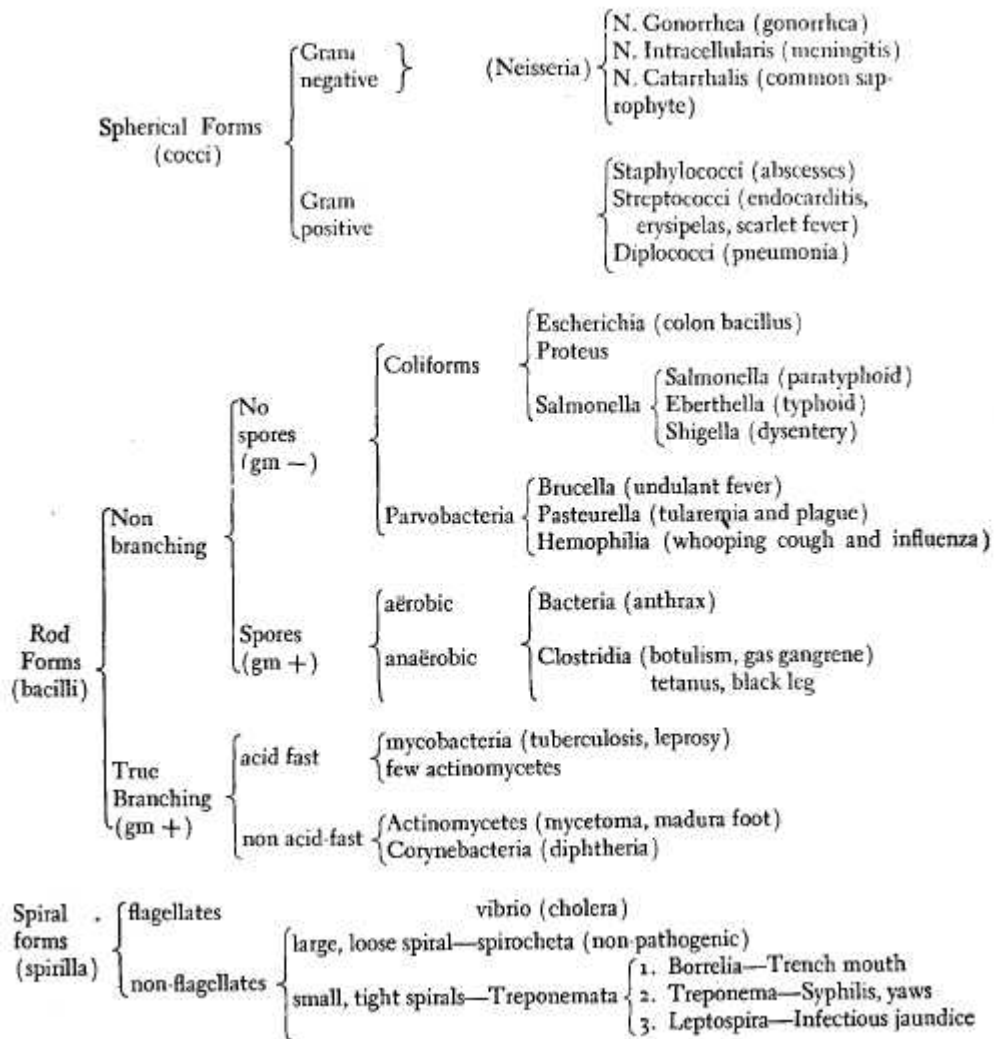
**S, R, and G colonies** these are colonies of smooth, rough or filterable variants.

Smooth colonies are usually virulent, motile and unstable. They tend continually to revert to rough, avirulent, non-motile, stable forms, which may in turn become "G" or filterable forms, considered by some as conidia.

This is enough of the detail needed for state board examiners, but the real issues are dealt with in later sections of this book.

## Classification of Bacteria—Artificial Key

We present now a very useful key to bacterial classification. It is much more practical for board examination preparation than is the accepted biological classification in Chapter IV. Familiarity with this key will be a big help in studying the various species.



## Historical Data

*(Items frequently asked by state board examiners)*

Humoral pathology—the theory that disease is due to improper mixture of the vital fluids of the body: blood, phlegm, yellow bile, and black bile. This theory was generally accepted in the time of Hippocrates.

Hippocrates (460-370 BC)—called “The Father of Medicine” because of his scientific approach to problems.

Spontaneous generation—the doctrine that living things began spontaneously without parents.

Leeuwenhoek (1632-1723)—made first microscope and described bacteria.

Spallanzani (1729-1799)—observed that boiled sealed infusoria give no evidence of decomposition.

Fracastorius in 1546 connected communicable diseases with a “contagion vivum.”

Jenner (1749-1823)—“Father of Immunology,” introduced smallpox vaccination.

Ehrenberg (1795-1876)—noted for early bacterial classification.

Pasteur (1822-1895)—called “Father of Bacteriology” by virtue of work on fermentation, and immunizations against anthrax and rabies. (See Tissot’s comments)

Ehrlich (1854-1915)—noted for “side-chain” theory of immunity and “606,” arsphenamine, used in treating syphilis.

Lister (1827-1912)—“Father of Aseptic Surgery.”

Semmelweis in 1846 proved childbed fever to be due to transmission of filth.

Koch (1843-1910)—noted for his postulates and for discovery of the tubercle bacillus.

Metchnikoff (1845-?)—noted for discovery of phagocytosis.

Virchow (1821-1902)—noted for “Cellular Pathology.”

Schaudinn (1871-1906)—noted for discovery of *Treponema pallidum*.

Noguchi (1876-1928)—noted for research on Spirochetales.



## 2 METHODS OF STUDYING BACTERIA

### *Culture media*

The first requisite for cultivating bacteria so that they may be studied is a suitable nutrient medium. The substances commonly employed for this purpose are beef broth, blood serum, milk, gelatin, potato, and agar (a gelatinous material obtained from a Japanese seaweed). The medium, after sterilization, if a liquid, is placed in a sterilized test tube which is then sealed with a cotton plug; if a solid, it can be placed in a covered glass dish called a Petri dish. We can introduce bacteria to be cultivated by exposing the medium to the air, by blowing the breath upon it, or by mixing it with a sample of any material such as milk, water, blood, saliva, pus, etc., which it is proposed to investigate.



FIG 5. Petri dish

Many bacteria require highly specialized media in order to present “fixed” characteristics. Thus, media are prepared in different fluid, semi-fluid or solid states. Aërobes will usually grow on plates, but anaërobes grow better in “stab” cultures prepared by inoculating deep into the solid medium, thereby excluding the air. Some media must be filtered. Others are first cleared by the entangling mesh of coagulating albumin. Viruses and Rickettsiae are so dependent upon a living host that minced chick embryo is commonly used. The pH of the medium must be carefully adjusted in nearly every case.

### *Incubation*

When the bacterial garden has been planted in this way, it is transferred to an incubator. The incubator is a kind of oven the walls of which are filled with water. The interior can be kept at a constant temperature by an automatically regulated flame below. Electrically heated and controlled incubators are also made. The bacteria of water and soil are usually incubated at about 60 degrees F.; bacteria of the sort found in the human body are cultivated at 99 degrees F.

After twelve to twenty hours the medium, which was originally clear, will be found covered with small rounded spots varying from the size of a pinhead to a half inch in diameter. Each of these spots was originally a single bacterium, which by multiplication has produced this accumulation of organisms exactly like itself. Such an accumulation is known as a “colony.” The number of colonies is the number of the original bacteria. This is the regular method of counting the bacteria in a measured volume of water or milk. If incubation proceeds long enough, the colonies coalesce and the medium becomes uniformly cloudy, or takes on the appearance of a slimy mass.

### *Preparation of a pure culture*

When a given colony has been identified by examining a sample under the microscope, the particular germ may be obtained in pure culture by using a platinum wire which has been sterilized in a flame to transfer some of this colony to a new

sterile medium. Cultures prepared in Petri dishes are called plate cultures, and the process of picking out specific types of bacteria from individual colonies for separate cultivation and study is spoken of as “fishing” a plate.

#### *Effects of bacterial growth upon the medium*

What happens to the medium, when a particular germ is cultivated in or upon it, constitutes a valuable aid in the identification of the organism. Gelatin, for example, may or may not be liquefied. Milk may be soured, coagulated, putrefied, or remain unchanged. Sugar may or may not be fermented. Some bacteria produce acids; others do not. Gas is sometimes evolved.

This is the basis for nearly all determinative procedures of morphologically similar organisms. All the enzymes found in the human digestive tract occur also among the many bacterial species. Many of the differentiations are based on fermentation of dextrose, lactose, rhamnose, mannitol, xylose, inositol, or indole. Such fine differentiations are important to a bacteriologist. Yet even these procedures may not be adequate for differentiation. Recourse may then be had to animal inoculation to determine virulence. The various determinations will be included at appropriate points whenever there is adequate demand by state boards for such information.

#### *Anaërobic cultures*

When anaërobic bacteria are to be cultivated, the process may be carried on under a Novy jar. Such a jar somewhat resembles a bell jar, but is equipped with connections through which gases may be introduced or withdrawn. Hydrogen or nitrogen is generally employed. A simpler and very common method is that of making “stab” cultures as mentioned above.



FIG. 6 Novy jar



FIG. 7 Fermentation tube

#### *Preparation of bacteria for microscopic examination*

The material to be examined (culture, pus, sputum, exudate, etc. ) is spread thinly upon the slide and allowed to dry. It is then “fixed,” by passing the slide a few times through a flame. This kills the bacteria, coagulates the material, and prevents it from being washed off in the staining process. The slide is now flooded with a watery solution of a dye, methylene blue, gentian violet, and fuchsin being the dyes most often used. After several minutes the stain is poured off and the slide washed with water and blotted dry. It is now ready to be examined.

Sometimes a special substance known as a mordant must be supplied to make the bacteria take the stain. For instance, carbolic acid is required in staining tubercle bacilli with fuchsin. Ordinarily acids remove the stain. Bacteria which retain the stain after treatment with acid are spoken of as “acid-fast” bacteria.

#### *Staining methods*

The Gram method is very commonly used in identifying bacteria. It is named for Gram, who devised it. The procedure is as follows:

1. Fix gently in dry heat by passing through a flame
2. Stain two or three minutes with gentian violet
3. Flush and stain with Gram's iodine one minute
4. Wash with water and blot; do not dry
5. Decolorize with acetone or alcohol and blot
6. Counterstain ten seconds or more with safranin or Bismarek brown
7. Wash with water, blot, dry, and examine

If the violet color is retained, the organism is said to be Gram positive; if, on the other hand, the violet stain is lost, the bacterium is Gram negative. One of the most common questions is to name five or ten Gram negative and five or ten Gram positive organisms. As a help the following chart is included:

#### GRAM POSITIVE

##### Cocci

Staphylococcus  
Streptococcus  
Pneumococcus

##### Spore formers

Bacilli—anthracis, subtilis  
Clostridia—tetani, botulinum, perfringens, etc.

##### Branching

Mycobacteria—tubercle bacillus, M. leprae, etc.  
Corynebacteria—Diphtheria  
Actinomycetes—Bovis and hominis

#### GRAM NEGATIVE

##### Neisseriae

Gonococcus  
Meningococcus  
Catarrhalis, etc.

##### Coliforms

Esch. coli  
Typhoid  
Paratyphoid  
Dysentery  
Proteus, etc.

##### Pseudomonaceae

Vibrios  
Pseudomonas

##### Parvobacteriaceae

Brucella  
Pasteurella  
Hemophilus

The Ziehl-Neelsen carbol-fuchsin method is used to stain the “acid-fast” organisms which have a heavy waxy sheath that resists ordinary stains. It is used for staining tubercle, leprosy, and smegma bacilli.

1. Flood with carbol-fuchsin and steam for five minutes

2. Wash and decolorize with acid-alcohol solution (2% HCl in 95% alcohol)
3. Wash with water and blot
4. Counterstain with methylene blue, wash, and dry.

### *Sterilization*

It is obvious that it frequently becomes necessary in handling bacteria in the laboratory to have sterile apparatus and materials. Platinum wires, as already stated, and many other pieces of apparatus can be sterilized directly in a flame. Glassware is first cleansed by boiling in soapsuds and then heated in a hot-air sterilizer at a temperature of 170 to 190 degrees C. for fifteen to twenty minutes. Before placing in the sterilizer, articles made of glass are wrapped in paper or placed in metal cans. Culture media are sterilized by steam under pressure in an apparatus called the autoclave. As the very high temperature of the autoclave (about 120 degrees C. ) renders some media unsuitable for use, another method known as discontinuous sterilization is sometimes used. This is carried out in a steam sterilizer, an apparatus somewhat similar to a double-boiler. The boiling is continued for fifteen or twenty minutes and repeated on one or two succeeding days. While spores are not killed by boiling, it is thought that they will, in the interval between boilings, pass over into the ordinary vegetative, spore-free form of bacteria which can be killed at the next boiling. This process is sometimes called fractionation.

### *Glossary*

**thermal death point** is that temperature required to kill a young culture of an organism with ten minutes' exposure to moist heat.

**thermostabile** able to withstand 55 degrees C. without alteration.

**thermolabile** destroyed by a temperature of 56 degrees C.

**disinfectant** an agent which destroys infective agents; a bactericide.

**antiseptic** an agent which prevents decay—inhibits bacterial growth. (Hypertonic saline is more effective than most commercial antiseptics. )

**bacteriostat** an agent which inhibits bacterial growth. Penicillin, aureomycin and the rest are typical examples. They are not bactericides.

**bactericide** an agent which kills bacteria. Seldom used internally because it would also kill or seriously injure the host.

**germicide** an agent which kills germs.

**germifuge** an agent which expels germs.

**phenol coefficient** the ratio of the bactericidal action of a disinfectant to that of phenol. Actually it is an inverse ratio of dilutions capable of killing typhoid bacilli in five minutes. (Hugh Nicol in *Microbes by the Million* regards this as a poor term. He describes an experiment whereby anyone, in his own kitchen with only a few water glasses, can demonstrate that bacteria will thrive and grow abundantly upon phenol. )

### 3 PATHOGENICITY, INFECTION AND IMMUNITY

This chapter is to be studied and understood, not because it is the whole truth, as is demonstrated throughout this book, but rather because it presents the half-truths in the generally accepted medical hypothesis on the subject. (Notes in parentheses are editorial comments on the academic material and are NOT a part of the medical hypothesis. ) Unless otherwise noted, quotations are from Jordan.

#### *Pathogenicity*

“The conception of a pathogenic micro-organism is a relative, not an absolute, one; that is to say, no microbe is known that is capable under all conditions of producing disease in all animals. The power of a microbe to produce morbid effects or changes depends, therefore, as much upon the nature of the host as upon its own characteristics.”

Virulence is extremely variable to different animals. A micro-organism generally considered virulent to man is usually totally non-virulent to other animals. The converse is also generally true. Virulence is extremely variable for man, the same organisms being present in both healthy and diseased individuals. Diphtheria bacilli and pneumococci are frequently found in normal, healthy persons. “The attempt to control the spread of meningitis by quarantining carriers is like sweeping back the Atlantic Ocean with a mop. If the policy of quarantining all proved carriers had been carried out in certain army camps the number of quarantined soldiers would have far exceeded those left in the ranks.” Virulence is extremely variable even within the same culture. A fresh culture may be strongly virulent, but after it ages for a few days on the same plate the virulence disappears. Aside from the age of a culture, there are still many other qualifications necessary for “infection.”

#### *Infection*

There are five requisites to “infection” of a host by a microorganism:

1. The micro-organism must be present in sufficient numbers.
2. The micro-organism must be of sufficient virulence.
3. The micro-organism must be introduced through a suitable portal of entry.
4. The micro-organism must be introduced into a susceptible host.
5. The susceptible host must have a lowered resistance.

(Such a combination of conditions, each being variable, must be present. The last, which is by far the most important, is treated lightly by medicine as a matter of malnutrition, excessive fatigue, etc. We will return to this subject in connection with our discussion of Tissot. Of particular interest, however, is the following note on the inoculation of monkeys with syphilis: “Subcutaneous, intraperitoneal, and intravenous inoculations, even of the most virulent material, are without effect, but cutaneous inoculation, particularly upon the eyebrows and genitals, is usually followed by typical primary lesions.” This phenomenon clearly demonstrates the role of the antigen as a specific nerve stimulus. )

#### *Body defense*

There are generally considered to be three lines of body defense:

1. Skin and mucosa. Myriads of pathogens are present upon them at all times.
2. Leukocytes and “natural immunity.”
3. Antibodies of “acquired immunity.”

The various secretions of the skin and mucosa have been demonstrated to possess bactericidal properties, though often extremely slight. Still as long as they remain unbroken, “there is a more or less impassable barrier. Under some conditions, not clearly understood, the natural defenses of the lungs are broken down and infection seems to occur rather readily. Well known pathogenic bacteria like the typhoid bacillus and the cholera spirillum may exist in the human intestine, . . . without having induced disease.” This mixture of many saprophytes, commensals and a few pathogens on any location is called the “normal flora”; that is, many normal, healthy mouths and throats harbor staphylococci, streptococci, and *B. subtilis*, some pneumococci, *Endamoeba gingivalis*, *Treponema microdentium*, *Corynebacterium diphtheriae*, *Klebsiella*, and others.

### *Immunity*

This topic, though generally speculative as to mechanisms, is divided into several headings: natural, actively acquired, and passively acquired immunities and their mechanisms. Natural immunity is observed but not generally understood. Various species of animals are quite immune to organisms which are pathogenic to other species. Cold blooded animals, for example, are normally immune to tetanus, but if they are warmed up and maintained at a temperature close to 37 degrees C, then their immunity disappears. Field mice are highly susceptible to glanders, while house mice are almost completely immune. Certain races of mankind are much more susceptible than others to particular diseases, although such differences are now attributed largely to environment and mode of living rather than to an inherited factor. (It is hoped that complete study of the present work will afford clearer insight into the matter of natural immunity. )

Acquired immunity is a resistance to bacterial infection which was not inborn. When bacteria and other parasites invade the tissues, they evoke many remarkable reactions in the body of their host. One of them is the development of a new resistance to further infection by the same micro-organism. The mechanism of this new resistance has been studied at length. It involves the phenomenon of antibody formation. Almost any foreign protein, when introduced into the body, elicits a similar reaction. The stimulating foreign protein is called an antigen. The substance developed by the host to counteract further invasion by the same antigen is called the antibody. When the antibody is actively produced by the host as an antigenic response, the new resistance is called an actively acquired immunity. When the antibody is produced in some other host and then introduced second-hand into an animal, the recipient has done no active work in antibody production, and any new resistance the recipient may have thereafter is called passively acquired immunity.

### *Antibodies*

The observed phenomena of antigenic response are quite varied. Their mechanisms have been explained in the following terminology:

*Antitoxins* are substances in the serum which nullify the toxin. They have often been isolated, and their ability to chemically neutralize the toxin has been demonstrated in vitro. Usually, the toxin is thermostabile, while the antitoxin is

themolabile. Because of this it is possible to neutralize a toxin in vitro and inject the mixture as an antigen into an animal without pathogenesis; but if the mixture is heated above 56 degrees C. to destroy the antitoxin, then the mixture, when injected, will be pathogenic. Diphtheria toxin-antitoxin supplies an example of this “non-toxic” antigen. It is 85% neutralized, and since the mixture is antigenic but not highly toxic, it has been used as an antigen to stimulate development of actively acquired immunity in children. (An evaluation of this phenomenon appears later in this book. )

*Lysins* are those agents which cause dissolution. Hemolysin breaks down red cells. Bacteriolysins break down bacteria. Cholera spirilla introduced into the peritoneal cavity of an immunized animal lose their motility, swell up, and crumble into small fragments. This was first observed by Pfeiffer and is called “Pfeiffer’s phenomenon.” Much investigation suggests that there are two substances in action here: one that is present in normal serum, which is called alexin or Ehrlich’s “complement,” and a second substance, which is present in small amounts in normal serum but in much greater quantity in the serum of immunized animals. The latter is the lysin, Bordet’s “sensitizer” or Ehrlich’s “amboceptor.”

*Phagocytes* are leukocytes which according to Metchnikoff are “devouring cells.” These are the little “white soldiers” which “police” the area, pounce upon any nasty little pathogens and gobble them up. In the blood stream the neutrophils are the main phagocytes, while outside the blood stream macrophages (reticulo-endothelial cells or large mononuclears) are the major phagocytic agents. The power of leukocytes to engulf and destroy micro-organisms is greatly enhanced by the addition of serum from immunized animals.

*Opsonins* (Gr. —to prepare food for) are the sensitizing agents involved in this process.

*Agglutinins*: If the serum from a recovered typhoid patient is added to a suspension of typhoid bacilli, the bacilli become motionless and clump together. This agglutination is supposedly due to a substance, agglutinin, developed as an antigenic response. Many serological tests are based on this phenomenon. The Gruber-Widal test for typhoid is one of the classic examples.

*Precipitins*: This phenomenon is almost like agglutination except that a fine powdery precipitate is formed, rather than the fluffy clumps.

Remember that these phenomena are all utilized in the hypothesis commonly given to explain immunity. Much evaluation is attempted later in this book. The most elaborate and ingenious hypothesis to explain these phenomena was that of Ehrlich; it is known as the “side-chain” or receptor theory.

*Ehrlich’s side-chain theory*: Various body cells (in Ehrlich’s imagination) act as though they had “mouths” to extract food from fluid environment. These mouths are the “cell receptors” or side-chains, so-called because the cells are imagined as analogous to complex organic molecules with a stable central structure like the benzene ring and numerous “side-chains,” the “mouths.” The receptors may be simple or complex. Certain toxic substances “fit” into the chemical (structural) formula of the cell receptors. If the antigen does not damage the cell too much, the cell overcompensates and grows many receptors. The surplus receptors are discharged into the blood stream where they function as free antibodies. In trying to explain the various phenomena Ehrlich postulated the existence of receptors of first, second, and third orders. His terms “amboceptor” and “complement” are listed in the glossary. The details of his theory can be found in most reference texts for

bacteriologists. The Wasserman test for syphilis is based upon his “complement-fixation,” even though the antigen used (a lipoid beef-heart extract) has absolutely nothing to do with syphilis, and though any febrile disease at certain stages may show a positive Wasserman reaction.

Since the examining boards occasionally ask questions concerning the three orders of immune bodies, a few words of explanation are included. The concept is a development of Ehrlich’s side-chain theory. Immune bodies of the first order have one combining portion only. The antitoxins are the only members of this group. Immune bodies of the second order have a combining portion (haptophore) and a ferment (zymophore). Since the ferment is thermolabile and the immune body must be complete to function, heating above 56 degrees C. will inactivate immune bodies of this order, to which belong agglutinins, precipitins and opsonins. Immune bodies of the third order have two combining affinities, one for the antigen and the other for the complement which supplements or completes the specific action of the immune substance. The complement is sometimes referred to as linking the antigen with the immune body. Complement is similar to an enzyme and is present in normal serum, but is inactive by itself as an immune body. Some authorities report that complement (alexin) is trypsin reabsorbed by the intestinal mucosa. The complement is non-specific and thermolabile. Bacteriolysins and hemolysins are of this third order. “Recent investigators... discard Ehrlich’s hypothesis. . . .”

#### *Allergy and anaphylaxis*

Theories of anaphylaxis are generally either proteolytic or colloidal. The first supposes that the dire results are from splitting proteins into toxic and non-toxic portions. The second supposes that there is a clumping of plasma proteins due to colloidal disequilibrium. The phenomenon of anaphylaxis is that observed when the first injection of a foreign protein has sensitized the host to a second injection which causes shock and often death. This is part of the usual explanation of “serum accidents.” Allergy is a rather mild, but relatively continuous hypersensitivity to various antigens. Atopy is a term used to designate inherited human hypersensitivity. McDonagh makes a fine colloidal explanation (J. E. R. McDonagh, The “Nature of Disease, Heinemann Ltd. ) but Tissot makes an equally suitable alternative explanation, which is outlined in this text.



## 4 BACTERIAL CLASSIFICATION

From the day that Leeuwenhoek first saw bacteria he noted their various shapes and actions. Since that time there has been an endless attempt to classify bacteria, just as trees and birds are classified and named. The science of biological classification is called taxonomy. The taxonomic position of bacteria is midway between plants and animals. The taxonomic position of the Spirochetales is midway between the true bacteria and the protozoa. Müller in 1773 made the earliest simple classification and naming, but Ehrenberg followed up in much more detail.

Morphology was the earliest criterion, to which was soon added motility. Staining characteristics provided further differentiation. Better microscopy and better staining methods showed flagella, but the cultural characteristics soon caused a dizzy inflation of the number of species. Filter-passing ability brought a new order, the Virales. Serological characteristics have again vastly increased the number of species and have added subspecies, types, and strains.

Confusion was inevitable in this cataloguing. Repeated revisions and reclassifications have corrected the most glaring faults, but there are still far too many loopholes. Repeated revisions mean that present day bacteriologists have had to learn as many as four accepted names for the same organism. To illustrate: *Serratia mareescens* was only a few years ago the *Bacillus prodigiosum*; *Malleomyces mallei* was *Actinobacillus mallei* and before that was an *Actinomycete*; *Fusobacterium plauti-vincenti* was *Bacillus fusiformis*; *Micrococcus pyogenes* has always been *Staphylococcus*, but the “Staph” is no more; *Salmonella typhosa* was *Eberthella typhosa*; while *Pseudomonas aeruginosa* has had three previously accepted names. There are many other examples.

Glaring faults in the latest accepted classification which are openly admitted include these: The *Rickettsiales* are non-filter-passing while the *Virales* are. Still the filter-passing *Cowdria* is classed as a *Rickettsia*. The *Eubacteriales* are “non-branching,” while the *Actinomycetales* are “branching and mycelial.” “Long filaments with swollen ends and true branching place the glanders bacillus {*Malleomyces mallei*} in the order of *Actinomycetales*.” (Jordan) Nevertheless, this organism has been moved into the *Eubacteriales* as a genus under the *Pasteurella* tribe. The *Corynebacteria* were previously classed with the tubercle bacillus as *Actinomycetales* because of their clubbing and branching, but are now placed as a separate order of *Eubacteriales* (non-branching). *Mycobacteria* are keyed as acid-fast but many strains of *M. tuberculosis* are not acid-fast. Polar flagellation is the criterion for listing under *Pseudomonaceae*, but there are several species of polar flagellates now listed as *Rhizobia*, *Micrococci*, while one entire genus of these polar flagellates is listed under *Corynebacteria*. *Streptococci* and *Corynebacteria* to be so named should be non-motile, but several of each group are motile. *Neisseria* are keyed as obligate parasites needing body fluids for culture yet *N. Catarrhalis* grows easily and abundantly on any ordinary media. Some of the *Sarcina* form spores, while among the spore-formers many strains at times simply refuse to sporulate under any conditions. Instead of being hypercritical of this impossible task of the complete cataloguing of bacteria, let us briefly consider the generalities of classification.

The Orders are differentiated largely by morphology.

The Families are differentiated about one-third by morphology and two-thirds by cultural characteristics.

The Tribes and Genera are differentiated almost entirely by cultural characteristics.

The Species are differentiated partly by cultural characteristics and partly by serological characteristics.

In order to be familiar with the major attempts at classification which are so often requested on board examinations, one should look over the general keys to the classification of the orders and families which are included here. This material shows the present consensus of authoritative biological opinion. For more elaborate details of taxonomy refer to Bergey's *Manual of Determinative Bacteriology*, 6th ed.

### **subdivision of classes into orders**

CLASS: Schizomycetes (Nageli)

*Order 1* Eubacteriales—rigid cells, not branched or mycelial

Suborder I—Eubacteriineae—unattached to substrate, no photosynthesis

Suborder II—Caulobacteriineae—attached by stem to substrate, no photosynthesis

Suborder III—Rhodobacteriineae—photosynthetic pigments in the cells

*Order 2* Actinomycetales—rigid branched and mycelial with spores and conidia

*Order 3* Chlamydoxiales—sheathed rigid cells

*Order 4* Myxobacteriales—flexuous cells which creep on substrate

*Order 5* Spirochetales—flexuous cells, free-swimming by flexion of spirals

Supplemental Orders:

I—Rickettsiales—obligate intracellular parasites, over 0.1 micron (non-filterable)

II—Virales—obligate intracellular parasites, under 0.1 micron (usually filter-passing)

III—Borreliales—grow in cell-free media and develop highly polymorphic rings, globules, filaments and minute reproductive bodies

### **subdivisions of the virales**

*Suborder I*—Phagocytivales—infected bacteria (D'Herelle's bacteriophage)

*Suborder II*—Phytophagocytivales (infected higher plants—mosaic, wilt, etc.)

*Suborder III*—Zooprophagocytivales (infected animals—includes the viruses of poliomyelitis, influenza, etc.)

### **division of orders (or sub-orders) into families**

*Sub-order eubacteriineae*

1. Nitrobacteriaceae—Autotrophic (all others, except many Bacteriaceae, are heterotrophic).
2. Pseudomonaceae—Gram-positive polar flagellate rods (Pseudomonas are straight, while Vibrios are curved.)
3. Azotobacteriaceae—Pleomorphic nitrogen fixers, free-living in soil
4. Rhizobiaceae—less pleomorphic nitrogen fixers, nodule bacteria
5. Micrococcaceae—cocci easily grown in masses or packets, usually gram-positive Gaffkya in tetrads

- Sarcina in cubes  
 Micrococcus in plates, groups or masses
6. Neisseriaceae—gram-negative diplococci, obligate parasites, need body fluid.
  7. Lactobacteriaceae—gram-positive microaerophilic sugar fermenters
    - Diplococci: parasites, usually paired
    - Streptococci: in chains
    - Leuconostoc: saprophytes
  8. Corynebacteriaceae—gram-positive, usually aerobic, clubbing and branching rods
    - Corynebacteria: pleomorphic rods with few polar flagellates
    - Listeria: one to four flagella
  9. Achromobacteriaceae—gram-negative rods, fermenting sugar with little gas
  10. Enterobacteriaceae—gram-negative rods, producing gas from glucose
  11. Parvobacteriaceae—small, pleomorphic, gram-negative rods, usually needing body fluids for growth
  12. Bacteriaceae—any non-sporing gram-negative rod not classified above
  13. Bacillaceae—gram-positive spore-forming rods
    - Bacilli—aerobic
    - Clostridia—anaerobic

*Order—Actinomycetales*

1. Mycobacteriaceae—gram-positive, acid-fast rods with rudimentary mycelium
2. Actinomycetaceae—true mycelium fragmenting into rods and cocci
3. Streptomycetaceae—usually undivided mycelium

*Order—Spirochetales*

1. Spirochetaceae—large loose spirals with definite protoplasmic structures
2. Treponemataceae—small tight spirals with no definite protoplasmic structures
  - a. Borrelia—stain easily with aniline dyes
  - b. Treponema—stain with difficulty, strict anaerobes
  - c. Leptospira—stain with difficulty, aerobes

Upon review of the scheme just presented we notice that the previously mentioned faults are quite obvious. Equally glaring is the lack of a definite borderline between Rickettsia and Virus. The former are defined as being in excess of 0.1 micron and are non-filter-passing, while the latter are smaller and pass through filters. Yet one genus of Rickettsia is filter-passing, while several of the plant mosaic viruses are too large to be filterable. The *Vibrio comma* shows definite spiral forms in culture and was previously classed with the morphologically similar spiral organisms. Some of the Achromobacteria are chromogenic, and the *Shigella*, which do not produce gas, are Enterobacteria (gas producers). One of the largest of all rod bacteria, the fusiform bacillus, is classified as a Parvobacterium (tiny bacterium). Consequently upon survey we note that bacterial classification is in sad confusion despite continual improvement by revision. The bacteriologists who work with the micro-organisms every day know that there are as many exceptions as there are rules. This makes their work nearly as much an art as a science.

“We do not yet know enough about bacteria and their life histories to establish any classification and nomenclature based on ‘genetic relationships,’ and for some

time to come our names, 'types' and subdivisions will continue to conform to our convenience . . ." (Jordan)

Careful unbiased investigation shows extreme pleomorphism to be the normal state of affairs. Any strict classification is therefore use less. A differentiation of closely related groups does, however, give clues to the state of degeneration of a patient and often guides the general prognosis. Medical practitioners find some consolation in that guidance, although their current chemotherapy is extremely generalized and almost entirely symptomatic. This puts detailed orthodox bacteriology into the category of an academic hobby.

The real significance and interrelationship of bacteria, brought out clearly in the second section of this book, have been widely missed by orthodox bacteriologists. This has usually not been their own fault of commission, but rather a fault of omission—failure to undertake unbiased study. A familiarity with the first section of this book is, however, necessary to understanding the second section.

## 5 THE STAPHYLOCOCCUS

Staphylococcus, like streptococcus, should be thought of in connection with pus formation. It is as widely distributed as streptococcus, but is much more resistant to adverse conditions. In the human body, it appears much more frequently in osseous pathology than does streptococcus.

### *Historical*

Observed by Pasteur in 1880

Isolated in pure culture by Rosenbach in 1884

### *Characteristics*

Spherical cells 0.7 to 0.9 micron in diameter

Seen commonly in irregular clusters resembling bunches of grapes

Gram positive

Grows well on ordinary media

Liquefies gelatin, ferments milk and most sugars

### *Varieties*

There are twenty-two accepted species and 481 questionable species. Bergey's ninth variety is *Micrococcus pyogenes* with two subspecies, *aureus* and *albus*. The tenth species is *M. citreus*. Previous terminology for these three varieties of clinical importance was:

*Staphylococcus albus* (*M. pyogenes-albus*)—white colonies

*Staphylococcus aureus* (*M. pyogenes-aureus*)—golden yellow colonies

*Staphylococcus citreus* (*M. citreus*)—lemon-yellow colonies

### *Habitat*

Air, skin (constantly), mucous membranes, and pus

### *Resistance*

One of the most resistant of non-spore-forming bacteria

May withstand 140 degrees F. an hour

May live for months in dried pus

In pus, killed by bichloride of mercury, 1:1000, only after several hours

### *Pathological conditions involving staphylococcus*

Acute suppurative inflammations

Acute abscess

Boils and carbuncles

Empyema and influenza

Septicemia and pyemia

Endocarditis

Osteomyelitis and periostitis

Some cases of food poisoning

The numerous staphylococcic toxins include a dermatotoxic, an enterotoxic, a hemolysin (staphylolysin), and leucocidin.

## REMARKS (critical)

These remarks are not totally in line with medical ideology as will become apparent. Hence they are better unquoted on state board examinations.

The constant and intimate association of staphylococcus with all animal and human life denies any assertion that it is primarily and solely responsible for disease conditions. A deeper, more basic reason will always be found to account for the pathology in which this organism appears to have a role.

### *Other micrococci*

Two other classifications are very similar to staphylococci but due to slight differences are separated. Gaffkya, named after Gaffky, who first described them, were previously called Micrococcus tetragenae because they commonly are found in tetrads. Like streptococci they do not liquefy gelatin as do the staphylococci. The other group, Sarcina, are commonly found in cubical packets of eight or more. They are of only slight significance.

## 6 THE STREPTOCOCCUS

The streptococcus (called, when associated with pus formation, streptococcus pyogenes) is a nearly ubiquitous parasitic organism of which many strains of varying characteristics have been observed. Perhaps no other micro-organism has been blamed for so many and various pathological conditions.

### *Characteristics*

- Spherical cells about one micron in diameter
- Division takes place in only one plane, giving rise to chain formation.
- Occasionally seen in pairs
- Develops well on agar or gelatin to which dextrose or blood has been added
- Gelatin ordinarily not liquefied
- Milk usually curdled by fermentation of lactose
- Optimum temperature 37 degrees C.
- Insoluble in bile (thereby distinguished from pneumococcus)  
(Jordan admits that some strains of staphylococcus are soluble in bile)
- Not encapsulated
- Gram positive

### *Varieties*

There are thirty accepted and one hundred eighty questionable species, mostly of little consequence to man. These are placed in eleven serological groups, seven of little interest. The usual names given to the two types of any consequence are: streptococcus hemolyticus, which dissolves erythrocytes, and streptococcus viridans, which produces a greenish coloration about its colonies on blood-agar. The distinction is not sharp, since both types have both actions, but the former, the beta type—streptococcus hemolyticus—by means of two lysins, Streptolysins O and S, produces more hemolysis. The alpha type—streptococcus viridans—produces more green and less hemolysis. A third, rather insignificant type, gamma streptococcus, has no effect on blood agar media. The most common type found in pathological conditions is the S. hemolyticus (beta) with its various subtypes. This type (S. hem.) is quite commonly called streptococcus pyogenes, the two names being freely interchangeable.

### *Habitat*

Air, milk, healthy skin, mucous membranes of throat, intestines, vagina, etc., and pus

### *Agents which kill streptococcus pyogenes*

- Bichloride of mercury, 1:5000
- 1% carbolic acid
- ½ % lysol
- Temperature of 130 degrees F. (10 to 20 minutes)

### *Pathological conditions involving streptococcus*

- Septic sore throat
- Scarlet fever
- Erysipelas
- Puerperal fever
- Rheumatic fever

Arthritis  
Endocarditis  
Numerous suppurative inflammations

#### *Serology*

The Lancefeld grouping method, commonly used today, is a precipitation reaction using specific cultured streptococcic antigens. The eleven groups are lettered alphabetically, but only the first three groups are of any consequence. Nearly all the human pathogens are in group A.

#### REMARKS (on the significance of streptococcus)

These remarks are not a part of the accepted medical ideology, but are comments of evaluation which become clearer throughout this book. Thus it is well to keep them apart from state board examinations.

It will be noted that streptococcus pyogenes is an organism of low resistance. Women in childbirth appear to be especially susceptible to streptococcus infection via the genital tract, but there is every reason to believe that this bacterium is a normal inhabitant of the skin and mucous membranes, since it can nearly always be found on these tissues in healthy people. It is often spoken of as a "secondary invader." We might interpret this to mean that it is not until the body has been decidedly weakened by some such condition as diphtheria or broncho-pneumonia, that bodily resistance against streptococcus breaks down. On the other hand, the theory of Béchamp that bacteria are products of human tissue cell degeneration would fit the facts quite as well. Such a theory would seem a particularly fitting explanation for the fact that large numbers of streptococci may be found in the blood stream of a diabetic patient shortly preceding death. The streptococcus is not a basic factor in pathogenesis.

#### *Streptococcus not a basic factor in pathogenesis*

Inflammation of cardiac tissue does not follow injection of streptococci into the circulation, unless this tissue has already been damaged by mechanical or chemical agencies. (Jordan, p. 219)

#### *Focal infection*

This term has assumed such prominence in modern medical literature that it deserves special mention. It refers to a general toxic state supposedly due to pollution of body fluids by localized foci of infection like the teeth, tonsils, nasal passages, middle ear, and gall-bladder. Streptococcus pyogenes has been regarded as the principal agent in the process. While such infection doubtless contributes in some cases to a symptom complex like rheumatism, the many cases in which eradication of the supposed focus (for example, by removing the teeth) has failed to yield appreciable benefits to the patient show to what extent this theory has been overworked.



## 7 PNEUMOCOCCUS—DIPLOCOCCUS PNEUMONIAE

### *Historical*

Discovered by Frankel

### *Synonyms*

Diplococcus pneumoniae (current preferable name)

Diplococcus lanceolatus

Micrococcus lanceolatus

Fränkel's pneumococcus

### *Characteristics*

Ovoid pairs or short chains

Lance-shaped pairs

When found in exudates, surrounded by a gelatinous capsule thought to protect it from antibodies

Non-motile and non-spore-forming (like all cocci)

Gram positive

Grows best on media containing blood serum

Strongly ferments most sugars

### *Resistance*

Relatively low

Killed by sunlight, drying, and mild disinfectants

### *Varieties*

There are seven species of the Diplococci, five unimportant anaërobes and two aërobes. Of the two aërobes, *D. mucosus* is probably Type III pneumococcus. It was previously called streptococcus mucosus capsulatus. The other aërobe is the type species, *D. pneumoniae*, the pneumococcus of old. Under **Serology** are mentioned the seventy-five types and subtypes of pneumococci.

### *Infections with which pneumococcus is associated*

Lobar pneumonia and bronchopneumonia

Pleurisy, otitis media, mastoiditis, meningitis, endocarditis, tonsillitis, rhinitis, arthritis, conjunctivitis, and keratitis.

### *Serology*

Typing of pneumococci is done with an antigen-antibody reaction in which there is a capsular swelling with homologous immune serum. This is called the Quellung reaction. Since Neufeld did the original typing, it is called Neufeld typing or the Neufeld reaction. Another method resulting in the same types depends on agglutination. As a result there are seventy-five types and subtypes. Type III is distinguishable by cultural characteristics, while all others are distinguishable only by serological typing. Originally there were types I, II, and III as specific types. All others were thrown into type IV, the scrap heap, which is now elaborately subtyped. Types I and II are found infrequently in normal, healthy throats, but the other types are much more frequently found. In addition to these various types there is a "dissociation" of even the virulent types I and II (the other types being much less virulent). In cultures the dissociation soon results quite consistently in a loss of virulence, loss of capsule, and loss of type specificity.

According to Jordan, early attempts to employ an antipneumococcus serum resulted in chills and hyperpyrexia, a difficulty which has been only partly overcome (by concentrating the serum). No ill effects, on the other hand, have been noted in the use of natural methods.

#### CRITICAL REMARKS (not the orthodox medical viewpoint)

##### *Relation to streptococcus*

According to the bacteriologist Rosenow, pneumococcus and streptococcus are interconvertible

Pneumococcus is ordinarily distinguished from streptococcus by being

- (1) soluble in bile,
- (2) able to ferment the carbohydrate insulin

“Insolubility of certain pneumococcus strains has been reported, but is believed to be comparatively rare.” (Jordan) With such an admission here and the equally confusing admission that streptococci occasionally are soluble the dogma of monomorphism is dealt another severe blow.

##### *Prevention of transmission*

It has never been possible to show conclusively that a given case of pneumonia could be traced to any other case. This is generally admitted by clinicians. Moreover, hospital attendants in pneumonia wards harbor no greater number of pneumococci than persons not in contact with the disease. Sanitary practice, however, calls for the disinfection of the sputum of pneumonia cases and advises against close contact with patients by elderly persons not in robust health. In no other so-called germ disease is it more obvious that the most important prophylaxis is attention to the general health.

##### *The cause of death in pneumonia*

It is of considerable theoretical interest that the actual cause of death in fatal cases of pneumonia is to be found not in any phenomena directly associated with the pneumococcus but in failure of nervous control. John M. Johnston, M.D. states in *Archives of Pathology*, Vol. 14, No. 4, in an article on “The Brain Stem in Pneumonia” : “There has been for many years a feeling that death in acute infections, particularly in pneumonia, is determined primarily by a failure of control of respiration and circulation by the central nervous system. There is considerable physiologic evidence to support this theory.”

He then tells of post mortem examinations of the fine structure of the brain in cases which had just died of pneumonia. The conclusions which he reaches follow:

“In death from pneumonia (six cases) there was marked damage to the cells of the medullary and pontile centers, the cells of the various nuclei showing varying degrees of change. . . . Cells of the reticular gray matter, which has been described by others as the probable site of the respiratory and vasomotor centers, showed the most severe degenerative changes.”

*The cause of pneumonia pathology*

In the section on Tissot it is shown quite definitely that the pneumococci are not the cause, but the result of the pathology. The cause is established as being an intrinsic degeneration due to vasomotor disturbances. Hence it is related directly to the factor of nerve trauma initiating pathological reflexes. Most chiropractors who have been in practice for some time agree that pneumonia responds even more readily than sciatica. They generally get better and quicker results.



FIG. 8 (a) Staphylococci (b) Streptococci (c) Diplococci pneumoniac

## 8 GRAM NEGATIVE COCCI—NEISSERIAE

The three preceding chapters dealt with staphylococcus, streptococcus, pneumococcus—all Gram positive cocci. We now consider two important cocci which are gram negative pairs, the gonococcus and the meningococcus.

### The Gonococcus—Neisseria Gonorrhoea

#### *Historical*

Discovered by Neisser in 1879

#### *Characteristics*

A diplococcus

Commonly seen inside the cytoplasm of pus cells

Flattening of adjacent sides gives coffee-bean appearance

Gram negative

Difficult to cultivate

Grows best on very moist agar mixed with ascitic fluid

Does not ferment maltose (thereby distinguished from meningococcus)

Vitality of cultures soon lost at room temperature

“Morphologically the gonococcus is very similar to the meningococcus.”

(Jordan)

#### *Habitat*

Urethral and vaginal pus of patients with gonorrhoea

Pus from the conjunctiva in ophthalmia neonatorum

Synovial membranes in gonorrhoeal rheumatism

Endocardium of persons harboring chronic infection, especially in the valves of the heart.

#### *Resistance*

Very sensitive to drying

Withstands exposure to air but a short time

Killed by temperature of 45 degrees C. in a few minutes

May live several weeks in masses of dried pus

#### *Modes of transmission*

Sexual intercourse (most common)

Contact of the eyes of the new-born with exudate of infected vaginal membrane of the mother during parturition

Carelessness in use of towels, wash cloths, etc.

#### *Importance*

Infection very widespread

Much damage incurred by tendency of entire genito-urinary tract to become involved in the female, often with consequent sterility

10% of all blindness attributable to gonorrhoeal conjunctivitis (12,000 children in United States)

Endocarditis and synovitis common complications

Immunity not conferred by recovery from one attack

#### *Prophylaxis*

The use of 2% silver nitrate in the eyes of the new-born

The use of condomata in intercourse

Note: Vaccines and serums have proved entirely unsuccessful.

#### *Non-pathogens*

There are eleven accepted and twelve questionable species of the Neisseriae. Morphologically they are almost identical. Distinction is made by their fermentation of various sugars. Even so, *N. flava* is often confused with *N. gonorrhoea*. Frequently a commensal, the so-called prostatic organism, is found in smears. Even serologically there is little distinction between several of the Neisseriae, because there is marked cross-agglutination, antibodies developed within the host being able to agglutinate several other "species."

Adding to the confusion are the Veillonella (two species and six subspecies), which are non-pathogens in the mouth and intestinal tract of man. Occasionally they are found in pairs but more often in masses.

### **The Meningococcus—*N. Intracellularis*, *N. meningitidis***

#### *Historical*

Discovered by Weichselbaum in 1887

#### *Characteristics*

A diplococcus (but occasionally in groups of four)

Flattened somewhat like the gonococcus

Grows best on media containing blood serum or ascitic fluid; usually grown on blood agar

Gram negative

Ferments maltose (unlike the gonococcus)

Like gonococcus, seen inside pus cells

#### *Habitat*

The mucous membrane of the naso-pharynx

The exudate from the meninges in epidemic cerebro-spinal meningitis (not other forms of meningitis)

#### *Mode of transmission*

Supposedly by way of nasal secretions

Exact mode unknown

#### *Resistance*

Readily killed by drying or exposure to sunlight

#### *Serology*

The meningococci do not constitute an entirely homogeneous group. At least four serological types are recognized, but in addition *N. flavescens* has been isolated without any of the meningococci present in many cases of meningitis and is listed with the meningococci types. "Serological typing of meningococcus strains has proved a difficult and laborious process, partly because of the merging of agglutination types . . . partly because of the antigenic instability of many meningococcus cultures." (Jordan) Consequently the current custom is to use polyvalent serum prepared by injecting horses with a number of different meningococcus strains.

### REMARKS

So frequently is the meningococcus an inhabitant of the nasopharynx of healthy people that any attempt to quarantine such people as carriers would paralyze civilization. Had such a rule been applied during World War I, Jordan says, there would have been more soldiers quarantined than on duty. Resistance to the meningococcus is believed very high in adults. Outbreaks of meningitis are likely only under conditions of overcrowding, long exposure, fatigue, and mental strain. "The attempt to control the spread of meningitis by quarantining carriers is like sweeping back the Atlantic Ocean with a mop." (Jordan)

#### OBSERVATION

Both gonorrhoea and cerebro-spinal meningitis respond readily to non-medical methods.

Greek army treatment: 6 weeks in bed—nothing else.

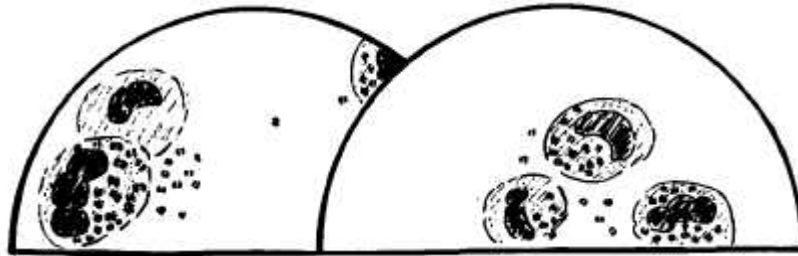


FIG. 9 Gonococci - Meningococci

#### *Other Neisseria*

As mentioned under "gonococcus" there are twenty-two species and their various types. *N. catarrhalis* is the most common of all the *Neisseriae*, very frequently found in the nasopharynx of healthy individuals. It grows readily on ordinary media. *N. catarrhalis* and *N. sicca* are frequently found in a simple leukorrhoea as incidental findings. This is often confusing and misleading. Moreover, the meningococcus commonly shows many involution forms of several shapes and sizes.

## 9 THE COLIFORM GROUP— ENTEROBACTERIACEAE

Gram negative, non-sporing rods, usually motile by peritrichal flagella make up this group.

A considerable variety of bacteria are found regularly in the intestinal tract of man and the higher animals. The greater portion of the solids contained in fecal matter consists of living and dead bacteria. They fall roughly into two groups; namely, a group of facultative anaerobes characterized by fermentation (production of acids from carbohydrates) and a group of true anaerobes characterized by the production of putrefactive changes in proteins or products of protein digestion. The consensus is that the former group exert a somewhat beneficent influence by holding the latter in check, since an acid medium is unfavorable to the growth of putrefactive bacteria.

The main group of non-pathogens thriving in a normal intestine is the Escherichiae tribe, the chief benefactor being the type species, the common colon bacillus. The Salmonella tribe, on the other hand, thrive best in a morbid intestinal tract. They leave the body in the bowel discharges, and sometimes in the urine. When sanitation is inadequate, as in the case of pollution of drinking water by sewage, the organism may again gain entrance to the human alimentary tract. Milk containers washed in sewage-polluted water become an effective vehicle of transmission. Outdoor closets are also often implicated, as they enable flies to pick up the germs and carry them to food. Typhoid bacilli have been transmitted by oysters bred in sewage-polluted water. Infection, when it occurs, is considered as taking place always by way of the mouth.

The five tribes of this order are as follows:

1. Escherichiae—beneficial non-pathogens
- 2, 3, and 4 Erwinae, Proteus, and Serratia—unimportant non-pathogens
5. Salmonella—often pathogenic—typhoid, paratyphoid, dysentery group

### *Escherichiae (tribe)*

The eleven species of this tribe are grouped into three genera, *Escherichia*, *Aerobacter*, and *Klebsiella*. Previously thirty-five species were recognized, but many are now classed as variants of other species. The type species of the first is the colon bacillus outlined next in order. The *aerobacter* (type species—*A. aërogenes*) is found in the intestinal tract as an incidental organism, but it is widely distributed in soils and waters which are not contaminated with human excreta. Hence the determinative tests are essential to sanitary engineers. “*Klebsiella* merely represents coliform organisms which have exchanged their customary habitat in the bowel for residence in the respiratory tract which they maintain by virtue of their capsular defense.” (Laboratory Methods of the United States Army, by Simmons & Gentzkow, Lea & Febiger, 1944) *K. pneumoniae* is frequently found in patients with lobar pneumonia, and having been found by Friedlander was called Friedländer’s pneumobacillus. It was also called *Diplobacillus pneumoniae*. Essentially it is a typical coliform bacterium with a heavy mucoid capsule.

### **The common Colon Bacillus—*Escherichia coli***

(*Bacterium coli*)

### *Historical*

First described by Escherich in 1886 Isolated from the dejecta of breast-fed infants

### *Characteristics*

- Form variable, sometimes short and coccus-like
- Dimensions 2 to 4 by 0.4 to 0.7 microns
- A few spiral flagella; moderately motile
- Curdles milk
- Ferments dextrose and lactose with gas production
- Does not attack proteins, but acts upon peptones, producing indol
- Gram negative
- Forms leaf-shaped colonies, as does also the typhoid bacillus

### *Habitat*

- Conspicuously found in the intestinal tract
- Found occasionally in air, soil, and water, in attenuated state
- In water taken as sign of sewage contamination

### *Relation to pathology*

- Normal to intestines; probably does not penetrate healthy intestinal walls
- Sometimes associated with infections of gall-bladder, bile duct, and urinary tract
- Can precipitate cholesterol and is occasionally found in center of gall stones.

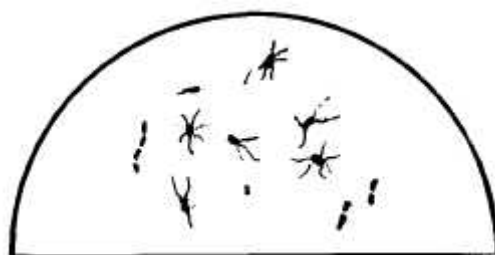


FIG. 10 *Escherichia coli* (the common colon bacillus). Note coccus-like forms.

### *Other fermentative bacteria*

Other common inhabitants of the intestines more or less similar in behavior and characteristics to the colon bacillus are *Lactobacillus acidophilus*, *L. bifidus*, *L. bulgaricus* (the lactic acid bacillus), *A. aërogenes*, and *Proteus morganii* (*B. metacoli*). The first two are especially prevalent in healthy infants. *A. aërogenes* is often found in sour milk. *L. bulgaricus* is the organism the eating of which was advocated by Metchnikoff. Further investigation, however, revealed that it was the sour milk of the cultures to which most of the benefit might be ascribed, and that *L. acidophilus* is much better adapted to implantation in the intestines than *L. bulgaricus*.

### *Putrefactive bacteria*

These bacteria, of which there are many varieties, carry on an anaërobic attack upon products of protein digestion, notably the amino acids, and convert them into such toxic substances as cresol, phenol, indol, and skatol. It is to the last two of these that the odor of feces is principally due. An excessive protein diet with low carbohydrate intake favors putrefaction, as does also sluggish intestinal activity.



*Differentiating features (important for state boards only)*

*Aërobacter aërogenes* is a frequent non-pathogenic contaminant of water unexposed to human excreta. *Escherichia coli*, although nearly identical, indicates human fecal contamination, which would mean a possible *Salmonella* contamination. Hence the three following tests are used for differentiation in any water showing coliform organisms.

	Methylene Blue Reduction	Methyl Red	Voges- Proskauer
<i>A. aërogenes</i>	rapid reduction	—	+
<i>Escherichia coli</i>	slow reduction	+	—

**Salmonella (tribe)**

This is the tribe of which a few members are human pathogens. The pathogens are those often found in the morbid intestinal tract associated with typhoid, paratyphoid, or bacillary dysentery. The organisms were divided into the three genera: *Eberthella*, *Salmonella* and *Shigella*. The present classification, being based on cultural and serological characteristics, places the *Eberthella* with the *Salmonella*. However, because of historical considerations and board examination requirements, they will be considered separately here.

**The Typhoid Bacillus—*Salmonella Typhosa*  
(*Eberthella typhosa*)**

*Historical*

Discovered in 1880 by Eberth, who, however, was unable to produce typhoid fever in animals by feeding them the bacilli.

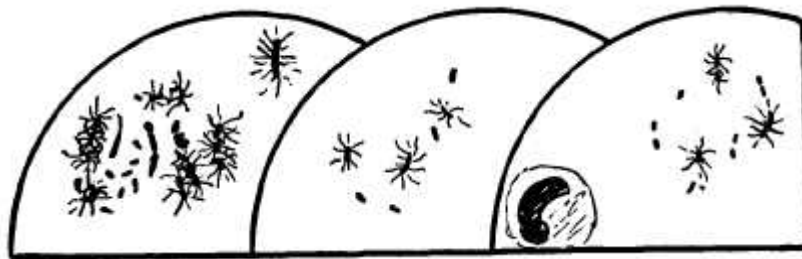


FIG. 11 *S. typhosa*, *S. paratyphosa*, and *Shigella dysenteriae*

*Characteristics*

- A short, plump rod
- Length: 1 to 3 microns; width: 0.5 to 0.8 micron
- Actively motile (numerous, long, wavy flagella)
- Gram negative
- No spores
- Gelatin not liquefied
- Milk-sugar (lactose) not fermented
- Dextrose fermented without gas production
- Colonies leaf-shaped

### *Habitat*

Chiefly the intestines of typhoid patients

Found also in the blood, the gall-bladder, the rose spots, the urine, and the feces of the patient

Unable to live long in water or soil

### *Sanitary precautions*

1. Disinfect the stool by covering well with chloride of lime and allowing to stand for an hour before disposal (or, use a cup of quicklime followed by a cup of hot water).

2. Soak the patient's underclothing, night clothes, handkerchiefs, etc., in 2 to 5% carbolic acid.

3. After handling patient, disinfect the hands in 5% carbolic acid or 1:1000 bichloride of mercury.

4. Keep flies out of sick room.

### *Typhoid carriers*

Carriers are well persons in whose stool the typhoid bacillus can be found. According to Jordan, 0.5 to 11.6% of all persons who get typhoid fever become carriers. Carriers are regarded as an important element in the transmission of the disease. The observance of rigid personal cleanliness by such persons, however, is all that is needed to prevent their being a menace to any one.

### *Serology* (these details are not significant for drugless practitioners)

Fourteen accepted Eberthella species were previously recognized by Bergey, but he currently accepts only one species, and that is classed as the sixty-sixth species of Salmonella. He does list twenty-three Eberthella species but not as fully accepted. The agglutination phenomenon (Gruber-Widal reaction) is still used extensively in diagnosing typhoid fever. The ability of the blood serum to agglutinate is usually noticeable by the fifth day of the disease, but not always. The technic involves mixing the serum of the patient being investigated with an authentic culture of typhoid bacilli in a dilution of at least 1:50. Microscopic agglutination will appear within twenty-four hours, often within two hours. There are difficulties to this test. Some strains of genuine typhoid bacilli are inagglutinable. Also, spontaneous clumping occurs in broth cultures. Any of the paratyphoid group, and even the proteus group, as antigens will incite an agglutinin response which will give a positive Widal reaction. Hence the significance of a positive Widal is that the patient either now has, or has had, an infection with or an inoculation against typhoid, paratyphoid, a proteus or occasionally the colon bacillus. "Isolation of the specific organism is a much surer means of making a correct diagnosis than is dependence on any sort of agglutination test." (Jordan)

Vaccination against typhoid is commonly practiced by the United States Army. Their statistics, offered for favorable propaganda, would indicate that vaccination has practically caused a reduction of the disease to less than one half of one per cent. Statistics from the Surgeon General's records quoted later in this book show quite the contrary, but this fact is not advertised by the medical profession. The currently used serum is TAB (typhoid, para A and para B) or "triple typhoid." It is standardized to contain one billion typhoid bacilli and 750 million bacilli per cc. of each of the paratyphoid types A and B. The bacilli have been washed with normal saline from agar media and then killed by heating to 55 to 56 degrees C. for one hour. After standardizing the TAB proportions it is diluted to contain two and one

half billion bacilli per cc. and preserved by addition of 0.25% tricresol. The vaccine is given in three weekly injections. The first is 0.5 cc. and the succeeding two are one cc. each. "As a rule, the reaction following inoculation is not severe, although occasionally fever, chills, nausea, and some nervous symptoms are observed." (Jordan) The "Rawlings strain" of typhoid bacilli was formerly widely used in preparation of serum, but "the Rawlings strain was found to be lacking in immunizing properties and hereafter a more virulent strain is to be used in the preparation of vaccine for the United States Army." (Jordan)

#### *Related forms*

Remember that twenty-four species are recognized, but that only one is pathogenic to man.

### **Paratyphoid Bacillus—*Salmonella paratyphosa***

(*B. paratyphosa*—type A)

"*Salmonella* is . . . our most complex genus. At least 109 types are known." (*Laboratory Methods of the United States Army*) Bergey lists 151 accepted types plus 40 accepted subtypes and uses the word type in this case as being synonymous with species. The serological typing seems endless. The typical species is *S. paratyphosa*. The main other species with their synonyms are listed below.

#### *Historical*

*S. enteritidis* isolated by Gartner in 1888.

#### *Characteristics*

Exactly like the *S. typhosa* except that there is usually gas production on dextrose media, and that the bacteria are frequently a bit smaller.

#### *Habitat*

Again like that of *S. typhosa* except that it is found in paratyphoid fever patients.

#### *Clinical detection and sanitary precautions*

Like those for *S. typhosa*. Distinguished by cultural differences. Both typhoid and paratyphoid are so uncommon that the average doctor will not have more than one case in a lifetime. (Thanks to sanitation. In spite of, and not because of vaccination or other medical "benefits."-R.W.)

#### *Varieties*

Of the nine or more species and over 109 types only three are of interest here:

*S. paratyphosa*— (*B. paratyphosa*—type A)

*S. schottmulleri*— (*B. paratyphosa*—type B)

*S. hirschfeldii*—(*B. paratyphosa*—type C)

These are still called types A, B, and C, although they are recognized as separate species.

#### *Related forms*

Only two other species, *S. aertrycke* and *S. enteritidis*, have been found to be of consequence to man, and then only in cases of food poisoning.

#### *Serology*

Refer to the material on Gruber-Widal reaction and TAB vaccine given under Typhoid bacillus.

## **Dysentery Bacillus—*Shigella dysenteriae* [Eberthella dys.]**

### *Historical*

- Discovered in 1898 by Shiga in Japan
- Second type discovered by Flexner in Philippines
- The Sonne type discovered by Duval in 1904

### *Characteristics*

- Somewhat smaller than the typhoid bacillus
- Gram negative
- No spores
- Generally non-motile without flagella
- Dextrose fermented
- The sugar mannitol fermented by Flexner's type, but not by Shiga's

### *Varieties*

Although there are eleven accepted and nineteen questionable species we list only three species with their old names.

- S. dysenteriae*—(Shiga dysentery bacillus)
- S. paradysenteriae*—(Flexner dysentery bacillus)
- S. metadysenteriae*—(Sonne and Schmitz types)

Although one or more of these species are found in cases of bacillary dysentery they are not common. Their etiological significance becomes more apparent under the chapter on Bacteriophage, which was discovered by d'Herelle in recuperating dysentery patients.

*S. cholera-suis* was once thought to cause hog cholera but is non-pathogenic. The disease is now classed as a virus disease.

## **Miscellaneous Coliform group**

*Proteus* is a separate tribe of four accepted and twenty-eight questionable species. They are almost identical to *B. coli*, but are almost entirely saprophytic or commensal organisms commonly present in the normal human intestinal tract.

*Serratia* is an interesting tribe of six species. Again non-pathogenic, but of historical interest because it includes the *S. marcescens* or *Bacterium prodigiosum* (the miraculous bacillus). They normally grow in bright red, glistening, raised colonies, having a remarkable resemblance to drops of fresh blood. Even when Alexander the Great was besieging Tyre in 332 B.C., "drops of blood" were found inside the loaves of bread. This was a miracle to be interpreted by the wise men. Many times this phenomenon caused unrest. Another feature worthy of note is that the name *Serratia* was given in honor of Serafina Serrati, who was the first to ply a steamboat on the Arno in Italy.

*Alcaligenes fecalis*, a normal intestinal inhabitant, closely resembles the typhoid bacillus and the other coliform organisms, morphologically and culturally. Found in feces and in water, it was once classed with the coliform group. However, since it has polar instead of peritrichal flagella, it was decided to place it entirely apart in another family. Now it is regarded as a member of this new family, the *Achromobacteriaceae*, despite its association with the coliform group.

Characteristics of part of the coliform group  
(useful on some difficult state board examinations)

		Dextrose	Lactose	Sucrose	Maltose	Mannite	Xylose	Indole-forming	Motility
Salmonella	typhosa	a	o	o	a	a	x	-	+
S "	paratyphosa	ag	o	o	ag	ag	o	-	+
S "	schottmülleri	ag	o	o	ag	ag	ag	-	+
Shigella	dysenteriae	a	o	o	o	o	x	-	-
Sh "	paradysenteriae	a	o	o	a	a	x	+	-
Sh "	metadysenteriae	a	o	a	a	a	x	-	-
Escherichia	coli (communis)	ag	ag	o	ag	ag	x	+	+
Esch "	coli (communior)	ag	ag	ag	ag	ag	x	+	+
Esch "	coli (acidilactici)	ag	ag	o	ag	ag	x	+	-
Aërobacter	aërogenes	ag	ag	ag	ag	ag	x	+	-

Code: a, acid produced  
ag, acid and gas produced  
o, no reaction

+, present or positive  
-, absent or negative  
x, not needed for identification

## 10 SPORE-FORMING ANAEROBES— BACILLACEAE

This vast group of spore formers are all Gram positive rods. There are only two genera; the *Bacillus*, of which there are thirty-nine accepted and 499 questionable species, and the *Clostridium*, of which there are a mere sixty-one accepted species. Only three are of sufficient clinical importance to concern us.

### The Tetanus Bacillus—*Clostridium tetani*

#### *Historical*

First described in 1884 by Nicolaier, who saw it in pus Obtained in pure culture by Kitasato in 1889

#### *Characteristics*

Slender rods 0.3 to 0.5 by 2 to 5 microns

Motile, with 20 to 30 flagella

Gram positive

Spore-forming

Spore located at one end of the rod, giving the bacillus the appearance of a nail.

Strictly anerobic

Grows readily in broth, brain tissue, meat, gelatin and agar from which air is excluded or in which it has been deeply imbedded.

Liquefies gelatin

#### *Habitat*

Garden soil, manure, street dust, hay dust, feces, and occasionally vaccines and serums

Normal to the vertebrate intestinal tract, especially of horses

#### *Resistance*

Withstands steam at 100 degrees C. 40 to 60 minutes

Killed by heating to 105 degrees C. for 10 minutes

Destroyed by 5% carbolic acid only after 10 to 12 hours

Killed by bichloride of mercury 1:1000 in 2 to 3 hours

Killed by 1% silver nitrate in 1 minute

#### *Conditions favoring infection*

Deep, dirty wounds with much local tissue destruction and the presence of other bacteria; pure cultures harmless

#### *Mode of action*

Multiplies locally without invasion of blood stream Secretes a powerful toxin which circulates through the blood

#### *Serology*

The toxin, an exotoxin, consists of two components:

Tetanolysin: disintegrates red corpuscles

Tetanospasmin: absorbed by motor nerve endings, migrates along peripheral axones

Increases conductivity, and gives rise to spasm

The toxicity is so high that 0.0001 cc. is more than adequate to kill a guinea-pig. This is called the M.L.D. (minimum lethal dose). The general medical opinion is that the toxin is absorbed by peripheral nerves and travels up the axis cylinders to the

central nervous system which then initiates the symptoms. (Speransky has proved otherwise.)

The antitoxin is prepared in a manner similar to the preparation of diphtheria antitoxin.

Strength up to 900 units per cc, the unit being ten times the least quantity of antitetanic serum necessary to protect a guinea-pig against 100 minimum fatal doses of tetanus toxin.

Possesses a probable prophylactic value, but no curative value whatever. (See A. Geoffrey Shera, *Vaccines and Sera in Military and Civilian Practice*, London, 1918.)

REMARKS (The following remarks are not sufficiently orthodox for state boards)

*Dangers in connection with tetanus antitoxin*

Atypical or chronic cases of tetanus have been known to occur following the prophylactic use of tetanus antitoxin. (Jordan, p. 415)

Speransky has shown conclusively that the antitoxin is worthless as a specific antibody and acts only as a nerve stimulus. In fact, a second injection of the tetanus toxin is as effective a protection as is the antitoxin. His statistics show that non-specific novocaine injections are much more effective than the T. A. T. (tetanus antitoxin).

*Tetanus following vaccination*

In *La Pediatria del Medico Pratico* of Turin, Italy, (Vol. VII, No. 8) Dr. Fausto Colo describes a fatal case of tetanus which developed in a previously healthy child of three, twenty days after vaccination against smallpox. He states that the evidence is clear and unmistakable that the vaccination constituted the mode of infection. Antitetanic serum was used to no avail. In a bibliography which appears at the end of the article nine different references are cited dealing with either tetanus or encephalitis traceable to vaccination.

### **The Bacillus Botulinus—Clostridium botulinum**

This organism is associated with a variety of food poisoning which has often been mistaken for so-called ptomaine poisoning. Ptosis, dysphagia, and aphonia are prominent symptoms. Double vision is common and death from asphyxia due to involvement of the respiratory center is frequent.

*Historical*

Discovered in 1894 by van Ermengem in Belgium

Named from the Latin botulus, sausage, the food in connection with which botulism was first noted

*Characteristics*

Dimensions about 1 by 5 microns

Gram positive

Subterminal spores

Motile, with 4 to 8 flagella

Anaërobic

Liquefies gelatin and digests coagulated protein with gas production

### *Varieties*

Of this one species there are fifteen different serological types listed and described separately by Bergey. This makes serum therapy more definitely a gamble.

### *Foods in which the bacillus has been found*

Sausage, pickled ham, fish, canned beans, corn, spinach, beets, asparagus, pears, apricots, and olives

### *Toxin*

Unique among all known bacterial toxins in not being destroyed by the gastrointestinal secretions

Considered analogous to the toxin of poisonous mushrooms This is one of the most powerful poisons known to man. The

M.L.D. varies from 0.0001 to 0.000,001 cc. depending upon the strain.

It is thus much more toxic than strychnine or cyanides.

### *Ptomaines*

Protein decomposition frequently yields a toxic fragment. These toxic products and some non-toxic products are grouped together as ptomaines. They have been blamed heavily for any and all food poisoning. Actually they are of little consequence. Staphylococcic and streptococcic contamination with botulism are the usual "food poisons."



FIG. 12 Clostridia tetani and clostridia botulina

### *Other Clostridia*

Clostridium perfringens (Cl. welchii) is the common microorganism found in gas gangrene. Cl. oedematiens (Cl. novyi) is another mentioned with gas gangrene as is Cl. septicum (Vibron septique). Cl. chauvei is the one often found with blackleg in cattle. Cl. sporogenes is a non-pathogen found in soil, feces, and in some contaminated wounds. The others of the sixty-one species are of almost no importance to man.

### **Anthrax Bacillus (Bacillus anthracis)**

#### *Historical*

First described in 1850 by Davaine and Rayer

Demonstrated by Koch in 1876 to have causal relation to anthrax

#### *Characteristics*

Large rod (one of the largest pathogens):

.4.5 to 10 microns long and 1 to 1.25 microns across

Non-motile

Single or short chains in infected host

Very long chains or filaments on artificial media



Gram positive—many of them are gram negative, especially in young cultures  
Spore formers, strictly anaërobic  
Liquefies gelatin slowly; rapidly ferments dextrose without gas formation



FIG. 13 B. anthracis

#### *Resistance*

While the bacilli themselves are not especially resistant, the spores are highly resistant

Spores killed by dry heat at 140 degrees C. for three hours

Spores killed by steam or boiling water in five to ten minutes

Spores withstand even a 60% creolin solution

#### *Conditions favoring infection*

Anthrax is called “wool-sorter’s disease,” because it is usually acquired from imported, infected hides and fleeces. Routes of entry are: skin, respiratory and alimentary tracts. The most frequent, however, is through cuts on the skin into which are rubbed the bristles or hairs of an infected hide or an infected shaving brush.

#### *Mode of action*

There is occasionally such a heavy bacteremia in some animals that it was once supposed that death occurred through capillary blockade. Yet fatal human cases show almost no bacteremia at all. Since there has never been either an endotoxin or an exotoxin demonstrated, the manner in which anthrax bacilli cause death is still a mystery. (Medically, that is.)

#### *Sexology*

“No antitoxin has been demonstrated in the blood of naturally immune animals.” Even so, Pasteur devised a method, still used, of inoculating animals with attenuated cultures. “The mechanism by which the protective serum exerts its action is not certainly known.” (excerpts from Jordan)

#### *Related forms*

“B. subtilis is morphologically and culturally indistinguishable from B. anthracis. There are many species of aërobic sporulating bacilli closely related to and indistinguishable from the anthrax bacillus on any basis other than pathogenicity . . . the more common of which are: B. subtilis—very common; B. vulgarus, also widely distributed; B. mesentericus, a common soil form; and six others. . . . No cultural or biochemical characteristics serve to differentiate the anthrax bacillus from these non-pathogenic saprophytic sporulating bacilli; animal inoculation to determine pathogenicity is essential.” (Jordan)

## CRITICAL REMARKS

This raises the question, are these actually different organisms or are they the same organism in different environments with consequent differences in virulence? Buchner in 1880 maintained that he had succeeded in changing the virulent anthrax bacillus into *B. subtilis* and vice versa. Remember, also, that there are hundreds of species in this genus, only one of which is a pathogen. The incidental remarks of this sort throughout this entire section on bacterial characteristics are to be remembered, because of their pertinence to the evaluation which comes later.

## 11 CHOLERA—THE VIBRIOS

Twenty-two accepted and eighty-one questionable species of Gram-negative polar flagellates comprise this genus. They are short, curved and twisted rods. In cultures they are found in long spirals, but old cultures exhibit many long, straight or spiral threads. These mycelia are considered involution forms. Only one species, the so-called comma bacillus is of importance as a human pathogen.

### The Spirillum of Cholera—*Vibrio comma* (*V. cholera*)

#### *Historical*

- Discovered in 1884 by Koch
- Often called the “comma bacillus”

#### *Characteristics*

- A short, slightly curved and twisted rod
- Gram negative, strongly aërobic
- No spores
- Motile (one terminal flagellum)
- Grows well on ordinary media at room temperature

#### *Habitat*

- Warm, damp soil
- The intestinal tract of cholera patients and carriers

#### *Serology*

A toxin went undiscovered for years. Later an exotoxin was demonstrated. Since the serum of actively immunized animals neutralizes the toxin, it is

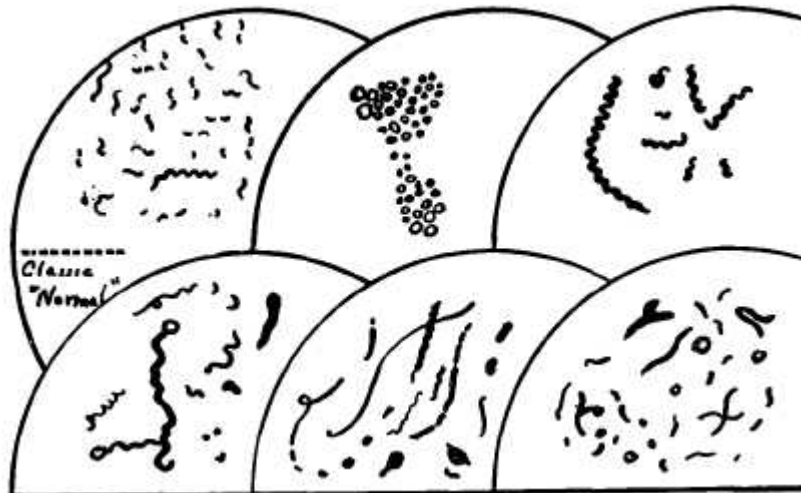


FIG. 14 *Vibrio cholera* (“normal” and involution forms)

presumed to contain a true antitoxin. This is the basis of the “Pfeiffer phenomenon” of bacteriolysis within the peritoneal cavity of the guinea-pig.

Vaccination is carried out by injection of living bacteria, either in full virulence or attenuated. Filtered germ-free cultures possess no immunizing power. Thus in practice there is a series of injections of increasing numbers of bacteria. Since the reaction is bacteriolytic and not antitoxic, an “immune” animal succumbs just as

readily as an unprotected one to injection of dead bacteria, although able to dissolve some live bacteria. The bactericidal power is specific and will not destroy any of the other vibrios.

#### *Varieties*

There are twenty-two accepted and eighty-one questionable species. “The majority of the cholera-like vibrios, so far as studied, however, are probably essentially saprophytic forms, or, at most, possessed of slight pathogenic power.” (Jordan) “The cholera vibrio is imitated precisely in its morphology, staining and cultural characteristics by vibrios which are found in the intestinal tract of (normal, healthy) man and also as free-living forms in nature. At the height of an epidemic, the characteristics of the ‘true’ cholera vibrio are well marked and it can be distinguished by utilization of Pfeiffer’s phenomenon or by specific agglutination tests. Non-agglutinable varieties predominate in the early and late stages of an active outbreak of cholera.” (Cecil—Textbook of Medicine) The following sketch of the many forms of the “true” cholera vibrio is to be considered. The evaluation comes later in this book.

#### REMARKS

(Again, not acceptable for state boards—even though they are authentic quotations of accepted authority.)

#### *Relation of the spirillum to cholera*

W. P. Mason, in his standard work on *Water Supply* states (p. 58):

“Pettenkofer and Emmerich each swallowed pure cultures of the comma bacillus, with the result of producing only temporary diarrhea, and they thereupon claimed that the germ is not to be considered as the cause of cholera. As opposed to this, Roux points out that the pure cultures referred to above may have been attenuated and very far from the point of virulence. Moreover, he shows that, even when truly virulent cultures are swallowed, the disease does not surely result.

“Tersely stated, a disease follows when the active pathogenic organisms invade the body and find there not only suitable soil for their development but also the absence of antagonistic conditions which prevent their multiplication.”

Mason also quotes the president of the National Health Society of England as declaring in an address:

“It is stated on the authority of the head nurse that not a single case of cholera originated in the hospital of Hamburg during the recent epidemic in that city, though the sick were often placed two in the same bed and the dead in long rows. Amid the gloom and excitement scores of suspects were hurried off to the hospital who were afterward found to be suffering from some other disease. Not one of these persons contracted the disease from the cholera patients with whom they were forced to associate. It would seem as if the safest place at the time of a great epidemic of cholera would be where there is the most sickness. All of these statements point to the fact that cholera is not infectious, and that the danger has been very greatly overestimated.”

The foregoing statements will be found applicable to practically all other so-called germ diseases.

### **The Pseudomonas**

These are half-brothers to the vibrios, being strongly aërobic, Gram-negative, motile, polar flagellates. They differ in being quite straight rods, instead of being curved. Also they are chromogenic and of very doubtful pathogenicity, being found in some chronic sinusitis excreta and in pus from otitis media. The only species of possible clinical significance is the *Ps. aëruginea*, previously called the *B. pyocyaneus*.

## 12 DIPHTHERIA BACILLUS— CORYNEBACTERIUM DIPHTHERIAE (Klebs-Löffler bacillus)

Twenty-five accepted and eighty-one questionable species comprise this genus. Formerly included under the Mycobacteriaceae, it is now placed in a new independent family. They are slender, slightly curved rods with tendency to club, and exhibit branching forms in old cultures. They are Gram positive and frequently barred. Only one species is of clinical importance.

### Historical

Discovered by Klebs in 1883

Isolated in pure culture by Löffler, who, however, did not claim that it is the cause of diphtheria, as he failed to find it in 25% of his clinical cases and did find it in healthy throats.

### Characteristics

A slender rod 1 to 6 microns in length

Beaded or striated in appearance when stained with Löffler's methylene blue



FIG. 15 *Corynebacteria diphtheriae*

("Normal," 5-hour and 12-hour involution forms)

Often clubbed at the ends

Occasionally branched

Non-motile

Gram positive

Strictly aërobic

### Habitat

The false membrane of diphtheria

Mucous surfaces, particularly tonsils and palate, nares, pharynx, and larynx

Occasionally the conjunctiva and the mucous membranes of the genital tract

### Clinical detection

First method: Piece of false membrane spread on slide, fixed, stained with Löffler's alkaline methylene blue, and examined under microscope

Second method: Sterile swab rubbed over membrane and then over surface of a tube of sterile Löffler's serum, which is incubated 12 to 18 hours; growth then examined under microscope

### Toxin

An exotoxin; that is, given off by the living bacterial cell

Chemical nature unknown

Destroyed by sunlight and by boiling

Production hindered by presence of acid

Circulation through the blood followed by fatty degeneration of heart muscle, the myelin sheath of peripheral nerves, and the white matter of brain and spinal cord; kidneys also damaged

#### *Antitoxin*

Prepared by injecting horses with gradually increased doses of diphtheria toxin over a period of several months until as much toxin can be given as would ordinarily kill several hundred horses; horse is bled, the blood allowed to clot, and remaining light-yellow fluid used as antitoxic serum.

Marketed at a strength of 500 to 1500 units per cubic centimeter, a unit being the amount that will just protect a guinea pig against 100 fatal doses of diphtheria toxin.

#### *Serum treatment*

Antitoxin alone has been used to "cure" diphtheria. "There is no evidence that antitoxin can repair tissue already damaged by the toxin." (Jordan) Hence it was used to neutralize toxins already present to prevent further damage. The reactions were so dangerous that a mixture of toxin-antitoxin was substituted. This TAT was also used to produce active immunity prior to exposure. Here again the reactions called for a modification with the result that currently a toxoid is used for immunization. This toxoid, or anatoxin, is prepared by treating toxin with formalin and incubating it at 37 degrees C. for a month. It has been still further modified by precipitating it with potash alum, the result being called alum-toxoid. Another modification has been partially to neutralize the toxoid into a toxoid-anti-toxin. Less serious reactions are claimed for the antigens thus obtained.

#### *Sanitary precautions in diphtheria*

Disinfect mouth and nasal discharges by carbolic acid

Disinfect spoons, glasses, and other objects coming in contact with the mouth

#### *Related forms*

The other twenty-four species are often called "diphtheroid" bacilli. "Bacilli morphologically similar to the diphtheria bacillus, but which are apparently quite harmless, are frequently observed in the throat. No absolute distinction can be made on morphological grounds, however, and the diphtheria bacillus must be identified by the virulence test." (Jordan) (Are they different species or the same organisms simply modified by their normal or abnormal environment? This is a question to which we shall revert.) Four polar flagellates are included now as Corynebacteria, although they are really Pseudomonas.

### CRITICAL REMARKS

#### *Immunization by inoculation*

Antitoxin gives only temporary immunity.

A mixture of toxin and antitoxin (TA) is said to be more effective. A large number of fatal cases of diphtheria in children previously so treated, however, are reported to have occurred in Berlin (*Journal of the American Medical Association*, March 22, 1930)

It is noteworthy that the editors of *Chemistry in Medicine* state in their Foreword: "Even some of our greatest triumphs have not yet been made altogether safe. The child that has been saved from an attack of diphtheria by diphtheria

antitoxin may be sensitized to horse serum, its vehicle; should a common accident of childhood, a torn hand or leg require protective antitetanus treatment for the same child, he may stand in positive danger of death by anaphylactic shock from the vehicle, horse serum.”

*Adverse symptoms and death following antitoxin*

Jordan, in his *General Bacteriology*, states that skin rashes and pains in the joints due to unknown substances in the horse serum may follow the administration of antitoxin. Occasionally, also, the use of antitoxin has been followed by sudden death.

*Value of antitoxin exaggerated*

An editorial in the *Journal of the American Medical Association* of January 7, 1933, dealing with an address by the late Dr. Jordan, who was at the time professor of bacteriology at the University of Chicago, makes the following significant statements:

“Diphtheria, Dr. Jordan points out, has always been subject to cyclic fluctuations, being unimportant in the early half of the nineteenth century in Europe, but becoming widespread between 1850 and 1860, so that Hirsch characterized it as ‘a new phenomenon in the history of pestilence.’ A decline set in about 1870 and continued through the rest of the century, during the last decade of which the discovery of the antitoxin was made by von Behring and Roux. Jordan holds that comparison of death rates for the twenty years just before antitoxin with those immediately after may give an ‘exaggerated impression of therapeutic efficiency.’ During the same time a declining trend was manifest also in scarlet fever mortality without any definite therapeutic advances of a specific nature. In 1926 and the following years the diphtheria mortality showed an increase in the cities of Germany, Italy, France, Spain and the United States, and the number of cases reported to the League of Nations from thirty-two countries scattered over the world increased fifty per cent between 1923 and 1929. Even in New York City, where antidiphtheria measures had been most intensively employed over the longest period, the rate of mortality increased.

“The explanation for increased mortality and greater prevalence may be found in a number of factors. Jordan dismisses as unlikely the possibility that any change occurred in the mode of transmission, or that serum treatment was neglected ... As probable factors in the changing picture, he discusses the soil on which the infection falls; namely, the population involved and its immunity, which might conceivably vary in different generations . . .”

*Diphtheria under drugless practitioners*

Drugless healers have been highly successful in handling diphtheria without serums. These groups now include many who have previously practiced medicine.



## 13 THE TUBERCLE BACILLUS— MYCOBACTERIUM TUBERCULAE

Thirteen species and nine subspecies of these acid-fast, aërobic, branching rods comprise the genus *Mycobacterium*. Only two are of clinical importance in man, *M. tuberculosis* and *M. leprae*.

### *Historical*

The tubercle bacillus was discovered in 1882 by Koch

Important as the bacillus in connection with which Koch laid down his famous postulates

### *Characteristics*

A very slender rod, occasionally clubbed or branched

Length: 2 to 4 microns

Width: 0.3 to 0.5 micron

Capsular substance much increased by artificial cultivation Non-motile Gram positive

No spores

Stains with difficulty.

The Ziehl-Neelsen method is used.

Acid-fast (non-acid fast forms are encountered occasionally)

Cultivable only on special media to which glycerol has been added (blood serum, whole egg, and meat extract often used)

Strictly aërobic

### *Resistance*

Unusually resistant to drying Lives in dried sputum 6 to 8 months In air, as dust, however, dies in 8 to 10 days

Withstands dry heat at 100 degrees C. for an hour

In fluid, killed by a temperature of 60 degrees C. in 15 to 20 minutes

Not killed by gastric juice

Quickly destroyed by direct sunlight

Able to resist 5% carbolic acid up to 24 hours

### *Pathological conditions in which the bacillus is found*

Pulmonary tuberculosis

Pott's disease

Tuberculous meningitis

"Hip-joint disease"

Scrofula

Tuberculous affections of any organ of the body

### *Tuberculosis in animals*

Cattle and swine, birds, and even some cold-blooded animals are subject to tuberculosis. The bacillus identified with the disease in these animals, however, presents certain slight, but constant differences from that found in man. Authorities differ on the relation between bovine and human tuberculosis. The opinion prevails, however, that it is only young children who may be harmed by milk from tuberculous cows. In subjects under five years of age, there is the possibility of acquiring tuberculosis of the intestines, the peritoneum, and the cervical glands.

### *Life cycle*

Mellon, Kahn, Much and others have extensively studied the life cycle of the bacillus. Filterable forms are well known, usually called "Much granules." Granules or gonidia with special reproductive functions have been thought to exist. Since they are frequently found in various forms such as long branched filaments, chains of rods, single rods, short rods and tiny cocci, investigation continues. and there is much diversity of opinion on this subject.

### *Serology*

Tuberculin, a concentrated broth culture filtrate of the bacilli, contains a poisonous nucleoprotein (animal type—not vegetable) which is the toxin.

Diagnostic tests are based upon the antigen reaction. The Moro test consists simply in rubbing tuberculin into normal skin. The Mantoux test is an intradermal injection. Local erythema and edema are evidence of a positive reaction. This reaction is accompanied by fever, pain, increased cough, and other symptoms. The von Pirquet test is a cutaneous scratch inoculation. The Calmette reaction is the development of conjunctivitis after dropping tuberculin into the eyes. Protective inoculations have been advocated by three methods:

- (a) Inoculation of living virulent bacilli. The risk is great.
- (b) Inoculation with attenuated bacilli. Successive cultivations on special media yield an attenuated organism. This is the BCG (bacillus Calmette-Guerin) which some consider safe and as giving "some protection." (Tissot denies that it gives protection.)
- (c) Inoculation with dead bacilli. This seems no more effective than BCG. "The use of antisera in the treatment of tuberculosis has proved of no value." (Jordan) (As for immunization, this subject will be treated in a later chapter.)

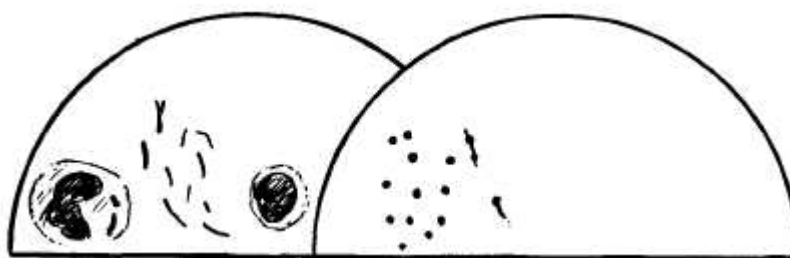


fig. 16 *Mycobacteria tuberculae* and Much granules

### *Other Mycobacteria*

Although thirteen species are now accepted as authentic, over forty different varieties have been described, nearly all saprophytic. "Saprophytic" applies to organisms living on dead organic matter. "Commensal" applies to parasites living on a host without benefit or harm to the host. Thus most of the saprophytes mentioned are forms free-living in nature; a few are commensals being temporarily dependent on a host. Of the tubercle bacillus, there are the human, bovine, and avian forms, all different. *Mycobacterium leprae* is very similar and is the organism found in leprosy. *Mycobacterium smegmatis* is a harmless commensal found in preputial secretion and the labial folds of the vulva. Thus it confuses the interpretation of urine samples. "The smegma bacillus is often difficult to distinguish from the tubercle bacillus on

morphological grounds.” (Jordan) “Differential diagnosis MUST be by inoculation for virulence.” (Stitt, Clough and Clough). “Grass” and “butter” bacilli, almost identical saprophytes, are confusing. “Animal inoculation does not always surely differentiate these organisms (numerous free-living forms—RW) from the tubercle bacillus, since some varieties produce histologic changes closely simulating those of true tubercle formation. Inoculation from these lesions into guinea-pigs, however, fails to cause infection. The rapid growth of the ‘grass’ and ‘butter’ bacilli in artificial media, best at about 20 degrees C, is the principal differential feature.” (Jordan) (Now, isn’t that a shock to “monomorphism” and a really scientific differentiation of *M. tuberculosis* and the saprophytes—by their speed of growth? Mmm.-RW)

Regarding classifications, the inclusion of *M. tuberculosis* with the vegetables (as a schizomycete) has no scientific justification whatsoever. “It is of great interest that the tubercle bacillus contains a typical nucleic acid of the animal type found in thymus, pancreas, sperm and spleen. It does not contain nucleic acid of the so-called plant type found in yeast and wheat.” (Jordan) Tissot clarified this discrepancy.

## CRITICAL REMARKS

### *Pasteurization*

Bovine tubercle bacilli have lived in human cutaneous tissues for many years without acquiring characteristics of the human type. Koch declared that the susceptibility of man to bovine tuberculosis is slight, and that infection from dairy products, including infected meat, is a very rare occurrence. Still there is some “evidence” that in small children there is some bovine tuberculosis. “More than one half of the cases of cervical adenitis and abdominal tuberculosis (in children under five years) are due to bovine infection.” (Jordan)

“Pasteurization kills the majority of harmless souring organisms, together with tubercle bacilli, while tougher micro-organisms escape. Hence it is a boon to wholesalers, because it enables them to ‘handle’ originally dirty milk; it simplifies buying and selling. That pasteurization should be altruistically welcomed by a large section of the medical profession, who thus become allies of the merely commercially-minded wholesalers, may seem strange, unless one understands medical mentality. On the question of the relation of bacteria to disease, the profession has not advanced beyond the stage of regarding the bacterial agents as the whole story of infection. Hence the insistence on killing bacteria in milk . . . Pasteurization is a confession of failure to secure decent milk to start with.” (So states Nicol, an outstanding bacteriologist.) Pasteurization also renders the phosphates unusable to the body.

### *Tuberculosis not contagious in adults*

Dr. Auguste Lumière summarizes in the *Revue Générale des Sciences Pures et Appliquées* of February 15, 1933 the evidence that tuberculous contagion is virtually a myth. His facts are as follows:

First, it is impossible to find any record of contamination of a specialist in tuberculosis by his patients.

Second, among the personnel of hospitals and sanatoria, the frequency of tuberculosis is no greater than in other groups.

Third, the directors of sanatoria are unanimous in maintaining that there is no case of contagion in their establishments, which, nevertheless,<sup>1</sup> are environments particularly rich in the incriminated germs.

Fourth, in the hospitals where tuberculous patients are not separated from other patients (as is usually the case), contagion does not occur despite the poor resistance of the patients who happen to be the neighbors of those who spit the bacilli.

Fifth, the wives and the husbands of tuberculous patients, despite the constant opportunity for contagion created by conjugal promiscuity, do not contract the disease. Those who are not of tuberculous stock invariably remain unharmed. "For several years we have sought in vain for a duly controlled case of marital contagion, and we have, without success, asked physicians about us, to cite examples of this contamination. On the contrary, the cases are innumerable in which one of the pair being phthisical, the other has remained indefinitely uninjured, despite the repetition of the most intimate contacts."

Sixth, when the death rate from tuberculosis is known it is found that the frequency with which man and wife are both victims is no greater than might be predicted from the calculus of probabilities and the laws of chance. Contagion does not enter into the phenomenon.

Seventh, "in the past, the remarkable clinicians of the time of Laennec never observed this contagion in the hospitals nor elsewhere, and the patients were not the object of any measure of isolation whatever. It is difficult to incriminate the physicians of that time for their ignorance or inadequacy, for having neither x-rays, nor laboratory resources, they must have relied upon clinical examination for their diagnosis and thereby acquired a sense of observation of the first order, thanks to which the facts of contagion could not have escaped them. Then, the masters of phthisiology, the practitioners and the whole Academy of Medicine were unanimous in proclaiming tuberculosis to be non-contagious."

Eighth, animals cannot be infected experimentally under the conditions to which human beings are exposed, even when the animals most sensitive to tuberculosis are chosen for the experiment. To infect animals one must have recourse to expedients to which man is never exposed. When inoculation is employed, it leads to an affection in animals that is always identical, without any polymorphism, that develops without remissions, without regression and never heals—an affection which has not, in fact, any of the characteristics of human tuberculosis common to the adult.

Tubercle bacillus found in absence of tuberculosis

In the article just cited, Dr. Lumière calls attention to the fact that the tubercle bacillus is often found in the sputum of persons who give no evidence whatever of having tuberculosis. More important, perhaps, is the report in the *Annales de l'Institut Pasteur* for November, 1932 of three patients in whose blood tubercle bacilli were found, although there were no clinical indications of tuberculosis. In two of these cases it was possible to make very searching postmortem examinations without finding a trace of tuberculous lesion. Such findings as these certainly invalidate the claim that this bacillus is the real cause of tuberculosis.

## 14 THE SPIRAL FORMS

These organisms are corkscrew-shaped microbes now classified as midway between true bacteria and the protozoa. Actually they are much more animal than vegetable. Because of this there is much argument about classifying them with the schizomycetes, which are placed in the vegetable kingdom. For that matter, the inclusion of *M. tuberculosis* with the vegetables has no scientific justification either. Eventually the schizomycetes may be classed as all being intermediates, neither animal nor vegetable.

The large, loose spirals, the Spirochetaceae, are of no clinical significance. The small, tight spirals, the Treponemataceae, do include a few pathogens. At one time all of them were called spirochetes but this terminology has been clarified.

### **Spirochete of Syphilis—*Treponema pallidum***

(*spirocheta pallidum*)

#### *Historical*

First observed in 1904 by Schaudinn and Hoffmann

First grown on artificial media in 1911

#### *Characteristics*

Extremely slender spirals

Length 4 to 20 microns

Number of turns variable, usually 3 to 12

One terminal flagellum

Forward, backward, and rotational movement

Grows on medium of blood serum, water, and sterile tissue

Strictly anaërobic

Very difficult to stain. Examined in “dark field.” A special condensor for oblique lighting illuminates the vital spirals by refraction against the dark background.

Inexpensive imitation is effected by flooding the slide with India ink through which the spirals swim and are seen brightly.



FIG. 17 *Treponema pallidum*, *Borrelia Vincenti* with “fusiform” bacillus, and *Leptospira icterohemorrhagica*

#### *Resistance*

Killed in 30 minutes by a temperature of 50 degrees C.

Cannot withstand drying

#### *Habitat*

The chancre, or primary lesion of syphilis

The papules of the secondary state and the mucous patches  
Various internal organs of infected individuals

#### *Modes of transmission*

Sexual intercourse

Direct contact of a syphilitic lesion with skin or mucous membrane, particularly if either of the latter is unhealthy or presents abrasions

Transmission from parent to offspring congenitally

Vaccination with human serum from syphilitic subject

#### *Prophylaxis*

Education in sex hygiene

Registration of cases and supervision until cured

#### *Serology*

Wassermann, et al, demonstrated the presence of specific antibodies in the serum of syphilitic patients. The Wassermann test is based upon the "complement fixation," which is complicated and easily invalidated. Complement fixation occurs when an antigen and its specific antibody are mixed in the presence of free complement. All three, antigen, antibody, and the linking complement are locked together. This change is invisible, so a colored indicator is added in the form of sensitized erythrocytes. Hemolysis is visible and as noted in Chapter 3, hemolysin is dependent upon a complement. Hemolytic serum is prepared by injecting a rabbit with sheep erythrocytes. The rabbit is bled, serum separated and inactivated by heating to destroy the complement. The serum is mixed with washed sheep erythrocytes. Hemolysis can then take place only by addition of free complement. In a separate tube the patient's serum is mixed with syphilitic antigen and free complement (guinea-pig serum). If the patient is syphilitic, thus having the specific antibody, the complement is fixed. Otherwise the complement remains free and will precipitate hemolysis of sheep erythrocytes in the indicator tube.

Disturbing facts are as follows: Any lipid-rich substance from normal man or other animal acts as a syphilitic antigen. Beef heart extract is commonly used in the test, although definitely not syphilitic. Also any febrile disease at certain stages will give a positive Wassermann. Several other chronic diseases also render a positive reaction, so it is far from being conclusive. Malaria, jaundice, tuberculosis, infectious mononucleosis, leprosy, and smallpox vaccination all give a positive Wassermann reaction.

Precipitation tests are based upon the fact that syphilitic serum will precipitate (or flocculate) colloids more than will normal serum. This is the basis for the Kahn test, which is the most generally used diagnostic test for syphilis. Kline, Ide, Mazzini, Eagle and Hinton tests are quite similar. The Kolmer test is a modification of the Wassermann complement fixation test. Another popular flocculation test is the gold colloid test based upon this same phenomenon, but used primarily to differentiate types of syphilitic pathology; hence the terms, "paretic" and "luetie" curves. This test is performed with ten test tubes of different concentrations of the cerebro-spinal fluid to which is added Lange's gold colloid. Color changes are the basis of interpretation. Meningitis has a typical curve, also. Encephalitis lethargica and poliomyelitis have shown "tabetic curves."

#### *Variants*

Yaws is a tropical disease, nearly endemic in places. It is caused by *Treponema pertenue*. "*Treponema pertenue* cannot be differentiated from *Treponema pallidum*

by microscopic or serological examination.” (Laboratory Methods of the United States Army) Yaws is not a venereal disease, most lesions being on the foot and ankle. Bejel is an Arabian disease caused by an identical treponema, but it is an endemic children’s disease like measles with papular eruptions. It is not venereal, nor does it have any nervous pathologies. Still another disease caused by an identical treponema, Treponema carateum, is called Pinta in South and Central America. A number of other treponemata are known. Three are found in the mouth and are non-pathogenic. “Treponema microdentium, which occurs in normal mouths, can be distinguished from Treponema pallidum only immunologically.” (Stitt, Clough and Clough) Some of these are definitely accepted as variants of the same species. In addition there are eight separate species. Many of them are strictly non-pathogens, such as the confusing Treponema calligyrum frequently found in smegma.

## REMARKS

McDonagh maintains that the Treponema pallidum is only the adult male phase of the “Leukocytozoon syphilidis,” a protozoon. He has also rendered syphilitics with positive Wassermanns quite negative simply by diet and colonic irrigation—no drugs at all.

“In monkeys, subcutaneous, intraperitoneal, and intravenous inoculations, even of the most virulent material, are without effect, but cutaneous inoculation, particularly upon the eyebrows and genitals, is usually followed by typical primary lesions.” (Jordan) This is a rather clear-cut way of saying that it is only a specific nerve stimulus. Speransky showed that very well.

## **Borrelia**

Seventeen accepted and ninety-two questionable species make up this genus of short spirals. Only three are even slightly significant clinically. B. recurrentis (Spirocheta obermeieri) is frequently found with relapsing fever, a louse or tick-borne disease. “Leishman et al believe that in the tick the spirochetes undergo a metamorphosis into granules. These are taken up by the cells and can be transmitted by the eggs through several generations.” (Jordan) This sounds interesting here, just follow it up through the rest of this book. A questionable condition, rat-bite fever, is supposedly caused by the spirillum, minus which Bergey has not fully accepted it as a bona fide borrelia. B. vincenti is found in trench mouth or Vincent’s angina. Of special interest is the fact that with it is always found the Fusobacillus plauti-vincenti, the fusiform bacillus. Many authors consider these two, a bacillus and a spirochete, to be varied forms of the same organism. Apparent transformations have been observed. Some consider them to be only symbiotic. “Borrelia refringens (genital) may be mistaken for Treponema pallidum.” (*Laboratory Methods of the United States Army*) It usually has looser spirals.

## **Leptospira**

Four pathogenic species and many saprophytic species make up this genus. One, the *Leptospira icterohemorrhagica*, is an item found at times on state board examinations. It occurs in the body in "infectious jaundice." "Leptospira closely resembling *L. icterohemorrhagica* have been isolated from water. These non-pathogenic forms are named. Their relation to the pathogens is uncertain. Some have suggested that they are avirulent forms of *L. icterohemorrhagica* whose virulence may be restored by animal passage." (Jordan) *L. hebdomadis* is a Japanese counterpart found with Japanese seven-day fever.



## 15 ANIMAL PARASITES

These are not bacteria but are usually included in bacteriology texts and in bacteriology examinations. Various of the protozoa are included, and the few of clinical importance are outlined in order. The metazoa include all multicellular animals. In this class come the various worms and flukes which are often considered in public health topics. They are not included here for discussion.

### Animal Classification

#### *Animal Kingdom*

- I. Metazoa (sub-kingdom)—all multicellular animals
- II. Protozoa (sub-kingdom)—all unicellular animals
  - A. Plasmodium (pseudopodia or flagellates)
    1. Rhizopodia—pseudopodiaters  
Include the various amoebae listed under Amoebic dysentery plus five coprozoic free-living and nine unclassified amoebae (all non-pathogenic) found in human intestinal tracts.
    2. Mastigophoria—flagellates  
Include the Trichomona, Trypanosoma and Leishmania plus four entire families of non-pathogens.
    3. Cnidosporidia—pseudopodiaters with filamentous spores  
—(NP to man)  
Include the Pebrine of Béchamp.
    4. Sporozoa—usually with no organelles for movement. Reproduce by schizogony; after syngamy produce sporozoites.  
Include the Plasmodia of malaria, the Coccidia (parasites of lower animals) and several others, non-pathogenic to man.
  - B. Ciliophora (ciliates)
    1. Ciliata—ciliates throughout life.
    2. Suctorina—ciliates when young, suctoriates as adults;  
— (NP to man)

### SOME PROTOZOA

#### **I The Malarial Parasite—Plasmodium**

The micro-organism of malaria is not a bacterium, but a protozoon. The fact, however, that malaria is a fairly common and widespread disease has resulted in the inclusion of a study of this parasite in the majority of textbooks on bacteriology.

#### *Historical*

Parasite first observed by the French army surgeon Laveran in 1880

Life history of one variety described by Golgi in 1885

Interrelations of the parasite, the mosquito, and man worked out by Manson, Ross, and Grassi

#### *The malarial parasite within the body of man*

Introduced by the Anopheles mosquito

Invades the red blood cells

First appears as a shiny, oval body with amoeboid movement.

Grows at the expense of the blood cell  
Undergoes segmentation into small rounded bodies  
Blood cell disintegrates setting free these bodies  
Fever occurs while these are circulating in the blood stream  
New red corpuscles are invaded and the cycle is repeated

*The malarial parasite within the mosquito*

Some of the parasites, while free in the human blood stream, become sexually differentiated. When the Anopheles mosquito bites the patient, such specialized cells may be withdrawn. These germ cells find their way to the stomach of the mosquito, where fertilization takes place. The fertilized cells become encysted and greatly enlarged

Rupture of the cyst releases innumerable minute bodies which soon infest the entire insect.

*Varieties of the parasite*

1. Plasmodium vivax

Actively motile in earliest stage  
Characterized by a yellowish brown pigment, very fine  
Reaches a size nearly double that of the red blood cell  
Completes its cycle in the human host in 48 hours  
Associated with the so-called tertian type of malaria, in which fever occurs on alternate days

2. Plasmodium malariae

Slightly motile  
Very glistening  
Coarse, black-brown pigment  
Size not in excess of that of red corpuscle  
Cycle completed in 72 hours, giving rise to the so-called quartan form of malaria

3. Plasmodium falciparum

Smaller and more actively motile than the other two  
Very little pigment  
Tends to be confined to the blood of internal organs, often the spleen  
Cycle variable  
Found in the estivo-autumnal type of malaria

4. Plasmodium ovale

Almost identical to Plasmodium vivax in every way except that the affected erythrocytes are characteristically oval

Clinically Plasmodium vivax and Plasmodium ovale considered the same, although technically separate species

*Mode of infection*

Aside from experimental methods, transmission is exclusively by the bite of an Anopheles mosquito which has already bitten a malaria patient. Whether man or the mosquito was the first to harbor the parasite, and how the parasite itself originated, are matters concerning which nothing whatever is known.

**Distinguishing features between the Anopheles and the non-malaria carrying Culex mosquito**

*Culex*

*Anopheles*

**LARVAE**

Nearly at right angle to surface of water

Parallel with surface

Feed below surface

Feed only at surface

**ADULTS**

Unspotted wings

Spotted wings

Body parallel to surface on which it rests

Body at sharp angle to surface on which it rests (abdomen elevated)

*Prophylaxis*

Drainage of breeding places of Anopheles

Treatment of water with oil or Paris green

Large-scale spraying with the newer insecticides

Use of screens, especially to prevent contact between a patient and the mosquito

Improvement of the general health by rational methods

(All exposed persons do not contract malaria)

*Note:* The tertian type of malaria is commonest in temperate climates;

The estivo-autumnal, in the tropics.

The quartan type is comparatively rare.

**REMARKS**

Quinine has been considered a specific cure for years. Recently atabrine has been used. Even though chemotherapy has been regarded so optimistically, Cecil's *Textbook of Medicine* states, "Malaria is a most difficult condition to eradicate completely." Statistics show that there is a very small percentage of medical cures. Actually the prognosis under chiropractic, with nothing but straight adjustments, is better than any present-day chemotherapy.

## IN FEMALE MOSQUITO

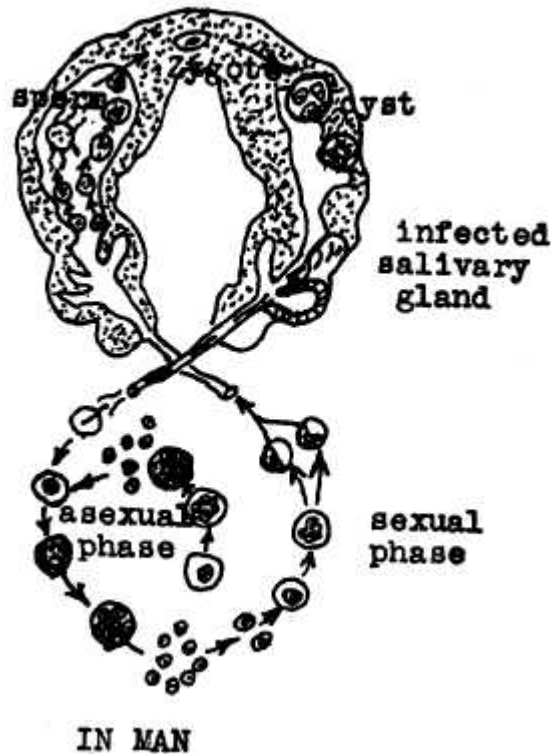


FIG. 18 Diagram showing life-cycle of malarial parasite from mosquito to man and back to mosquito. (From Wheat and Fitzpatrick: *Advanced Biology*)

## II Amoebic dysentery—*Endamoeba histolytica*

Six species found in man make up the order Amoebida. Only one is pathogenic, the type species described. Amoebic dysentery is essentially a tropical disease, brought to the temperate zone by military personnel and occasional travelers.

### *Historical*

Observed and described by Losch in 1875

### *Characteristics*

#### Active form

- 20 to 30 microns in diameter
- Amoeboid movement conspicuous
- Consumes erythrocytes and tissue elements
- Consumes bacteria in cultures

#### Cystic form

- 5 to 20 microns in diameter
- A shrunken form which secretes a heavy capsule, becoming a uninucleate cyst
- By nuclear division then becomes a mature quadrinucleate cyst
- Expelled in feces, may be ingested as contaminant

Life cycle outside man unknown

Difficult to culture (has been cultured on egg and blood agar flooded with Locke's solution and serum)

### *Habitat*

Morbid intestinal tracts of patients and carriers

Contaminated food and water

*Mode of Action*

Erosion of intestinal wall to form undermined craters

Abscess formation in deeper tissues, especially the liver

*Resistance*

Pathology highly resistant to chemotherapy

Prognosis poor under any type of therapy

Endamoebae easily killed in vitro, but inaccessible in a patient

*Related forms*

There are at least four other species of non-pathogenic amoebae living in the intestine and one living in the mouth. *Endamoeba gingivalis* (a commensal) is extremely common in the mouth and is indistinguishable from *Endamoeba histolytica* except for its lack of virulence. The intestinal non-pathogens eat bacteria, yeasts and feces but never tissue elements. They are *Endamoeba coli*, *Endolimax nana*, *Iodamoeba williamsi* (*I. butschlii*) and *Dientamoeba fragilis*. "There are strains of *Endamoeba histolytica* differing in virulence. A strain which produces acute dysentery in some individuals may give rise to symptomless carrier conditions in others. The relationship among (strains of) *Endamoeba histolytica* is extremely complex. Cultures of *Endamoeba coli* (a normal saprophyte) resemble *Endamoeba histolytica* so closely that differential diagnosis is often very difficult." (Jordan)

**CRITICAL REMARKS**

This inter-relationship is fascinating double talk, a "scientific" who-done-it.

**III Trichomona**

A group of flagellate amoebae. *T. hominis* is a non-pathogen of worldwide distribution in normal colons. An indistinguishable form, *T. vaginalis*, is occasionally found with vaginitis and more rarely with male urethritis.

**IV Giardia lamblia**

This is a binucleate flagellate amoeba found commonly in the upper part of the small intestine. It has been suspected of causing a few cases of dysentery. A bacteriology professor once asked a student to tell what he knew about *Giardia lamblia*. "Well," was the reply, "he was once mayor of New York."

**V Trypanosomes**

A group of free-swimming protozoa found in blood plasma and body fluids. *T. lewisi* is a normal parasite found in rats. Several other species are recognized, but only one, *T. gambiense*, is of human clinical importance, because human trypanosomiasis is African sleeping sickness. It is a tropical African disease, not to be confused with the worldwide, virus-type encephalitis lethargica.

**VI Leishmania**

Kala-azar (dum-dum fever or tropical splenomegaly) is due to the presence of *L. donovani*. "Donovan bodies" are inclusion bodies associated with this condition.

## **VII Balantidium**

Among numerous non-pathogenic ciliata is *Balantidium coli*, a common intestinal parasite. Hosts are usually symptom-free, but occasionally a pathology occurs, almost identical to that with *Endamoeba histolytica*.

## 16 RICKETTSIAE

Typhus and related diseases finally yielded a possible causative agent. The Rickettsiae are small pleomorphic units, either cocci (single or double) or short diplo-bacilli 0.3 micron or more in length. They are considered as midway between a bacterium and a virus. They grow only on living cells and are easily killed. The twelve recognized species fall into four groups:

- |   |               |
|---|---------------|
| 1. Classical typhus and Brill's disease | —R. prowazeki |
| 2. Rocky Mountain spotted fever         | —R. nipponica |
| 3. Tsutsugamushi disease                | —R. burneti   |
| 4. "Q" fever, and Trench fever          | —R. Quintana  |

Bergey has grouped these forms partially by vectors; as follows:

### Rickettsia (non-filter-passing forms)

1. Louse borne  
Rickettsia prowazeki (classical typhus)  
\* R. pediculatis (R. quintana of trench fever)  
(R. pediculatis and many others are not definitely accepted as yet.)
2. Flea borne  
R. typhi (R. mooseri) (endemic murine typhus)
3. Tick borne  
R. rickettsiae (Rocky Mountain spotted fever)  
R. conorii (boutonneuse and Kenya fever)
4. Mite borne  
R. tsutsugamushi (including R. akamushi, R. orientalis and R. nipponica)  
R. akari (rickettsialpox)

### Coxiella (filter-passing form):

- C. burnetti (R. burnetti of Q. fever —RW)

Bergey says that there is too little evidence of the relation to trench fever.

All these diseases are commonly vectored by ticks and lice. "The Rickettsia of Rocky Mountain spotted fever seems to have undergone a series of adaptations to successive hosts. It is thought that the organism was first a plant parasite; then became adapted to the mites which fed on the plants; then to the rodents to which the mites had become adapted; and finally to the ticks which infest the rodents." (Stitt, Clough and Clough)

Of particular interest is the Weil-Felix reaction. Serum from Rickettsial patients will agglutinate certain strains of *Proteus vulgaris*. It is a diagnostic test very similar to the Gruber-Widal reaction. "The significance ... is not clear." (Jordan) [How could it be to an orthodox bacteriologist?—RW] A much more vital comment is: "Rickettsiae are essentially intranuclear organisms and they appear in the cytoplasm after intranuclear development." (*Laboratory Methods of the United States Army*) This set of "puzzles" is made very clear throughout the latter section of this book.

## 17 THE FILTERABLE VIRUSES

There is a fairly extensive group of diseases which because of their communicable or epidemic character have traditionally been looked upon as germ diseases, despite the fact that no germ has been isolated for which any experimental evidence has been adduced indicating an etiological relationship to the disease. The channel of invasion of the infectious agent is generally thought to be the respiratory tract, and it is frequently in the exudates of some portion of this tract of the patient that the infectious agent can be demonstrated. When such an exudate is filtered through an unglazed, burnt clay filter of the sort used in separating bacteria from their toxins, it is found that the infectious agent is present in the filtrate, although no micro-organism can be seen in these filtrates, even when the highest power of the optical microscope is employed. Many "submicroscopic" particles have been observed under the electron microscope. The toxic agent has, for this reason, been called a filterable or filter-passing virus.

### *Nature of Filterable Viruses*

There is no uniform opinion among bacteriologists concerning the nature of these viruses. Up to the present time it has not been possible to cultivate them in the laboratory media used in growing bacteria. In fact, nothing simulating a culture has been obtained apart from living tissues. There are several interesting conjectures regarding the filterable viruses, among them the following:

1. They are inanimate substances or colloidal particles
2. They are a hitherto unknown subcellular form of life
3. They are bacteria of submicroscopic size
4. They represent a certain phase in the life cycle of ordinary bacteria
5. They are enzymes of unknown origin
6. They are products of cellular degeneration capable of inciting further degeneration. "Since these agents are able to increase in the body of the host (and not outside), it would appear that they stimulate the host cells to form more virus." (Jordan)

In the present state of our knowledge one cannot afford to be dogmatic in this matter. The fourth conjecture was suggested by the experimental fact that some common forms of bacteria may, under certain conditions of cultivation, develop a filterable stage which may later revert to the ordinary non-filterable form. It has not been demonstrated, however, that ordinary bacteria can arise from any of the known viruses, nor that the converse is true. Undoubtedly, the sixth conjecture is the most interesting to the drugless practitioner of whatever school. It represents a point of view held by them long before bacteriologists ever proposed it, a suggestion which until recently would have met with nothing but ridicule in orthodox scientific circles. Incidentally, it will, if proved true, make perfectly understandable the spontaneous appearance of a filterable virus disease without previous contact of the patient with another.

The latest authoritative reports at the International Congress of Cellular Biology, 1950, by Lwoff et al fully support the sixth conjecture as did a still later symposium of international authorities. *Viruses—1950* by Delbrück is a compend of this symposium and states that "Virus is produced by ... the virus infected cell."



“Only a fraction of a percent of the cells ... are wounded in the right way and to the right degree for entry and establishment of the virus . . .” Well known spontaneous degeneration of cells and consequent spontaneous virus formation, as mentioned with anterior poliomyelitis in Cecil’s *Textbook of Medicine*, is further support of the sixth conjecture. Tissot’s work, summarized later in this book, makes the most plausible explanation of viruses and their significance.

#### *The filterable virus diseases*

The following grouping includes the more important diseases with which filterable viruses are associated:

1. Neurotropic—Anterior poliomyelitis (virus named Legio debilitans), rabies, lymphocytic chorio-meningitis, equine encephalitis, encephalitis lethargica (St. Louis and Japanese types), ascending myelitis, West Nile fever, Herpes Zoster (shingles)
2. Dermatropic—Variola (smallpox), varicella (chickenpox), Alas-trim, vaccinia, herpes simplex (cold sore), foot and mouth disease, trachoma, epidemic kerato-conjunctivitis, inclusion blennorrhoea, molluscum contagiosum, verruca vulgaris (common warts)
3. Respiratory—Common cold, influenza (A, B, and X), psittacosis (parrot fever)
4. Intermediate—Rubeola (measles), rubella (German measles), lymphopathia venereum (lymphogranuloma inguinale), dengue, Rift Valley fever
5. Viscerotropic—mumps, yellow fever, hog cholera

#### *Inclusion bodies*

The pathology of the filterable virus diseases is essentially an intracellular pathology, the virus usually appearing first in the nucleus, where it probably originates. Also, in the majority of these diseases there appear minute corpuscles within the affected cells. These corpuscles may be sufficiently characteristic to identify the disease. Thus, in the brain cells of rabid animals it is possible to detect bodies known as Negri bodies. Inclusion bodies is a general term used in referring to structures of this kind found in various virus diseases and some parasitic infestations.

#### *Transmission of the virus diseases*

As already stated, these diseases are believed to be most commonly transmitted by exudates from the respiratory tract. In the case of hydrophobia, the saliva of the rabid animal contains the virus, and the bite of a mad dog, wolf, cat, or other animal may be followed by the disease. It is noteworthy, however, that statistics indicate the occurrence of hydrophobia is only about 16 per cent of the persons bitten by mad dogs. Yellow fever and dengue are carried by mosquitoes, while in the case of trench fever it is probably the body louse that performs the role of carrier.

#### *Other viruses*

Tobacco mosaic was one of the earliest pathological manifestations to be definitely attributed to a filterable virus although the virus is too large to pass present filters. With the advent of the electron microscope there has been a scramble to see who can find and name the most tiny particles. By referring to Bergey’s *Manual of Determinative Bacteriology*, Sixth Edition, one can find listed a host of plant diseases with the names of associated viruses. Plant diseases, however, are outside the scope of this work.

Bacteriophage, discussed in the following chapter, and now classed as a virus, is subdivided into forty-six named species. This classificatory tendency reminds one of an article by Ian Stevenson, M.D., in *Harper’s Magazine* on “Why Medicine Is Not

a Science.” He said that medical treatment of pathology is simply an increasingly complex cataloguing procedure with very little knowledge of how the pathologies started, their interrelationships, or any broad understanding of the subject. This is exactly the reason why you find Rational Bacteriology so different from orthodox texts. We are attempting a practical evaluation.

## 18 THE BACTERIOPHAGE

### *Twort's discovery*

“No bacteriological discovery in the last twenty-five years,” writes Gardner, in his *Microbes and Ultramicrobes*, “has aroused such a widespread interest as that of the mysterious agent that destroys bacteria and regenerates itself in the process.” It came about in the following manner. The British bacteriologist F. W. Twort had a culture of *Staphylococcus albus* in which he observed small patches where the bacteria appeared to have dissolved. He filtered some of this culture through porcelain to remove the bacterial cells, and then suspended in the filtrate a fresh culture of *Staphylococcus*. To his surprise the new culture disappeared and very few living bacteria remained. When he experimented with the filtrate to see whether it would exert a similar action on other cocci, he found that this was the case, although not to the same degree as with the *Staphylococcus*. Here, then, was a filterable something that exerted marked destructive action on bacteria. Twort reported his findings in *The Lancet* in 1915. His researches were interrupted by World War I.

### *D'Herelle's discovery*

The following year d'Herelle found that whereas, in the early stages of dysentery, the stool of the patient yields bacilli which can be readily cultivated, there comes a time just preceding convalescence, when no colonies can be produced from this source. Furthermore, if the stool at this time is filtered through porcelain, and cultures of the dysentery bacillus are added to the filtrate, these cultures promptly dissolve. When the resulting material is again filtered and additional bacteria added, the solvent action is further intensified. In fact, each successive addition of bacilli and subsequent filtration enhances the power of the filtrate to bring about the strange effect. D'Herelle's discovery is obviously the same phenomenon which Twort had already observed and has come to be known as the Twort-d'Herelle phenomenon.

### *Appearance of a colony undergoing bacteriophage action*

The accompanying illustration from Gardiner, based upon a study of colonies of the *Shiga* bacillus, shows the process at work.

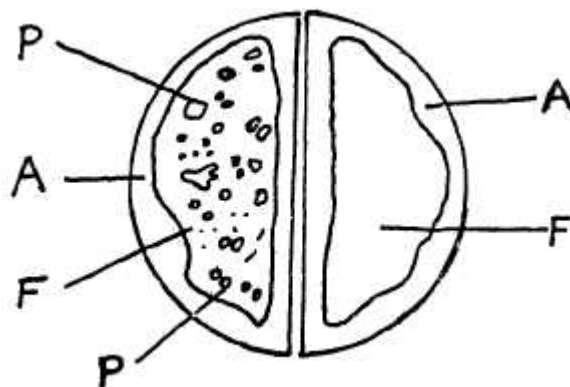


FIG. 19 Left side—Action of bacteriophage  
Right side—Control of growth without bacteriophage  
A—Agar medium  
F—Film of bacterial growth  
P—Plaques of lysis

*Appearance of individual microbes in bacteriophagy*

Bacteria in the presence of bacteriophage are seen under the microscope either gradually to fade away or else to swell up, become granular, burst, and finally liberate, in this manner, their granular contents. A barely perceptible shell of the original bacterium may remain for a time. Gardner illustrates the process in Fig. 20.

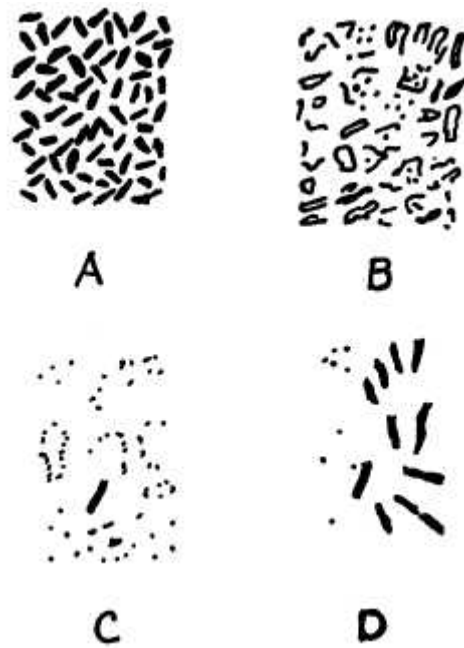


FIG. 20

A—Before                      B—During  
C—After lysis                D—Secondary growth beginning

### *Sources of bacteriophage*

Bacteria closely related to the common colon bacillus (for example, the typhoid and the dysentery bacilli) can frequently be made to manifest the Twort-d'Herelle phenomenon by filtrates obtained from the intestines either of man or of animals. The blood of both man and animals, especially during recovery from an infectious disease, will also at times induce the phenomenon. Other sources of bacteriophage are sewage, certain soils, and many natural waters. Old cultures of bacteria sometimes *spontaneously* become lysogenic; that is, productive of substances which exert a solvent action on bacterial cells.

It must be remembered that bacteriophage is not known to multiply outside specific cells in the manner that free-living bacteria exist and reproduce in soil and water. Bacteriophage is believed not to reproduce; it is produced by and within degenerating cells. Thus the phage is a mutant. For details refer to *Viruses—1950* by Delbrück. Bacteriophage cannot infect perfectly healthy cells but can be absorbed by a crippled cell in which it acts as a pattern to condition further degeneration. The subsequent new phage particles are then terminal end products of a degenerating cell and may be widespread in nature to the degree that organic waste is widespread.

### *Interpretations*

The bacteria-destroying agent was named bacteriophage by d'Herelle in the belief that he was dealing with an ultramicroscopic living parasite of bacteria. D'Herelle has never abandoned this view, and he has many staunch defenders. Many, and probably the majority of bacteriologists, however, incline to other interpretations. Among the arguments advanced against the idea that the bacteriophage is a living entity is that it is not destroyed by even so high a temperature as 120 degrees C. There are many other cogent reasons for rejecting d'Herelle's interpretation which it would be beyond the scope of this work to

consider. The reader will find them well presented in Gardiner's *Microbes and Ultramicrobes*. Suffice it to say that the bacteriophage originates within bacteria themselves, that it is in the nature of a gene, that it exerts suicidal tendencies, so to speak, in the bacterial cell of which it is a part, and that it is able, when liberated from such a cell, to impart its property to other adequately injured cells. Hence the notion long ago advanced by Béchamp that bacteria may originate through a *degenerative metamorphosis in living tissue cells* seems worthy of re-examination and rigid test, and we may look for an important contribution to the problem of the origin of communicable diseases. Nor should one forget that the development of bacteriophage within the human body coincident with convalescence may well signify the inherent power of the body so to modify bacterial activity as to render the bacteria innocuous.

*Present classification*

Bacteriophage is now considered by most orthodox bacteriologists as a virus infecting a bacterium. Such reasoning regarding "infecting" is untenable in that a bacterium must be of a susceptible strain and be adequately injured before infection; also that bacteriophage appears spontaneously in isolated cells without previous contact. There are now forty-six accepted separate species "preying" upon many different bacteria. From the first observations on Staphylococci and Shigella the phage has been observed affecting several other species including many of the Coryne bacteria. The forty-six "species" of the phage have rather common morphological characteristics. The following sketch is typical of the appearance under an electron microscope. The phage, usually a tailed coccus, is seen being adsorbed onto an injured bacterium (just before being absorbed and incorporated into it).

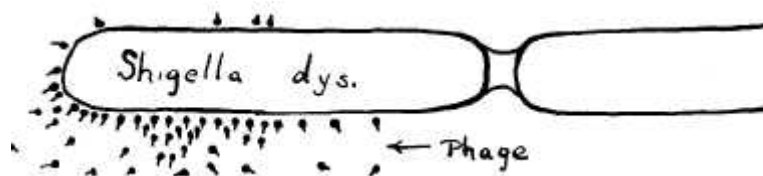


FIG. 21 Bacteriophage under electron microscope

## 19 MISCELLANEOUS PATHOGENS

A number of micro-organisms of relatively small importance are included here in brief survey. Some are bacteria and some are not. The non-bacterial forms are included only because of the tradition of bacteriology texts and of state board examiners to include them with bacteria, simply because they are considered pathogenic organisms.

### *Bacterial forms*

*Parvobacteriaceae* is the family which includes many of these leftovers. They are small pleomorphic, Gram negative, non spore-forming bacilli or cocco-bacilli. The various relationships can be seen easily in the chapter on classification. This family includes the next eight bacteria listed below.

*Pasteurella tularensis* is the very pleomorphic agent found in tularemia. It cannot be cultured on artificial media. An agglutination test is used.

*Pasteurella pestis* is the “plague bacillus” of the “Black Death.” Very pleomorphic, it takes the forms of gigantic straight rods, chains or rods, tiny cocco-bacilli, etc. It can be grown on ordinary media. Old cultures show a heavy capsule.

*Malleomyces mallei* is the “glanders bacillus”, which used to be classed as an Actino-bacillus. It was discovered by Löffler and Schutz in 1882. Morphologically it is very similar to the tubercle bacillus. “Long filaments with swollen ends and true branching place the glanders bacillus in the order of Actinomycetales.” (Jordan) Now it is classed with the *Pasteurella*. Actually glanders can almost be considered as tuberculosis of the horse. It affects the respiratory mucosa, and the glanders nodule is very similar to the tubercle, although more degenerative. Diagnostic tests like those for tuberculosis are used. Mallein is the equivalent of tuberculin; it is prepared in the same way, and the “mallein tests” are carried out just as the tuberculin tests, intradermally or via the conjunctiva.

*Brucella melitensis*, discovered by Bruce in 1887, is found in Malta fever in goats and in undulant fever in human beings. A tiny coccobacillus, 0.5 micron in diameter, it quite commonly causes a bacteremia. It is easily isolated from the spleen. A specific agglutination test is used by laboratories as a diagnostic procedure. The organism was formerly called *Micrococcus melitensis*.

*Brucella abortis* is practically identical with *Brucella melitensis*, but it is found with infectious abortion in cattle. Inoculation into monkeys produces symptoms like human undulant fever.

*Fusobacterium plauti-vincenti* is the new name for the old fusiform bacillus found with *Borrelia vincenti* in Vincent’s angina (trench mouth). The two are often considered as varied forms of the same organism.

*Hemophilus pertussis* is a tiny pleomorphic bacillus found with whooping cough. It was once called the *Bordet-Gengou* bacillus.

*Hemophilus ducreyi*, (*Ducrey’s bacillus*) is the organism, found in chancroid.

*Hemophilus influenzae*, *Pfeiffer’s bacillus*, is a tiny pleomorphic bacillus, often thread-forming, associated with influenza. It is one of the smallest pathogenic bacilli, seldom over 1.5 microns long and 0.3 micron in diameter. The *Koch-Weeks* bacillus, found in some cases of conjunctivitis, is often considered to be identical with it, although now called *H. conjunctivitis*.

*Moraxella* is a new genus with but one species, the *Morax-Axenfeld bacillus*, found occasionally with conjunctivitis and previously called *Hemophilus duplex*.

*Actinomyces hominis* is a branching rod form found in Madura foot. There are thirty-five species of *Actinomyces*, each of which is “a much-branched mycelium, which may break up into segments that function as conidia.”

*Borrelomycetaceae*—(not to be confused with *Borrelia*—a genus of spirochetes, or with *Borrelia*—a genus of viruses infecting insects, or with *Borreliota*—a genus of viruses with the typical pox diseases). This is the third supplemental order of *Schizomycetes* (after *Rickettsiales* and *Virales*). A single genus is definitely classified as *Asterococcus*. One of the seven accepted species is *A. mycoides* found with bovine pleuropneumonia. There is another species, probably of this genus which is a non-pathogen found on human genitalia. Four other species of uncertain classification develop within pure bacterial cultures. These, like all *Borrelomycetaceae*, are small, soft, fragile, pleomorphic micro-organisms, but “peculiarly,” they develop within uncontaminated, pure bacterial cultures. (Béchamp and Tissot would be amused at this naive separate classification.)

*Bartonella bacilliformis* is a unipolar flagellate (3 to 5 microns) which parasitizes erythrocytes in “Carrion’s disease,” Peruvian Verruca, and Oroya fever. They are found as cocci, as vibrios or as straight rods and are classed tentatively with the *Rickettsiae*.

### **Molds—Eumycetes**

These filamentous forms are more specialized forms than the bacteria and are consequently morphologically more complex. Some pulmonary infections with *Mucor* or *Aspergillus* occur in man, but more commonly in birds. Histoplasmosis, pandemic and usually symptom-free, is usually a microscopic granulosis of the reticulo-endothelial system and lungs. The fungus is *Histoplasma capsulatum*. The granulations of both histoplasmosis and aspergillosis closely resemble tuberculosis on x-ray and under a microscope. Thrush is an infection of the oral mucosa of infants probably by *Monilia albicam*. Herpes tonsurans, or barber’s itch, is usually associated with two or more species of *Trichophyton*. *Blastomycosis* is a localized pustular dermatitis found occasionally in man. Other than these the molds are of little concern clinically. Tissot, however, brings these molds into a new highlight.

### **Worms**

Many varieties of these are dealt with at length in texts on hygiene and have no place in this text at all. It is true, however, that many state boards seem to confuse their subjects and mix them up indiscriminately.



## 20 STATE BOARD EXAMINATION QUESTIONS

If possible, get series of questions asked previously by the board which you anticipate taking. Some boards offer them readily; for example, Minnesota. In certain other states (like Ohio) the questions are listed after each examination in the State Medical Journal, a practice which requires some library research

Examples of the more difficult type of question frequently encountered are grouped below. This is not to scare anyone, but to show the extreme variety of viewpoints held by the examiners. Some basic science examiners expect us to be experts in every subject.

### A Drugless Board: Two-Hour Examination

1. (a) Define bacteria  
(b) Would the tubercle bacillus normally be stained differently from the streptococcus? If so, why and how would you go about staining each?  
(c) Name the three basic forms of bacteria  
(d) Differentiate ptomaines and leukomaines (See Dictionary)
2. (a) When would you use the Widal test?  
(b) What is the causative organism in: syphilis, estivo-autumnal malaria?  
(c) Wolfhugel's method of determining bacterial count is used for what?  
(d) Would milk with a bacterial count of 9,000 per cc. be fit to drink? Why?
3. (a) Name two portals of infection each for gonorrhoea and tuberculosis.  
(b) At what temperature is milk pasteurized and for how long?  
(c) What are pyogenic bacteria?  
(d) Which of the following stain Gram negative: pneumococcus, bacillus influenzae, bacillus typhosis, bacillus botulinum.
4. (a) Differentiate antiseptic and disinfectant.  
(b) Which is easiest to culture on ordinary media, gonococcus or diplococcus-intracellularis meningitidis? Which media are usually used for these?  
(c) Define pleomorphic bacteria.  
(d) State whether the following tests are skin, agglutination, complement-fixation or precipitation tests:—Schick, Wassermann, Dick, Kline, Widal, Kahn.
5. (a) Name different modes by which bacteria propagate.  
(b) Which of the following conditions are caused by pyogenic cocci: Anthrax, osteomyelitis, siderosis, erysipelas, byssinosis, tuberculosis.  
(c) How is yellow fever transmitted? What is its cause?  
(d) What has pulmonary aspergillosis in common with blastomycosis?
6. (a) What are Rickettsia bodies?  
(b) State whether Koch's dicta are fulfilled in the following diseases: measles, typhoid fever, croupous pneumonia, diphtheria

- (c) What is the cause of Vincent's angina?  
 (d) State the incubation period in vaccinia, parotitis, varicella, rubella.

#### Assortment of Questions from Various Boards

1. Give the approximate dates of the following and their outstanding contributions:  
 Noguchi, Koch, Ehrlich, Lister, Ehrenberg, Pasteur, Metchnikoff, Fracastorius.
2. What is the size range of bacteria? How does heat, light and drying affect them?
3. Define bactericidal, bacteriostatic, antibiotic, antiseptic, germicidal, disinfectant.
4. Define and discuss attenuation, adaptation, and drug fastness.
5. State Koch's postulates. With what organisms have they been fulfilled?
6. List the external defenses of the body and briefly discuss each.
7. Discuss "normal flora" and resistance.
8. Discuss anaphylaxis.
9. Name six culture media and five examples of organisms grown on them.
10. Name ten Gram negative and ten Gram positive organisms.
11. Describe the following technics: Gram stain, Ziehl-Neelsen, Widal test.
12. List five neurotropic virus diseases; discuss transmission and control of one.
13. What diseases utilize the following reactions and tests (list the significance of each):  
 Widal, Weil-Felix, Schick, Dick, Mantoux, Shultz-Charlton, Quellung, Voges-Proskauer, mallein, and oxidase.
14. A patient has a sore throat of four days' duration. Name the organisms that may be responsible for this condition.
15. Differentiate *S. typhosa*, *S. paratyphosa*, *S. schottmuleri* and *Esch. coli* by laboratory methods.
16. Discuss the significance of acid-fast mycobacteria in a urine sample.
17. Name the diseases caused by (1) *Rickettsia prowazeki*, (2) *Histoplasma capsulatum*, (3) *Brucella suis*, (4) *Malleomyces mallei*, (5) *Plasmodium vivax*.

#### Questions from Basic Science Examinations

(5 questions usually comprise a two-hour examination)

#### BACTERIOLOGY (answer any 5)

1. Describe the method of performing a Gram stain, naming all reagents (do not merely list), giving the general precautions of the same and list five (5) Gram negative and five (5) Gram positive organisms pathogenic to man.
2. Distinguish the four (4) chief blood groups in man. Distinguish their significance.
3. Give the scientific name and describe the morphology, staining, cultural and biochemical reaction of the organism causing a) typhoid fever, b) mumps, c) diphtheria, (skip a line between each part).

4. Describe the influenza bacillus (*Hemophilus influenzae*) in regards to morphology, culture, physiology, pathogenicity for man and discuss its relationship to influenza, (skip a line between each part).
5. Define the unit of diphtheria antitoxin. How is the potency of a sample of diphtheria antitoxin determined?
6. Describe two (2) methods of isolating anaerobic micro-organisms. Name three anaerobic micro-organisms pathogenic for man.
7. Define: a) haptane, b) toxoid, c) Weil-Felix reaction, d) somatic antigen and e) anticomplementary action, (skip a line between each part).

1. Define the following (a) mycosis, (b) morphologic, (c) toxemia, (d) fusiform, (e) antigen, (f) opsonin, (g) Pasteurella.
2. Give the classification of elementary bacteria.
3. Explain the thermal death-point of bacteria. How and where does it take place?
4. In clinical diagnosis of tuberculosis by the use of tuberculin name three methods of making the test.
5. State the characteristics by which each of the following may be identified: (a) Shiga's bacillus, (b) Hemolytic streptococci, (c) Anthrax bacillus.
6. Describe the preparation and use of diphtheria toxin.

1. Draw and explain a culture growth curve.
2. Name two pathogenic and one non-pathogenic acid-fast bacteria giving their morphological and staining characteristics.
3. Discuss generally the morphology, motility, sporulation, and the taxonomic position of spirochetes.
4. Define four types of antibodies.
5. Contrast bacteria, yeast, and virus as to morphology, pathogenicity and cultural characteristics.

1. Discuss pleomorphism, monomorphism and oligomorphism.
2. State the morphology, cultural characteristics and pathogenicity of the pasteurilla, salmonella and the clostridium groups.
3. Discuss active and passive immunity.
4. How would you prove conclusively whether or not a culture of *Corynebacterium diphtheriae* was virulent?
5. Why is it important in the bacteriological analysis of drinking water to differentiate the index organism, *Escherichia coli*, from *Aerobacter aërogenes*?

1. Discuss the serological response and laboratory procedures involved in determining a diagnosis of Rocky Mountain spotted fever.
2. What are the chief characteristics which differentiate the Kahn test from the Wassermann test as used in the diagnosis of syphilis?
3. Give the binomial name of the etiological agent for the following infectious diseases:  
tuberculosis, cholera, anthrax, bacillary dysentery and a bacterial food poisoning caused by a Gram positive anaerobe.
4. How would you establish a laboratory diagnosis of bacillary dysentery?

5. Name and describe at least three of the eight toxins produced by *Staphylococcus aureus*.

1. Briefly discuss the humoral and cellular theories of immunity.
2. Give the correct binomial name for the seven orders of bacteria; characterize each order briefly. (Bergey listed seven in 1939 but now lists only five.)
3. Give the bacteriological diagnostic procedures to be used in establishing a diagnosis of diphtheria in an adult.
4. Give a procedure for the cultivation, isolation and the complete identification of *Neisseria intracellularis* (meningococcus).
5. Name one type of hypersensitivity and explain the reaction with regard to antigen and antibody.

(Answer any 5 questions)

1. Mention at least one significant contribution of each of the following men to the field of bacteriology:
  - (a) Edward Jenner
  - (b) Louis Pasteur
  - (c) Robert Koch
  - (d) Paul Ehrlich
  - (e) Walter Reed
2. Distinguish between the following terms:
  - (a) Autotrophic and Heterotrophic
  - (b) Saprophytic and Parasitic
  - (c) Aërobic and Anaërobic
  - (d) Psychrophilic and Thermophilic
3.
  - (a) To what class of the plant kingdom do bacteria belong?
  - (b) What order in this class constitutes true bacteria?
  - (c) Name three families of this order.
  - (d) Describe the morphological characteristics of each of these families.
4.
  - (a) Name five “portals of entry” for disease germs into the human body.
  - (b) Mention one disease whose causal agent enters through each of these portals.
5.
  - (a) Distinguish between antigen and antibody.
  - (b) Name five kinds of antigen-antibody reactions.
  - (c) Give an example of each of the reactions mentioned under 5(b).
6. For each of the following tests tell:
  - (a) For what disease it is used.
  - (b) Whether it is a diagnostic or susceptibility test.
  - (c) What kind of antigen-antibody reaction is involved.
    - 1) Wassermann, 2) Widal, 3) Kahn, 4) Schick, and 5) Dick.

Answer in table form as follows:

: Name of Test :	Disease :	Diagnostic or Susceptibility :	Kind of Reaction :
: a) Wassermann :	:	:	:
: b) :	:	:	:

7. In parallel columns compare active and passive artificial immunity as to:
- Source of antibody that produces the immunity
  - Whether antigen or antibody is administered
  - Duration of the immunity
  - Time required before onset of immunity
  - Whether it is primarily curative or preventive

## 21 GLOSSARY

See also glossaries with Chapters 1 and 2.

**agglutinin** an antibody found in immune serum which causes clumping when added to a homogeneous suspension of the micro-organism for which it is specific.

**alexin** a term synonymous with complement.

**amboceptor** see sensitizer.

**amphitrichous** provided with tufts of flagella at each end.

**anaphylaxis** an extreme state of allergy, hypersusceptibility, protein sensitization.

**antibiotic** destructive to life.

**antibody** a substance produced in an animal body against an antigen; e.g., antitoxin, agglutinin, precipitin, lysin, and opsonin.

**antigen** a substance which may induce antibody formation—a foreign protein.

**antitoxin** a substance found in body fluids, specifically antagonistic to a toxin.

**atrichous** without flagella.

autoclave an instrument for sterilization with steam under pressure.

**autolysin** a substance present in an organism which is capable of disintegrating that organism.

**autotrophic** requiring neither organic carbon nor organic nitrogen, but able to build both carbohydrates and proteins without them.

**bacteria** (L. little rods) the Schizomycetes or fission fungi. Minute unicellular organisms which multiply by division and which have no chlorophyll.

**bactericide** an agent which destroys bacteria.

**bacteriemia** bacteria in the blood (in appreciable amount). bacteriolysin an antibody which destroys bacteria.

**bacteriostatic** an agent which prevents bacterial growth.

**B.C.G.** Bacillus Calmette-Guérin, specially attenuated tubercle

bacilli and/or a vaccine therefrom.

**Bordet-Gengou phenomenon** same as complement fixation, catalase an enzyme which decomposes hydrogen peroxide liberating free oxygen.

**commensal** an organism capable of independent existence but which depends partially upon a host without benefit or harm to that host; a facultative parasite.

**complement** Thermolabile, ferment-like body normally present in blood serum which plays a significant role in lysis where it is fastened by an amboceptor on the cell to be dissolved. (Alexin is the preferred term.)

**conidia** asexual spores.

**cytolysis** dissolution of cells.

**Dick test** scarlet fever susceptibility test, based on the Schultz-Charlton blanching phenomenon.

**Donovan bodies** inclusion bodies in Kala-azar.

**drug fast** resistant to the usual action of drugs.

**endotoxin** a toxin within a bacterial cell liberated only after bacteriolysis.

**exotoxin** a toxin excreted by a bacterial cell.

**flagella** whip-like processes for motility.

**Forssman antigen** a partial or heterophile antigen; must be completed to act.

**Frei test** a diagnostic skin test for lymphogranuloma inguinale. Intracutaneous injection of heated pus, or brain of an infected mouse, results in an inflammatory necrotic area.

**gamete** mature germ cell—unfertilized ovum or mature sperm.

**Guarnieri bodies** inclusion bodies in the cells of affected tissues in variola and vaccinia regarded as a product of cell reaction to the virus.

**gymnobacterium** one which depends for movement upon mechanisms other than flagella.

**H-antigen** antigen from the flagella of an organism as contrasted with the O-antigen from non-flagellate bacterial bodies.

**hemolysin** an agent which dissolves hemoglobin or which destroys erythrocytes.

**hepten (haptane)** partial antigens. They do not, in an isolated state, act as functional antigens, but when united with any antigenic protein endow it with their own chemical specificity.

**heterophile antibody** an antibody having an affinity for more than the one antigen for which it is specific. One is found at the height of infectious mononucleosis. Detected by agglutination of sheep cells.

**heterotrophic** dependent upon organic carbon and organic nitrogen. See autotrophic.

**inclusion bodies** intracellular corpuscles characteristically found with various virus diseases and parasitic infestations.

**mallein** concentrated glycerol broth in which glanders bacillus has been grown. See *Malleomyces mallei*. Prepared like tuberculin.

**MLD** minimum lethal dose.

**monomorphism** idea that bacteria are rigidly fixed, immutable species.

**mordant** a substance which serves as a chemical bond between a dye and the material to be dyed, thus creating a “fast color.”

**mycelia** a meshwork of thread. Typical mold formation.

**Neisser-Wechberg phenomenon** although bacteriolysis takes place at an optimum concentration of immune serum, either too little or too much immune serum fails to cause bacteriolysis.

**Neufeld reaction or typing** A method of typing pneumococci.

**O-antigen** see H-antigen.

**oligomorphism** idea that organisms pass through only a few forms.

**opsonin**—a “seasoning” an antibody which enhances phagocytosis.

**oxidase test** a test to demonstrate Neisseria colonies on a mixed plate.

**parasite** an organism living at the expense of another.

**peritrichous** having flagella around the periphery.

**Pfeiffer’s phenomenon** bacteriolysis of *Vibrio cholera* in an immunized guinea-pig.

**pleomorphism** idea that bacteria pass through many forms.

**polyvalent vaccine** a vaccine prepared from several strains of a species.

**precipitin** an antibody which causes precipitation of the bacteria in question.

**ptomaine** a product of protein decomposition, sometimes toxic.

**Quellung reaction** capsular swelling used in typing pneumococci.

**receptor** a “mouth” of a tissue cell. See Ehrlich’s theory under Immunity.

**saprophyte** an organism living only on dead or dying organic matter.

**Schick test** a diphtheria susceptibility test based on an intracutaneous injection of toxin.

**Schultz-Charlton blanching phenomenon** the bright rash of a scarlet fever patient fades away completely (blanches) about 6-8 hours after injection with 1 cc. of convalescent serum. Serum from a patient with the active scarlet fever lacks this blanching power.

**sensitizer** a substance supposedly found in blood serum, serving as one of the active elements in cytotoxicity, the other element being the complement. It serves to connect the invading cell with the complement. (Same as amboceptor.)

**septicemia** a condition with poisons (toxins) in the blood, somatic antigen same as O-antigen.

**stab culture** a culture of anaerobic organisms prepared by a deep stab carrying the inoculum deep into the solid medium to exclude air.

**symbiosis** the living together or close association of two dissimilar organisms.

**T.A.B.** a vaccine of typhoid, paratyphoid A and B organisms. thermal death point that temperature needed to kill a young culture in ten minutes.

**thermolabile** destroyed by temperatures of 56 degrees C. thermophilic growing best at 45 degrees C. but tolerating temperatures to 65 degrees C.

**thermostabile** unaltered by temperatures of 55 degrees C. titer highest dilution in which serological reaction occurs, toxin (Gr. poison) any poison produced by bacterial action (including bacterial degeneration). Soluble and thermostabile. toxin-antitoxin a mixture of both, the toxin of diphtheria being about 85% neutralized.

**toxoid** a tempered toxin, usually treated with formalin. trichobacterium one which has flagella, a flagellate, tuberculin a concentrated filtrate of a broth culture of tubercle bacilli. Twort-d’Herelle phenomenon that of the bacteriophage.

**univalent vaccine** a vaccine containing only one variety of an organism in pure culture, vaccination inoculation with a virus or bacterium with intent to develop protective reactions.

**vaccine** (L. cow) a bacterial preparation supposedly capable of producing active immunization by antibody formation.

**vi-antigen** Vi for virulent; a distinct antigen found with virulent strains (typhoid). See H-antigen.

**Vi-antigen vector**

**virulence** disease-inciting ability.

**virus** (L. poison) a living virulent cause of disease as distinguished from toxins and vaccines. Some bacteria are unfilterable viruses, while many filterable viruses, so-called, are non-pathogenic and hence not viruses at all. Filterable viruses may not even be living. Smallpox vaccine, however, carries a living virus! In common usage the word virus, however, refers to a filterable virus. In order that biological classification may include the non-pathogens, Luria (“Viruses—



1950,” Delbrück, Caltech Bookstore.) defines virus as “an exogenous sub-microscopic unit capable of multiplication only inside specific cells.”

**Weil-Felix reaction** an agglutination reaction diagnostic for Rickettsiae by agglutination of *Proteus vulgaris* X19.

**Widal reaction** Gruber-Widal reaction is a diagnostic agglutination for typhoid.

**zymogens** micro-organisms active in fermentation; acting as enzymes.

## 22 SUMMARY

Anyone who has studied the first twenty chapters of this outline on conventional bacteriology will have many questions in mind. It is obvious that the very numerous inconsistencies make bacteriology much more an art than a science. Where each rule has more exceptions than conformities the “rules” must be seriously questioned. A science is built upon organized knowledge. New findings are fitted into the general plan of previous findings. When inconsistencies become numerous the basic hypothesis needs critical examination. Perhaps the accepted axioms are faulty. This frequently requires some drastic overhauling of fundamental concepts. Ptolemaic cosmology was replaced by that of Copernicus. The phlogiston theory was scrapped when Lavoisier proved the oxygenation basis of combustion. It is not a disgrace to discard an untenable theory just because of sentimental tradition, but it is criminal to perpetuate an erroneous tradition especially when it results in frequent crippling injuries.

The second section of this book was written with the express purpose of examining the many inconsistencies of conventional bacteriology and of the possible alternative fundamental concepts which may be much more consistent. If, for example, we find a basic law or axiom which will cover all or most of the observed phenomena and have few or no exceptions, then we must adopt that new law and scrap the faulty old one. Fortunately there are many bacteriologists who are not afraid of the truth. These true scientists are willing to put their own ideas to test every day. When a new and better idea arises it will replace the old inadequate concept. Several such scientists are quoted and lauded in this book. From their collective works arise new concepts which the authors find to be much more factual and fruitful than the conventional dogma.

The authors are not setting themselves up as final authorities on the subject, but are making an evaluation of the old and of the new. They are recommending the concepts which are more comprehensive and which have fewer inconsistencies. As still better concepts arise, the authors will not hesitate to discard their present ideas. They do, however, leave the material in this book as a challenge. They replace with better material than that which they tear down and invite still more comprehensive concepts of bacteriology. Show them something better and they will gladly follow the light of truth in further progress.

With thorough familiarity of the material included in the first section of the book one will have no trouble studying and understanding the next section. Unless the preceding material is familiar, return and study, because it is a necessary prerequisite to understanding the evaluations set forth from here forward. With comprehending and critical minds let us proceed with the studies.

## **II: The French Revolution**

**from Béchamp to Tissot**

## 23 INTRODUCTION

The late Dr. J. Tissot was Honorary Professor of General Physiology, National Museum of Natural History, Paris, France. His death occurred in June, 1950. He was a scientist in the fullest sense of the word. For several years he occupied himself with a series of cancer studies in collaboration with medical colleagues. He did not allow popular dogma to influence his interpretation of findings; he simply investigated, goaded on with the eternal question: Why? Preliminary findings were published in 1926, when he had gathered enough new material to warrant a book. For another ten years he continued his researches on the constitution of animal and vegetable organisms. During this period he made discoveries closely paralleling those of Béchamp. Further extensive research over the next twenty years confirmed Béchamp's findings and elaborated upon them with technics more refined than any available to Béchamp.

These revolutionary findings of Tissot have been published in French under the title *Constitution des Organismes Animaux et Végétaux, Causes des Maladies Qui les Atteignent*. They are published in three separate volumes, each with an accompanying catalog of photomicrographs which clearly illustrate each point. Since the last volume went to press in 1946, it is definitely not outdated. As the work has not yet been published in the English language, it is necessary that we present an extensive analysis of it in order to show the need for a revision of bacteriology. We proceed, therefore, with the salient points of this vast generalization.

As biologists, we are fully aware of broad biological problems and concepts. As chiropractors, we are concerned primarily with human welfare, and hence may seem to speak with an anthropocentric orientation, but this is merely a matter of emphasis.

We must remember that the "infectious" process is universal. There are "germs" everywhere, "germs" in every process. Where there is life there are "germs." They are absolutely essential to life as we know it. (Notre Dame University experiments questioning this will be discussed in connection with "The Biont Cycle.") In the very ground we walk on there are from 10,000,000 to 100,000,000 micro-organisms per gram of soil, and that is only about one quarter of a thimbleful of soil. Without "germs" we would have no cheese, cider, or any fermented beverages. Rubbish would not rot and plants would not grow. Without the "germs" to break down the material to simple forms there would be no vegetation on this earth, and we would not eat. Of the many varieties of "germs" over ninety percent are beneficial, and many are absolutely vital to life. Only a few ever cause any trouble to man, and the manner whereby this occurs is described in this book. What are "germs"? We believe that we have the answer to that question.

We find that there is no ultimate distinction between plant and animal organisms. Still more simply we find that there is no fine distinction between organic and inorganic matter—no ultimate division between living and non-living material. Size cannot be a differentiating criterion. The very organism that on one occasion passes through a filter may later grow so large as to filter out. Rod-shaped bacteria easily become cocci. Mutation and variation lead many eminent bacteriologists to say that all bacteria are simply variants of *Bact. Coli*.

As we go through this book together, it becomes obvious to us why some persons maintain that there is only one disease (or very few diseases). McDonagh of

England has long been teaching a unitary theory of disease and conducting a successful practice based thereon.

These few remarks make us wonder whether perhaps we “know” too many things that are wrong, and whether we should not consider the new findings and remodel old ideas to conform to the truth.

Dr. Tissot spent nearly thirty years of research in this one field. His findings are such as to revise fundamental concepts of biology. We must begin with a clear mind and be willing to investigate with him without prejudices. Most persons are inclined to scoff at revolutionary changes in basic ideology, but, as we repeat and verify his experiments, we see that we are long overdue for an overhauling of ideas. In science, a single theory which unifies a vast number of phenomena is to be preferred to limited theories inconsistent with each other.

The basic building units of both animal and vegetable cells are identical living entities.

Similarities—Tissot (correctly) demonstrated that a cereal culture spontaneously develops structures which resemble mycelia and conidia. Although cells of each species of cereal are distinctly different, they are composed of the same living subcellular units or bionts.

Differences—Béchamp (correctly) demonstrated, however, that there are vast differences both in the cells and in the cellular products of all animals—in the enzymes, for example.

These contributions by Béchamp and Tissot are very important for both biology in general and bacteriology in particular.

## 25 STRUCTURE

Every cell (both animal and vegetable) is composed of just two types of living units, knobbed rods and granules, both demonstrated by Tissot. These two subcellular components, or intra-cellular bionts, may be reduced ultimately to simply one living entity, the microzyma of Béchamp. When we consider that Hofmeister has estimated that there are 200,000 billion molecules in a cell (a liver cell, for example) we get some notion of the range of possibilities at the molecular level.

Extensive studies of normal animal and vegetable cells show that the two subcellular, primal components are both capable of independent existence, growth, and multiplication. The first is a structural component, many of which are joined to form the stroma, the fibrous mesh of all protoplasm. This mesh is constructed out of “sticks” of varying lengths with knobbed ends. The union of several chromophilic knobs is easily seen. They have been variously named, but usually are called simply chromophilic granules. The free, mobile mitochondria often described are simply artefacts produced by improper preparation of the specimen, which destroys and clumps several of the normal sticks into large chunks. The normal “dumb-bell” units (so named from their shape) have a typical formation and growth which are everywhere maintained. As such they form the real structural component of all cells. A diagram of this microscopic structure is included in our section on Tissot’s work at the end of the chapter on Cellular Pathology.

## 26 FUNCTION OF THE “DUMB-BELLS”

The knobbed sticks (“dumb-bells” of Tissot) form a reticular framework throughout both the nucleus and the cytoplasm of the cell. This framework (linin) may be thought of as somewhat resembling a three-dimensional spider web. In the tissue fluid within the interspaces of this stroma are the chemically active granular microzymas. Many stoichio-chemical changes and changes in colloid chemistry occur within the cell. Some molecules are large:

Albumin,  $C_{1428}H_{2244}O_{462}S_{14}$ . There are more than 1000 enzymes in every cell.

### The Granular Biont

The second subcellular, primal unit is a granule, free and mobile, having Brownian movement. It is the active agent of all enzymes of the body and is the functional component of all the body fluids and secretions. Hence the “dumb-bell” unit is the structural agent of all cells, while the granule is the chemist of the cells. There is a possibility that the “dumb-bell” unit is a more advanced stage of the granule; it possesses a stalk, and a new granule grows at the tip of stalk. In this case there is but one primal life form as envisaged by Béchamp, called by him the microzyma, but it functions in the two elementary forms, the stick and the granule, which perform the basic work of structure and function. They are the builder and the chemist. All cells are composed exclusively of these two formed and viable units.

## 27 CELL DIVISION

*Mitosis.* The single fertilized cell from which the adult organism develops is composed of nucleus and cytoplasm with a separating membrane. As the cell grows and prepares to divide, the “dumb-bell” units in the nucleus which are responsible for that function line up as several filaments laid side by side to form the commonly known chromosomes. Most of the chromosomes show the characteristic form of “dumb-bells” at their tips as the usual “satellites.” The “dumb-bell” units multiply and thicken the chromosome until it appears to split longitudinally. This stage is followed by formation of two nuclei and then of two adult cells. Basically all this is effected by multiplication of the “dumb-bell” units and granules with their precise concerted action in this special work of mitosis.

*Amitosis* (budding). In addition to the mitotic method of cell multiplication, there is another routine followed by many of the embryonic cells and their grown-up relatives, the fibroblasts. Their increase in numbers is accomplished by sending out a filament upon which grows a knob. This little knob contains both the “dumbbell” sticks and the microzoma granules which are the fundamental life forms. By increasing these subcellular units the new knob grows up to form a new cell. This is a process quite common to vegetable cell multiplication and is known as budding. The animal cells have both means of multiplication, splitting and budding.

Tissot showed that connective tissues are developed directly from the granules, which are free-living. The details are given in connection with the discussion of platelets. The bionts are really “Life’s Primal Architects” in the fullest sense.



## 28 SPECIALIZATION

*All cells secrete.* The best known secretions are

- enzymes and diastases
- hormones
- blood plasma
- cerebro-spinal fluid
- milk
- saliva
- perspiration
- urine
- pus
- toxins
- antitoxins and other antibodies

All the secretions are produced out of and by specialized intracellular bionts, and all the secretions contain numbers of these living microzymas, which, if filtered out, inactivated, or killed, leave the secretion impotent.

Each of the cells in the body is specialized to a certain degree and fulfills the specific function of the tissue. This implementation is dependent upon the granule, which also provides specialization of enzymic function characteristic of each area. The little granules are the active agents, the basic chemists, of all the digestive juices; they are the active agents of blood coagulation. In fact, the blood platelets are just small clumps of the granules. In the clumps their action is diminished because their exposed surface is reduced. When they are scattered out as free granules, they rapidly hydrolyze the prothrombin (which is a combination of lecithins) and set free the fatty acids. These in turn neutralize the sodium portion of the fibrinogen, thus reducing its solubility. Hence there is a precipitation with the formation of a web of fibrin which entangles the granules.

The active granules can be isolated, and when they are removed from any of the enzymes, the enzymic action is lost. When they are taken from the blood, it does not coagulate. When they are removed from milk, it does not clot to form a curd. When the granules are removed from saliva and the other digestive juices, they lose their enzymic action. When the granules are removed from toxins, they lose their antigenic properties and become totally impotent. The same is true of antitoxins. They are utterly functionless without their granules. Without the little chemists there is no function of secretions. Their functions vary with their environment, but they are basically the same chemist wherever found and whatever they do. Further discussion of the variation of function will be dealt with later.

The preceding material has been devoted exclusively to normal healthy organisms, their structure and their function. The essential point is that all cells, both animal and vegetable, are composed of not many kinds but only two subcellular units, one the structural unit and the other the chemical activator. These are the "dumbbell" sticks which form the framework of all cytoplasm and nucleus, and the tiny granules that are present in all the normal cells and their products.

## 29 PATHOGENESIS

The beginnings of disease are the beginnings of alterations of the normal state of harmony with the environment. Actually, then, the total form and function of all the factors of both heredity and environment will determine the nature of the resultant disease process on the one hand and of the state of health on the other. Since a thorough discussion of this matter would take us too far afield, we must consider here only the disease processes in their relation to bacteriology, which is the theme of Tissot's work. Some disease processes are referred to as specific, some as non-specific; some constitutional, some functional; some acute, some chronic; some reversible, some irreversible; some communicable, some not. Actually the names of the various diseases hardly ever give any clue to their real cause, but are merely descriptive in a symptomatic way and usually refer to the terminal stages rather than to the early beginnings. It will remain for some future genius-philosopher to reclassify the pathologies as to their causes and actual relations. Let us proceed now with some specific details of pathogenesis.

### 30 BACTERIOLOGY

In response to variation in environmental influence there are many characteristics which the intracellular bionts may assume. The accompanying diagram summarizes the possibilities.

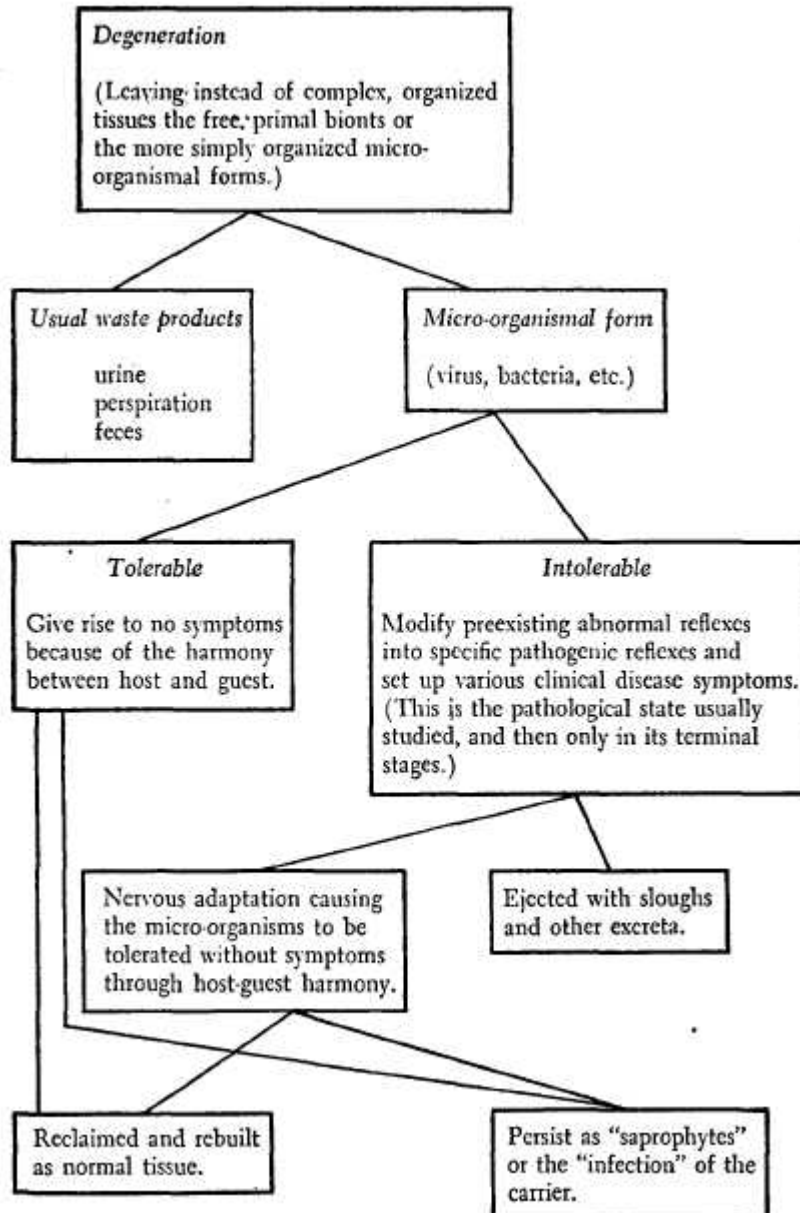
If a pathogenic micro-organism is present as a part of that stirring experience which initiates the disease process, then the metamorphosis of intracellular bionts will tend toward assuming form and function similar to that of the "invader."

In the course of the disease process the bionts may be modified and go through the following stages: clumping, sprouting, spore formation, virus formation, development of any bacterial form, and/or interchange of forms. Tissot studied and described these stages at length.

The clumping of the bacteria produces the leukocytic forms described elsewhere, which under favorable conditions begin to sprout and send out filaments. These clumps are the germinal centers of the mold forms frequently seen in laboratories.

These germinal centers send out filaments or mycelia which usually fragment soon into spores and various bacterial forms, or, if the nervous system has been sufficiently normalized, they fragment into normal granules and are then used as building material for normal, healthy body cells.

If the fragments are abnormal and submicroscopic in size, they are called virus forms. Jordan and other bacteriological authorities state that the filterable forms of bacteria are well known, because under certain conditions the bacteria do break down to granular form and are capable of re-integration to again become typical bacteria.



All standard bacteriology texts devote considerable space to admitting that cocci and bacilli are interchangeable; they devote a section to the non-pathogenic forms of each bacterium and almost admit that there is only one bacterium with many disguises.

Response to an injurious experience causes a modification of the cells and of their various components, and various degenerative changes occur. As the cells degenerate and disintegrate, they set free the two primal units of life and these in turn are capable of independent existence. This degeneration and the consequent perversion and modification of the two primal units thus gives us a real insight into pathological processes. Tissot studied the various modified forms intensively and found that they usually take on a bacterial nature. These new, perverted forms of the granules are seen as cocci of various types: staphylococci, streptococci, micrococci, diplococci, pneumococci, and others; as rod-shaped organisms known as the colon bacillus, tetanus bacillus, proteus vulgaris, the various clostridia found with gangrene, and other rods.

These are now modified forms acting as free agents. In pathological tissue preparations and in pure cultures they are seen gradually to enter a clumping routine. As they clump together, they fuse into a single unit. This fused unit of a bacterial clump then sprouts and emits long filaments, until it becomes a typical mold. Such molds are commonly seen in laboratory examinations, but they are discounted as contaminants. Hence the pathologists and bacteriologists are naively missing the truth, because they have learned too many untruths. It is not only what we do not know that hurts us; it is the many things we do “know” that are not true that damage our efficiency and make us do foolish things. The sad result is that many persons are killed by others who are sincerely trying to help.

In order to demonstrate clearly this metamorphosis of typical bacteria into molds and the reverse, it may help to discuss the technics that Tissot used. Molds ordinarily flourish at twenty to twenty-five degrees centigrade on rather dry media. Bacteria (pathogens) “prefer” thirty-seven degrees and fluid medium. Hence a gradual lowering of temperature and a drying of the solid medium will encourage the change from bacterial form to mold form. Seeding the spores of any of the vegetable molds on a suitable fluid medium will lead rapidly to a growth of mycelia. As these mycelia (filaments) proliferate, the temperature can be gradually raised to approach thirty-seven degrees.

After the typical molds have developed, the tips of the filaments at a certain stage of their development break off as tiny spores which are identical with the perverted granules of the degenerated cells. At some stages they break off as matured bacterial forms, various cocci or bacilli. The major feature of this startling picture is the fused clump which is the germinal center of the mold: It is identical with the usual mononuclear leukocyte. Many observations have shown that pure bacterial cultures undergo this same type of bacterial clumping, a fusion into a mononuclear “cell” and subsequent mold formation. The last of the bacteria approaching fusion into the mass give it the appearance of phagocytosis. This indicates that phagocytosis is really not a defense mechanism of bacterial destruction, but a stage of maturation of the subcellular primal life forms after the cellular disintegration has occurred which released them temporarily as free agents. All the other antibodies, the antitoxins, agglutinins, opsonins, and so forth, are simply phases in the maturation of bacterial forms into molds. Hence the body is not combatting the bacteria and attempting to kill the germs, but rather is speeding their maturation into a more tolerable form from which a subsequent reclamation is made to return the “delinquent” granules to a normal useful form.

Tissot demonstrated a similarity between animal tissue metamorphosis and vegetable tissue metamorphosis. In their normal states the cellular components of animal and vegetable tissue are distinctly different as units, and they carry on different functions. They are, however, basically the same bionts, either the sticks or the granules. They become specialized and adapted to their different environments. It is this specialization that makes them distinctly animal or vegetable tissues. Since they are basically the same bionts, they can metamorphose into identical bacterial forms. This is discussed at length by Tissot in one section of his work describing how he developed typical diphtheria pathology and the typical Klebs-Löffler bacillus from a few grains of bacteriologically sterile barley (chemically sterilized surface).

Tissot demonstrated the ability of bionts taken from either an animal tissue culture or a cereal culture to initiate an identical metamorphosis in a culture of the other. He performed experiments showing that aseptic biopsies of normally healthy

animal tissues metamorphose directly into typical molds which, if they are kept under optimum conditions, are vegetable in nature. If these aseptic biopsies are kept more moist and warmer, then the normal healthy animal tissues metamorphose directly into typical bacteria. Remember that this happens by degeneration of tissue and perversion of the bionts, the subcellular living units, in response to a “perversion” of their environment.

One caution is this: Do not consider all bacteria as pathogens. Over ninety per cent are quite harmless, and the majority of them are absolutely indispensable in the overall ecology. Without them there would be no life as we know it. Even plants cannot use free nitrogen from the air without bacterial help. They cannot use manure, until the bacteria break it down for them. Bacteriologically sterile soil will not support plant life. Tissot says that the bionts are of a bacterial nature, although they are not the usual familiar bacteria of laboratory studies, At any rate, without them there would be no action of the body enzymes; in fact there would be no body processes at all. Without the bionts there would be no clotting of blood and no healing of wounds (as discussed later).

In case there is a modification of the bionts into a typical bacterial form which can be recognized and named as a pathogenic organism, still no recognizable disease will develop unless the nervous system of the host is perverted. “The power of a microbe to produce morbid effects or changes depends, therefore, as much upon the nature of the host as upon its own characteristics.” (*General Bacteriology*—Jordan and Burrows) Much more will be said upon this subject when we come to discuss etiology.

Extensive studies of the bacteria show definitely that there are no fixed species. The cocci become bacilli and spirilla, and vice versa. Streptococci and pneumococci interchange. All bacteria either acquire or lose virulence depending upon their environment. Bacteria change to molds and vice versa, in response to adequate environmental stimulus. Furthermore, they can resolve into their smallest form, a sub-microscopic granule. The bacterial “toxins” (which were thought to be entirely free of particles) are not really homogeneous solutions at all, but actually contain the tiny granular stages of the bacteria which broke down to form them.

There is another almost identical type of fluid which, when passed through a porcelain filter, carries along with it an ultra-microscopic form of life. This is known as a filterable virus in contradistinction to the many forms of larger viruses, known as cocci and bacilli, which do not pass through fine filters. These tiny granular forms of bacteria which pass through a filter may reintegrate or grow up to form again microscopic bacteria, once the environment is favorable for such response.

Jordan writes that *in vitro* the azotobacter and the rhizobiae present a rather constant appearance of “fixed” species, while in the nodules of leguminous roots they are found in many varied forms entirely different from the “typical” laboratory forms. Which then is to be considered the normal typical form, the natural or the artificial? Jordan shows slides to illustrate vibrio cholera in many forms including cocci, short rods, long and short spirals, chains of rods, and long filaments.

“Sudden changes in bacterial cultures do occur; often these changes apparently overstep boundaries of ‘species’ and ‘genera’ formerly thought impossible.” “Connected with the variability of bacteria is their remarkable plasticity or adaptability to diverse conditions of life. By a series of inoculations or transfers it is possible to so alter bacteria that qualities originally present are sometimes accentuated, sometimes abolished.” “It may be urged that the ability of cell

fragments to regenerate is no new thing in biology and that it is quite plausible to regard minute filterable forms of cocci or tubercle bacilli as portions of fragmented cells or as gonidia.” (Selections from Jordan)

Bacteriologists in general admit that there is a great deal of variation in form of the same species, but their interpretation is restricted by the Pasteurian dogma of monomorphism. Tissot demonstrates that there is the greatest pleomorphism throughout the bacterial world, and that monomorphism is only another mistaken assumption of Pasteur which has been accepted as an axiom. Stitt, Clough and Clough, eminent authorities, write at length on the Much granules of tuberculosis, minute viable, filterable granules which reintegrate to form again the typical tubercle bacillus. They also discuss at length, the adaptability of Rickettsia. By passage through guinea pigs or rabbits the pathogen changes its characteristics markedly. Then inoculation into mice (after such passage) causes entirely different pathologies from the usual mice problems with “typical” unmodified virus. They even note that the nervous system must first be injured, before the disease can develop.

The environmentally perverted forms of the bionts, whether virus, bacteria, or molds, have their varied size, varied form, and varied function dependent upon their abnormal environment. If the environment is “normalized,” they can be reformed and will take on the characteristics of normal granules. In the human body this reformation is a “re-education” process supervised by a normal nervous system. Remember that even these normal granules, the cell and body fluid chemists, have varied sizes and varied functions depending upon their environment. It is a well-known fact that enzymes and hormones circulate in the blood in an inactive form and assume their characteristic function only at the destination where “expectant” cells await specific “supplies.” The potential local need will provide that particular stimulus which is adequate to activate the hormone or enzyme in the characteristic fashion. The type of reaction depends definitely upon the environment.

There are many examples, but the simplest one is this: Adrenalin has no effect on the clotting time of shed blood. However, when injected into the blood stream, it acts as a specific nerve stimulus, and a reflex is activated which appreciably shortens the clotting time. Thus the adrenalin is active only at its destination, and its function is utterly dependent upon the nervous system.

Zondek says, “Apparently the hormones circulate in the blood in an inactive form; not until they reach the organs of their destination, to which, they are attracted by special electric affinities, are they activated and is the intensity of their action adjusted to the right pitch. In no circumstances can hormonal influence be conceived to be constant. It is variable, depending in the highest degree upon the character of the medium in which it acts.” It is well known that the pancreatic juices and others are not active within the glands of origin or their ducts, but become active only at their destination in the gut. Tissot showed definitely that the granules are fundamentally the same in any “normal” environment, but by changes of that environment they can metamorphose into various bacterial forms or toxins thereof. They can revert and become “normal” granules again, having the normal characteristics which the words imply, “normal granules in a normal environment.” There is a constant interrelationship and interdependence between agent and environment.

### 31 CRYSTALLIZATION

Observe, if you will, that filterable viruses retain the ability to initiate the same type of process with which they were associated, even after they have been crystallized. And, at the same time, the crystals of what were formerly certain filterable viruses can, in an appropriate, abnormal environment cause the metamorphosis of intracellular bionts into a form identical with their own form previous to crystallization. "There seemed to be no escape from the conclusion that the virus must be a living organism capable of multiplication, or if not that, then some inanimate agent capable of self-reproduction. The close association with, and dependence upon, the living cell (to multiply) must be reckoned one of the most striking properties of the viruses." "It may be pointed out, however, that the terms 'living' and 'dead' are meaningless in a world of near molecular dimensions." "Their properties are unaffected by repeated 'recrystallization!'" (Excerpts from Jordan) These points are brought up so that we can more fully evaluate the bacteria, the viruses, and their interrelationship. This evaluation will be more fully discussed under "The Biont Cycle."



## 32 INFECTION

Assuming that the material just considered under bacteriology is well understood, it is an obvious conclusion that the term "infection" has taken on an entirely new meaning. The mere presence of pathogenic micro-organisms in a pathological animal or human patient does not mean that these pathogens are the first cause of the disease, nor does it mean that they have been initially received by infection from some pre-existing patient with the same disease. The pathology and the pathogens can be and frequently are totally endogenous. We could say that the pathogenic bacteria are in these cases totally spontaneous in that there is no outside source. The word "spontaneous," however, implies that there is no known outside cause, and that the pathological degeneration is automatic. That would be a fatalistic conclusion. Since there is a known external cause, adverse environmental factors resulting in various types of nerve trauma, we should use the word endogenous. Endogenous means that the situation, here a pathology with pathogens, began materially within the nervous system of the animals, and not as an infection.

This endogenous pathology raises a serious question as to the fitness of the words "infection" and "infectious." Several of the so-called infectious diseases are totally endogenous every time and should, therefore, be classed as degenerative diseases, even though pathogenic micro-organisms are usually found as end-products of the degenerative pathology. Many other so-called infectious diseases occur only occasionally as totally endogenous processes. This group usually occurs according to the following second pattern:

First there is a definite cellular degeneration, as in the beginning of the totally endogenous pathology. The cellular debris metamorphoses into various virus and bacterial forms. At this transition phase there can be a specific nervous guidance in the form of pathogenic reflexes, initiated in this case by a specific antigen. Hence if the patient, already half sick, contacts another patient with an abundance of specific pathogens, there can be an infection in which the pathogens from the outside source act as a specific antigen—a specific nerve stimulus. This antigenic nerve stimulus will modify the already abnormal reflexes to become specific pathogenic reflexes. The pathogenic reflexes in turn influence the pre-existing degeneration of tissue so that the bionts metamorphose to become pathogenic micro-organisms similar to or indistinguishable from the antigen. This type of pathology, therefore, has a totally endogenous beginning modified by an exogenous process. The end result of this mixed etiology may be indistinguishable from the end result of a totally endogenous etiology. Anterior poliomyelitis is a classic example of a disease which can be either endogenous or mixed. The revolutionary fact is that all these diseases, either totally endogenous or mixed endo-exogenous have totally endogenous beginnings, which means that no disease can be called wholly infectious.

Further details of this subject are found under "Cellular Pathology." The practical significance, however, is continued here under "Resistance."

### 33 RESISTANCE

Speransky has shown that regardless of “health,” an infectious process is very readily inflicted in the laboratory in the presence of what is discussed later in this book as “Double Trouble.” This double trouble is the double etiology just considered under “Infection,” wherein there must be two distinct traumata to the nervous system in order to establish a pathology. Both may be non-specific traumata resulting in a totally endogenous disease, or one may be non-specific and the other a specific antigenic nerve trauma resulting in the mixed endo-exogenous disease process. At times this first trauma, this “lowered resistance,” is established only an instant before a bacterial antigen is added to become the second injury of the “double trouble” and the specific nerve stimulus which differentiates the final symptoms. A classic example is an inoculation. The needle puncture is obviously a nerve trauma which affects the entire nervous system. The second and specific insult to the nervous system is the antigen. There is no immunity against such an experience.

Vaccinia is inflicted in 100% of attempts—barring technical error. The problem is very much confused by the vague terms, “health” and “resistance.” Of course, mucous and serous membranes or skin may be depleted, enervated, or abraded, in which case infection may readily occur in the presence of a pathogenic agent, which in this case acts as the second nerve trauma—the specific exogenous antigen. This accounts for the exogenous, non-laboratory disease, usually called infectious or contagious. Is the endogenous disease a true “infection”? No, in that there is no antigen from an outside source. The pathogenic agents here are only incidental end products of the endogenous pathology.

“A. G. Molotkov has already accumulated a large amount of material on the treatment of tuberculous ulcers of the tongue by neurotomy of n. glossopharyngeus. A case stands out especially clearly where an extensive and dirty ulcer of the tongue rapidly healed and became covered with epithelium, while isolated tubercles remained visible in the depths of the tissue. For some reason, these tubercles ceased producing additional reactions. Biopsy showed the presence in them of typical tissue elements and microbes which subsequently only gradually disappear. First, the inflammation came to an end, and only after this came the liquidation of the ‘causes’ which had produced it.” (Speransky, p. 255) Tissot showed endless examples of this phenomenon.

There seem to be at least four distinct categories for consideration:

a) The best known phenomenon in which an observed double experience occurs to cause a pathology—as in a dog bite, mosquito bite, vaccination, or exposure to a sick patient by one who is definitely run down and is already half sick. In this class there is the double nerve trauma which makes the pathology an endo-exogenous disease.

b) The Speransky phenomenon, wherein a recognized double nerve trauma is inflicted, both traumata being non-specific in character—no specific exogenous antigen. This was exemplified by causing staphylococcal kidney abscesses with the double trauma of laparotomy and a drop of formalin on an ovary, or by causing typical tubercles with similar double non-specific traumata. This is again the mixed, endo-exogenous type.

c) The Béchamp-Tissot phenomenon, wherein the process is totally endogenous and intracellular. Here again there is no “invasion.” In addition to the many examples recorded by both Béchamp and Tissot, the classic experiment of Servel of producing “spontaneous” bacteria (quoted under “asepsis” with “Pasteurian Dogmas”) belongs here. The “spontaneous” cases of anterior poliomyelitis “generally known but unexplained” (Cecil) are quite typical of this process of definite degeneration. In this type of pathology part or all of the double nerve trauma may be unrecognized, but it is definitely a case of a totally endogenous process. The fact that several of the so-called “infectious” diseases are often in this classification throws further light on the term “infection.”

d) The most common phenomenon of unrecognized first trauma and recognized second trauma. The unrecognized portion may be nose picking, violent sneezing or coughing, or tissue depletion as in vitamin deficiency, which degrades the skin or mucosa so that it just about disintegrates all by itself and is thus susceptible to exogenous agents. Here there is a definite invasion and infection. This phenomenon is again a case of mixed endo-exogenous pathology.

Resistance now becomes a little clearer when we realize that there must be a double nerve trauma before the pathology develops. It is not difficult to see how subluxations so often fit into this picture as the initial nerve trauma which “lowers the resistance” and allows the second trauma to have more immediate effect as the final differentiation of the degeneration. Suppose that a patient does have a subluxation which allows an antigen to initiate an endo-exogenous “infection.” In the chart, “The Biont Cycle” under “Cellular Pathology,” you will see very readily how normalization of the original subluxation may sufficiently stabilize the nervous system that it is able to “abort” the case by a reclamation of the perverted bionts and speed a rapid recovery of health.

In the final analysis the topic of resistance is one of nervous integrity. If the reflexes are perverted and allowed to remain abnormal, then it takes only a single additional trauma to begin a pathology. If the reflexes are maintained at their normal integrity, then it will take “double trouble” to start a pathology. Again that brings us to the word “health,” which can be best defined as a state of mechanical, chemical, and mental balance characterized by excellent adaptation to the environment.

There are other aspects of this subject which require further elaboration in succeeding pages.

## 34 PATHOLOGY (GENERAL)

As the environment of the cell changes, the intracellular activity also changes. Of course there are degrees of change and degrees of response. As long as the basic living units, the bionts, are in harmony with their environment, all is well and the state of health prevails. If the environment becomes too severe and the bionts cannot be well adapted to a harmonious condition, then a state of disease begins and the bionts change. Since they cannot continue living in discord, they become perverted and assume modified forms. As the basic units are perverted, so the entire organism built of myriads of bionts becomes degenerate. The many forms of this degeneration are called disease and have many descriptive names. If an environmental change is sufficiently drastic, the very form and function of the intracellular bionts will change radically, and the cell will decompose. Some types of cell can never be regenerated, if the degree of damage has been sufficiently great.

The term environment includes nutrition and oxygen, drainage, temperature, injurious agents, and anything else which in any way influences cellular well-being. Since each cell has a direct nerve supply (a few have indirect nerve supplies), the nervous system is an informer of distant environmental changes. Actually this is one of the most important parts of cellular environment. Without this intact nervous system only the most primitive form of physiology can exist. Even a starfish has a well developed nervous system. (This matter is discussed more thoroughly under "Special Pathology" and under "Neurology".)

One particularly fascinating and appropriate subject is that of phagocytosis). So long as this mold stage endures, the relative immunity persists. We shall have occasion to return to this subject in connection with acquired immunity. It is, however, something which can and does occasionally misfire, as noted under **Anaphylaxis**.

Eventually the "cells" are fragmented to normal microzymas, unless a more specific product, such as connective tissue fibers, is required, in which case the "cell" takes on the characteristics of a fibroblast, actually becomes one, and builds the fibers. For more specific data read through the section **Platelets and Coagulation**.

### 37 CARRIER

If what has been said under **Recovery** is recalled, then the carrier problem will be clarified. Pathogenic organisms may continue to exist in the “recovered” person. The erstwhile patient has reached a new “normal” by compromise between the host and the micro-organism amounting to mutual tolerance between them. It is a case of adaptation permitting them to live quite happily together for a while. Usually the organisms are modified and reformed to become the normal granules, although occasionally they retain their pathogenic characteristics. A homely illustration is provided by shoes. In this country we are accustomed to wearing shoes, and this practice has become normal for us. In Australia the aborigines are barefooted and have tough feet. If one of them were fitted with shoes, he would be temporarily crippled and worthless in his society. Probably he would throw them away, but he might possibly get used to them and go on wearing them until he became agile on his feet and could again take his place in society. He would then have become a carrier of shoes. He would get along well with them, but if he were to put shoes on another native, then the second person would be crippled. This is because other persons in that society are not adapted to this new condition. Depending on their adaptability they may or may not suffer. So the carrier is an example of an unusual adaptation. For all purposes he is like other folks, but he is carrying something that if forced on another person would possibly cause suffering.

## 38 CELLULAR PATHOLOGY

Cellular pathology here means something quite different from Virchow's term. Virchow maintained that the cell is the ultimate unit of life. Béchamp and Tissot showed that the cell is not ultimate but that the cell is made up of even more primitive units. Tissot showed that either the stroma or the parenchyma of the cell can become diseased: Thus there are two fundamental types of disease process, according to Tissot, as already explained in the section on Pathology. The intracellular aberration of the two subcellular components, the bionts, is a response to abnormal environment and assumes several interesting forms.

Both Béchamp and Tissot demonstrated that under certain conditions the germ is a product, a result, an effect of the disease process: The disease comes first, then the germ from the tissues—a metamorphosis. There need be no “invasion” and no “migration.” Under certain conditions the “infection” is exclusively an endogenous process. The most accurate presentation of this aspect of a complicated biological phenomenon has been achieved by Speransky, but both Speransky and Béchamp must be understood in order to grasp the full significance and the detail of this highly and vitally important phenomenon in its totality. On the other hand, the infection, which always begins as an endogenous, degenerative process (the so-called lowered resistance) may be secondarily exogenous. The lowered resistance is established by a nerve trauma at times occurring shortly before an antigen is added to become the second injury of the “double trouble.” A classic example is an inoculation.

Examples of the totally endogenous process as demonstrated by Speransky include: (1) the formation of typical staphylococcal abscesses complete with bacteria in a kidney as a result of nerve stimulus in the form of a drop of formalin applied to an ovary; (2) the appearance of typical tubercle bacilli of endogenous origin under conditions excluding all means of entry from the outside; and (3) the development of myriads of *treponema pallida* of syphilis within the body as a result of abnormal nerve stimuli. The specific stimuli in each case caused the degeneration of the normal cells in such a way as to develop the “typical” micro-organisms generally believed to be totally responsible for the diseases. Other series of experiments performed by Speransky and by Tissot showed that once the initial degenerative process has started in the body and disease is initiated, it can be further guided by specific nerve stimuli in the form of bacterial inoculation, as antigens (or similar use of their toxins), with the result that the symptoms of the disease are finally altered to fit a pattern typical of that antigenic stimulus. Vaccinia is one of the many instances of exogenous disease.

Remember that Tissot showed that all cells, both animal and vegetable, are composed of the same two primal units. There is, then, no fundamental dividing line between animals and plants. This concept was carried much further by him. In checking the variations of plant cell granules he sterilized the surface of grains of barley. These were aseptically ground to meal and then cultured. They grew a typical mold. The spores of the mold were implanted on a rabbit's cornea, where there then developed a typical false membrane containing the typical diphtheria bacillus. This experiment was suggested because the cultured molds of pure cultures of diphtheria bacillus appeared to be identical with the molds of barley meal. Further checks always showed that the grain would grow molds which transform into organisms identical to *Corynebacterium diphtheriae*, the Klebs-Löffler bacillus of

diphtheria. When the organisms recovered from the cornea implanted with barley mold were sent to the bacteriological laboratory at the nearby medical college for identification, they were pronounced by the bacteriologist in charge to be typical *Corynebacterium diphtheriae*, for they responded characteristically to all the morphological, cultural, and pathogenicity tests. Not only barley, but also wheat and rye developed the typical bacteria and pathology of diphtheria. Hence Tissot concluded that epidemics originate not from contagion, but from eating degenerate cereals. Children never get diphtheria as small infants, while exclusively on the milk diet, but acquire it later, when they start eating cereal. Thus epidemiology should start with sanitation and horticulture, not with inoculation, isolation, fumigation, and disinfection.

Consider now the following quotations from an abstract of the seventh international congress of the International Society for Cell Biology at Yale University (1950). (Their next meeting will be in 1953.) “The report by Dr. Andre Lwoff, head of the Department of Microbial Physiology at the Pasteur Institute in Paris, was one of the highlights.” His studies show that, “a substance normally part of the internal structure of an organism, and causing no harm of any kind [obviously the normal bionts are benign], is metamorphosed by external causes into a virulent virus-like agent that multiplies itself about a hundred fold in a short time and causes complete dissolution of the cell that gave it birth.” He took a “fixed” species of a bacterium (grown from a single cell), “irradiated it with ultraviolet light and placed it in a culture consisting of yeast extract. In less than a half hour all the host bacilli dissolved and were replaced by about ninety bacteriophage per bacterium. The application of the shock of ultraviolet irradiation and the change in its nutritional medium cause the metamorphosis. Starving the bacilli for two hours following their irradiation enabled them to grow normally without dissolution. This indicates that both the ultraviolet rays and the nutrient change are essential to the metamorphosis.” This sounds as though it were lifted bodily from Verner’s *Science and Logic of Chiropractic* under the heading of “double trouble”.

Drs. Billingham and Medawar of Birmingham, England, in experimenting with the cellular heredity of pigmented cells in guinea pigs found that extracts from the black pigmented cells when injected into white cells caused new pigmentation. These suggest “that the cells of an adult mammal owe their inheritable difference to the possession of the different types of particulate, self-reproducing enzyme systems.” This sounds as though they had been studying Tissot’s works on the granular bionts, the microzymas of Béchamp.

Dr. Melnick of the Yale School of Medicine did “studies suggesting that the nucleus of cells of the spinal cord may be the place where polio virus is produced. Dr. Melnick displayed photographs, obtained with the electron microscope, showing the structure of nucleoprotein, in which segments only two millionths of an inch long could be seen clearly. ‘The experimental data we have obtained,’ Dr. Melnick said, ‘suggests that the virus may be manufactured in the nucleus and then released into the cytoplasm where it is stored in higher concentration. The relationship of fatigue to polio may now be seen in a new light.’ “ One might think that Dr. Melnick had been chatting with Dr. Raskin, a Connecticut chiropractor whose studies led him to these same conclusions many years ago.

The extracts quoted serve to demonstrate that the scientists, having been prejudiced by Pasteurian errors, are hitting all around the truth, but never quite arrive. Worse yet, they do not hesitate to ridicule any findings coming from outside

their own clique. In each of the scientific investigations cited they were definitely looking for fixed species of pathogenic organisms, or the seed of a pathogen, without ever once suspecting or admitting that these seeds might be the normal sub-cellular bionts which can be perverted. They will not yet admit that bacteria are simply cellular debris which through perversion has metamorphosed into microorganismal forms composed of the primal bionts.

Since there are only the two subcellular bionts, there are, as might be expected, two forms of cellular “degeneration.”

1. The dumb-bell (haltère) or knobbed stick form (structural biont), when perverted, metamorphoses into tubercle bacillus, mycobacterium leprae, and the organisms found in cancerous growths.

2. The granules (microzymas of Béchamp), fibriniferment or micrococcic forms of *B. coli* (Tissot), when perverted, metamorphose into any of the various cocci, the enteric group of bacilli, the various clostridia (tetanus and gangrene), the various *Corynebacteria* and many others.

Both forms of bionts can and do go through a cycle, parts of which are associated with the following two major phases of disease:

A. Acute (bacterial) phase, in which there are generally symptoms of a recognizable disease in a more or less acute form. There is a fine distinction of terminology here which might be confusing, because many of the diseases progress into a “chronic” stage as far as symptoms are concerned, despite the continued presence of an abundance of bacteria. Tissot uses the terms acute and chronic to designate the developmental stage in the cycle of the bionts rather than to characterize the frequently confusing clinical symptoms of the patient. The forms in the biont cycle can be demonstrated in vivo. Bear in mind that Tissot is using “acute” and “chronic” with relation to phases of development of the bionts and not to the clinical symptoms of an incidental host.

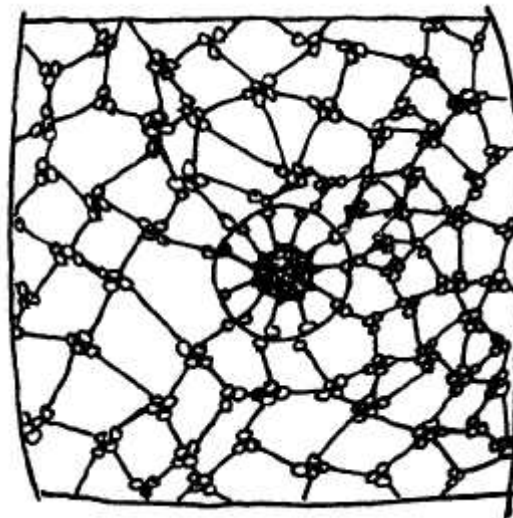


FIG. 22 Constitution of a normal cell. The stroma is enlarged for clarity. The spaces are filled with myriads of granules in the fluid matrix. The Golgi apparatus, Nissl's granules, and the nucleolus are all easily discernible.

B. Chronic (mycelial) phase, in which there is generally a minimum of clinical symptoms, but in which the bionts have always progressed in their developmental cycle to the vegetable, mold phase. In this phase the perverted bionts of the acute



phase have now clumped into germinal centers. Newly introduced bacteria clump rapidly into these germinal centers, giving the appearance of phagocytosis. Hence there are few free bacteria to be found.

Remember that Tissot uses the term “chronic” with reference to the bionts themselves, which can be demonstrated in vitro without a host and without clinical symptoms. Thus it happens that acute bacterial forms are found in some so-called chronic diseases. Actually when these so-called chronic diseases reach the chronic stage, the bacteria clump into the germinal centers of molds, and clinical symptoms almost completely disappear.

### 39 THE BIONT CYCLE

The phases “acute” and “chronic” are only transitional high spots in a cyclical development of bionts. This cycle, which is discussed under Bacteriology, is the same for all the various end-products of pathology and their attendant bacterial by-products. Although there are an infinite number of fine distinctions in these end

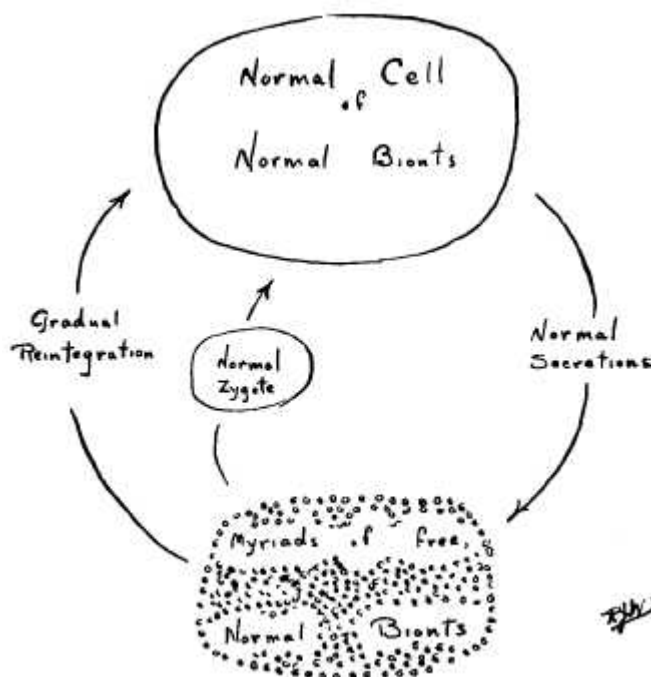


FIG. 23 Normal biont cycle

results (since no two sets of environmental circumstances are identical) all pathology and all co-existent bacteriology come within the one cycle.

With the aid of the accompanying diagrams of the biont cycle we have a simple graphic basis for understanding the interrelationship between the various phases of this cycle. Tissot published a wealth of material to illustrate the abnormal cycle (sketched here in an attempt to help us properly to orient ourselves in bacteriology). Since it is obvious that bacteria are only a stage in the cycle, we see that, unless the entire picture is realized and understood, bacteriology can become a frustrated and a frustrating science. All bacteriologists know that it is difficult to maintain a “fixed” species, and that it can be done only under rigid artificial conditions. To date the bacteriologists have been classifying bacteria on the basis of the hundreds of fine differences in form which occur. Hugh Nicol, a progressive English bacteriologist, writes in his book *Microbes by the Million*:

“There are numerous textbook divisions of bacteria into classes depending upon shape and modes of arrangement, but most of these divisions are based on the older teachings of medical bacteriology which assumed that the behavior of bacteria was so far invariable that they could be grouped into species, as plants are, according to definite descriptions. In the newer branches of bacteriology less reliance is placed upon constancy of bacterial characteristics; hence it seems unnecessary to indulge here in hackneyed explanations of technical terms which I seldom use myself. As a

bacterium undergoes a life cycle both in the soil and in the nodule, it cannot be said to have one special form. They never do anything invariably.

“Good bacteriologists are often content to speak of ‘a short rod,’ and give their bacterium a number or initials. ‘Y.B.’—i.e., ‘yellow bacterium’—is a species or strain which some of my colleagues have kept alive and under observation for years, scorning to give it a pseudo-scientific name.”

This is one reason why the first section of *Rational Bacteriology* does not go into lengthy and boresome details, which are pointless. Familiarity with the general body of material is necessary, but the details (interesting to the bacteriologist, but useless to the general practitioner, medical or chiropractic) are kept separated for use in passing state board examinations. Multiplicity of form characterizes the bacteria, the molds, the normal cells, and the normal free granules. Histology, for example, is a science devoted to the multiplicity of normal cell forms; half the science of physiology is devoted to the various normal secretions and their actions; mycology, to the multiplicity of molds; and pathology, to the perverted, disrupting cells and their debris. One should not, therefore, conclude that because Tissot often speaks in general terms he is belittling these related fields of science. On the contrary, he is enriching their meaning by establishing their relationship with each other.

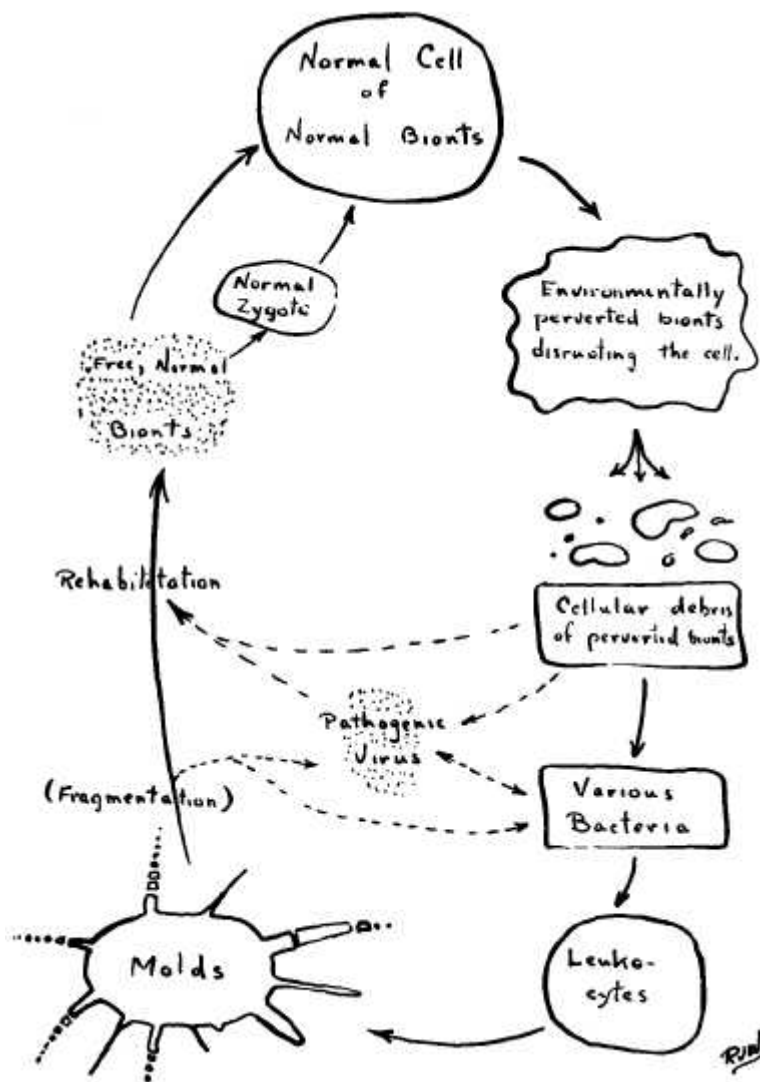


FIG. 24 Abnormal biont cycle

At this point we should mention the Notre Dame experiments on “aseptic” animals. If we keep the biont cycle clearly in mind, these experiments should present no problem. For years bacteriologists have found that animals delivered aseptically by Caesarian section, kept in aseptic cages, and given sterile food and water do not live more than a few days. This made it apparent that contamination by exogenous bacteria is absolutely essential to their lives. For a number of years, however, there has been an experiment going on at Notre Dame University in which laboratory animals have been kept alive for several generations without any exogenous bacterial contamination. Autopsies show that the lymphatic tissue in these animals is almost non-existent. Moreover, the post mortem decay of specimens is delayed.

In view of Tissot’s material and his insistence on the “bacterial nature” of all living beings, we can see that, so long as the animals remain healthy, the normal bionts are present and active throughout their bodies and are able to carry on the normal physiology without difficulty. If, after an animal is killed, the specimen is slow to decay, that is only what we should expect, in that there is only a meager amount of degenerate material present at that time. If the specimen is maintained under optimum conditions—still aseptic—it does, nevertheless, definitely decay after a time, forming “spontaneous” bacteria. Actually then, these experiments are just one more substantiation of Tissot’s work.

## 40 ETIOLOGY

The intracellular biont which has undergone a specific metamorphosis (in situ) becomes a free agent when its cell has decomposed. Coming in contact with a new environment, under certain circumstances and conditions this organism (or its product) is able to initiate the same type of process with which it was itself recently associated and thus induce an identical degeneration perverting other bionts. Thus the first modified biont acts as a “blueprint” pattern for the others to follow. The process is like the addition of one rotten apple to a barrel of good ones. Bacteriologists in general have agreed that the virus multiplies in this way, not by reproduction, but by inducing degenerating cellular components to follow its pattern and become more virus. Thus the modified biont acts as a pattern and induces in other bionts which it is now able to influence the same type of metamorphosis which it has recently experienced.

Tissot has concluded after much study and evaluation of the pneumococci that, “Since pneumococci are NOT found in the alveoli during the initial stages of the disease, but only well after the fever is under way, the pneumococci must be NOT the cause but the consequence of the disease. The studies of chilling in relation to pneumonia made by Wurtz are good. Chilling of the thoracic wall (without bacteria) or trauma to the wall—rib fracture—easily causes pneumonia as a result of severe vasomotor changes in the alveoli. The traumatic origin alone is sufficient proof of the lack of specificity of pneumococci. I conclude that it is borne spontaneously (more properly, endogenously—R.W.) within man’s body.” (Vol. III, pp. 239, 240)

Etiology must include all the known causes of disease. As was mentioned before, these include any environmental circumstance to which the organism fails to become adapted. Severe chilling, starvation, crushing injuries, burns, contact with violent poisons, psychic trauma, or any other harmful agents can be factors in the cause of disease. In order properly to evaluate the role of bacteria in this sense we must first study *Neurology and Double Trouble*. Then you can draw your own conclusions in the light of the evidence presented.

It has been asked, “Where do the bacteria go, when the person gets well?” They pass into clumps as leukocytes, then to the mold stage, and finally are fragmented into re-formed, normal healthy granules which are of constant use in all normal tissue. Studies of Speransky, Kuntz, Pottenger, and many others who are in position to know, reveal that the environment within the body is utterly dependent upon the nervous system. Many experiments show that the various immune reactions, agglutination, and similar phenomena are all produced through the mediation of the nervous system. Speransky has shown numerous histological experiments conclusively demonstrating that the tissues are sick first. There is degeneration through nervous incoordination and the various bacterial forms are the result of the disease, not the cause of it. The bacteria can be collected from other degenerate patients, and may be followed by pathological phenomena, but they act only as “blueprint patterns,” as specific nerve stimuli which modify an already degenerate or perverted reflex.

Thus the nervous system maintains a position of supremacy. Its integrity is a *sine qua non* for health. The disease starts in the nervous system, and recovery starts in the nervous system. The bacteria are bystanders, innocent for the most part, and they disappear after the patient has started to recover. This disappearance is by “re-

education” of the little rascals and their reformation into normal, healthy, primal units to be used as parts of normal, healthy tissues.

## **41 HEMATOPOIESIS: THE RED BLOOD CORPUSCLE**

The red blood corpuscle is formed in the red marrow and in other places in the manner mentioned under the discussion of mitosis. This process has repeatedly been demonstrated to be definitely under the control of the autonomic nervous system. After about three weeks of hard work the red blood cells are decomposed in the blood stream by their own intracellular bionts. This is a normal, not a pathological decomposition. Hence, as the cell disintegrates, the bionts are liberated in a normal healthy form. Iron is set free in the process and quickly reclaimed. The bionts are now free healthy individuals. Both Béchamp and Tissot demonstrated their abundant presence in the blood. Free bionts may now enter into the composition of other cells, or platelets, or simply function as a relatively simple but vitally essential constituent of the normal blood plasma.

## 42 HEMATOPOIESIS: THE WHITE BLOOD CORPUSCLE

Tissot has demonstrated that leukocytes (the mononuclear) are not normally formed in fixed locations, but are “spun” out of the clumping of free microzymas and germs in the circulating blood. Health and disease are interpenetrating processes. The greater the shift from health in the direction of disease, the greater the decomposition of white blood cells, for example, and the greater the number of more or less “perverted” free microzymas or germs. Leukocytes are composed of “scrapped” material and are discarded as pus, in feces, and in other ways.

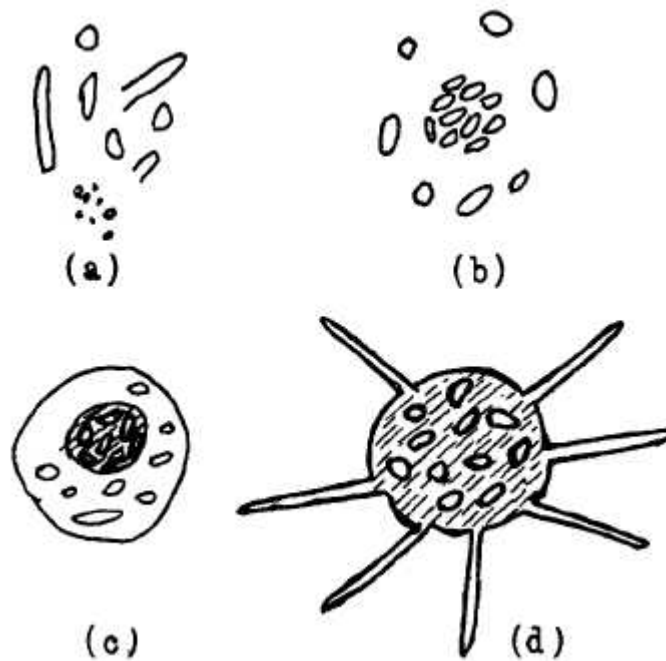


FIG. 25 Interrelation of bacteria and leukocytes, according to Tissot: (a) Isolated bacteria and cellular debris (b) clump together and then (c) fuse to form leukocytes, which subsequently (d) emit filament, becoming germinal centers. In certain locations, under appropriate environmental stimuli, they give rise to connective tissue fibers.



### 43 PLATELETS AND COAGULATION

Platelets are composed of groups of microzymas. These microzymas are the normal healthy granules called by Tissot fibrin ferment or micrococci forms of *B. coli*. Being in the form of small groups and therefore having a diminished exposed surface, their activity is temporarily limited. If all the free granules were separated as single individuals, the entire blood stream would be suddenly coagulated. From this massive coagulation the granules would “spontaneously” develop areolar tissue.

We mentioned earlier, under **Specialization**, the coagulation of blood by the precipitation of fibrin and with it the granules, all as a function of the granules. Tissot showed that connective tissues are developed directly from the granules. One experiment described was that of taking a drop of dog blood, sealing it in a collodion capsule and implanting it into the dog’s peritoneum. After eight days, examination showed that the plasma granules (free granules and small clumps of granules called platelets) had clumped and fused to form the leukocytes—germinal centers—which then grew a heavy mesh of fibers. At this stage the leukocytes are identical (many of them) with the fibroblasts, which give rise first to areolar tissue and ultimately to scar tissue. Others of the leukocytes are in the form of plasma cells, mast cells, and histiocytes, which at their full development generate many new granules. This replaces the continual loss through feces, urine, perspiration and skin friction.

This recalls to mind the statement by pathologists that a sterile clean wound without any bacteria, leukocytes, or clot will not heal. Introduction of a few staphylococci will start the healing process, because in such an environment the staphylococci revert to the normal granule form and function; they multiply and fuse to form leukocytes and fibroblasts, which in turn construct the new tissue. (See *Boyd’s Pathology*, 3rd ed. p. 132 or 5th ed. p. 125—R. W.)

This is a clear answer to the puzzled pathologists who still wonder how so many leukocytes and fibroblasts get on location as fast as they do. Also it clearly answers the old question as to where the histiocytes originate, and why they appear in such profusion after injuries.

## 44 PHAGOCYTOSIS

The romancing of Metchnikoff on phagocytosis leads into a trap. The imagined phenomenon of diapodesis is more romance.

In discussing disease processes in general, there are two stages: an acute bacterial stage and a chronic mold stage. Intrinsic cellular decomposition is accomplished by the intracellular bionts. Upon adequate stimulus they metamorphose into a bacterial form, the specificity of which is determined by the sum total of environmental factors. These early changes constitute the acute phase of disease.

Under continued environmental stimulus, still slightly abnormal as found in the usual "clinical" case, the free bionts clump. Then the clumps of bionts and incidental bacteria begin to sprout mycelia. This is the chronic stage, in which the granules are sufficiently modified to become compatible with the body. Although Tissot has not observed and hence not speculated on the "aborted" cases, it is undoubtedly true that through a rapid complete normalization of the nervous environment, either the acute stage with bacteria or the "prebacterial" stage can clear up rapidly by bacterial fragmentation and subsequent normalization of the individual modified granules. Hence the perverted forms can take either of the two routes of normalization, the complete abnormal cycle or a by-pass through the clump and mold phases, depending upon the nervous environment. Tissot has, however, put great stress on the bacterial "maturation" through the mold stage. The overall picture, of which this phagocytosis and leukopoiesis are only a small part, has already been clearly depicted under *The Biont Cycle*.

## 45 NEUROLOGY

The fact that modified bionts, similar to those which have initiated “infection” on the exterior of the body, appear internally—and even suddenly within remote, centrally situated nerve cells—indicates that the nervous system is competent to transmit a quality of excitation which will cause intracellular bionts to undergo a metamorphosis into a form similar to that of the irritant, as described in the section entitled **Cellular Pathology**.

Speransky (p. 261) reports the definite production of the tuberculous pathology complete with the bacilli in the kidneys in every case in which the specific nerve stimulus in the form of bacilli has been applied to the parenchyma of the testicle, in spite of the lack of communication between the two areas except in the form of nervous stimuli. He also cites (p. 262) experiments in which typical bacteria were developed in the kidneys exclusively through nervous stimuli by applying a drop of formalin to an ovary. Even the typical organisms of syphilis were developed in a similar bizarre manner. (p. 274)

If we were limited to developing bacteria endogenously by perverting the nervous system, we would be without practical application, but the clinical results of chiropractic over the years have shown the reversal of the process to be of very general occurrence, happening in thousands of persons daily; that is, normalizing the innervation is followed by disappearance of the bacteria. Speransky has added his results to these by handling cases of quinine-resistant malaria with a form of general nerve massage, and in ten out of eleven cases has obtained complete clinical recovery with prompt and complete disappearance and non-recurrence of the parasites.

In view of these examples of a commonplace occurrence we must conclude that a microorganism is primarily a specific nerve stimulus which can only slightly modify an already perverted nervous system. If, on the other hand, the nervous system is in excellent condition with good adaptability, the organisms (although acting as stimuli, it is true) are either modified by their nervous environment and undergo metamorphosis into the normal granules, or else they are completely ejected from the body. If, however, the nervous system is crippled and consequently has poor adaptability, then it is already in a state of degeneration, the reflexes are not normal, and the added specific stimulus of the microorganisms may sufficiently modify the reflexes through spatial and temporal summation to alter the symptom pattern and bring about a clinical picture of disease. In this instance, by breaking up the consequent pathogenic reflexes and re-establishing the normal reflexes, the body is able to regain its state of harmony with its environment, and health results. The few drugs that are at all effective in helping recover health are simply specific nerve stimuli that set up counter-reflexes and help normalize the nervous function. Many methods have been used. The few antitoxins that are effective act only as specific nerve stimuli. Speransky showed that novocaine injections were much better than specific antitoxins, (p. 296) Still he discouraged even the novocaine blockade, because it blocked the normal reflexes as well as the abnormal ones.

The adverse experience which initiates pathology is always through the nervous system. To demonstrate this some rabbits were given lethal doses of cyanide salts. One group was first given a general anesthesia to cancel out the possibility of setting up pathogenic reflexes. Controls were given the same dosage of poison. The

controls died suddenly and characteristically, while the other animals with their sensory nerves blocked and the autonomic system working efficiently suffered no ill effects. Their bodies simply eliminated the poisons through the usual channels while their sensory nerves were blocked, and upon awakening there was no change in their behavior, the reason being simply that the specific stimuli were unable to initiate the pathogenic reflexes. A similar series of experiments consisted in freezing guinea pigs. When the freezing started pathogenic reflexes, there was pathology as a result, but the anesthetized animals were unaffected. These violent adverse experiences, having their effect ONLY through the nervous system, should make it adequately clear that minor adversities are brought about likewise through the nervous system.

#### 46 DOUBLE TROUBLE

On the basis of what has been said under **Neurology**, it is evident that among the conditions required for the initiation of infection (that is, actual infection of exogenous origin), two things are absolutely necessary: a certain kind of nerve trauma plus a specific infectious agent. This “double trouble” is the *sine qua non* for the initiation of infection. Remember, however, that identical pathology with identical bacteria can and does develop *without* the “invasion” of a pre-existing germ. It develops intrinsically and *only* by aberration of the normal nervous reflexes. This is of utmost significance, for it shows the *Nervous Supremacy* over all physiological and pathological processes. The bacteria then are not primary causes at all. They often act as patterns which influence the degenerating cellular debris to metamorphose into more bacteria, thus slightly altering the final symptom complex. Influenza and anterior poliomyelitis provide fine examples. They are half-brothers. Occasionally polio is “spontaneous,” with no history of prior contact with the virus. Then it is totally endogenous. More often it is the final perversion of a case of influenza. The patient is first run down, his tissues already degenerating and manifesting abnormal reflex patterns which gradually take on the picture of influenza. To that is added the specific nerve stimulus of the polio virus, which conditions the pathogenic reflexes further and causes finally a typical case of poliomyelitis.

## 47 ANIMAL-VEGETABLE RELATIONSHIP

Studies of typical animal tissue preparations which included the various nerve endings in striated muscles, connective tissue stroma, myelinated nerves, and other tissues convinced Tissot that cultures of pure molds elaborate identical structures. The Ranvier's nodes, Lantermann's clefts, neurolemma nuclei, axons with collateral branches, motor end-plates, spiral endings, and other animal structures all showed up in these vegetable cultures. They were not in profusion, because they occurred at random and were not, coordinated as in the body. Since Tissot was a real scientist, he reported what he saw, and did not speculate on the ultimate nature of the coordination, but he noted that in a living normal body there is a definite coordination which is not present in laboratory preparations. Neither did he speculate on the internal environmental factors of the body.

One of the questions which he considered was that of the actual relationship between animal and vegetable tissues. Since the tissue extracts containing the elemental granules taken from either animal or vegetable cells develop identical structures, is it not possible that animals are groups of perfectly adapted, symbiotic vegetables? Are we then modified vegetables? He puts that as a basic question in biology. He states definitely that the primal units of life, the stick "dumb-bell" units and the granules, are identical in both animal and vegetable cells and grow identical molds when cultured. His work is replete with references to typical bacterial forms and typical vegetable mold forms developed by direct metamorphosis from aseptic animal tissue biopsies, and again he arrives at the conclusion that there is only one disease—degeneration.

## 48 IMMUNITY (NATURALLY ACQUIRED) .

The immunity theory is so full of holes that it looks like Swiss cheese. One doctor was still enthused about it after his daughter died of a third attack of scarlet fever. Based on the current statistics a mathematician, Vogt, showed that a person who has once had smallpox is sixty-three per cent more likely to get it again in a subsequent epidemic than a person who has never had it. Statistics cited later in this book show that, after the use of inoculations against various diseases, not only is the number of cases increased, but a higher percentage of persons die from them: The mortality rate is increased by inoculations. The reasons are as follows:

During the acute bacterial stage of a disease the incoordinated bionts have been sufficiently perverted to become typical bacteria, which at that stage act as specific nerve stimuli and set up reflexly the usual symptoms of the condition. Gradually they clump and fuse into the leukocytes. As they all fuse, the bacteria decrease, while the leukocytes increase in number. At a certain developmental stage the leukocytes appear to “degenerate” into basket cells and then sprout into typical molds, which are discounted in laboratories as contaminants. The molds then develop many filaments which break off usually into fragments, the rehabilitated normal granules. If the normal process is upset at this point, they may be renegades, breaking off as typical bacteria. It is well known that there are a certain number of bacteria in any healthy blood stream, but their presence has never been adequately explained.

Immunity is, therefore, relative and acquired, when it is of this type. Its actual mechanism is that of rapid flocculation of newly introduced microorganisms of the same type. Thus they do not linger in the system in their bacterial form to act as abnormal specific nerve stimuli and reflexly set up the typical symptom pattern, but rather are hurried through this acute bacterial stage too fast for any symptoms to develop and incorporated into the pre-existing germinal centers of the mold stage, the leukocytes. Thus Tissot says (p. 332) “Actual immunity against disease does NOT exist. It is only relative.” (The acute stage is by-passed.) The period of time during which the mold stage of a particular type of organism does exist in the system is the time during which there is immunity to the corresponding disease. If this process of rapid resolution goes wrong (as it does occasionally) there is anaphylactic shock, which is the result of a few clumps large enough to cause capillary blockade and shock. This has been described by McDonagh and by Tissot; it is discussed further under ***Allergy and Anaphylaxis***.

As soon as the mold stage is completed and all the filaments fragmented into the rehabilitated normal granules, then there is no speeding through the acute process, and not only can the acute disease develop again, but it is more likely to develop than in a person who never has had the disease. This predisposition has been shown several times to be statistically true and is easily explained, if one understands the neurobiotaxis and facilitation involved in any reflex action, whether physiological or pathological. It is simply that once the nervous system has been conditioned to perverted reflexes, a developed habit pattern exists which it is easier to repeat than it is to initiate a new pattern.

The complete re-educational reformation by which these perverted forms are rehabilitated by a normal environment (nervous—R.W.) to return to their normal healthy forms takes a varying length of time ranging from a few weeks to about seventy-five years. During this reformation period of passage through the mold

stage, fragmentation of filaments into granules, and their final incorporation into normal tissue cells, there is a period of relative immunity. This “acquired immunity” then means that any perverted granules in bacterial or sub-bacterial form which are spread from another degenerate (sick) person will be transformed very rapidly into the chronic mold stage so that the acute stage will be partly or entirely skipped. The bacteria do not remain in bacterial form with the capacity to act as specific nerve stimuli. When this reformation is complete and there are no more germinal centers or mold stages of this particular type of mold, then the “acquired immunity” is over, and the person is again normal and healthy. By again degenerating and becoming sick he can have the acute bacterial stage of the disease reproduce the clinical picture once more. This type of relative immunity is specific. If diphtheria were obtained from degenerate barley, then the “immunity” would be worthless against diphtheria from degenerate wheat. Thus, to be effective, an inoculation would have to be made directly into the person to be immunized (not through a horse or even another person), and the preparation would have to consist of the one specific type of perverted granule.

Since there are at least six different types of perverted granules of degenerate cereal which can modify the symptoms of an already degenerate, sick patient into those of diphtheria, it is obvious that it is almost impossible to inoculate against diphtheria. What is actually done in the classical attempt is to inoculate the horse and thus modify the intracellular granules of that animal as its body reforms them into normal horse granules. This can at most effectively immunize the horse for a few years against the same species of diphtheria, but the procedure is absolutely useless as a means of protecting man, and the horse is no more worried about diphtheria than a hog is worried about a holiday.

Further than that, the preparation has become a foreign protein, horse serum, which is dangerous to man in that it sensitizes him for possible anaphylactic shock, if successive inoculations are used. Tetanus toxin, anti-toxin, and toxoid are all developed in similar fashion. It is well known by all who have seen tetanus “shots” used, that they produce more or less mild symptoms of anaphylactic shock in nearly all the “victims” inoculated. The tetanus bacillus is a perverted form of the granules and the anaërobic relative of the aërobic colon bacillus. Thus the tetanus inoculation might protect the horse from human tetanus, but is worthless for man, as well as dangerous to him.

Many unbiased physicians (granting that there are a considerable number) have discarded toxin-antitoxin completely in favor of simple hygiene. They get better results, fewer cases of tetanus, and no “serum accidents.”

See also in this connection the chapters ***Immunity (Artificial)*** and ***Inoculation***.



## 49 IMMUNITY (NATURAL)

There is a natural indifference to many agents. A healthy person is naturally immune, because there are no perverted granules of cellular debris to follow the example of a foreign agent and become bacteria. If this were not true, then everyone would be affected in an epidemic, instead of the usual one per cent. That is the reason wild animals are immune to practically all the diseases of mankind, but when they are placed in captivity, with their body flexibility and their natural diet limited, they begin to fall prey to every disease of mankind. Again the entire problem is reducible to only one disease—even including the diseases of subhuman species—the disease of degeneration.

Again, there is a sub-clinical response to many agents (serology proves this fact). The requirements for infection are not quite as simple as most persons think they are. Both Speransky and Béchamp must be understood, if one would be well informed on natural immunity. The problem is quite clear, if one thoroughly understands what has been said under *Infection* and *Resistance*.

## 50 IMMUNITY (ARTIFICIAL)

If the material under the heading of **Biology** has been clear, it will be apparent that artificial immunization is not a rational practice.

It was shown by Béchamp and confirmed by Tissot that the biological differences in all animal species are sufficiently great to invalidate the theories of immunology. The additional feature of the delicately balanced, highly-organized body on which the practice of artificial immunity is inflicted, indicates a wide range of potential damage. Artificial immunity is both worthless and injurious, says Tissot.

First, there is a vast difference between test tube phenomena and reactions in a living body. Second, a standard reaction in a horse, guinea pig or rabbit is absolutely different from the corresponding reaction in man. Third, since there are no two human beings who are the same, no two persons can have identical reactions to similar environmental changes (including inoculations). Fourth, the introduction of any product of one living organism into another organism, even of the same species, is the introduction of foreign material and always causes some reaction, frequently producing very dire results.

The close ultimate similarity among pathogenic agents has been stressed herein. But now the converse feature (difference) must be discussed. Although it is possible to cause diphtheria through the agent taken from a cereal culture, it is not rational to attempt to immunize with horse serum, because of the great biological differences between man and horse. The horse serum then becomes injurious to man, no matter how it is modified. It would not be injurious to re-inject it into the same horse, but that would be pointless.

Recall that “acquired immunity” is the reformation stage during which bacteria are passing through the mold phase. At this time any newly injected bacteria are speeded through the clumping into the mold stage, almost skipping the acute phase. Anaphylactic shock is the extreme case of this mechanism. Here a sensitization is produced by the first injection, which causes a clumping of the injected granules into germinal centers. The second injection adds bulk to these clumps so rapidly that the clumps, now greatly enlarged by newly infected granules, cause a capillary blockade with the typical symptoms of shock.

## 51 INOCULATION

There are two points to be kept in mind whenever inoculation is considered. First, it is not possible to limit the technical procedure to an isolated feature: one cannot avoid inflicting a trauma at the time of inoculation. (See section on ***Double Trouble***.)

Again, it is not possible to isolate the result of the procedure: As Herbert Spencer pointed out, when you change the body in relation to one disease, you change it in relation to all. Statistics and history show that the change is for the worse.

## 52 ALLERGY AND ANAPHYLAXIS

Both allergy and anaphylaxis are caused by body response to specific agents. Tissot wrote, "Lambling in *Biochemistry Abstract* says, 'All diastases act like antigens because animal injections provoke a corresponding antidiastase.' It is not a true antidiastase at all. Each diastase (enzyme) is a form of *B. coli* micrococcus (biont granules). Thus each foreign diastase is a foreign protein, and the experimental disease develops either as the acute bacterial stage or the chronic mycelian (mold) stage. This agglutination of the bacteria or virus to form germinal centers for the chronic mycelian stage marks the beginning of the sensitized state. Newly injected virus then agglutinates much more rapidly than before to keep pace with the earlier injection. This high-speed clumping (too few and too big granular accumulations) is what blocks the capillaries and induces shock." (p. 258) "The albuminoids play only a passive role in developing the injected antigen and the production of agglutinin." (p. 331)

McDonagh made a very similar explanation on the basis of the size of the serum protein particles and the consequent capillary blockade, but he did not pin the tail on the right donkey. Tissot says that the albuminoids are only passive in this show.

Inability to regulate the clumping of the antigen is an added risk in any form of inoculation. All inoculations inflict the disease, usually in a chronic stage and for varying lengths of time.

Allergy is the same general type of reaction to a specific agent as anaphylaxis, with the difference that in allergy the agent in question does not usually enter the body. Hence it is not a problem of clumping and passing to the chronic stage, but rather a problem of unstable physiological reflexes easily perverted by external stimuli, so that they become actual pathogenic reflexes. It is a problem of nervous adaptability and thus an easy one for chiropractors to solve.

## 53 SPECIFIC SERA

Diphtheria antitoxin and toxoid are both not only worthless in practically every case, but also virulent and injurious in all cases. The reasons for this are obvious in that typical diphtheria pathology with pathogens can be consistently developed from barley cultures, as well as from wheat and rye cultures. With so many antigens it is impossible to make specific sera. Furthermore, the manufacture of these sera from horse serum brings into the picture the added danger of a foreign protein inoculation in every case.

Rabies, which in man is practically unknown, is the only excuse for the Pasteur treatment with a serum made from the spinal cords of rabid rabbits. Tissot has shown beyond all question that the serum is utterly useless. Statistics quoted later show that this serum has actually killed many more patients than has rabies.

(How many benefited?—Verner)

Typhoid serum is completely exposed by Tissot as worthless, but he adds, “Anti-typhoid serum, especially dangerous, inoculates one hundred per cent of its victims with chronic typhoid and its dangers, although very few are immediately or seriously impaired.”

Tetanus is strictly an endogenous disease accompanied by endogenous organisms. It is often inadvertently caused by surgeons with bandages that are too tight or sutures which constrict the circulation. The clostridium tetani is an anaërobic brother of the B. coli developed in situ from the debris of the cellular breakdown. The perverted granules take this bacterial form and thus get blamed for the disease, whereas, actually, they are the result of the disease. The entire family—Welschii, Chauvii, etc. is made up of almost identical end products of anoxemia and its consequent cellular breakdown, the variations being attributable to slightly different environments. In castration, tetanus is frequently caused by veterinarians through localized anoxemia. Since tetanus with its bacteria is often caused by the aseptic quinine injections of physicians who use them, there is no question about the endogenous origin of the disease.

Since tetanus occurs as an anaërobic degeneration and is totally an endogenous process, there is not the slightest excuse for the toxin-antitoxin which is so indiscriminately peddled. The false assumption that the clostridium tetani is a causative agent again stems from Pasteur’s obdurate refusal to admit error. Only education in this matter of biological inter-relationships can obliterate this dangerous fad that is still foisted upon the people.

Vaccinia is deliberately inflicted upon as many people as possible by unscrupulous or woefully ignorant inoculators.

Sera for “immunization” against tuberculosis have been offered and one, BCG, has been exploited in Europe. To understand this we must first investigate the pathology itself.

Intensive studies of tuberculous tissue showed that the disease begins as a degeneration of the lung some time before any bacilli are present. An adverse experience inflicts damage upon the cell (see *Neurology*), thus disturbing the welfare of the intracellular bionts. These units now undergo physico-chemical change in very specific response to their intrinsic and extrinsic environmental situation. In the disease process at present under discussion, they metamorphose into a characteristic

form, and they decompose the cell. In each specific disease, the intracellular bionts metamorphose in specific response to the, environmental situation.

As the cells degenerate and disintegrate, the stroma or “dumbbell” units become perverted and metamorphose to become the tubercle bacilli (Koch’s bacillus, *Mycobacterium tuberculosis*). Hence, bacilli are found in the amorphous mass of cellular debris at the center of the tubercle. The epithelioid cells, the lymphocytes, and the giant cells, all characteristically found in a tubercle, are formed by direct “budding” from the preexisting embryonic cells in the denuded stroma of the degenerating alveolar walls. Here, as in all other purulent reactions, the leukocytes are developed on location; they are not migrants from the blood stream nor are they produced by hematopoietic tissue. Diapedesis is probably a non-existent phenomenon fabricated to explain the presence of great numbers of leukocytes present in the tubercle. The problem of explaining the sudden appearance of extravascular leukocytes has worried pathologists for years. They admit their ignorance of the answer. Tissot has clearly provided that answer.

With the knowledge that tuberculosis is an intrinsic degeneration not caused by external invaders, and that the tubercle bacillus is a perverted and modified form of the “dumb-bell” ~ unit, it becomes clear that to vaccinate successfully against tuberculosis is impossible. It is foolish to use one type of bacteria (bovine) to attempt to immunize against another (human), and the BCG serum advocated by the pharmaceutical houses for compulsory vaccination is from bovine organisms cultured on bile media. Furthermore, since the human “tubercle bacillus is of intrinsic origin, it is impossible to immunize against it. We cannot immunize against our own cellular components without destroying parts of ourselves. Besides this, there is the introduction of foreign protein—a very harmful practice.

BCG serum is vauntingly held forth as a certain prevention. Such devastatingly injurious propaganda is advanced by individuals who are entirely vicious, because no one with supposed medical education could be sufficiently ignorant to believe that BCG serum could be effective.

This particular topic arouses the ire of Tissot to great heights. Details of the results with BCG are found under Statistics.

Of the so-called specific sera just mentioned (typhoid, diphtheria, tetanus, rabies, and tuberculosis) Tissot writes, “THESE FIVE INOCULATIONS SHOULD BE PROHIBITED!!! There must never be, under any pretext, reason for inoculating any living organisms, attenuated or killed, or a serum or other product of living organisms. It is the principle adopted by England and Holland to refuse to make these compulsory. With the inefficacy and the dangers of these sera known will the French people continue to use their children as guinea pigs for the sole purpose of furthering the financial prosperity of the Pasteur Institute?”

“Numerous cases of encephalitis, nephritis, blindness, and deaths have been such that Holland has outlawed smallpox vaccinations, and for several years, it has been optional in England, with the intelligent folk refusing it.”

## 54 CHEMOTHERAPY

In concluding a thorough analysis of the problem, Tissot writes, "This method of treatment (chemotherapy) is able to be successful in some cases, but unfortunately they are quite rare. Many of the diseases are autogenous and intrinsic (exclusively), and the extrinsic diseases have a bacterial phase which is only one small part of their life cycle; that small portion ONLY is attacked." (P.-295, vol.1) He is speaking here of strict chemotherapy into which the sulfa group, penicillin, and the other "miracle" drugs fall. Of serology with its inoculations and vaccinations he is much more critical.

McDonagh, an eminent physician and research expert, states: "In much the same way as we have discarded local manifestations of disease as being disease entities, and now consider them as representing symptoms or end points of disease only, so we now discard the specific action of drugs as playing anything but a very minor part in their activity." He attributes their action to their qualities of altering vegetative nervous system balance. He even demonstrates that the use of glucose and of insulin in treating many things entirely divorced from sugar is based simply upon its effect on the vegetative system. He says also, "Vitamins are not really specific substances, nor the results of vitamin starvations specific entities." Again, their action is only on the vegetative nervous system.

Speransky has gone even further in demonstrating the effects of chemotherapy. "The ancient methods of treatment . . . equally with more recent methods—subcutaneous injections of foreign substances, radium, x-ray, diathermy, and nerve blockade—all find the explanation of their action in those characteristic changes which the nervous system undergoes on encountering processes of irritation. There is every reason to believe that drugs such as sodium salicylate, quinine, arsenic, mercury, and many others as well, owe their action to the same mechanism." Thus he states that specific drugs are only specific nerve irritants. "Pathology is turning to chemistry because, not having good methods of its own, it hastily seizes on methods which may be good but are foreign to it, without always taking into account the limits of their applicability. This is not the first time that pathology is passing through such a period. Contemporary chemistry can deal only with small things."

Voltaire hit the mark when he said, "Doctors put drugs of which they know little into patients of whom they know less, to cure diseases about which they know nothing at all." Oliver Wendell Holmes, a great physician, rightly said, "If all the medicines were poured into the ocean it would be better for mankind but bad for the fish." Speransky wrote, "The medicine of Virchow, Pasteur, and Ehrlich is approaching exhaustion and cannot cope with the contradictions that have arisen."

As for the modern "miracle" drugs, one after the other has been withdrawn from the market, as its turn came to pass out of style. Time magazine reported several such exhaustions in connection with a report on penicillin. After a year's research and spending a million dollars, the Carnegie Foundation reported that powdered chalk was just as effective as penicillin in controlling colds, and that it was a lot cheaper. They called penicillin "useless." In 1950, some communities noticed that most of the poliomyelitis cases had followed intensive penicillin therapy. Marked reduction of penicillin usage in 1951 was accompanied by marked reduction in the number and severity of poliomyelitis cases.

## The Forerunners of Tissot

Tissot reviews the work of Béchamp and criticizes him mainly for missing the “dumb-bell” stick units and recognizing only the granules. He mentions that Turpin in 1835 described the granules capable of reproduction and transformation into molds and back again to granules. Hallier and others also had done a lot of work with this material, but Béchamp was the most competent investigator. Tissot, however, carried it further and in much more scientific order.

The intrinsic origin of bacteria was shown classically in 1874 by Serval, who took fresh, living kidneys and livers, ligatured them immediately upon opening the abdomen, and then suspended them in one per cent chromic acid solution for eight days. The surfaces, which were almost embalmed by the bactericide, were sterile and quite unaltered, but the central mass of degeneration was teeming with bacteria of many varieties. The Pasteurian school maintains that the *B. coli*, *proteus vulgaris*, and similar organisms found in the viscera at autopsy are migrants from the intestinal tract. Instead, they are in the vast majority of cases endogenous.

Such migration would take several days, eight at least, but many pathologists have found these bacteria in the liver, spleen, and kidneys in the majority of autopsies within forty-five minutes after death. The same bacteria were demonstrated many times in these organs *before* death during the crises of the intoxications by cantharide or arsenic poisonings. As the body temperature altered, all the granules changed and frequently became bacteria. An additional drop in temperature further metamorphosed these degenerate bacterial forms into other degenerate bacterial forms; that is, staphylococci and streptococci changed to *proteus vulgaris* and then to *B. coli*.

Normal, clean milk and normal, clean saliva both contain the granules which change these secretions and curdle the milk in time. Normal blood plasma contains these granules, and all three of these fluids develop the various bacterial forms when cultured. In fact, normal blood will show them without culturing. Direct examination of the white layer in the test tube immediately above the centrifuged cells shows granules in profusion, cocci, rods, germinal centers, molds with filaments, and other particulate material.

“The thrombin of blood coagulates milk; milk coagulates blood; colon bacillus, staphylococcus, streptococcus, pneumococcus, *Bacillus lactis aërogenes* all coagulate both blood and milk. . . . Injections of staphylococcus cause *B. coli* abscesses. Introduce streptococcus and colon bacillus side by side into a medium of bouillon, and only the colon bacillus develops; the pneumococcus is transformed into colon bacillus; chains of *B. coli* develop spontaneously in oxalated plasma. Little chains of streptococcus develop spontaneously in serum, urine, and milk kept at room temperature.

“Add to these facts a whole group of properties common to all these aspects of the colon bacillus and we get a collection of such convincing proofs that one can no longer deny that staphylococcus, streptococcus, pneumococcus, enterococcus, *Bacillus lactis aërogenes* are all forms of the organic colon bacillus, have no fixed specific character, and ought not to be catalogued as different species nor, with even stronger reason, as genera.” (Tissot, pp. 237, 238)

Thus, in summary, Tissot has established that the Pasteurian school has fallen into gross error by following four major blunders of Pasteur. First, living animal tissues are not aseptic, but are composed entirely of subcellular primal units which are capable of independent existence. When they become perverted due to faulty



environment, they decompose the cell and become bacteria of various forms. This corrects Pasteur's second error of bacterial monomorphism and a third in which Pasteur insisted that putrefaction and all disease processes are caused by the "invasion" of external, inferior germs. This is false. Pasteur drew this erroneous conclusion from the fact that autoclaved media will not spontaneously brew bacteria. This fact really proves only that roast beef, for example, is dead; it does not reveal the characteristics of living cattle.

Place upon autoclaved media a bit of tissue fluid or a few living cells, aseptically removed from a healthy, living animal and such tissue or fluid will, under suitable conditions, definitely develop into luxuriant bacterial cultures. Then by modifying the temperature and the fluidity of the media the specimen can be transformed into a mold.

The major errors of Pasteur which have had such deleterious effects on mankind in general and on medicine in particular are discussed later under ***Four False Dogmas of Pasteur.***

## 55 STATISTICS

“Does diphtheria antitoxin immunize against or even retard diphtheria? All physicians know that the incidence in immunized and NON-immunized is about the same. Ruttgen and Fischer showed thirteen cases of diphtheria out of forty-eight inoculations. (Med. Bull. 3 Dec.) Here the incidence was much higher than with non-immunized patients.” (p.277) “Results published by Medical Syndicate of Doubs show: (.1) One child received three diphtheria inoculations. Two years later he was hospitalized with severe diphtheria. His three playmates, un-immunized, did not get it at all.

(2) Child immunized. Next year had diphtheria. His younger brother and sister, not immunized and not isolated remained healthy.

(3) Child immunized and soon had diphtheria. His three younger brothers, not immunized or isolated remained healthy. (4) A mother refused general immunization of her child because her other two children had died with diphtheria soon AFTER they were inoculated against it.” (p.278)

“Statistics published in Greece and France prove that immunization does NOT decrease the number of cases, rather they INcrease. In Greece general immunization of everyone was carried out from 1926. In 1929 they had 750 MORE cases than previously and in 1934 1840 more. In France the incidence per 1,000 was 11.033 in 1923, when everyone was immunized. By 1927 it rose to 14.259, in 1928 to 18.898 and in 1933 to nearly 21. In Germany at the time of general immunization in 1926 the incidence was 30.3, in 1933 up to 77.3; in 1934 to 119.1, and in 1937, up to 146.7. Dr. Renard wrote in *Revue Française* of June 1938 ‘School children were practically all immunized, but the incidence was rising, and often the immunized children had it much more severely than the non-immunized.’ If the people accept this without protest they will also have compulsory T. B. vaccination (BCG) which does NOT protect.” (p.278)

“ ‘These and smallpox vaccination are dangerous. Numerous cases of encephalitis, nephritis, blindness and deaths caused Holland to outlaw smallpox vaccination, and for several years it has been optional in England, with the intelligent folk refusing it.’ Is it possible that the drug houses can continue their bald LIES about the disappearance of diphtheria due to their ‘efficient’ *nostra*, when in spite of their almost total coverage the cases doubled in France and quadrupled in Germany during the ten years of heaviest immunization?” This is Tissot’s condemnation of the matter. He summarizes by saying, “Vaccination (inoculation) by the antitoxin is ineffective and illusory. Worse yet, it is harmful and can kill . . . Since it is worthless, it must be abandoned.”

An excerpt from the *Congressional Record* reporting statistics from the Surgeon General’s files notes that in the Philippines the heaviest epidemic of smallpox followed soon after almost universal inoculation, with only the non-immunized area escaping. Even the military personnel with their repeated “protective” vaccinations were hit hard, and the mortality rate rose to 45.93%, while in the far less vaccinated United States the mortality rate never exceeded three per cent. “During the epidemic of 1918, with the Philippines almost universally immunized against smallpox by vaccination, the case-mortality rate rose to over sixty-five per cent.” (Hume p.174)

Tissot was considerably impressed by three statements in Dr. Bourget's work *The Errors of Modern Medical Science*. Dr. Secretan (who never used toxoid) handled some 12,000 wounded, many of them from the stable, in his polyclinic without ever having a case of tetanus. Dr. Reiffel told the French Surgical Society that toxoid has no effect on tetanus or its progress. Moreover, Professor Berger collected thirty-five cases of tetanus which developed AFTER serum inoculation. These experiences caused Tissot to conclude that tetanus antitoxin and toxoid, along, with diphtheria antitoxin and toxoid, ought to be outlawed, since they are not only worthless, but injurious.

Tissot has collected great numbers of statistics which show that during the first ten years of compulsory inoculation against diphtheria the incidence in France was doubled and in Germany quadrupled. This was during the time of most intensive inoculation. There were more fatalities among the inoculated than among the non-immunized."

He made similar studies on the statistics of rabies and the Pasteur treatment. First he showed that the Negri bodies are identical with the well-developed spores of a mold, *Aspergillus*. Then he pointed out that the "street" rabies from a mad dog bite are characterized by convulsive symptoms, while the symptoms of a rabbit with rabies are paralytic. Patients dying after the Pasteur treatment exhibit the same paralytic symptoms as the rabbits used to prepare the serum. Thus the statistics show that the Pasteur treatment is not only absolutely worthless, but also virulent: It inflicts upon many patients a type of rabbit rabies, so that they die of paralysis—in contrast to the convulsive symptoms of rabid dogs—a very serious charge. Further statistics are hardly needed to point up the futility of current practices.

## 56 FOUR FALSE DOGMAS OF PASTEUR

1. **Panspermism** Pasteur held that germs are found everywhere in the air, and that these atmospheric organisms are the cause of fermentation, putrefaction and many of the diseases of mankind.

2. **Monomorphism** Pasteur held that each type of bacterium is a distinct species; that this species and this alone causes a corresponding specific disease; that there is no transmutation of bacteria-cocci cannot possibly become rods.

3. **Asepsis** Pasteur held that a normal healthy animal has bacteriologically sterile tissues; that there are no bacteria normally found within the body proper, and that putrefaction is caused exclusively by contamination, the invasion of external germs.

4. **Contagion** Since Pasteur believed that animal tissues are aseptic, then any disease must be caused by invasion of external germs through direct or indirect contact with a pre-existing case of the disease. There was to him no such thing as an endogenous disease.

Tissot has presented voluminous material in the nature of indisputable evidence that Pasteur was not only absolutely wrong about these four points, but also vicious in the promotion of these falsehoods exclusively for the benefit of Pasteur, his own fame and fortune. We shall consider portions of this evidence point by point.

1. *Panspermism*. Quoting Tissot (Vol. III, p. 10), we repeat: "These new ideas definitely destroy the Pasteurian dogmas and render them untenable in the future. They are going to be violently attacked by a school which for three quarters of a century has defended the false and nefarious dogmas above enumerated and wants to maintain them at all cost despite all appearance of truth to the contrary.

"That will be nothing new; it will be only a continuation of the attacks of which I was the object in 1926 and 1936, upon the occasion of the publication of the first two volumes of this work, and a continuation of the attacks upon all those who have published ideas contrary to these dogmas: Frémy, Béchamp, Galippe, Portier.

"How was such a situation, so prejudicial to the progress of science, ever created?

"Primarily the origin of this situation resides in Pasteur's obstinate denial of the intra-cellular origin of the ferment in the juice of the grape and his insistence upon its atmospheric origin.

"As a consequence of this false principle, he maintained similarly that the ferments which cause animal matter to putrefy are of atmospheric origin, denied their intra-organic origin, and created the false dogma of the asepsis of living organisms, asserting that the body of animals is closed, under ordinary circumstances, to the introduction of germs from lower beings.

"The result of these false principles was that, in applying them in his memoir *Recherches sur la Putrefaction* (1853), he drew from them false conclusions totally devoid of foundation. Béchamp, by experiments clear, precise, easy to control, demonstrated beyond all doubt the falseness of these conclusions, first on meat, then on blood, milk, and urine; he proved that the cause of the putrefaction of these materials is due to the granulations of intra-organic origin which these materials contain, and which he called 'microzymas.'

"Out of this contradiction were born the often violent discussions in which Pasteur was unjust to Béchamp, discussions which lasted more than twenty years.

Despite the formal proofs which were placed right under his eyes, Pasteur upheld obstinately the false principle of the atmospheric origin of ferments, denied their intra-organic origin, and sustained the false dogma of the asepsis of living organisms.

“Following him, his school, his pupils and collaborators, have continued to maintain and defend the same errors and, at the present hour, more than three quarters of a century after their elaboration, his school keeps vigilant watch to keep them untouchable and attacks every idea which may injure them, thus delivering themselves to a continuous struggle against the truth.”

On pages 244-6 Tissot reports many experiments by various pathologists which show that tetanus and gangrene (with their organisms) are endogenous. Anaërobic blood cultures commonly develop these organisms. On pages 247-8 he shows many reports of the “putrefactive” organisms being entirely endogenous. They even develop within the body before death with severe crises of various poisonings. Streptococci, sareina, M. tetragenus, proteus vulgaris, and nearly always B. coli are found in the viscera during these crises. Other classic experiments performed repeatedly are mentioned under the heading of asepsis, but would be equally appropriate here. The experiments of Pasteur upon which he based this erroneous and seriously misleading conclusion showed only that it is impossible to develop organisms spontaneously in media that are heat-sterilized, and that the organisms found later on that medium come from external contamination, principally from the air. That proves nothing except that boiled beef is no longer living. It shows absolutely nothing about the characteristics of living cattle. Aseptic biopsies in culture consistently yield bacterial forms which are absolutely endogenous.

Lord Lister (a medical “saint”) recommended phenol spraying of the air in surgeries. This Pasteurian “theory that the *causa causans* of septicism in wounds rested on micro-organisms in the air was an altogether mistaken theory.”—Of this Lister made an honest recantation before the Medical Congress in Berlin: “I feel ashamed that I should ever have recommended it for the purpose of destroying the microbes in the air.” Dr. Wilson continues: “The real source of all the mischief was the unclean or putrefying matter which might be conveyed by hands, dressings, or other means, to freshly made wounds.” (Final Report of the Royal Commission on Vivisection—p. 90) Louis Pasteur was responsible for misleading almost the entire world of science. His errors are still responsible for much suffering and many deaths.

2. *Monomorphism*. The present-day bacteriologists being financially supported by the Pasteurian school must uphold the political front, but they all admit that there are truly no fixed species except when held under rigid artificial laboratory environment, and even there the bacteria will change both form and function quite readily. It is difficult to maintain fixed “typical” cultures. They find even the germinal centers and filaments of the mold phase of the bacteria quite commonly as “contaminants” in old cultures. These are usually Aspergillus or Penicillium. Tissot writes (p. 156), “(a) Each bacterial species is only one provisional form of basic living material; (b) each one can be modified and changed severely by environmental changes; (c) constant types of these species can be changed and are only those held under rigid control; (d) these pathogens have both bacterial and hyphomycetic (mold) forms, and generally the hyphomycete is the original form, which in the infected organism gives rise to the secondary bacterial forms.”

“General Conclusions (p. 161): (a) Polymorphism is one of the principal properties of living matter; it is adaptability, (b) Bacteria transform through these stages: micrococci (granules and filterable forms), bacilli, mycelian web, hyphomycete, organized fungus, (c) These forms do not exist in normal tissues, but only in perverted tissues as either endogenous or exogenous ‘guests.’ They are parasites at the ‘invitation’ of the host.” Actually to anyone who knows much about bacteriology there is no such thing as argument on this matter. Pasteur’s errors are being perpetuated by scientists who are fully aware of the damage he inflicted on the human and animal world.

3. *Asepsis*. Laboratory experiments demonstrating endogenous bacteria are classic, (p. 169) “A dog is sacrificed by femoral hemorrhage; the abdomen opened, the liver and kidneys ligatured and removed aseptically; then suspended in a one per cent solution of chromic acid (a bactericide) and kept at 15 to 20 degrees C—just normal laboratory temperature. After five days microscopic examination will show the periphery to be perfectly sterile, no bacteria and only the normal microzymic granules; the center by contrast is filled with bacteria which are active. As soon as they hit the chromic acid they stop. This experiment was a classic done by Servel in 1874 and vigorously denied by Pasteur. Altmann, Béchamp, and many others repeated these experiments and refuted Pasteur.”

These endogenous bacteria are developed by perversion of the normal tissue granules. Since the normal granules can be transformed into bacteria quite easily in any laboratory, Tissot uses a rather questionable term in stating that the normal granules are of bacterial form. Actually he is correct, but the term has a prejudicial meaning to most persons. Tissot concludes this question of asepsis on p. 11: “The climactic stroke that closes the indictment of Pasteurism is the demonstration of the fibrin ferment (normal granules) and its relation to *B. coli*. By obstinately insisting on the asepsis of living beings, which are wholly constituted by the haltère unit (the structural stick) of bacterial nature and of *B. coli* (in its various subforms as normal granules), Pasteur made the worst error in biological sciences of which I know. Consequently the Pasteurian school has, by supporting these blunders, blocked the discovery of (a) the bacterial nature of living beings, (b) the original source of *B. coli* and its role in living beings, (c) the real nature and original form of endogenous viruses (often of vegetable origin), (d) the existence of endogenous, spontaneous virus and bacteria, (e) the identity between all the cocci, *B. lactis aërogenes*, *Vibrio septic*, *Clostridium tetani*, and others, (f) the bacterial nature of enzymes and ferments. These blunders have appreciably blocked scientific progress and perverted bacteriology. The insistence on asepsis in living beings, the ignorance of *B. coli* and its role, and the blaming of Koch’s bacillus for tuberculosis are responsible for antibacterial chemotherapy, which has claimed many victims, especially the tuberculous, by trying to destroy the bacterial units which are vital components of all animal organisms. They are responsible also for the creation of vaccines and sera for the purpose of combatting the other of the two vital components of ourselves. They have impeded research into the cause of disease and have only ‘perfected’ their blunders by developing other nefarious sera.” Continuing on p. 12 Tissot writes: “By the arrest of scientific progress, by the continued accumulation of a mass of errors which they have provoked, the false Pasteurian dogmas have slowly directed medicine for three quarters of a century into an extremely grave impasse, a situation of incredible ignorance and incoherence, and led bacteriology into a decline. I have

been accused of being a 'destroyer' and of making sport of biological principles. I can easily reply that I destroy only the false and unfounded, and that I immediately replace the false with exact notions of capital importance which could never have been discovered without the demolition of these false dogmas. Thus I have opened new, fertile fields of scientific research. With what result? After twenty years of this research I have been officially denounced by the Academy of Medicine in writing and mobbed and grossly insulted during a public lecture to make these things known to the scientific world."

4. *Contagion*. If the preceding has been understood, there is no need presenting the case against contagion as Pasteur advocated it. It is not an argument, because Pasteur has already lost. Since it has been definitely proved over and over again that tuberculosis is endogenous, we only mention it. Both tuberculosis and pneumonia begin through pathological reflexes causing degeneration within the alveoli. (Vol. II, p. 38) Tissot states: "Pulmonary tissue is destroyed and replaced by tuberculous tissue in this order: (a) disintegration of the blood capillaries, (b) destruction of the alveolar epithelium with the free cells falling as debris, (c) proliferation by pediculation of the stroma\* of the alveolar wall forming many embryonic cells." p. 41 reads: "The development of pneumonia follows the identical process, except that there is very little development of new embryonic cells, and those made are used in regeneration, while in tuberculosis they further degenerate to form the typical tuberculous tissue."

Many series of photomicrographs verify these findings and show the endogenous development all the way through, including typical bacteria. Tissot continues in Vol. III, p. 239: "Since pneumococci are NOT found in the alveoli during the initial stages of the disease, but only well after the fever is under way, the pneumococci must be NOT the cause, but the consequence of the disease. Chilling of the thoracic wall, or trauma to that wall (rib fracture) easily cause pneumonia by the severe vasomotor changes in the alveoli. The traumatic origin alone is sufficient proof of the lack of specificity of pneumococci." Of pneumonia he writes (p. 240): "I conclude that it is born spontaneously within man's own body."

Cecil's *Textbook of Medicine*, Fifth Ed., p. 77 states: "The paradox of the isolated case (poliomyelitis) remote from civilization, on the one hand, and on the other the epidemic focus which offers the picture of mass group infection is well known but not explained." Naturally, the isolated case is not explained by Pasteur's erroneous approach to the problem. As soon as they take the prejudicial scales from their eyes and become really scientific, they must necessarily agree with Tissot, Béchamp, and the many others who have studied endogenous bacterial origins.

It would be fallacious to say that all disease processes are ENTIRELY endogenous, even though they have an endogenous beginning. This is why we are opposed to vaccination. Certainly there are symptom patterns that are superimposed upon an already abnormal nervous reflex pattern by specific nerve stimuli in the form of specific antigens. Some of these are bacteria, some are toxins, and some are filterable virus forms, but all of these were previously degeneration products from perverted bionts of another diseased person. These same bionts were once normal healthy cellular components. All of them can be developed in an exclusively endogenous manner without a previous antigen, but many of them frequently are guided in their metamorphosis from normal bionts to bacteria by an exogenous antigen. Thus Tissot does not say that there is no such thing as contagion, but he

does say that Pasteur's insistence on contagion as the only cause of disease is a figment of a perverted imagination. Further, Tissot maintains that in view of all the evidence presented to Pasteur which was indisputable and pointed out, Pasteur's errors would indicate that Pasteur was much more interested in the fame and fortune of Pasteur than in the truth and the welfare of humanity.

Referring directly to contagion and tuberculosis, Tissot writes (p. 301): "In 1865 Pidoux wrote, 'Tuberculosis is a spontaneous degeneration.' Today he stands justified in spite of later errors by the Pasteurian school." On p. 306: "The principle of BCG immunization is false, since tuberculosis is autogenous and intrinsic. BCG being a bovine bacillus, specifically different from human, it is NOT able to vaccinate at all against human tuberculosis; it is virulent for man." On p. 309: "Lignères said of calves, 'In every case, one must not forget that vaccinated subjects become totally tuberculous if they submit later to injections.' What can happen here to calves can happen to man. Watson, in Canada, notes that of the guinea pigs inoculated by the hundreds with BCG, certain ones after a year or so show a series of active tuberculous lesions. Military statistics show ninety to ninety-five per cent and sometimes ninety-eight per cent having active tuberculous lesions. If so many really were tuberculous, the human race would be long gone."

In summarizing the pseudo-scientific actions and the hypocritical propaganda of the Pasteurian school, their insistence on their own infallibility, and their immediate persecution of any ideas not originating from within their own inner circle, Tissot is rather blunt in stating the case. Of the four false dogmas of Pasteur he writes: "These have been nefarious in completely falsifying the reasoning of scientists and have imbued them with incorrect ideas on living matter and started them up blind alleys of research. This is the cause of many fruitless research programs."—(p. 9) He concludes this discussion on p. 12: "By the arrest of scientific progress, by the continued accumulation of a mass of errors which they have provoked, the false Pasteurian dogmas have slowly directed medicine for three quarters of a century into an extremely grave impasse, a situation of incredible ignorance and incoherence, and led bacteriology into a decline."

Tissot has done magnificent work in clarifying the picture in bacteriology and immunology. We have kept its significance for chiropractic separate and confined to the **Conclusions**, so that Tissot's work can be seen strictly on its own merits and in terms of the basic problems with which it deals.



## 57 COMMERCIALISM

Hume (p. 211) states: “Had it not been for the sale of sera and vaccines, nowadays grown to such vast proportions, Pasteur’s germ theory of disease might before this have collapsed into obscurity.” Tissot states (p. 347), “Dr. Charles Nicolle, professor at the College of France and Director of the Pasteur Institute of Tunis, said in an interview published February 3, 1934, ‘The Pasteur Institute has a chance of considerable financial aid with the preparation of sera and vaccines. It is a chance to return a favor to the state for endowments. Then it is much better to have the government as a customer than a protector. It assures more regular and better receipts.’ The Pasteur Institute is built on sera and vaccines and hence MUST sell. The Institute is THE pharmaceutical house of France. It is incredible to think that they don’t know how useless the nostra really are after all their ‘research.’ “

With regard to the French law of June 25, 1938, of compulsory inoculation against diphtheria, Tissot says, “This law is illegal, unconstitutional, because it violates the supposed freedom of the people. Dr. Chavanon, in *They Can Kill Your Child*, says, ‘A surgeon has no right to perform a necessary operation to save the life of a child without written permission of the parents, but does have the right to force an injection without permission, which is harmful and can kill a healthy child.’ And to top it off the stuff is totally useless. This law was passed by intense medical propaganda and lobbying with the line that total vaccination would cost the government less than the cost of really stamping out diphtheria.

“This is an odious and unconstitutional law. NO, the vaccines (inoculations) of anti-tetanus and anti-diphtheria do NOT vaccinate. Because they are totally useless and confer only a chronic stage of the disease, and because of the liability of ‘accidents’ their usage should be prohibited. Because anti-rabic inoculation is useless and is dangerous it ought to be outlawed to avoid killing more victims.

“It is impossible to vaccinate man effectively with BCG, and since it is virulent to man, it should be outlawed. At the time of publication (June, 1946) the Pasteur Institute was circularizing propaganda through the medical profession to curry favor for this vaccination and try to make it compulsory. A part reads, ‘It is possible to state most formally that the vaccination is without any danger and it is effective. To pretend otherwise one must be either misinformed or of evil intent.’ The Pasteur Institute put out similar propaganda on the diphtheria immunization, but it has caused many deaths since then. They also said that diphtheria immunization would cause diphtheria to disappear from France, but actually the number of cases has increased. The virulence of BCG is established clinically by THE NUMEROUS ‘accidents’ provoked, but Calmette systematically denies it in spite of the evidence.

“There must never be, under any pretext, reason for inoculating any living organisms, attenuated or killed, or a serum or other products of living organisms. It is the principle adopted by England and Holland to refuse to make these compulsory. With the inefficiency and the dangers of these sera known, will the French continue to use their children as guinea pigs for the sole purpose of the financial prosperity of the Pasteur Institute?”

Still, to this very day, “Pasteur’s school is vigilant and does not hesitate to attack fiercely any ideas that may unseat Pasteurian dogmas.” “They have impeded the research of the cause of disease and have ‘perfected’ their blunders only by developing other nefarious sera.” “The Pasteur Institute, the pharmaceutical house

of France is built on sera and vaccines. They must sell their product. They put out propaganda of bald lies in order to make vaccinations compulsory.”

Tissot finishes, as mentioned on an earlier page, with huge capital letters that: “These five inoculations ought to be prohibited.” None are effective; all are virulent, and many are very dangerous.

## 58 CONCLUSIONS OF TISSOT

Many years of intensive and extensive research revealed that prior information on cellular anatomy was erroneous due to technical artifacts. By studying undamaged cells Tissot discovered them to be made up entirely of two subcellular bionts. These tiny living units and their function really revise fundamental biological concepts. This in turn explains many previous enigmas of physiology. Quite obviously Tissot had to go on to study the significance of these revolutionary facts on abnormal, pathological states. Here he found that bacteriology must be brought up to date on the nature, function, and total pleomorphism of microorganisms. Their true interrelationship throws Pasteur's "germ theory" into discard, because usually the bacteria are incidental end products of an endogenous, degenerative pathology. The "germs" are the result, rather than the cause of disease.

The most inconceivable situation is that much of the true nature of disease and of microorganisms was already known, and had been published and expounded by A. Béchamp at the time when L. Pasteur was misleading the scientific world, first through sheer ignorance, but later through utter disregard for any one or anything except his own fame and fortune. By favoritism, power politics, and ruthless savagery, Pasteur foisted his false dogmas upon the world, where they are still a scourge upon mankind.

Terrible damage has been inflicted upon the human race and upon subhuman victims of Pasteur's inquisition, and the end is not in sight. Tissot writes, "By obstinately insisting on the asepsis of living beings —Pasteur made the worst error in biological sciences of which I know. Pasteur remained adamant to his death, after over twenty years of arguing in spite of actual proof of his errors before his own eyes." The Pasteurian school, by the arrest of scientific progress, has slowly directed medicine into an extremely grave impasse.

All due to the blundering over simplification of Pasteur and the vicious determination of men high in the world of science, irreparable damage has been inflicted—and the juggernaut still rolls on—and on —and on! Our contemporary scientists have been thoroughly indoctrinated. Untrained in the canons of rigorous thinking, ignorant of the history of their subject (fewer than half a dozen universities have chairs in the history of science), unaware for the most part of the existence of evidence controverting the dogmas to which they cling, lacking independence of judgment, and fearful of antagonizing either their institutional superiors or the financial interests which subsidize them, they flounder along, the hapless victims of a sub-culture which has perhaps irretrievably conditioned them. Willingly or unwillingly, consciously or unconsciously, they perpetuate the errors to which they are committed.

The situation is one of abysmal ignorance on the part of most physicians and bacteriologists, who are guided in their damaging efforts by an unscrupulous hierarchy who are simply running a big, an immense business in human lives. Some of these deluded "cat's paws" are sincerely trying to help relieve suffering humanity, but many of them are aware that "There is something rotten in Denmark," and that the pharmaceutical industries stink to high heaven. For this conclusion, we can readily understand why Tissot, who had the courage to call a skunk a skunk, quickly became *persona non grata* to the pharmaceutical monopolies, the Drug Trust. It is a

crying shame that there are not many more real men in this world with his courage and understanding.

## 59 TECHNOLOGY

Tissot demonstrated vividly the need for better technology. Heretofore the information on cellular anatomy has been derived largely from microscopy of cells prepared with harsh fixatives. The resultant artifacts are still taught as factual truth by histologists, while actually they do not even exist in living cells. The “mitochondria” are a fine example of this. Hence it is essential that these fundamental studies be rechecked for verification to avoid the continued teaching of errors.

Another vital principle is that what may be true in a test tube as a chemical reaction may be exactly the opposite in a living body, because of the nervous environment. This type of artificial, erroneous research when added to the faulty Pasteurian dogmas is just one more reason why there is so much fruitless “research” in pathology. All research men admit this fact, but continue to waste their time and our money.

The next big point is that normal horse serum and normal guinea pig serum are both virulent to man. They are foreign proteins. Two injections (remember Double Trouble) of simple egg albumin can kill a man. It, too, is a foreign protein. Yet, in spite of this well known fact, the pharmaceutical houses persist in peddling a nostrum which is not only a foreign protein, but one that has been otherwise poisoned. The amazing thing is that mankind has been able to survive in spite of this brutal punishment so well and so long. The wonder is that not more have been crippled and killed. Here again we have a vivid demonstration that the human body can be badly damaged and still survive.

## 60 TERMINOLOGY

Bacteriological and biological terms should not be interchanged. It is better to coin words in order to avoid confusion. Tissot's use of the term "B. coli" where he is actually referring to the granular biont is damaging to his case. The term *colon bacillus* should be confined to the function given it by bacteriologists. Perhaps it is better to speak of the basic organism, the microzyma, one elaboration of which is the colon bacillus. You will recall that Tissot suggested the possible development of the "dumb-bell," *haltère*, stick-shaped biont as a direct outgrowth of the fibriniferment, the micrococcic granule, the biont which is the primal chemist. Thus we use here the term microzyma as Tissot often does, and as Béchamp always did, to refer to the granular biont.

The term "bacterial form" has come to mean to most persons a mature bacterium. For that reason Tissot unknowingly confuses the issue when he says that all living organisms are made up entirely of the two bacterial units. Actually he is correct in the terminology in its pure sense, but because of the current connotation of the word bacteria, it would have been much better to coin a word such as biont. Bacteria are elaborate forms of perverted bionts and can be reconstructed into normal bionts again; so there is a very close relationship between the normal and the abnormal. Remember these few points when again studying this text.

Again a matter of popular connotation leads us to suggest the use of the term "filament stage," rather than mold. Actually the terminology of Tissot is exactly correct, but again it leads to confusion. Recall that the word "insane" originally meant inspired, which was a desirable and honored condition. Now by popular connotation it has a much less honored significance.

The same remarks are applicable to his use of the words "acute" and "chronic." The perverted biont form was observed to follow a definite cycle which usually can be correlated with the symptoms in the patient. Tissot, in his research, was primarily interested in the phase through which the bionts were passing. Consequently his use of "acute" and "chronic" is primarily in reference to the biont phase which usually does, but often does not, coincide with acute and chronic symptoms of the patient.

## 61 THERAPY

What is Tissot's therapy? This question can be countered with another: Did Tissot not already contribute much more than his share to the general human welfare? We could not expect to have him solve all our problems for us. Since he was primarily a research expert and did not care to damage his work with diversions of energy, he offered no therapy. In view of the work he did present, however, and its significance in general, it would be most appropriate to mention Speransky's therapy. Tissot demonstrated that the biont cycle is directly influenced by environment; the immediate environment overrides any significance of bacterial agents. Speransky demonstrated the nearly identical finding that the immediate environment of the cells (their nervous influence) is the governing factor. Throughout all pathology and in all therapies Speransky has demonstrated beyond question the supremacy of the nervous system. Speransky's ideal therapy is not neurosurgery, although he tried it extensively, nor is it novocaine blockade, though he used that, too. His objections are that these both cut the normal reflexes along with the pathological reflexes. He suggests that any therapy directed toward breaking up the pathological reflexes and re-establishing the normal reflexes will approach his ideal. He tried a general form of nervous massage, pumping, and criticized it because it is non-specific.

If we evaluate chiropractic in the light of these criteria, it stands up exceptionally well.

## 62 SIGNIFICANCE TO CHIROPRACTORS

Chiropractic is put on a more substantial basis by a thorough understanding of Tissot's work. Our clinical findings have always pointed toward bacteria as the result and not the cause of disease. Some of the chiropractors have philosophically ignored bacteria as though they were non-existent. Others have theorized that bacteria are scavengers. They have compared the "germ theory" ideology to that of flies as the cause of the manure pile. Those who just declare that bacteria are non-existent are like the toad beside the railroad track who remarked, "There is no such thing as a train and besides, the things can't go that fast anyhow."

Chiropractors can now rise above the confusion of bacteriology and properly evaluate that science, instead of disregarding it, as a few have done. Each and every chiropractor should take it upon himself to help educate the general public on this subject in a constructive way—not by derogatory remarks to the effect that nothing medical can be right any way, but by clear, sober, scientific evaluation of current teaching and practice in the light of the basic biological facts presented by Tissot and summarized in this text.



### **III Critique of Applied Bacteriology**

## 63 THEORY OF INFECTION

Mode of Entrance of Bacteria into the Body—Natural Defenses of the Body—  
Manner in which Bacteria Produce Pathology and Symptoms—Critical Comment.

“Not as adventitious, therefore, will the wise man regard the faith which is in him. The highest truth he sees he will fearlessly utter; knowing that, let what may come of it, he is thus playing his right part in the world—knowing that if he can effect the change he aims at—well; if not— well also; though not so well.”—Herbert Spencer: *First Principles* (p. 106).

This work is confined to those microorganisms which are termed pathogenic—those which, coming in contact with an environment favorable to them, aid in establishing symptoms. Let us examine the theory of infection. Theoretically, the human body, when in a state of perfection, is impervious to the entrance of these microorganisms; but, should a break occur in the skin or mucous membrane, an opportunity for entry is afforded. The skin is literally covered with these minute forms of life, and the digestive tract is even more so. But the body cavities lined with mucous membrane are considered external to the body. It is only when entry is obtained through the skin or mucous membrane that the interior is reached—and it is thus that microorganisms associated with pathology make their entry. Thus the skin and mucous membrane are passive physical barriers to the multitudinous bacteria in the air, food, and water, which are consumed or contacted. It should be remembered, however, that some bacteria, as in diphtheria, develop on the surface of mucous membranes and, without reaching the interior, produce symptoms by the toxin they secrete.

At various points in the digestive tract there is what may be termed a competitive bacterial activity wherein one form restrains another from gaining ascendancy.

Entry once gained, certain reactions take place. If the environment is unfavorable it is so either because of existing chemical conditions or else because of chemical reactions which are stimulated by the germ's presence—the specific nature of these reactions being determined by the many factors entering into the phenomenon. Normal lacrimal fluid and normal gastric juice are unfavorable media for bacteria. They are, of course, considered outside the body, but just as they are poor culture grounds, so are the blood and lymph normally—or may become so upon adequate or specific stimulation. Thus, there is both a passive and an active chemical reaction to bacterial entry. This is known as the factor of resistance and is responsible for the different types of immunity which will be dealt with in a later chapter.

If the environment is favorable, bacterial metabolic and reproductive activities are initiated. Feeding is accompanied by bacterial end-products; reproduction, by a geometric multiplication of these toxic materials plus a conversion of body tissues into less complex forms, and in some cases the accumulation of such material. The disturbance is called an infection, and it can give rise to nearly every discomfort and symptom in the category of disease. Circulating toxins cause lassitude, dizziness, weakness; localized, they cause inflammation (redness, heat, pain, swelling). When

excreted, disturbances are manifested such as diarrhea, skin eruptions, abnormal urine, and germ- or toxin-laden respirations. In anthrax, the tissues are deprived of oxygen. In tetanus, the toxin alters normal nerve conductivity. In milk-leg, an embolus of bacteria blocks the blood vessel. In botulism, the bacteria themselves are as poisonous as some varieties of mushrooms. In ptomaine poisoning, the patient is poisoned by decomposition products derived from food through the action of bacterial enzymes. The modes of action of bacteria that may be detrimental to the body are thus seen to be numerous. It is definitely established that bacteria are associated with many diseases and have a causal relation to these diseases under certain conditions.

To the critic who is a logician a glaring error in the major premise of the theory of infection is at once apparent: a cause must precede an effect. Yet the opening paragraph states that germs cause disease and at the same time postulates perfection by stating that a sound body is impervious to disease germs. Of two contradictories at least one must be false. Either disease precedes germ entry, or germs per se do not cause disease. This is an inescapable dilemma, and it is obvious that another approach must be made. Fundamental premises underlying a popular theory are herein involved. Such a challenge must not be ignored, for it endangers the superstructure of practice which has been erected on the theory.

Since Koch's postulates have served bacteriologists as a support for this premise, they must be critically examined. Citations used herein are from recognized authorities. When diametrically opposite conclusions can be drawn from the same evidence, as is done herein, the time is propitious for critical thinking, which alone will demonstrate the misapplication of the term "science" in certain quarters. As Koch's postulates will serve equally well two contraries, it is advisable to restate them in more rigorous form; otherwise, they are scientifically useless. This shall be dealt with in the chapter ***Bacteriology and the Scientific Method***. For the present we shall confine our attention to the theory.

Gould's Medical Dictionary, 1926, gives Koch's law or postulates thus:

"The specificity of a microorganism is conclusively demonstrated when the following conditions are fulfilled:

1. The microorganism must be present in all cases of the disease.
2. It must be cultivated in pure culture.
3. Its inoculation must produce the disease in susceptible animals.
4. From such animals it must be obtained and again cultivated in pure culture."

To which we add a contribution from Park in Stedman's *Reference Handbook of the Medical Sciences* (Vol. III, p. 601): Failure to produce the disease by means of any other microorganism.

Plus an additional postulate which is frequently overlooked and an indirect reference to which is made in Jordan's *General Bacteriology* (8th Ed., 1924, p. 274): The microorganism must never be found apart from the disease.

Without in any way violating these postulates (and the appended requirements of Park and Jordan) we wish to call attention to the fact that the major premise of the theory of infection (that germs cause disease) fails to consider or adequately account for the possibility of infectious disease arising spontaneously; that is, the evolution of virulent microorganisms from non-virulent forms—said evolution

occurring after the disease process has started and resulting therefrom. It must be understood that spontaneous generation is not the issue.

The phlogiston theory “was erroneous only in regard to one question” (*Encyclo. Brit.*), but note that that simple error set back scientific advance for a century by substituting a dogma for a fact. We hold this error is paralleled in the germ theory, the speciousness of which is nowhere better demonstrated than in the half-truth that “the specificity of a microorganism is conclusively demonstrated when these conditions are fulfilled.” In spite of the term “conclusively,” the case remains open—for all the facts are not adequately accounted for by Koch’s postulates. They are acceptable as far as they go, but they are incomplete and scientifically inadequate.

This will immediately become clear by drawing an analogy from the phenomenon of spontaneous combustion. We shall use this analogy as a control on our reasoning processes—nothing more. To deny the reality of this phenomenon merely because artificial fire-making is so very much more common or because the former may never have entered into one’s direct experience would be a ridiculous position to take. Yet that is exactly what is done in the case of diphtheria, for example. Because the symptoms of this disease may be artificially produced by inoculation of the germ or its toxin, therefore the spontaneous appearance of the disease is considered a superstition. Examination of the matter in the light of Koch’s postulates, however, reveals that there is no discrepancy between such a view and these postulates. If the bacillus evolved from the tissues, then, of necessity, it would be present in every case of the disease—as well as sometimes present without the disease (always a weak spot in the postulates with abundant facts to support the charge); in addition to being recoverable after the disease, and producing the disease in another susceptible subject. An infinite number of experimental fires would not prove that spontaneous combustion never occurred, and an infinite number of artificially-produced diphtheria cases does not discredit the possible spontaneity of disease. To lay down postulates dealing with such items as heat, light, flame, gas, etc., always present in combustion, does not adequately deal with the origin of combustion, which may have a multiple cause. And the postulates of Koch, likewise, do not eliminate all the possibilities in the cause of infection. There is more than an academic question involved herein, and it is essential that it be clearly understood. Upon this ambiguous theory sciolistic practices have been built, and in the light of this inquiry we propose examining them. If the major premise of the theory of infection falls, then the practice based upon it will likewise fall. This is an instance of *falsus in uno, falsus in omnibus*. Further, Koch’s third postulate as given by Gould, takes on quite a different significance when viewed in the light of our spontaneous combustion analogy. If the artificial production of disease by inoculation is identical with disease produced in the ordinary way, and if it disproves the possibility of spontaneous disease, then artificial fire disproves the reality of spontaneous combustion—and this, of course, is a fallacy.

Diphtheria has been selected, as it is the disease which orthodox bacteriologists concede is their best example, and the only one which immunologists cite as having almost the unanimous support of the medical profession in the matter of inoculations. Let us analyze some remarks by Jordan on diphtheria, in his *General Bacteriology*:

“Although Loeffler’s observations favored the view that the bacillus cultivated was the cause of diphtheria, Loeffler expressly disclaimed the assumption that this was actually the case (1), largely on the ground that the bacillus was not found in all

cases of clinical diphtheria (2), while, on the other hand, it had been found by him in the throat of a perfectly healthy child (3). The significance of such findings is now more clearly understood. A similarity of clinical symptoms does not always betoken causal identity (4). So far as the local manifestations are concerned, streptococci can produce a condition apparently indistinguishable from that in which the Klebs-Loeffler bacillus is found (5). Again, it is now known that the diphtheria bacillus is occasionally present in the healthy throats of persons associated with diphtheria patients (6). Continued investigations by various observers showed that the Klebs-Loeffler bacillus was always present in the false membrane of diphtheria (7), and in 1888-89 Roux and Yersin triumphantly demonstrated the etiologic relation (8) of the bacillus to the disease by showing that it formed a toxin which was capable of reproducing with singular fidelity the characteristic symptoms and lesions (9). (p. 274) . . . Diphtheria of the conjunctiva sometimes occurs as the result of a diphtheritic patient coughing or sneezing into the eyes of the attendant physician or nurse (10).” (p. 280)

1. The insinuation here is that Loeffler was incompetent to draw a conclusion from the evidence. As he was well grounded, this is an affront. His difficulty was due to the fact that

2. His findings did not corroborate or meet the requirements of Koch’s first two postulates, and he, therefore, was in a quandary, as it is evident that he was too honest to make the facts fit the theory. Note that Jordan does not directly comment on the absence of the bacillus in some cases—in spite of the statement: “The significance of such findings is now more clearly understood.” Furthermore,

3. This (never found apart from the disease) is not a part of Koch’s postulates as given by Gould or Conn but has been eliminated, as we shall prove later, for the convenience of explaining the “carrier” theory. Note, however, that this postulate is included in the *Lancet* editorial quoted subsequently.

4. Loeffler, the master, is here accused of confusing identity with similarity. He was led astray, Jordan thinks.

5. “The specificity of a microorganism . . . !” says Gould. Suppose a patient has both streptococci and Klebs-Loeffler bacilli present. Which germ, if either, is the “cause” in the situation? It is Jordan’s opinion that the expert Loeffler did not recognize his own bacillus, but was muddled by streptococci. This reads like a specious argument—almost as much so as his remark on

6. Loeffler’s finding of the bacillus “in the throat of a perfectly healthy child,” which should be cited in favor of Loeffler’s position rather than against him. He is fully vindicated by Jordan’s admission that “the diphtheria bacillus is occasionally present in the healthy throats of persons associated with diphtheria patients,” although Jordan understates the facts by saying “occasionally present” when he should say “frequently present,” and the phrase “associated with diphtheria patients” is not justified in many instances. Jordan may wish it so, but the facts contradict him.

7. This is an indirect reply to the absence of the bacillus in some cases. Thus he evades the issue by dealing with an effect and not a cause. That the bacillus is always present in the pseudo-membrane does not establish its causal role at all—any more than flames prove incendiarism. Why not refute Loeffler and show the germ always present prior to the onset of the disease—as a causal agent must be—and not deal with an effect? The burden of proof is upon Jordan and his school, and his case is not proved but confused. The case for diphtheria is still quite vulnerable in spite of the naive contention that it is the best example of the germ theory of infection.

8. The term “etiologic relation” is an unwarranted assumption, and the climax is reached with

9. A *post hoc ergo propter hoc* argument—so far as the major premise of the germ theory of infection is concerned, while the case taken from

10. Jordan (p. 280) shows how a spark might start a fire in inflammable material, but, of course, does not invalidate the phenomenon of spontaneous combustion—or spontaneous infection.

London *Lancet*, March 20, 1909 (Vol. 176, p. 848): “The organism must always be discoverable in cases of the disease and not in other diseases or in health; that it can be cultivated for many generations outside the body; that when injected into another animal it will cause the disease; and that it is then to be found in this animal.

“It must be acknowledged that all these postulates are complied with very rarely indeed, if ever. It is not at all rare to fail to find the causal organism in an individual case of disease, and the explanation usually given is that the search has not been as yet sufficiently thorough. Again, many organisms which are considered to be causal are frequently to be found in healthy persons. The organisms of enteric fever, of cholera, and of diphtheria may be cited as examples of this, and to explain these facts we have to invoke the idea of healthy ‘carriers’ of disease. When a causal organism is injected into an animal often it happens that it gives rise to a disease bearing no clinical resemblance to the original malady. When the pneumococcus, isolated from a typical case of pneumonia, is injected into an animal, such as a rabbit, it will produce, not a pneumonia, but a general septicemia, and this even if the injection is made into the lung itself. Thus we cannot rely on Koch’s postulates as a decisive test of a causal organism . . . The question then naturally arises: Are these so-called causal organisms truly causal, or are they only ‘secondary invaders’? They may, perhaps, be normal inhabitants of the body, only assuming importance in disease . . .”

Note that although this last statement is at variance with the proposition that germs cause disease, yet it does not violate Koch’s postulates. This is definite evidence for the contention that Koch’s postulates are scientifically inadequate. They are insufficiently rigid and serve only one school of bacteriologists. Says Hadwen: “If the postulates of Euclid cannot be accepted, how can we prove his problems?”

In summary, we see that Koch’s first postulate assumes that the mere presence of the germ proves that the germ caused the disease—a pure assumption. Further, Jordan throws this postulate into confusion, showing that “streptococci can produce a condition apparently indistinguishable from that in which the Klebs-Loeffler bacillus is found.” Further, on Jordan’s admission, we have disease without germs, and germs without disease. This leaves the case for bacterial infection as presented by Jordan’s school unproved. However, medical practice is based on Louis Pasteur’s dogma, “A germ is a disease and a disease is a germ,” the converse statement of which is: No germ, no disease. Millions of facts show the absurdity of this notion—and impeach the practice based upon it as irrational.

Specific instances of disease arising spontaneously within the body—the germ evolving after the inception of disease processes and having its specific characteristics determined by the location and condition of the site of such process—are of daily occurrence. The records cite case after case of infectious disease wherein it is admitted that germ transmission was impossible, and yet with blind fanaticism an extraneous microbial prime solitary causal factor is dogmatically

insisted upon—or occasionally an intrinsic latent form admittedly becomes virulent. But never is the possibility of spontaneous biological mutation even considered.

Jordan says, in *Chemistry in Medicine* (p. 558): “In some instances the ‘germ theory’ plainly has only the status of a theory and can claim little or no basis in fact.” On page 550, he says: “It will be generally conceded that as regards diphtheria the theory that a microbe is the cause of the disease has passed from the realm of theory into that of observed fact.” The fact that millions of infants and children have diphtheria bacilli in their throats and do not have diphtheria should be sufficient to destroy the unmodified premise that diphtheria bacilli cause diphtheria. And, of course, Jordan will modify it; and when he does, it will be seen that the additional factor which he allows will be the real, prime factor—and it will be recognized that this additional factor will in turn modify the practice which Jordan has superimposed upon the theory.

The admission that “the ‘germ theory’ plainly has only the status of a theory and can claim little or no basis in fact” would indicate that those who formulated or contributed to this theory took every advantage of the right of free men to theorize, a practice which readily becomes a habit, especially of those theorists who are but little or not at all concerned with the observations of the phenomena upon which they theorize. To them, the facts concerned therewith are of course quite irrelevant. One occasionally suspects them of deliberately attempting to lead serious-minded persons astray. More frequently, it appears that they indulge simply for the pleasure they themselves derive. Those falling under the latter classification are easily recognized and are quite harmless. It so happens that unscrupulous persons are capable of catering to the fancies of such theorists to the extent that, through clever persuasion they attract attention, and, seeming to possess certain elements of value, their theories may be thus foisted upon others and exploited. When such incidents are discovered, people are apt to place the responsibility upon the innocent theorist, whereas he himself has been victimized, and the only crime with which he might justly be charged would be with attempting to amuse himself, and either not realizing or not caring that sensible men regard theories as subordinate to factual knowledge.

A so-called theory that is not confirmed by all the observed facts, or, on the other hand, has not been applied satisfactorily in explaining the phenomenon to which it refers, aside from being purposeless and without utility, is not a theory at all. It cannot even be considered a hypothesis. It may, with propriety, be regarded as a postulate, but even thus, if any considerable exceptions are noted, it must obviously be rejected as a mighty poor one.

Warning is hereby issued not to confound the theory of infection with the practice of serum therapy. They are two separate and distinct propositions—either of which must stand or fall on its own merits. Serum therapy is nowhere justified in Koch’s postulates. Nor is it justified should error be shown anywhere in the foregoing. This practice is a separate and distinct subject and will be dealt with as such in a later chapter.

It should be apparent to a logical mind that the case for diphtheria has been fairly presented. All claims save one have been accepted at their face value. The exception is the interpretation of cause—which has heretofore been incompletely presented—how very incompletely can be judged by the present inquiry. The two-column chart at the end of this chapter may serve to clear up the matter even further—serving as a two-column summary of the conflicting theories and practices of two different schools on cause, treatment, and prevention.

Nor is the inquiry confined to this alone—the single phase of a complicated problem. Bacteriology has been studied and exploited from the anthropocentric point of view. Biologists have specialized in this field to such a degree that even the literature in one division is practically incomprehensible to workers in another, and the technic is impossible except for the few. And it is because of this narrow, overspecialized study of pathogenic bacterial forms for human benefit that many phases of bacterial activity remain mysteries. What ultimate disposal is made of a dead bacterium? Does its body invariably undergo complete autolytic disintegration? “We have but little satisfactory knowledge of the fate of the typhoid bacillus in the modern sewage purification processes,” says John F. Norton, Ph.D., University of Chicago, in *Chemistry in Medicine* (p. 329). He is interested in where the carcass goes, but it would be instructive to know what forces cause its ultimate decomposition, and whether it may not be composed of ultra-microörganic entities which become units in the natural order of events.

This closing of the case with the completion of some immediately utilitarian process is the habit of the specialist—an indictment which is rapidly taking form. “Unfavorable results of excessive specialization in the subject are strikingly revealed,” says Dr. C. Watson, in the London *Lancet*, November 29, 1924. (Vol. 207, p. 1150)

“Pathological bacteriology is now hampered everywhere by the absence of knowledge on fundamental biological matters . . . What is a species of bacillus?”—Editorial: London *Lancet* (Vol. 207, p. 609)

“Bacteriology is now sterile; no discovery of first-rate importance has been made for many years, and we are constantly being brought to a standstill for want of knowledge of how bacteria live and move and have their being—knowledge of a sort which may be called botanical. We do not even know how bacteria multiply and whether they have anything in their life history corresponding to a sexual process. Not knowing that, we cannot evaluate the possibilities of variation and mutation, questions which lie at the root of many problems of epidemic disease; nor can bacteria satisfactorily form the subjects of a number of biological experiments for which their small size and rapid multiplication so admirably fit them. And this state of affairs is due to the fact that bacteria have been studied as causes of disease; no one has loved them for themselves. Practice has outrun theory so far that practice itself is coming to a dead end.”—A. E. Boycott, Graham Professor of Pathology, University of London: London *Lancet*, November 15, 1924 (Vol. 207, p. 998).

Professor Boycott says that technological progress has been prodigious, but it must be evident that a task awaits some enterprising philosophical scientist who will collect and correlate and coordinate all the scattered facts in bacteriology and thus permit the subject to be viewed from a rational standpoint. Additional problems will be presented in later chapters. For the present, the inquiry on the theory of infection must rest while some of the elements comprising the superstructure are examined.



## Opposing views of germ diseases

### *Cause*

<p>Germ, from extraneous sources, enter the body, multiply, and by direct action on tissues, or indirectly by generating toxins, cause disease.</p>	<p>Disturbance in the metabolism of cells by trauma, exposure, faulty nutrition, altered innervation, etc., results in heat and congestion. Undrained waste matter acts upon cells, the cell entity disappears, and the microsomes (subcellular units) become microbes.</p>
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### *Treatment*

<p>Kill or inhibit the germ. Neutralize the toxins. If possible, extirpate the site of bacterial activity.</p>	<p>Realize that bacterial action is controlled by the laws of metabolism. By-pass the germ; strengthen the tissues by measures calculated to correct metabolic disturbance. Allow bacterial action to go on until it ceases spontaneously.</p>
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### *Prevention*

<p>Anticipate bacterial activity by the use of biological products (stimulating or suppressing, as the case may be, the normal reaction to bacteria). Extirpate organs which show signs of bacterial activity or are likely to show such signs in the future.</p>	<p>Live hygienically, modifying, in so far as possible, the environment in the interests of the individual and adjusting the machinery of the body for more efficient adaptation to environment.</p>
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### *Criticism*

<p>This is orthodox bacteriological theory. It gives no consideration to the possibility of disease of spontaneous origin. It is supported by many superficial observations, but lacks the rigid proof demanded by scientists in other departments of knowledge.</p>	<p>This is an outgrowth of the work of Béchamp, further elaborated by Tissot, discussed in an earlier chapter. While it is not fully verified, there are no facts which contradict it. It explains a multitude of facts which are incompatible with the orthodox theory and throws light on some of the most perplexing problems of modern bacteriology.</p>
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“All those who oppose intellectual truths merely stir up the fire; the cinders fly about and set fire to that which else they had not touched.”—Johann Wolfgang von Goethe

To deal with this phase of the subject will necessitate an examination of some material already presented, but, as there is a different objective herein, it is felt that the charge of redundancy will not be made. It is proposed to inquire into the validity of Koch's postulates as quoted by certain authorities for the purpose of determining how closely scientific procedure is observed by our contemporaries.

In scientific practice postulates are formulated for the express purpose of rigidly encompassing all the possible situations and problems which may arise in a theory. Postulates are inclusive and exclusive; they act as controls on the process of theorizing; and to do this effectively they must embrace not only an affirmative statement but also its converse, a negative statement—at least by direct implication. Koch's original postulates meet these requirements: they are all-inclusive; each one is a universal. The intent was to apply them in their complete and rigid form; otherwise, there was no scientific reason for formulating them. Imagine, then, the temerity of some authors in quietly modifying these rigid rules to suit their personal interpretation of the facts. It is true that the postulates in the original German are cumbersome and involved, but they contain several basic propositions which, taken altogether, preclude the possibility of error; their application either confirms or disproves the theory, and under no circumstances are they to be altered without an ample explanation being submitted. Note that in this literal translation from the German (very kindly provided to us in the original German of Koch by Koch's biographer, Dr. Bruno Heymann, of the Hygienic Institute of the University of Berlin), the first two comprise a positive statement and its converse. Briefly, to establish the pathogenic specificity of a microorganism, it must be

1. Found in each and every case of the disease
2. Not found with any other disease
3. Isolated in pure culture
4. Able to produce the disease again

Stedman, Goepf, Gould, and Conn delete the second element in the postulates, “not found with any other disease,” because it nullifies or complicates their theory—and effectively abandons the position postulated by Koch.

Note hereunder that No. 1, as given by Fomon, comes nearest to Koch No. 2, but, as it does not even *imply* its converse, it is obvious that Fomon's quotation is faulty. Fomon: *Medicine and the Allied Sciences* (Vol. I, p. 9):

1. The same organism must always be present in the same disease.
2. The organism must be isolated, and grown in artificial media.
3. On inoculation of culture in animal, must produce the same disease.
4. The same organism must be isolated from the lastly inoculated animal.

Stedman refers to the postulates as Koch's law. It is unfortunate that the advocates of the law have seen fit to introduce a joker into it so that a loophole of safety is provided for the violator. When a bacteriological law can be altered to suit one's need, it certainly does not speak very highly for the science. The literal observance and strict respect of this law is imperative and the penalty for its violation is exacted when a low evaluation is placed upon the work of the offender.

Stedman's *Medical Dictionary* (p. 487): To establish the specificity of a microorganism, it must be present in all cases of the disease, inoculations of its pure cultures must produce the same disease in animals (when it is transmitted to such), and from these it must be again obtained and propagated in pure cultures.

Understand that we are dealing with a science—not antediluvian folk-lore; with the precepts of a man who was contemporary with these authors; with modern German and not Sanskrit; with a quotation from a master's literature which requires no revision by his fellows. If they will take such liberties here, what will they do to statistics and other matters? We take exception to Arthur R. Guerard's statement that "all the conditions have been fulfilled for diphtheria," etc., and also to his inspired postulates which we are supposed to assume are Koch's in *Stedman's Medical Reference Handbook* (Vol. I, p. 845):

"All the conditions have been fulfilled for diphtheria which are necessary to the most vigorous proof of the causative relation of a given microorganism to an infectious disease, viz:

1. The constant presence of the organism in the lesions of the disease
2. The isolation of it in pure culture
3. The failure to produce the disease by any other bacteria
4. The additional demonstration of the immunizing value of the specific antitoxic substances developed in animals subjected to injections of diphtheria toxin.

"In view of these facts we are justified in concluding that all cases of true or primary diphtheria are due to the Klebs-Loeffler bacillus."

Note that Goepf devitalizes the postulates in the form which he substitutes for Koch. Goepf: *State Board Questions and Answers* (p. 314):

1. The microorganism must be found in the tissues, blood, or secretions of a person or animal sick or dead of the disease.
2. The microorganism must be isolated and cultivated from these sources; it must also be grown for several generations in artificial media.
3. The pure cultures, when thus obtained, must, on inoculation into a healthy and susceptible animal, produce the disease in question.
4. The same microorganisms must again be found in the tissues, blood, and secretions of the inoculated animal.

Gould invalidates the rigidity of Koch's postulates with his contribution; he calls it a law and strikes out a clause. *Gould's Medical Dictionary*:

1. The microorganism must be found in all cases of the disease.
2. It must be cultivated in pure culture.

3. Its inoculation must produce the disease in the susceptible animal.
4. From such animals it must be obtained and again cultivated in pure culture.

Conn adds his worthless substitution to confound the gullible. Conn and Conn: *Bacteriology* (p. 252):

1. Constant presence of the organism in animals suffering from the disease
2. Its isolation
3. Production of the disease upon experimental inoculation, and
4. Recovery of the same organism from experimentally inoculated animals.

Jordan neglects to give Koch's postulates, per se, but makes damaging concessions which are worth examining. In *Chemistry in Medicine* (p. 546), he writes:

1. The germ is present in the disease. ("It is sometimes found in healthy persons who have been in contact with diphtheria patients or in children recently convalescent from the disease. In many persons, too, microbes resembling the Klebs-Loeffler bacillus, the so-called diphtheroids, are present, but an experienced observer is generally able to distinguish them quite readily from the bacilli found in diphtheria. There is, therefore, a characteristic microbe invariably present in the characteristic membrane of diphtheria *and found elsewhere only under conditions where as a rule its presence can be readily interpreted.*") (Emphasis by JRV)

Jordan and Burrows, in *General Bacteriology*, (14th ed., page 567) list many non-virulent bacteria which resemble the diphtheria bacillus so closely that "*the diphtheria bacillus must be identified by the virulence test.*" Thus the only absolute identification is a pathological response when inoculated into a susceptible animal.

2. It can be artificially grown, and the inoculation of its pure culture will cause many of the typical symptoms of diphtheria in a guinea pig.

3. In fatal cases similar pathological changes occur in human diphtheria and its inoculated analogue in animals.

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4. The toxin in the Klebs-Loeffler bacillus will cause some of the most characteristic symptoms and lesions of diphtheria.

5. Small, non-fatal doses of diphtheria toxin produce antitoxin and immunity.

6. The Schick test demonstrates susceptibility or resistance, and is another bit of evidence as to causal connection.

Jordan devotes almost four pages to this specific subject and terminates with the reader bewildered. It is just a barrage of words to confuse and confound the student into accepting conclusions which are highly questionable.

These conclusions have been dealt with elsewhere in this book. The present task is to demonstrate that Koch's postulates have been altered and obscured in an unwarranted manner. .

Notice, in No. 1, how breezily Jordan deals with Koch's second postulate. His words show that he is aware of the requirement and does not want to evade it entirely, but, when he concedes that diphtheria provides his very best evidence, it is obvious how weak his position is and how necessary it is to rationalize or modify Koch's rigid postulates.

Postulates must embrace both poles of a proposition: otherwise they are worthless. While this may seem superficial or overexact or subtle, its importance becomes apparent immediately upon the propounding of the speculation, "Suppose the germ is found in a healthy person?" If the postulates as stated do not embrace or imply this possibility they are invalidated by the facts. The fact of the matter is that Koch's second postulate does, but recent authors have seen fit to delete or obscure it—a very regrettable violation of a supposedly true and inviolable postulate.

Jordan's remark (*General Bacteriology*, p. 274), "Although Loeffler expressly disclaimed the assumption (that the cultivated bacillus was the cause of diphtheria) largely on the ground that the bacillus was not found in all cases of clinical diphtheria, while, on the other hand, it had been found by him in the throat of a perfectly healthy child," is without point unless it is explained that Loeffler was trying to apply Koch's postulate, that the pathogenic microorganism must never be found apart from the disease. (Emphasis by JRV)

Note that Loeffler did not tamper with the postulates, but "expressly disclaimed the assumption" which he considered insufficiently substantiated—the attitude of a true scientist.

The attempt to rationalize Loeffler's dilemma betrays Jordan by interms "health" and "disease," that every individual is to some extent diseased. No person is anatomically and physiologically perfect. "People who have never had pneumonia, diphtheria, poliomyelitis, scarlet fever, and many other infections possess antibodies and antitoxins, the only explanation of which is that at some time they have had a light, unrecognized sickness or that they were for a time healthy carriers of microorganisms," says Dr. W. H. Park, *The Newer Knowledge of Bacteriology and Immunology*, (p. 934) If an examination fails to disclose symptoms and abnormalities, and if there obtains a personal feeling of well-being and the ability to carry on, the individual is ordinarily adjudged healthy. These factors may indicate an individual's efficiency, but it is quite evident that they do not satisfy our definition. There is, therefore, an ideal state of health which is never attained, and a real state of existence which consists of more grades and degrees of disease than it is possible to classify and enumerate. However, one may be quite efficient in spite of that fact. But personal efficiency is not the criterion of disease processes. The constant presence of these processes becomes more clear when we consider those diseases in which pathogenic microörganic metabolic activities are manifested.

The human body is a finely balanced and highly sensitive organism. Coordination between all its parts is imperative to perfect health, and yet how infrequently is this coordination observed! One hundred per cent coördination equals perfection, yet where do we see it? Incoördination is equivalent to disease—functional or organic. In the complexity of organic processes some measure of abnormality is constantly present. The universality and omnipresence of incoördination, or abnormality, or disease, is of *primary* consideration. Tissues must break down. The causes of their breakdown are many—one of them being ordinary catabolism, a physiological process. (Gerontological deterioration and normal catabolism are not here being confused with tissue degeneration, but the line of demarcation is so poorly defined that one merges into the other.) To postulate perfect function in a living being of man's complexity is simply to dream of ideals. It *does not* exist and *can not* exist. Further, to seek for extraneous causes, germs included, without first understanding this supreme factor is to indicate a lack of scientific knowledge; while postulating perfection and then calling an extraneous

agent, germs (which are admittedly impotent in the presence of perfection), a first cause, is pure obscurantism. Pasteurians assume that the healthy body is a chemical and physical unit of perfection immune to germs. How can foreign germs cause disease in such a body until *after* disease has broken down immunity?

It is implicit in the germ theory that the germ is the specific causative agent in diseases accompanied by bacterial activity. The fact, however, that the germ does not invariably initiate the disease has led the proponents of the theory to acknowledge that some other factor is involved. They have sought to clarify the matter by asserting that the other factor is a predisposing cause, and the germ, the exciting cause.

Pretense at simplifying the issue by such sub-divisions as predisposing and exciting causes has only added to the confusion. A predisposing cause which enters into the experience of every individual (either with or without the disease) is no cause at all, unless every individual is similarly affected (for example, age, season, climate). If it is shown that one of these factors—age, season, climate, etc.—has had a bearing on the production of the disease, then there must have been a prime reason why it was so in this particular instance. This, then, takes on the form of an idiosyncrasy or diathesis—of organic origin. When the age, season, or climate becomes a factor for certain individuals and not for all, then the prime cause must be resident in the individual's organic processes. He simply does not make the adjustment to ordinary environmental factors and hazards which work no hardship upon others. To raise any of the factors of ordinary environment up to the dignity of a primary cause is to attach unwarranted importance to these incidental factors and simultaneously to obscure the investigation of the primary cause. Such factors are not efficient causes, and to indulge in such glittering generalities is equivalent to ignoring the true cause.

Maud Slye, in the *American Journal of Cancer*, in speaking of the inheritance of cancer susceptibility, says that “an external factor acting with internal factors upon a susceptible soil is probably the cause of cancer.” (*New York Times*, July 15, 1933) So at last investigators candidly recognize a multiple cause of disease. Many years ago Tripier said: “A single cause of disease is no cause at all.” Some sections of science certainly move slowly.

William F. Petersen, in *The Newer Knowledge of Bacteriology and Immunology* (p. 1087), says:

“The establishment of the etiological epoch in the seventies of the last century brought with it an utter disregard of the reaction of the infected host. The simple equation, bacteria plus host minus disease, sufficed. But finally an impasse was reached; certain facts could no longer be ignored. There were differences in susceptibility; and among the susceptible there were differences in resistance, differences that could not be explained on an immunological basis.

“Even during the triumphant bacteriological era competent observers called attention to certain non-specific phenomena. . . . Not only was typhoid fever terminated abruptly by intravenous vaccine injections, puerperal fever by coli vaccine, erysipelas and arthritis by milk injections, paresis by tuberculin, skin diseases by autohemotherapy, but even diphtheria was cured by normal instead of antitoxic horse serum.

“Whatever may have been the clinical basis for this intense interest, psychologically probably it did represent a protest against the all-too-narrow view of the immunologist concerning the nature of recovery from disease. Specificity and

specific antibodies obviously play a role in recovery; quite as obviously they are not the sole factors concerned.”

Such being the case, are immunologists to be granted *laissez faire* in conditioning the internal factor and ignoring both the external factor and the susceptibility of the soil? The internal factor and the susceptible soil constitute the predisposing cause in the form of a morbid imminence—the presence and degree of which is proved by such tests as the Schick and Dick. Morbid imminence under the theory of infection is considered an incidental or secondary factor, the prime cause of infection always being the germ. It is perfectly factual and logical to say, however, that this morbid imminence or departure from normal, pathologically speaking, constitutes disease—and only predisposes to an additional factor, the germ. In other words, disease processes are under way before the germ’s presence is demonstrable; morbid imminence is equivalent to disease.

A break in the skin or mucous membrane is one of the factors known as lowered resistance. In view of the fact that only certain individuals take a specific disease, human beings are said to possess a vaguely-defined, all-inclusive resistance factor which is the determinant in whether or not infection occurs. The conception of resistance is extremely vague; it includes external and internal factors of either physical or mental origin. The factors may be so conditioned as to produce susceptibility to infection in practically every organism. In other words, any factor which promotes a departure from normal lowers resistance and predisposes to infection in proportion to its deviation or fluctuation away from normal. There is a factor in the processes of the immune individual which makes possible his adjustment to endocrine and other physical and chemical changes (of a physiological and anatomical nature) and to ordinary environmental changes; lack of this factor is the predisponent of environmental susceptibility primarily, and bacterial multiplication secondarily or sequentially.

If it be suggested that all this is generally known and admitted, the reply is that practice does not indicate any such thing. Popular therapy proceeds on the naive assumption that the germ is all that need be considered as a causal agent, while the prime factor of pathology which is of internal origin, is allowed to remain exclusively in the realm of theory. This predisponent arising internally is the factor which must be the bedrock approach in therapeutics. To give the germ primary consideration obscures determining the organic factors which make the germ’s presence possible. The germ is a concomitant of infection—not necessarily an antecedent. Walter R. Hadwen, M.D., maintains “that tuberculosis of the lungs is not a bacterial disease, but a physiological process.” In preventive therapy the natural factors of predisposition must be rationally determined and dealt with, while in treatment they must not be ignored, as is so commonly done.

Disease is a process, and not an entity. It has no static existence *per se*, but microorganisms, and the disturbance accompanying their metabolic activities are very much in evidence in certain diseases. Note the vastness and complexity of the subject: The power of *B. botulinus* to affect the tissues of almost every individual; the absolute harmlessness of the smallpox virus when swallowed; the apparent avirulence of *B. acidophilus*; the extreme virulence of the Klebs-Loeffler bacillus; the high potency of the anthrax and tetanus bacilli; the tenacity to life or vitality of *B. tuberculosis* contrasted with the avitality of others; the thermophilism of the malarial parasite in contrast to *treponema pallidum*. These are but some of the extreme characteristics of the microorganisms which are associated with man. And all

degrees intermediate between these extremes can readily be demonstrated. Add to this the variations—racial, geographical, and individual—to be found in the human being, and immunology assumes proportions so great and so complex as to be beyond the ken of any individual.

Multiply the number of individual humans by their idiosyncrasies, times the number of kinds of bacteria, and again by the characteristics of these bacteria, and you approximate the number of factors entering into the problem of immunity. Says Jordan: “Not only do the nature and state of the individual play an important part in determining the occurrence of infection, but the conditions influencing the infecting agent itself are also of great importance.” The first multiple, i.e., the personal equation or idiosyncrasy, is popularly known as immunity or insusceptibility. Says Jordan: “Natural immunity sometimes depends upon the simple fact that a microorganism which finds favorable conditions for multiplication in one species of animal meets with unsuitable conditions in another species.”

This is indeed a very simple definition of immunity; but is it not possible to simplify it still more? Is it necessary to modify it at all? Is it not a fact (almost a platitude) that microorganisms only multiply where conditions are suitable? Are not all the requirements met in the generalization that immunity depends upon the simple fact that a microorganism meets with unfavorable conditions? Of course, the specific details of enumerating and describing the many elements involved is far from simple.

To begin with, immunity or insusceptibility is divided into two classes: *natural* and *acquired*. When an individual fails to develop the symptoms of diphtheria and other members of the household are down with the disease, he is said to be immune to that disease at that particular time. If he has never had the disease and has not been inoculated against it, he is said to possess natural immunity. The same applies to typhoid, tuberculosis, septicemia, rabies, tetanus, or botulism—in regard to natural immunity. The reason given for this is that conditions are not favorable for multiplication of the germ, which would be evading the question were it not for the fact that it leads to the subject of acquired immunity.

Note the qualifying clause: “If he has never had the disease and has not been inoculated against it.” These are the conditions underlying the term “natural.” It is obvious, however, that acquiring immunity through experiencing the disease comes under the head of “natural,” whereas inoculation is an artificial means. Some authorities hold that they are identical—a question which we shall consider presently.

Biochemists and immunologists have created very complicated explanations of the mechanism of immunity. In some instances, the blood develops an antitoxin which neutralizes the toxin of the germ; in other cases, the antitoxin comes from the body cells. In either case, its formation is initiated by the presence of an antigen—which may be a bacterium or any of its products, or, in fact, any foreign substance. This antigen is thus responsible for the production by the body of antistances or antibodies. When produced by the blood, they were formerly known as alexines, and it was at that time supposed that the blood solely was responsible for all phenomena of this nature. It should be clear that in natural immunity some such chemical action and reaction as that just described occurs with no apparent inconvenience to the individual. Acquired immunity, on the other hand, is explained as follows: The action of the antigen is of such nature as to stimulate the organism chemically to produce antibodies so that at the cessation of the disease processes there is either a profusion of these antibodies or the habit of producing them resident in the



organism, which is thus left with an acquired facility to meet similar situations, should the need ever arise again.

### **Smallpox**

The ravages, of disease were formerly laid at the door of the gods. In the name of Sheetula-Mata, the East Indians performed propitiatory rites which consisted of smearing the exudate of smallpox on themselves. Later, Christians took up the practice and embellished it with a cut on the arm in the form of a crucifix and a ceremonial prayer to the Trinity as they smeared the wound with human pus.

This superstitious rite is credited throughout the medical world with being responsible for the decrease in smallpox. Of course, hygiene and sanitation have come in for a measure of praise, but it is believed by many that vaccination has been the factor of prime importance. That the opinion has not been advanced without some question is evident in the statement of Erwin Liek, M.D., in *The Doctor's Mission* (p. 39):

“Nowadays we recognize the shortsightedness of medical men who expounded bacteriology in the past. Formerly the conception was that the germs of disease enter the human body and cause disease and that the doctor destroys the disease germs and re-establishes bodily health. That conception was as foolish as was the conception held by the savages that disease is caused by the body being invaded by demons which have to be exorcised and expelled. Those physicians who are too easily overawed by the pronouncements of scientists should study the science of immunity. They will be amazed to find that, although entire libraries have been written on the subject, we know hardly anything about it. Our scientists are able to describe certain events which take place in the living body. However, we have no clear idea of how the body does it. We have a vague notion that important events take place in the struggle between the invading forces and the body. How and why the struggle takes place is an enigma and will always remain an enigma. Meanwhile, science has obscured the facts by inventing a new language for describing these processes and events.”

Valuable testimony that present-day practices have evolved as hit-or-miss, trial-and-error procedures is provided by Alfred Russel Wallace, the great scientist and statistician. In *The Wonderful Century* (p. 215), he says:

“The mild smallpox usually produced by inoculation was quite as infectious as the natural disease, and became quite as fatal to persons who caught it. Toward the end of the last century many medical men became so impressed with its danger that they advocated more attention to sanitation and isolation of patients, because inoculation, though it may have saved individuals, really increased the total deaths from smallpox.”

#### *Multiple “attacks”*

The question of multiple “attacks” is something which has caused great confusion and controversy. Whether it is merely chance that so many people have only one experience with a specific disease, or whether it is because the cessation of the disease leaves the body so conditioned that recurrences are prevented, is a question which has agitated the world for centuries. It was formerly held in orthodox circles that multiple experiences are impossible, and great efforts were made to establish the facts. This is purely a statistical problem. We quote from Wallace, *The Wonderful Century* (p. 276, et. seq.):

“The most efficient vaccination does not diminish the number of attacks, and does not mitigate the severity of the disease, but both these results follow from sanitation and isolation. . . .

“Dr. Adolf Vogt, University of Berne, expert statistician, proves that ‘A Previous Attack of Smallpox Does Not Confer Immunity. Second attacks occur more frequently than they should do on the doctrine of chances alone, indicating that, instead of there being any *immunity*, there is really a somewhat increased *susceptibility* to a second attack. . . .’

“Now the whole theory of protection by vaccination rests upon the *assumption* that a previous attack of the disease is a protection; and Professor Vogt concludes his very interesting discussion by the remark: ‘All this justifies our maintaining that the theory of immunity by a previous attack of smallpox, whether the natural disease or produced artificially, must be relegated to the realm of fiction.’ If this be the case, the supposed *probability* or *reasonableness* of an analogous disease, vaccinia, producing immunity wholly vanishes. . . .

“The taking it for granted that second attacks of smallpox, or of any other zymotic disease, are of that degree of rarity as to *prove* some immunity or protection, indicates the incapacity of the medical mind for dealing with what is a purely statistical and mathematical question. . . .

“Quite in accordance with this influence of smallpox in rendering the patient somewhat more liable to catch the disease during any future epidemic is the body of evidence adduced by Professor Vogt, showing that vaccination, especially when repeated once or several times, renders the persons so vaccinated more liable to take the disease, and thus actually increases the virulence of epidemics.”

Herbert Spencer quotes from *Memories*, by Kegan Paul (p. 270): “I had had smallpox when a child, in spite of vaccination, and had been vaccinated but a short time before [that is, before he had smallpox as an adult—JRV]. I am the third of my own immediate family who have had smallpox twice, and with whom vaccination has always taken.”

These remarks from Wallace and Spencer were shouted down as heresy by their contemporaries, but we are able to report that modern investigators confirm their position. We quote herewith an authority who, in addition to supplying material on the matter in hand, also provides support for other contentions which are dealt with elsewhere in this text. In an article on “Smallpox and Immunity,” *Medical Journal and Record*, August 2, 1933 (p. 80), Joseph A. Mendelson, Major, Medical Corps, U.S.A., says: (emphasis by JRV)

“The practice of medicine in China has disclosed the startling fact that *vaccination or a previous attack of smallpox does not confer the degree of immunity we have so long taken for granted* (1).

“A case in point was reported to me by the chief nurse at the Isolation Hospital. An Anglo-Indian nurse in the Indian Medical Service had had smallpox. During a subsequent epidemic of this disease she was vaccinated under protest because she believed she was immune in consequence of her previous attack of the disease. *She was the only one of the nurses who developed smallpox and she was seriously ill* (2).

“Another point of marked interest is the bizarre symptomatology frequently encountered in the early stages of smallpox cases. Atypical cases are quite common. We have seen cases of *smallpox, some of which at first appeared to be typical chickenpox, others measles, and still others meningitis* (3a). *Conversely, there have*

*been cases which looked exactly like smallpox in the beginning and proved to be mild chickenpox* (3b).

“. . . When first seen the case was *diagnosed chickenpox* because their lesions and their distribution were characteristic of that disease, *because* of the patient's history and clinical appearance, and *the presence of the large vaccinia scars*. After the fourth day he exhibited definite manifestations of *smallpox* and went through the complete cycle, making a good recovery except for extensive scarring.” (4)

1. The immunity theory is upset. What has been “so long taken for granted” by pseudo-scientists is found to be a very poor guess —after so much damage has been done that the Spanish Inquisition pales into insignificance by comparison.

2. A selected individual was vaccinated (“under protest”) to provide immunity—and was the only subject who contracted smallpox. Conclusion: Vaccination either caused smallpox or rendered the subject susceptible to smallpox. No other possibility is reasonable under the circumstances, for to doubt the diagnosis, invalidates all statistics and is fatal to the entire case for vaccination. Wallace, Spencer, Vogt, and their supporters are vindicated.

3a. Smallpox may have evolved or emerged from some other disease, he concedes.

3b. Some other disease may have evolved or emerged from smallpox, he also concedes.

4. Vaccination scar is accepted as evidence against a diagnosis of smallpox, which constitutes

4a. A violation of the rules of logic in that vaccination is first condemned for not providing immunity against smallpox, and then the scar of vaccination is accepted as evidence that the case can not be smallpox because of the vaccination, and

4b. A violation of those rules which make it possible to compile dependable statistics, by making a diagnosis on evidence which is justifiably discredited. This is an application of the will to believe.

What logic! What a science!

And so we see that there is much to question in the practice of immunization. The present method is one of attenuation and consists of infecting a heifer with smallpox and then vaccinating (vacca. L., cow) the human subject with the exudate containing the living virus. This is known as active immunization. Biochemists have found that there is a scientific fact underlying this former religious act. There is some similarity between it and the phenomenon of fermentation, the parallel being sufficiently close to allow an analogy to be drawn. As a matter of fact, such a comparison was made by Pasteur many years ago.

The product of fermentation by yeast, alcohol, when added to a fermentable substance, will prevent, slow up, or terminate the activity of the yeast to an extent determined by the percentage of alcohol added. And so also will the toxin produced by bacteria prevent, slow up, or terminate the action of other similar bacteria. And there the analogy ends. We are aware that the human body is not a barrel, and that living cells *in situ* are not the same as grapes in a barrel, and that the specific reactions following the two experiments are very dissimilar, but the principle underlying man's control of microörganic activities is sufficiently similar in both technics to permit this illustration. The problem is not nearly as simple as is here stated, but the intricate chemical mechanism is being studied by the biochemist and the immunologist and more skepticism is apparent each day.

Let us use a very well known example of the work of the bacteriologist with which to illustrate the point: In two herds of an identical number of cattle, will the 1953 descendants or counterparts of an 1853 herd have as large a quantity of sour milk in its record as the earlier group? It must be conceded that today the souring of milk has been greatly diminished and could, with very little effort, be entirely eliminated from our experience. But the question must be asked: Is that the only change brought about in the milk? Is it not the task of the bacteriologist who recommends the pasteurizing of the milk to prove also that the article is not thereby damaged or made deficient? It will be generally conceded that he has accepted the responsibility and admits that the milk is deficient and/or defective in vitamin content and prescribes orange juice to supply the deficiency. Next, is it not a fact that one bacterial change has been substituted for another? The lactic acid bacteria have been destroyed and those of putrefactive action now gain the ascendancy. Bacterial change has not been prevented. So we must concede both that the milk has been damaged, and that the destruction of the lactic acid bacteria is not the only change.

In the simple matter of controlling the bacterial content of milk, one bacteria] change is substituted for another, and the article is damaged. Imagine how much more complex is the work of the bacteriologist when dealing with the human body, and how much more likely damage is to result and one bacterial form given ascendancy over another. In the question of milk it is imperative that we know that Vitamin C has been destroyed. How much more important it is to require that the question of change be definitely and completely answered when the bacteriologist suggests doing tricks with the much more complex human body. This illustration is employed only in order to emphasize the complexity of the problem. The matter is not so simple in the human body. Nor does the phenomenon work in just this way in every case.

The classical example of serum therapy is supplied in diphtheria. It consists in introducing into a horse gradually increased doses of bacillus-free toxin of *B. diphtheriae* (which produces active immunity); then withdrawing a quantity of its blood and using the serum thereof either to prevent or terminate the process of diphtheria in the human. This constitutes passive immunization, for it is obvious that the antitoxin was not formed in the subject's body. Such immunity is generally of short duration.

The difference between vaccination and serumization is thus made clear. In the one case, germs, living or dead, virulent or attenuated, are introduced into the body to provoke the production of antibodies; in the other case, the blood serum of an animal so treated, and hence supposed to contain antibodies, is injected into the patient. Occasionally also, as in the malarial treatment for paresis, germs or their products which are not related to the specific disease are employed; or even, in some cases, substances unrelated to bacteria (chemotherapy). The intent is to secure a similar reaction to that produced when germs come in contact with a naturally immune individual.

### **Phagocytes**

The blood contains two figured elements—red and white corpuscles. The white show many variations in shape and chemical reaction—one of which is chemotaxis for certain bacteria. In some instances, virulent bacteria repel these leukocytes (negative chemotaxis). The phenomenon of bacteria being engulfed by leukocytes is called phagocytosis. It may be enhanced by conditioning the blood and leukocytes with

specific substances, known as opsonins, which some blood possesses naturally. The opsonic index is one of the means employed by some investigators to determine the condition of the body in relation to a specific disease, or as a guide in administering the indicated opsonin. Positive chemotaxis is a physico-chemical phenomenon and either bacterium or leukocyte, or both, may be altered in a physical and chemical way as a result of the contact; they may even lose their identity.

Concerning the statement made in a previous paragraph to the effect that when certain infections terminate there is a profusion of antibodies or the acquired characteristic of producing them, there are several theories, the most popular of which, that of Ehrlich, has been seriously challenged recently by many authorities. For example, F. P. Gay, in *The Newer Knowledge of Bacteriology and Immunology* (p. 881), says:

“In spite of the importance of antibodies as indicative of a protective reaction, a very marked immunity may exist without their presence; and their presence alone does not insure protection.”

(P. 882) *ibid.*, “[Ehrlich’s] theory, as is well known, rests on certain assumptions which are probably erroneous, or at all events unproved. ... In short, Ehrlich’s theory, while claiming to offer a complete explanation of antibody production, has never demonstrated that any particular organ or tissue is responsible for the formation of any given antibody. . . . The central nervous system of animals susceptible to tetanus toxin is not equivalent to, nor does it give rise to, tetanus antitoxin as Wassermann and Takaki thought.”

A. Besredka, in *Local Immunization*, says:

“The known antibodies should be without hesitation stripped of their importance; their function in immunity is in reality entirely secondary, or negative in certain cases.”

Besredka’s motion to strip antibodies of their importance is seconded without hesitation.

W. H. Manwaring, in *The Newer Knowledge of Bacteriology and Immunology*, says:

“My conviction that there is something radically wrong in the major assumption of the Ehrlich theory arose from a consideration of the almost universal failure of therapeutic methods based on these assumptions. ... In spite of millions of dollars spent in research and in the commercial exploitation of such antibodies, therapeutic sera have been lamentably unsuccessful except in a small group of relatively unimportant diseases.” (P. 1078): “The Ehrlich theory of the origin and nature of specific antibodies beautifully explains and coordinates all known immunological facts. For three decades this theory has been the accepted basis for immunological deduction and clinical interpretation by the majority of medical workers. Nevertheless, I am convinced that there is hardly an element of truth in most of the hypotheses incorporated in the Ehrlich theory, and that for two decades the wide acceptance of the theory has been a serious handicap to medical progress.”

A. D. Speransky in *A Basis for the Theory of Medicine* states, “The medicine of Virchow, Pasteur and Ehrlich is approaching exhaustion and cannot cope with the contradictions that have arisen.”

Unquestionably the most severe condemnation of the orthodox opinions on leukocytes and immunization is the evidence presented by Tissot as summarized elsewhere in this book.

In short, the tenets underlying modern “scientific” medicine have been guesses—romance instead of science. Because many practices worked out “as if” the theory were true, it was therefore assumed that they were true—a scandalous application of wish-fancy indulged in by men supposed to be grounded in science. We append Ehrlich’s theory for what it may be worth. Many state medical examining boards will continue to require applicants to answer questions “as if” the theory were well established. Fallacies are relinquished reluctantly.

## **The Mechanism of Immunity**

### *Antibodies*

The tissue cells are thought to be normally able to generate substances which in one way or another act as a protection against injurious bacterial activity or the action of foreign substances in general. These protective substances are known as *antibodies*. The germ or foreign substance which excites the production of an antibody is known as the *antigen* and is said to possess *antigenic* properties. The chemical formula neither of antibodies nor of antigens is known, and no antibodies have been obtained in a pure state, though there is evidence that both of these classes of material are proteins. Six kinds of antibodies have been recognized, as follows: antitoxins, antiferments, agglutinins, precipitins, lysins, and opsonins.

*Antitoxins.* The serum of a horse that has been inoculated with diphtheria toxin is capable of neutralizing the toxic properties of the filtrate from diphtheria bacilli. It is therefore assumed that a substance is present in this serum that is capable of combining chemically with the bacterial toxin in a manner which renders the toxin inert. Such an antibody is called an antitoxin. Very little is known of the exact nature of this chemical combination. One of the earliest theories was that of Ehrlich, who conceived the reaction to be similar to that between a strong acid and a strong base. A later theory interpreted the phenomenon in terms of the ionic theory, as the latter theory applies to the reaction between a weak acid and a weak base. At the present time the tendency is to seek an explanation in terms of colloid chemistry.

*Antiferments.* Antiferments are antibodies which counteract the activity of foreign enzymes. If, for instance, pepsin were to be introduced into the blood stream, digestion of the blood cells, the proteins of the plasma, and even of the walls of the blood vessels might conceivably occur, were it not for the generation of such protective antiferments.

*Agglutinins.* The antibodies of this class have the properties of causing specific bacteria to clump together and cease to multiply. Agglutinins are produced in typhoid and paratyphoid fevers. Very early in the course of the disease the blood of a typhoid patient contains a specific agglutinin. Use is made of this fact in the Widal test, the object of which is to determine whether or not the patient’s blood will agglutinate specimens from a pure culture of typhoid bacilli. If the result is positive, it is considered assured that the case may be correctly diagnosed as typhoid fever.

*Precipitins.* The action of this group of antibodies is to cause foreign proteins to be precipitated, thereby checking their diffusion through the circulation.

*Lysins.* These antibodies exert a disintegrating or solvent action on foreign cells. Lysins which dissolve ordinary’ cells are called cytolysins; those which dissolve bacteria are termed bacteriolysins.

*Opsonins.* Certain white blood cells appear to destroy bacteria and subject them to intracellular digestion. Such cells are called *phagocytes* and the process is known

as *phagocytosis*. Phagocytosis seems to be dependent in some measure upon the presence of a special type of antibody known as opsonins.

#### *Theory of Antibody Production*

As already stated, the opinion prevails that the place where antibodies are produced is in the tissue cells. According to Mathews, *Physiological Chemistry*, (p. 709): “Recent work indicates also that it [the skin] has a very important function in the production of the immune bodies; for Besredka has found that guinea pigs may be immunized by the injection of anthrax vaccine into the skin, but not if the injection is made elsewhere in the body.” Until recently there was very little experimental evidence of any kind on which to base a theory to explain this production. The cells were often regarded as independent purposive units endowed with an unlimited range of self-protective ability. Various physico-chemical theories were also proposed and continue to be held by many. Four bacteriologists of the Psychiatric Institute and Hospital in New York recently conducted some experiments which throw light on this question.

It was the aim of these investigators to determine, by experiments on human beings, whether a nerve mechanism is involved in the natural production of the antibody which agglutinates typhoid germs. They reasoned that, if this process is a reflex act, it can be conditioned.

A familiar example which illustrates the conditioning of a reflex is to show food to an animal and at the same instant ring a bell. After a few repetitions of the performance, the sound of the bell is in itself sufficient to cause a flow of saliva without the sight of food.

In *Proceedings of the Society for Experimental Biology and Medicine* (Vol. XXX, No. 1), these investigators tell how for twenty-one successive days they gave patients small doses of typhoid bacilli subcutaneously, immediately after applying an ice tube to the cheek. After a rest of two weeks, the patients were given the ice application and were pricked with the needle, but this time no germs were introduced. Nevertheless, the blood showed an immediate rise in antibody content, just as though more germs had been injected. In other words, the reaction was in the nature of a conditioned reflex. The only cases (three out of forty) in which no such rise was noted were those in which there had been no response to the germs in the first place.

These experiments constitute one of the most convincing bits of evidence on record for the drugless practitioner’s view that the body with a normally functioning nervous system makes its own protective serums.

Further confirmation of this theory is to be found in the work of S. Metalnikov, of the Pasteur Institute, Paris. In the *Bulletin de l’Institut Pasteur*, April 15, 1933, there was published a review of a paper by Metalnikov on “The Role of the Nervous System and Conditioned Reflexes in Immunity.” Translating, we read:

“Utilizing the methods of Pavlov, the author and his pupils have been able to demonstrate that it is possible to provoke the reaction of immunity, not only by antigenic injections (that is, the introduction of a germ or toxin), but also by excitations externally conditioned. To obtain these conditioned reflexes in a guinea pig, one must have recourse to repeated injections of some antigen, followed by external excitation (scratching or heating of some region of the skin). After 20 to 25 injections, followed by external excitation, the animals are left to rest for 10 to 15 days. After that it is enough merely to practice the external excitation previously employed, in order to see appear in the peritoneum or in the blood a typical defense

reaction: very considerable augmentation of the leukocytes and antibodies in the blood and very intense leukocytosis in the peritoneum.

“Continuing his research, the author was able to demonstrate that, in the guinea pig, with the conditioned reflex, under the influence of a corresponding excitation, defense reactions can be produced in the peritoneum which protect these animals in certain cases from a mortal infection.”

#### *Theories of Vaccines and Serums*

The practice of vaccination consists in introducing bacteria, either attenuated or dead, into the body on the theory that the tissues will then generate the antibodies for these bacteria. It is equivalent to giving the patient the disease in a mild form in order to render him immune from any virulent attack to which he might subsequently be exposed. Thus vaccination is presumed to produce a mild specific disease. This granted, how is it possible to re-vaccinate successfully; that is, reproduce the same specific disease, after the patient has been rendered “immune” to that very disease which vaccination produces in a “mild” form? This is an inconsistency which has been rationalized for generations. It is plain evidence against the theory of vaccination, and can not be suppressed.

If the serum instead of a vaccine is used, the serum is that of an animal which has received gradually increasing doses of the germ or its toxin with a view to inducing the production of antibodies. For details of such a process, consult the chapter on the diphtheria bacillus. A person in whom immunity has supposedly been produced by vaccination is said to be actively immunized, while one who has acquired immunity by the introduction of animal serum containing antibodies is said to be passively immunized.

Evidence is given elsewhere in this text of the fact that agents other than bacteria and their products have the power to change the course or even suppress the symptoms of a disease. Here is one instance reported in *The New York Times*:

“A powerful salt from castor oil which promises a new method of immunizing from diphtheria and tetanus also was reported. . . . Development from castor oil of a substance named soricin, a safeguard against cobra and snake venom and the poisons in tetanus and diphtheria was also described by Dr. Rider. . . . The most remarkable action so far observed is ability to kill the poison in bacteria without killing the bacteria themselves. This opens the possibility of immunizing against deadly diseases without risk of poisoning from the immunizing substances.”

We suggest that the immunologists try all these different methods on plant life and let the human race alone until such time as they have definitely proved that they can accomplish what they claim and also confine the organic changes involved to one specific reaction. Let them “inoculate” trees. We can keep a better check on them that way. We suggest this in all seriousness. Trees do not change their locations so often as human beings.

#### *Ehrlich's Receptor Theory*

This theory, according to Stedman, assumes that the protoplasmic molecule is analogous in constitution to the benzene molecule or benzene nucleus, with its linked hydrogen atoms capable of being displaced by various groups to form side chains. So, linked to the protoplasmic molecule are numerous side-chains, or receptors, capable of seizing upon certain bodies such as foodstuffs or poisons and incorporating them into the molecule. By receptor Ehrlich meant one of the side-chains of the cell which combines with foreign substances; this foreign substance



may be something needed for nutrition of the cell or it may be a destructive toxin. When certain receptors are bound by toxin, the remaining receptors are stimulated to overproduction; those in excess of the needs of the cells are thrown off and circulate in the blood. Here they seize upon any toxin present and bind it so that it is unable to attack the cell; these free receptors in the blood-plasma constitute the antitoxin. This theory has very few definite facts to support it and is not much in vogue among bacteriologists at the present time.

#### *Anaphylaxis and Allergy*

Upon occasion, instead of causing the production of specific antibodies, the organism is rendered hypersensitive to further inoculation of similar substances. This is known as anaphylaxis—the opposite of prophylaxis. Stedman's dictionary says it is the opposite of immunity. Anaphylactic shock is accompanied by unfavorable symptoms and sometimes death. Following the introduction of a foreign serum into the body the tissues frequently become in some little understood way sensitized to this particular serum, and any second introduction of the same serum is likely to set up a fatal reaction. Thus the matter of multiple inoculations has presented a problem to immunologists, for it is obvious that the possibility of hypersensitizing must be seriously considered. Instant death has been reported in many, many instances. Furthermore, heredity must be reckoned with, for hypersensitiveness may be transmitted through the mother.

Anaphylaxis is sometimes confused with allergy. It simplifies the matter to consider allergy as a reaction to proteins (all bacteriology is in the protein field) other than those employed medicinally. Thus, hypersensitiveness to pollen, animal dander, dust, and foods would constitute allergy, while serum hypersensitivity would be known as anaphylaxis. The difficulty in distinguishing between the two phenomena and the pitfalls awaiting the layman is nowhere better illustrated than in *The New York Times* of June 11, 1933, where Waldemar Kaempffert, science editor, says: "Since 'allergy' is not to be found in most dictionaries, it is as well to explain here that the word means the reaction of an immunized organism to a specific disease. The allergic condition often precedes immunity. Hay fever and bronchial asthma are now regarded as allergic diseases." One might assume from this that hay fever and bronchial asthma are the reaction of immunized organisms, thus inferring that an inoculation initiated the disease. Immunologists make no such incriminating generalization; their opponents may, but they do not. These diseases frequently appear in persons who have never been inoculated. Further, how can allergy be the reaction of an immunized organism, and at the same time often precede immunity? Dr. Kaempffert is betrayed into this net of word-weaving by interested persons who wish to modify the stigma attached to the term anaphylaxis. The terms must not be confounded; they are not synonyms, regardless of the lexicographers. Anaphylaxis is a protein reaction directly resulting from an immunological administration; allergy is a protein reaction directly resulting from some other cause. Anaphylaxis results from the multiple administration of serum from the same species; allergy has no such precise restriction.

#### *Summary*

In summary we would say that the ambiguity and limitless scope of such terms as lowered resistance, immunity, etc., are worthless generalizations. They allege so much that they define nothing. Precise definition and orderly classification are the foundation of science. Unless it can be shown that germs invariably initiate and

cause infectious processes, the premise of the theory of infection is not proved—and lowered resistance and immunity are shallow, rationalized substitutes for exact scientific knowledge, as subtle as any ever advanced on behalf of the phlogiston theory which delayed scientific progress for 100 years. The vast majority of “natural” disease processes precede germ activity, and are its prime cause, and the reasoning must start from this premise. Then the theory of infection and the theory of immunity will shape up in a rational fashion. Clear distinction must be made between “natural” disease and artificial, laboratory, inoculated disease. (See Speransky)

We close this chapter with some excerpts from an editorial in the *Medical Journal and Record*, May 3, 1933:

“Careful observers, like Crile, Cannon, Pavlov, Ellice MacDonald and others, have had a lot to say about what they have discovered in regard to the various tensions which exist in human beings. The studies they carried on were done in the laboratory and in the clinic, which after all has been considered a respectable enough field of operation for any scientist.

“Other workers have done considerable observing in regard to the autonomic tensions in psychopathic personalities; among them, men like Kempf, Lewis and Sullivan. They have come to the conclusion that in the psychopath there exist certain tensions of considerable intensity and furthermore that they exert certain influences on metabolism, growth, posture and even cause pathological conditions, anatomical and functional. . . . We may also ask the question, What is the secondary effect of this tension on pathological tissues, such as sclerotic conditions, which have primarily been caused by infection or toxic conditions? . . .

## 66 PHENOMENA WHICH DESTROY THE VALIDITY OF THE PRACTICE OF ARTIFICIAL IMMUNIZATION

Defective Theory—Non-Specificity of Bacteria and Serum—Viruses —Sanitation—Immunology a Cult—The Infectious Process—Carriers —Bacteria Omnipresent—Contagion—Sciolism—Disease Substitution—Dangers and Failures—Unapparent Infection—Allergy and Anaphylaxis—Laboratory Risks—Condemnation of an Expert—Non-medical Methods

“A fair judgment of any procedure can only be reached by an examination of the principle on which it is founded. When the principle is unsound, the procedure must fail in the long run. I therefore wish the practical procedures which I am now putting forward to be judged by the principle which underlies them.” F. M. Alexander: *Man’s Supreme Inheritance* (p. 46)

*Theory Underlying the Practice of Artificial Immunization:* The principle underlying artificial immunity will now be examined in the light of some of the questions which it raises. Obviously, artificial immunity rests upon the germ theory of infection and it will be necessary to examine the premises of this theory, and some others allied thereto, in order to obtain a proper perspective.

Jordan says, in *General Bacteriology* (p. 277): “It is stated that barred forms of bacilli are sometimes found in clinical diphtheria as the sole type, but this condition appears to be very rare. The relation of the solid type to clinical diphtheria is still obscure. By some authorities the solid forms are classed as pseudodiphtheria bacilli and are not regarded as capable of causing diphtheria. The barred and solid types are found much more commonly than the granular type in the nose and throat of healthy individuals.

“The granular type, on the other hand, predominates in clinically characteristic diphtheria. Several observers report that during convalescence the granular type is gradually replaced by the solid, a fact that has been regarded as pointing to a gradual morphologic alteration brought about by the influence of the body-fluids of an immune individual.”

Then why cannot the reverse of this occur; that is, why cannot the solid type found in healthy individuals change into the granular type as the individual fails in health? Does not the evidence indicate that environment regulates the germ, rather than that the germ causes the disease? Again:

“The diphtheria bacillus exhibits a marked tendency to the production of involution forms. These occur especially abundantly in cultures on artificial media; for example, on blood-serum after five to seven days. The biologic significance of the appearance of involution forms as well as of branching forms among the diphtheria bacilli and certain other groups of bacteria is not at present understood.”

Jordan’s statement, “The biologic significance of the appearance of involution forms,” etc., involves not alone an important problem of fact, but the more important problem of the scientific habit of mind. Again, his dogmatic assertion in *Chemistry in Medicine* (p. 550), “It will be generally conceded that as regards diphtheria the theory that a microbe is the cause of the disease has passed from the realm of theory into that of observed fact,” prompts the question: How? Have the

facts been twisted, or partially ignored to fit the assumption? On the admitted statement quoted herein how can one assume an attitude of finality—and be scientific? The pseudo-scientific thinker substitutes hunches, guesses, and wish-fancies for a realistic, demonstrable interpretation of fact. The popular tendency to make facts fit some *a priori* “philosophy” is a type of non-scientific thinking.

The case for diphtheria may be closed “philosophically,” but the scientific thinker will observe in the quotation from Jordan that fundamental problems of fact—verifiable knowledge—are still in dispute. It, therefore, behooves us to examine the theory critically and proceed cautiously with practice, lest we be building on fallacy.

*Etiology:* The generally accepted etiology of communicable disease comprises a major and a minor premise: 1) that germs cause infectious disease; 2) that some factor predisposes thereto—a factor commonly called lack of resistance, or susceptibility. The resistance factor may cease to operate for the protection of the individual in one of two ways: 1) a normal mechanism may be broken down by trauma, as in the case of the bite of an insect, a gunshot wound, an abrasion of a mucous membrane, or an experimental inoculation; 2) there may be constitutional idiosyncrasies or diatheses. It is possible to classify communicable diseases of known etiology into two groups corresponding with these two phases of resistance failure. When infectious diseases of unknown etiology are understood, it will probably be found possible to place them likewise into one or the other of these groups. On this basis we would have, for example:

- a) Conditioned by trauma:
  - yellow fever malaria dengue
  - bubonic plague tetanus
- b) Conditioned by diatheses or idiosyncrasies:
  - tuberculosis
  - arthritis
  - gonorrhoeal rheumatism
  - pneumonia
- c) Undetermined:
  - smallpox
  - scarlet fever
  - measles
  - influenza
  - anterior poliomyelitis

This scheme of classification makes somewhat comprehensible just what the factor of resistance is.

In each instance where infection takes place, the field is primed for that specific infection. Certain pathogenic microörganic metabolic activities are initiated when conditions are favorable, and present-day methods lean toward their suppression. This suppression, we hold, is irrational therapy. The rational procedure would be to prevent the development of those conditions which make possible adverse bacterial activity, or, failing this, institute physiological measures which would aid in the elimination of those elements in the field which make possible and inevitable the metabolic activity of pathogenic microörganisms—rather than introduce pathogenic end-products or the originators thereof into the tissues (or chemo-”therapeutic” agents).

*Non-Specificity of Bacteria and of Serums:* It must be pointed out that all vaccine and serum therapy is founded upon the assumption that bacteria are specific. There is abundant evidence, however, that bacteria vary greatly both in morphology and behavior. Bacteriologists of the United States Public Health Service, for instance, have shown that the coccus associated with encephalitis lethargica may spontaneously take on a rod-shaped bacillary form. It then becomes impossible to determine whether the culture is pure or not. The rod-shaped forms may indicate contamination with other bacteria or may have originated from the cocci themselves. Other investigators have made similar observations with many other types of bacteria. It has also been shown that many of the common types of bacteria may pass into an invisible state, in which stage they are able to pass through porcelain filters. Professor Kendall of Northwestern University has perfected a technic whereby he can cause bacteria to alter from the filterable to the non-filterable form, or vice versa, at will. It is, therefore, evident that bacteria possess no specificity of form.

What is still more important, behavior of the germ is also very variable. So-called pathogenic germs may exist either in a latent state with the virulent characteristics quiescent or in the harmless state of saprophytes. Dr. P. Jousset, in *The Pathogenic Microbes* (p. 17), describes the same germ in the pathogenic, the latent, and the saprophytic state, and indicates that the environment is responsible for the variation. Pasteur transformed the anthrax bacillus into a saprophyte by heating to 107 degrees F. and the virulent characteristic was never regained by descendants. Roux, by the use of antiseptics, eliminated the sporing characteristic in this bacillus. Permanent loss of two such distinguishing characteristics make it apparent that the environment is responsible for the change in behavior of the germ, and that the germ can be conditioned.

Even a casual survey of the current literature of bacteriology makes very evident these facts. Consider, for example, the following statements by W. H. Manwaring in an article on "Research Trend of Medical Bacteriology" in *Science* for July 15, 1932: "The static bacteriology of Pasteur, Ehrlich, and Koch led to diphtheria antitoxin, the most spectacular therapeutic victory of all ages. Each victory [Pyrrhic Victories: J.R.V.] of this type, however, was eventually paralleled by a half dozen diagnostic paradoxes and therapeutic failures. Tetanus partially surrendered, but tuberculosis and streptococcus mocked at nineteenth century infectious logic.

"The dissociation of a score or more pure bacterial strains into two or more morphological . . . variants is now well confirmed . . .

"There is accumulated evidence that departures from classic test-tube morphology are almost invariably accompanied by equally marked changes in chemical composition. . . . The same filterable virus, for example, injected into two different animal species is reported to transmute into two different vaccination specificities.

"One of the theoretical surprises of the past three years has been the fairly conclusive evidence that properties simulating specific serum antibodies appear apparently spontaneously in the circulating blood of both man and laboratory animals at the approach of sexual maturity. (Jungeblut and Engle, of Columbia University, have presented evidence, for instance, to show that immunity to anterior poliomyelitis is acquired with maturity as the result of normal endocrine balance and is not dependent upon exposure to the disease.)

“To those who have dreamed of the early test-tube synthesis of therapeutically useful antibodies, the most discouraging surprise, however, is the recent alleged experimental evidence that there is a specific immunity center in the brain. Without this integration center specific somatic antibodies are alleged not to be formed in the body or released into the blood stream. Specific immunological conditioned reflexes are seriously proposed to account for certain specific immunological phenomena.”

*New Views of the Viruses:* As the result of experiments on tobacco performed by Dr. Carl G. Vinson, of the University of Missouri, a special chemical theory is advanced; namely, that the so-called “virus diseases” of plants and animals are caused by a non-living chemical substance that can attach itself to crippled living matter, rather than by ultra-microscopic living organisms. Dr. Vinson’s work apparently supports the belief, held on theoretical grounds by many physiologists and pathologists during the past thirty years, that the causes of these mysterious diseases of plants and animals are substances analogous to enzymes, the digestive ferments of normal organisms, but malefic rather than beneficent in their effects.

It is suggested by T. M. Rivers of the Rockefeller Institute in an article on “Viruses” in *Science* of June 24, 1932, that the viruses may be products of cellular perversions capable of inciting similar perversions in other cells. This is very significant as it points to an internal rather than an external source for these agents. Lwoff and others made independent reports at the International Congress of Cellular Biology in 1950 proving definitely what Rivers had suggested in 1932 as previously mentioned.

*Study of Viruses*, 1950 by Delbruck shows a symposium of foremost authorities agreeing that viruses are produced by and within injured, degenerating cells and that a normal, healthy cell cannot be infected by any virus until after the cell is damaged. Degeneration is a prerequisite to infection.

*Sanitation versus Serums:* Dr. Rivers also makes the following statement: “Numerous diseases spread by means of water, milk, food, filth, and insect vectors have been controlled not by protective vaccines and curative sera, but largely through the improvement of sanitary conditions.”

Consider, in this connection, the communication of disease through insect carriers. This can occur only when the insect first bites a patient suffering from the disease and then bites and so infects a victim. Is not this primarily an argument in favor of screens and sanitary engineering, and secondarily for quarantine? It is certainly not an argument for inoculation. In order to discuss this subject more fully, we select from the three divisions on page 82 the following representative diseases with which pathogenic microorganisms or viruses are associated:

- yellow fever
- malaria
- tetanus
- pneumonia
- smallpox
- scarlet fever
- measles
- influenza

Now we re-classify this group according to their connection with insect carriers:

- A—Eliminated
  - pneumonia
  - tetanus

- B—Insect carrier
  - yellow fever
  - malaria
- C—Undetermined
  - smallpox
  - scarlet fever
  - measles
  - influenza

The diseases of groups B and C are all regarded as communicable —either directly or indirectly. They are therefore quarantinable; group B behind screens, and in vermin-proof environment. Further, sanitary engineering is indicated; in group B, not merely to eliminate the insect carrier by destroying its breeding grounds, but to eliminate the polluted matter on which it thrives. (Isolation is useless without this, for such matter may just as readily be the origin of infection as the biting of an infected person.) In group C, sanitary environment is indicated for obvious reasons. As is shown elsewhere, in none of these groups is inoculation indicated or justified.

Whatever there is of value regarding cause in the inoculable disease hypothesis obviously applies only because there is an intermediary host—an insect carrier or any similar penetrating and nerve-traumatizing agent. Just as obviously this argument does not apply to disease causation where this intermediary is not a factor. The point is: even though germs *may* be carried in another animal and be injected into the victim, pathogenic germs *cannot* travel themselves—and retain their virulence. One hour of direct sunlight kills all bacteria. Nor can they “invade” without the aid of an insect, for example. But organic matter and dejecta can be air-borne and *can* transport bacteria and spores. All of which shows the interrelationship of sanitary engineering, isolation, and screens, and the un-necessity of inoculations.

A South American moth has just been seen in New York—blown there by a recent storm. Under such circumstances, and they are not rare, even quarantine and sanitation have their limitations. Protests which stress some of the limitations of quarantine are well presented in a *New York Times* article:

“Asserting that the present highly involved system of plant quarantine in the United States has resulted in the immense expenditure of public funds for protective measures that do not protect’ and in ‘serious inconvenience and losses’ to legitimate business, the Merchants’ Association has filed with the Federal Department of Agriculture a long memorandum urging the appointment of a non-departmental, non-partisan commission of experts to study the whole subject, it was announced yesterday.

“The association pointed out that at the time of a survey two years ago the Federal Government was enforcing 19 foreign and 24 domestic plant quarantines, while 112 other quarantines were being enforced by local governmental agencies. Despite all these, it was pointed out, ‘insect pests and plant diseases not heretofore known in this country have continued to invade it and have spread here.’

“Maintaining that the present policy can only lead to one quarantine after another and will develop a ‘wholly impossible’ situation, the association urged that ‘relatively less effort and money should be expended upon quarantine inspections and other precautionary activities which serve seriously to obstruct trade and that relatively more attention should be paid to fundamental research and the practical development of methods of control which would constructively promote horticultural science and commerce.’ “

When men become as much interested in their children as they are in their business and the rights of trade, there will be effective protests against inoculations, and a demand for “fundamental research.” When they resist the coercive efforts of commercial organizations to tamper with children, and refuse to lead their trusting charges to have their tissues mutilated; when they protect their offspring as effectively as they protect their business through organizations functioning for that purpose, a brighter day will have dawned.

Concerning the demand for “fundamental research” made by this association as it applies to plant quarantine, no better approach could be made than through an investigation of the principles and theory underlying the use of organic dejecta as fertilizer. Spreading dung over the landscape strikes one as being a far-fetched theory (and quite at variance with our ideas of sanitation), especially in view of the fact that the most fertile land in the world is found in the valley of the Nile where such practice is not followed. The inorganic silt eroded from the mountains provides plant life there with all its necessary elements. Our system, on the other hand, is to use organic waste materials, slaughter-house by-products, and offal on the soil, and then use sharp tools and implements which cannot help but abrade the plant tissues and thus inoculate large quantities of the foulest and vilest of matter directly into the plant. Is it not apparent that plant resistance under this system has been depleted to the vanishing point? Is it not apparent that stimulation to growth is not equivalent to vitality and vigor? Does not high disease susceptibility accompany pituitary gigantism in the human? Then why confuse gigantism in the plant with vigor? They are not synonyms and they are not inseparable.

Between breeding out vital characteristics, and feeding organic dejecta when inorganic elements are probably required, man has evidently substituted size for vitality in domestic plant life.

This is not a condemnation of composting. Raw waste is questionable, because it is not reduced to normal states ready for usage by plants. Composted material, however, is broken down by bacteria into natural plant food. While the foul and odorous dejecta is in question we must mention that strong chemicals used as commercial fertilizer or as weed and bug killers are often equally harmful in reducing the vigor and food value of plants. Accumulating evidence shows that such foods do become poisons.

“Sports, or new varieties of plants,” declares a writer in *The New York Times* (June 25, 1933), “certainly appear more frequently in cultivated gardens than in the wilderness. Why this should be so has long been a puzzle. Sir Daniel Hall explained the cause very simply and plausibly. It is because environment is allowed to gain the upper hand over heredity.

“In the woods a plant must fight for its life. Many a seedling perishes because it came into the world with undesirable characteristics—undesirable from the standpoint of the struggle for existence. Transplant that same seedling to a garden where a biologist or a plant breeder defends it against the onslaughts of nature and it not only lives but characteristics that never had a chance in the forest are permitted to assert themselves. If the characteristics happen to please the breeder—a queer petal, for instance, a new flavor in fruit—he nurses the plant along. What does he care about such things as survival value? It is the prize at a flower show, the money to be gained by marketing a new melon or an addition to biological knowledge, in which he is interested.” In other words, there are great differences between wild and domestic plant life. And one of the differences is the forcing of growth by means of organic dejecta.



Further, considerable quantities of this decaying, putrid filth are wafted through the atmosphere through the world, and Krakatau proves that no place is secure from atmospheric pollution. Why prate about airplanes carrying insects when the air is being contaminated, and the practice being condoned and encouraged by the very school which prides itself on its scientific achievements in sanitation and hygiene? Even sub-human carnivora bury their dejecta—and the Merchants' Association owes them a vote of thanks for so doing. The rotting of leaves and the rotting of cellulose are quite different organic processes from the pollution which civilized man scatters or hoards. Authorities tell us that plant enzymes convert inorganic elements into organic elements and compounds—analyzing and synthesizing according to the needs of the plant. Inorganic elements insoluble in water are rendered soluble by plant enzymes. Thus it happens in the laboratory. Now then, where is the body of evidence to show definitely that the same thing cannot be done on the field; that plants cannot assimilate inorganic silt if it contains all the required elements, and grow and be vigorous as a result thereof?

The question of sanitation versus inoculations is nowhere better illustrated than in the case of typhoid. The following quotation from Walter R. Hadwen, M.D., could be supplemented by a great deal along the same line, but this will suffice:

“On November 8, 1918, a meeting was held at the Royal Society of Medicine, London, for the purpose of listening to an account of an outbreak of typhoid fever in a company of United States soldiers. Capt. Fred. M. Meader, M.C., told the story. The facts briefly stated were these: Out of 248 men, 95 were struck down with typhoid fever. Seventy per cent of the cases were ‘severe high-grade typhoid,’ and they had a death-rate of thirteen per cent. Capt. Clinton Hawn, M.C., said: ‘All the men had been inoculated, many of them several times and at different posts, and the agglutinins for typhoid and paratyphoid A and B in the cases investigated by Capt. J. G. Hopkins, M.C., reached a titre normal for inoculated men.’ So there was no mistake about their ‘perfect protection.’ Even Sir Wiliam Leishman expressed himself ‘satisfied by the evidence produced that the men had been properly inoculated.’ And yet these 95 ‘fully protected’ men on drinking polluted water, went down with a heavy death-rate. Sewage water laughed the vaccine fetish to scorn.”

*The Call for a New Immunology:* Curphey and Baruch, of Bellevue Hospital Medical College, New York, have written an article on “The Need for a New Experimental Approach in Immunology,” which appeared in *Science*, January 15, 1932. In this article the following statements are of great importance: “By degrees the top-heavy superstructure of immunological truths (?) has been crumbling under the attack of the more liberal-minded workers, who are seeking to rationalize the cult of immunology in the light of chemical investigation . . .

“No better evidence need be adduced to show the error of the older concepts of this branch of science than the repeated clinical failures with therapeutic agents prepared according to the established immunological theories. Even the long-suffering clinicians have ceased to clutch at the therapeutic straws that the immunologists have from time to time cast forth on the sea of hypotheses and look askance instead at any new therapeutic agent with an immunological background.

“It has ceased to be the fashion to study the disease in its entirety and to substitute instead a finer and apparently more fruitless study of the alleged agent of causation of the disease. The immensity of this potential error is appalling. What if these diseases that in the main show such clear-cut clinical manifestations as to enable the clinician to constantly classify them, should be caused by agents other than those that are now

credited to them! Think of the wasted immunological endeavor of recent years, if time shows that scarlet fever as it is clinically manifest, is due not to a single strain of streptococcus, but rather as recent workers are inclined to believe, to any one of a number of strains provided beforehand with a suitable environment and therefore producing a specific type of toxin. Contrast this with the greater progress that might have been made if the investigation of this disease had been conducted along lines controlled by fundamental biological facts, the chief of which being the close interrelationship between the disease producing agents and their environment. How uneasy must Sydenham, Jenner, Pasteur, and Koch rest when they view our repeated attempts to replace observation and experimentation *in vivo* by methods *in vitro*! One of the greatest assumptions that over-specialization in the field of immunology has sanctioned is that the test-tube is analogous to the living host. This has been a considerable handicap to progress and has led to more wasted effort than any other single factor. Fortunately the viewpoint is already changing and evidence is rapidly accumulating to, show what an important influence the host exerts in the fundamental biological characteristics of the invading organism in cases of infectious disease.”

*Analysis of the Infectious Process*

It was shown (Chapter 63, first paragraph) that three factors are generally involved in infectious processes—two of them absolutely necessary: the body, the germ, and those elements which combine to condition one or the other of these two. (Upon occasion, this third factor may be eliminated, but its seeming absence may, in all likelihood be due to our limited observational powers.) Any of these factors may be regulated, controlled or modified so as to affect the process under consideration. The dogma that germs cause disease, therefore, never stands alone. It must always be modified—not once, but every time—invariably.

*Hypersensitiveness:* Under what seem to be identical circumstances of exposure, infection may occur more readily to some subjects than others; i.e., they may be hypersensitive.

Carl H. Browning, Gardiner professor of bacteriology, University of Glasgow, in *Bacteriology* (p. 232), says: “The reaction in the living body which follows the introduction of an antigen may not always result in immunity, but may lead to the opposite effect, i.e., specific hypersensitiveness, the existence of which can be demonstrated under special conditions. Thus if an individual infected with tuberculosis receives, a subcutaneous injection of a small amount of tuberculin (the soluble products of the tubercle obtained from an old culture), there follows a marked inflammatory reaction at the site of the injection and also fever; a much larger dose of tuberculin administered to a non-tuberculous individual fails to produce any such reaction. This tuberculin reaction has proved of great value in detecting tuberculosis in cattle. Glanders in horses is diagnosed on the same principle by using mallein derived from cultures of the glanders bacillus. It has been proved by experiment that hypersensitiveness is the result and not the cause of infection, since a non-tuberculous animal which to begin with reacts negatively to tuberculin, after acquiring tuberculosis comes to react positively. A similar phenomenon is that of anaphylaxis in which a parenteral injection of foreign serum, which is quite harmless to a normal individual, may cause serious symptoms in one who has received a previous injection of serum; this result follows only when both injections consist of serum of the same foreign species.”

In the past it was considered sufficient to *define* hypersensitiveness. No cause for the phenomenon was advanced—least of all the one which is now put forward.

Now it is said that infection is the cause of hypersensitiveness. This is a new position. More, it is the reverse of what was formerly implied; namely, that hypersensitiveness was the cause or a contributing cause of infection. There are four aspects of the matter which we wish to emphasize:

1. That a body defect is present before hypersensitiveness is possible or occurs simultaneously with “exposure”—a position with which we concur.

2. That Browning differentiates between hypersensitiveness and anaphylaxis. Stedman erroneously intimates that they are synonymous.

3. That bacteriologists reverse their theories at will—a practice which is not tolerated in any other science and which does not speak very highly for its dependability.

4. That at one time it is said that hypersensitiveness causes infection and another time it is said that infection cause hypersensitiveness. Is this not circular reasoning? Is this not confusing cause and effect—infection being cause at one time and effect at another time; and similarly with hypersensitiveness? Are these terms to be used in different senses at different times? Is the phenomenon to be interpreted according to the whim of the theorist? Is this not reasoning in a circle, or using contradictory terms? Such loose application of terms leads to inconsistencies in the germ theory and makes it vulnerable. However, it works no hardship on our thesis. The fact is that infection, or disease, or lack of ideal health is a cycle which starts at birth and lies at the bottom of all pathogenic germ activity.

Exactly what causes hypersensitiveness? Certainly infection does. But are there other causes? Cannot the body, through normal growth and development, produce naturally a state of imbalance, resulting in hypersensitiveness? Given this condition, germ infection finds a fertile field for development. On this reading of the facts, not the germ, not infection, but the *de facto* condition of the body is the important consideration. A healthy, robust, normal body, adequately balanced, is impervious to infection. It follows, too, that imbalance may be caused by injections, trauma, etc., resulting in hypersensitiveness. In *Science*, August 15, 1933, Dr. Albert Claude refers to certain normal tissues of the body which contain a factor that has the property of breaking down resistance to infectious agents. Certain bacteria possess an extractable substance which has similar properties. This factor is a strange chemical manufactured in the body by the bacteria, or by certain normal tissues, that first weakens the body's resistance over a large area and thus prepares the ground.

This is the phenomenon of hypersensitiveness. It explains both susceptibility and “resistance.” Obviously, at no point in the situation outlined in the preceding paragraph can the body be perfectly normal. As pointed out in a previous chapter, there is no such thing as perfect health—it is a hypothetical state.

Resistance is that complex coordinate state of the body in which the metabolic processes are so integrated that infective processes at that moment are discouraged. Therefore, lack, loss, or lowering of resistance presupposes pathology. Before the infective process can be initiated, resistance must be reduced, and this reduction of resistance is a pathological process (sometimes of traumatic origin, sometimes initiated by changes in environment or nutritional deficiencies), which predisposes to germ activity, as will be shown in greater detail in the next chapter.

*What is Health?* Aschoff, in his *Lectures on Pathology* (p. 59), states: “As long as they satisfactorily fulfill their functions the vital processes or vital functions are considered healthy or normal and the state of the respective organism is designated as health. The organism seems to be adapted to the given vital conditions. We know,

however, that these external vital relations are extremely variable. ... The organism must therefore be equipped with certain regulatory mechanisms in order to insure its biological existence. A healthy organism is one possessing complete powers of adaptability toward the natural exchange of external vital conditions. . . . We can thus characterize a healthy life as one possessed of the capacity for adaptation.”

It is our contention that the universality of microorganisms places them within the realm of the natural exchange of vital conditions to which Aschoff refers, and, therefore, that a human body which is incapable of adaptation to such organisms (barring trauma) is not a healthy body. Note distinctly and definitely that it is the body and not the germ which is of primary importance. Maud Slye finds that in a family of mice with a high frequency of a certain type of cancer due to chronic skin disease, it is possible to prevent the occurrence of cancer by preventing the chronic skin disease. (*The New York Times*, July 15, 1933) In other words, *health* prevents cancer. Sir Arbuthnot Lane said: “Very many years ago I demonstrated unmistakably that cancer never affects a healthy organ.” Thus we have both laboratory and clinic arriving at the same conclusion on the most important question of disease today.

*Conditioning the Organism:* The external environment enters into the conditioning of the organism so that varying reactions to infection are presented at different times. Species and individuals naturally immune to certain infections may be so conditioned by such agents as cold water, heated air, etc., that immunity is lost. Chickens, normally immune to anthrax, become easily susceptible after being dipped in cold water. Modifications of the surroundings promote modifications in the organism, and, as we shall see later, in the microbe as well.

As indicated in Chapter 63, it is also possible to condition the organism by changing the internal environment (just as fermentable material is conditioned with similar products of similar activity) and so modify bacterial processes. Thus the toxin of *B. diphtheria* will serve as a deterrent to further activity or as a preventive. Vaccine or any other biologic product may be looked upon as an agent employed for the purpose of conditioning the human organism in any one of several different ways. Granting that the cell can be “fortified” against toxins or opiates or hypnotics, etc., such “fortification” is a modification of the cell—and that constitutes a departure from normal. (Drug addiction is a case in point.) “Fortification” of the cell will very likely produce or induce some injury to the cell.

The Kupfer cells in the liver are injured and altered by vaccination. It is a hazardous procedure which will be particularly evident when the potentials for harm are considered. The agent has great potential powers for altering the cell—for better or worse—and the risk is too great to compensate for the questionable benefits derived. The limitations in all respects are not known, and practice should be restricted in proportion thereto. *Primo non nocere*.

Herbert Spencer vigorously opposed this conditioning of the organism as practiced in vaccination. Says Spencer:

“Jenner and his disciples have assumed that when vaccine virus has passed through a patient’s system he is safe, or comparatively safe, against smallpox, and that there the matter ends. I will not here say anything for or against this assumption. [In the paragraphs immediately following hereunder he speaks decidedly against, and it should be particularly noticed that his opposition is not merely on the ground of human liberty, as certain obscurantists pretend to believe, but specifically on the ground that vaccination is an unwarranted, unscientific, and irrational procedure—

J.R.V.] I merely propose to show that there the matter does not end. The interference with the order of Nature has various sequences other than that counted upon. Some have been made known. [The statistics which Spencer gives and his comments thereon have been placed in the chapter on statistics which follows— J.R.V.] To the communication of diseases thus demonstrated, must be added accompanying effects. It is held that the immunity produced by vaccination implies some change in the components of the body; a necessary assumption. But now if the substances composing the body, solid or liquid or both, have been so modified as to leave them no longer liable to smallpox, is the modification otherwise inoperative? Will anyone dare to say that it produces no further effect than that of shielding the patient from a particular disease? You cannot change the constitution in relation to one invading agent and leave it unchanged in regard to all other invading agents.”— *Facts and Comments* (p. 270).

Again: “We have no means of measuring alterations in resisting power, and hence they commonly pass unremarked. There are, however, evidences of a general relative debility. Measles is a severer disease than it used to be, and deaths from it are very numerous. Influenza yields proof. Sixty years ago, when at long intervals an epidemic occurred, it seized but few, was not severe, and left no serious sequelae. Now it is permanently established, affects multitudes in extreme forms, and often leaves damaged constitutions. The disease is the same; but there is less ability to withstand it.

“There are other significant facts. It is a familiar biological truth that the organs of sense and the teeth arise out of the dermal layer of the embryo. Hence abnormalities affect all of them; blue-eyed cats are deaf, and hairless dogs have imperfect teeth. The like holds of constitutional abnormalities caused by disease. Syphilis in its early states is a skin disease. When it is inherited the effects are malformation of teeth and in later years iritis. Kindred relations hold with other skin diseases: instance the fact that scarlet fever is often accompanied by loosening of the teeth, and the fact that with measles often go disorders, sometimes temporary, sometimes permanent, of both eyes and ears. May it not be thus with another skin disease—that which vaccination gives? If so, we have an explanation of the frightful degeneracy of teeth among young people in recent times; and we need not wonder at the prevalence of weak and defective eyes among them. Be these suggestions true or not, one thing is certain: The assumption that vaccination changes the constitution in relation to smallpox and does not otherwise change it is sheer folly. . . .

“Is it changed for the better? If not, it must be changed for the worse.”

Dr. Farr, the great medical statistician, in 1872, said: “The zymotic diseases replace each other; and when one is rooted out it is apt to be replaced by others which ravage the human race indifferently whenever the conditions of healthy life are wanting. They have this property in common with weeds and other forms of life; as one species recedes another advances.”

Arrogantly boasting of “stamping out” smallpox, typhoid, and diphtheria with the biological products of disease, and standing helplessly amazed at the more than offsetting increase in cardio-renal-vascular diseases, tuberculosis, cancer, and syphilis, might better be replaced by a scientific investigation of the possibility of a causal connection between the two phenomena. Conditioning the body has always been opposed by rational people as unscientific in its ultimate effect.

Henry Carlton Bastian in *The Nature and Origin of Living Matter* (p. 324), dealt with the problem as follows:

“The injection of a small quantity of a germ-free chemical irritant into the subcutaneous tissue of a healthy rabbit has made it plain that pathogenic microorganisms may either be produced by heterogenesis in the focus of inflammation thus caused, or else that the germs of common bacilli, existing in the healthy animal on which the experiment has been made have been roused, rendered extremely virulent, and have been converted, in fact, into pathogenic bacilli, *henceforth capable of acting as contagia for the indefinite* propagation of this form of septicemia.” On page 329, “. . . just as the boiled dilute liquor ammoniae injected into the subcutaneous tissue of a guinea-pig or a rabbit produces, even within a few hours, swarms of the bacillus met with in Pasteur’s septicemia. . . .”

More recently, A. D. Gardner, in *Microbes and Ultramicrobes* (p. 66): “Common colds can be transmitted to chimpanzees by means of sterile filtrates of nasal discharge. As soon as the catarrh develops, large numbers of pneumococci (a pathogenic species of streptococcus) are found in nasal mucus. There were presumably a very few of these present in the healthy nose, as there often are in human beings.”

Of course, it will be claimed that the germ was present and was merely conditioned in situ by the filtrate, but this is an admission that sterile products of bacteria may initiate disease processes without the assistance of germs from extraneous sources. This has never been denied; it is merely one of the unchallenged inconsistencies of the germ theory. It constitutes a serious dilemma—either possibility of which will be fatal to the major premise of the theory. It has always been conceded that diphtheria toxin could cause all the symptoms of the disease—without the germ appearing. Now we see sterile filtrate either stimulating bacterial activity or causing a spontaneous appearance of bacteria. There is no other alternative, and either of these possibilities is ruinous to the popular exposition of the germ theory. Conditioning the human body with biological products may be the torch that starts a bacterial conflagration.

Consider, also, the possible effect on heredity. Although certain serums confer but a fifteen-day immunity, hypersensitiveness may be permanent—and be transmitted. E. O. Jordan, in *General Bacteriology* (p. 189), says: “The liability to react hypersensitively is transmitted from mother to offspring, the young of actively sensitized female guinea-pigs being themselves hypersensitive.” Herbert Spencer opposed the practice of vaccination on the ground that such conditioning of the organism could not be confined to the one objective. Now it is admitted that, not only are the undesirable effects to which Spencer referred a real hazard, but they are also transmitted to offspring. Whether this is brought about through the germ plasma, through the blood, or through some other channel we shall have to leave to geneticists to determine. The admission that hypersensitivity is transmissible is sufficient for the purposes of this inquiry, and serum therapy stands condemned on that account.

Again, A. D. Gardner, in *Microbes and Ultramicrobes* (p. 66), states: “In experimental work with vaccinia virus, widespread infection of the animal often occurs with bacteria of the Pasteurella group. Since these are certainly not introduced with the virus, either they must be already lying latent in the animal body, or, if they enter casually after the virus-injection, they must have a *far greater virulence for virus-infected* than for healthy tissues.” (Emphasis by J.R.V.)

Abstract of an address by W. H. Hay, M.D., on vaccination and diphtheria:

“We are never able to prove that vaccination saved one man from smallpox. When you have protected anybody, as we denote protection in medicine, you have at the same time destroyed your evidence. If that man doesn’t take the disease against which he is supposed to be protected, how can you ever know he wouldn’t have taken it if he hadn’t been protected? We have destroyed the evidence. . . .

“Perhaps it is safe to say that not more than ten per cent of the people ever would take smallpox if sleeping in the same bed with an infected smallpox victim. . . . Ten per cent under such conditions is highly laudable for the natural resistance factor.”

Hay knows of an epidemic of smallpox of over 900 cases in which 95% of the infected had been vaccinated, most of them recently.

Speaking of diphtheria. Hay states that but 15% of children are susceptible to the disease in general. After the various diphtheria inoculations, the average immunity is 85%. Still 15% are susceptible, and contract diphtheria. “Isn’t that the same 15%? We have no way of proving it isn’t.

“Cook County, Ill., Hospital decided to immunize one-half of its nursing staff and not the other half. Soon after, diphtheria [broke out among the immunized nurses, not the others. It] invaded both halves, but the number of cases was higher among the supposedly immunized nurses, than among those not immunized.

“The U.S. Government was going to eliminate smallpox in the Philippines. Six years after mass vaccinations, there was ‘the worst attack of smallpox, the worst epidemic three times over, that had ever occurred in the history of the islands and it was almost three times as fatal. The death rate ran as high as 60% in certain areas where formerly it had been 10 and 15%.’” The climax to this story is that the Province of Rizal (Manila was the center) was the most accessible. “The little rascals couldn’t get away there.” In Rizal some were vaccinated 3, 4, 5, 6 times in the 6 years. During the epidemic, Rizal had the highest incidence and greatest mortality of any part of the Arehipelago.

“The navy reported that in spite of vaccination, as regular as drills, the sailors had their usual percentage of smallpox.

“Vaccination is not prohibited anywhere. Those who are vaccinated, say they are protected, yet they inconsistently insist on universal vaccination; thus indicating that they have no faith in their contention.

“During those thirty years [of Dr. Hay’s practice], I have run against so many histories of little children who had never seen a sick day until they were vaccinated, and who, in the several years that have followed, have never seen a well day since.”

Hay says that in England where statistics are a little more “accurate and above-board than in this country [the United States] the actual official records show three times as many deaths directly from vaccination as from smallpox for the past twenty-one years. If they record three times as many deaths, I will guarantee you that there [still] are three times as many deaths that were not recorded that are directly traceable to vaccination.

“That still does not take into account the many, many cases of encephalitis, or sleeping sickness, and of this or that form of degeneration, that occur as a direct result of vaccination. Hay speaks of the derelicts (unemployable human beings) produced during the last thirty years directly by vaccination. . . . It is nonsense and outrageous to assume that pus (usually from dead smallpox victims, say the manufacturers) can be applied to a child and in any way improve its health.

“What is true of vaccination is exactly as true of all forms of serum immunization, so called. There is no such thing as immunization.

“You can’t change the vitality of the body for the better by introducing the products of any disease into it.”

So the potentialities for conditioning the organism in an undesirable manner are very great, and tremendous responsibility rests upon those who indulge in any such activities.

*Conditioning the Germ:* Jousset has already been quoted to show that so-called pathogenic germs may exist in either the latent state with the virulent characteristics quiescent or in the harmless state of saprophytes. Jousset, Roux, and Pasteur have already been mentioned in connection with conditioning the germ’s behavior and morphology. The activity of benign germs is necessary to certain physiological functions of the body, but we hold that the evidence indicates that these germs, when properly conditioned by their environment, may evolve virulent characteristics. When and how this happens is little understood, but it challenges very decidedly the validity of the major premise of the germ theory.

Bruce-Porter says: “The present artificial methods of rearing germs by passing them through other animals and then cultivating them on an entirely different food, alters the character of the germ.” “. . . purely saprophytic fungi can be educated to become parasitic by sowing the spores on a living leaf that has had injected into its tissues a substance positively chemotactic to the germ-tubes of the fungus experimented with. By similar means a parasitic fungus can be led to attack a new host plant. These experiments prove what has hitherto only been assumed, viz., that parasitism on the part of fungi is an acquired habit.” (*Nature*, No. 1792, p. 429, quoted by Schiller: *Logic for Use*, p. 328)

Dr. Robert Koch, *International Congress*, Vol. VI, said: “All competent investigators agree that the tubercle bacilli of human origin differ from the tubercle bacilli of cattle, and that, consequently, we must differentiate between a *typus humanus* and a *typus bovinus*. The British commission also admits the existence of these differences, but as some of their cultures showed definite changes in their characteristics after passage through animals and various cultivations, they have differentiated a third group, which they call ‘unstable.’”

More important still is the phenomenon of transformation in microorganisms commonly regarded as pathogenic for man. An early observer of such transformation was Dr. R. C. Rosenow, of the Mayo Biological Laboratory, Rochester, Minnesota. In *The Journal of Infectious Diseases*, 1914 (Vol. 14, pp. 1-32), he reported that he had been successful in making the streptococcus assume all the characteristics of the pneumococcus, and vice-versa, by appropriate changes in the laboratory environment. Nor were the experiments of this sort confined to the two organisms here mentioned. He was equally successful in similar attempts with other varieties.

Meanwhile the evidence accumulated that there is hardly any such thing as a specific germ. By 1932 various investigators had been able to show that a filterable-virus stage is, in some cases, merely one phase in the life-history of the organism. The crowning discovery in this field was that of Professor A. I. Kendall, of Northwestern University, who found that, by varying the culture medium, he could, at will, make many common organisms disintegrate into a filterable submicroscopic form or integrate again into the usual visible morphology. This, according to the



World Almanac for 1932, is regarded as the greatest bacteriological discovery since the work of Pasteur.

Evidence of bacterial transformation within the human body is furnished by Col. Nathan Raw, member of the International Committee for the Prevention of Tuberculosis. In British Medical Journal, August, 1933, he reports the case of a child whose glands definitely revealed the presence of tubercle bacilli of the bovine type and who eighteen years later developed pulmonary tuberculosis with tubercle bacilli of the human type.

The most significant contribution to the whole subject of the instability of bacterial forms, however, comes from the pen of Dr. W. H. Manwaring, professor of bacteriology and experimental pathology at Stanford University, writing in Science (May 25, 1934) on "Environmental Transformation of Bacteria." Among the amazing statements in this article are the following:

"The mere addition of sterile milk to a routine culture medium allegedly causes the acid-fast tubercle bacillus ... to transmute into a non-acid-fast coccus. . . . With proper preliminary selective care, this quasi-coccus can be grown indefinitely as an apparently stable new species. Non-flagellated typhoid bacilli, non-capsulated pneumococci, Gram-negative diphtheria bacilli, as well as a host of other unconventional species or genera can be produced at will. ...

"Of the eleven presumably unit characters thus far studied with certain bacterial species, there is apparently not a single character that is static under test-tube conditions, nor is there the least suggestion of a genetic 'linkage' between any two of these eleven characters. The currently assumed unit characters include such diagnostic properties as: size, morphology, staining reactions, colony type; power to ferment certain carbohydrates, pigment production, toxin production, racial specificity of fractional antigens and type of disease produced in laboratory animals. Even the specific virulences for two closely related animal species or for two different organs or tissues of the same species may vary independently of each other."

Hence, it appears that there is no such thing as a specific germ. It inevitably follows that there is no such thing as a specific serum, and the whole superstructure of the classic germ theory of disease and of serum therapy falls.

"There is apparently convincing evidence," adds Manwaring, "that hormones 'chemical organizers' or integrating enzymes play an important role in stabilizing bacterial populations, and in initiating or inhibiting dissociations or transformation phenomena."

This places the emphasis squarely where it belongs; namely, upon internal factors. Bacterial activity is purely secondary to glandular activity, and glandular activity, as is pointed out by Pottenger (*Symptoms of Visceral Disease*) is generally subordinate to nerve activity.

*Spontaneous Appearance of Virulence:* In case an organism with its ever-present microbes should sustain an injurious change in environmental factors, it is conceivable that the microbes would change with their environment or even, as we shall show in the next chapter, that body cells might degenerate into bacterial forms. This would constitute a spontaneous conditioning of the germ in situ or a spontaneous devolution of body cells. Infectious disease would thus develop spontaneously. Moreover, the very instability of bacteria renders it highly probable that forms ordinarily regarded as innocuous may readily transform themselves into so-called pathogenic types. *The Nature of Disease Journal* has published a

monograph tracing the descent of many disease germs from the common colon bacillus. Dr. R. C. Rosenow, of the Mayo Foundation, has announced that there is an unexplained and simultaneous increase in the virulence of certain germs in human throats, in the water, and milk supplies and in flies, which occurs in the autumn.

*Carriers:* The problem of the healthy carrier has proved to be insurmountable for the germ theorists. Typhoid, diphtheria, and infantile paralysis are some of the diseases which are supposed to be spread by immune individuals. The germ may remain latent in the bodies of these persons for years and, upon coming in contact with an environment favorable to such a change, become virulent.

Dr. Osier is authority for the statement that the bacilli of diphtheria, pneumonia, and many other virulent diseases are found in the bodies of healthy persons.

A celebrated French bacteriologist reported that he found in the mouth of a healthy two-months-old infant, almost every pathogenic germ. Another physician, appointed by the French government to investigate the causes of tuberculosis, informed the International Tuberculosis Congress in Rome that he found the bacilli of tuberculosis in ninety-five per cent of all the school children examined by him.

Bacteria are always present in the cow's udder. (Conn and Conn, *Bacteriology*, p. 115)

The germ of Malta fever "is found in the milk of goats which are perfectly healthy," says Hadwen.

Ninety-eight to ninety-nine per cent of all human post-mortems reveal tuberculous lesions, says Tice, indicating that this one disease is almost universal. The percentage of cows declared to be tuberculous is almost as high.

Dr. Park, testifying for the board of health in the Montclair case (*Record*, p. 1578), states that it is a fact that a large proportion of the human race has at one time or another had tuberculosis in some form; that a large percentage of humanity has had a slight infection at some time or other which, as a rule, has never been detected, that they have recovered from that infection and died of something else, and that the tendency of the human body is to fight diligently against any such infection.

Dr. Van Derslice, president of the milk commission of the Chicago Medical Society, testified that over eighty per cent of the human race at some time or other have an infection of tuberculosis, but that it is cured in most of the cases, and he argues that the same rule of infection should hold true as to cattle.

Dr. L. K. Shaw, of Albany, N. Y., testifying in the Montclair case (*Record*, p. 261), speaking of autopsies in the Bender Laboratory, says that in a series of over 1,100 autopsies, over eighty per cent showed some tuberculous lesions, healed in the majority of cases. The autopsies were performed on all classes of cases. Any physician or any institution can have the Bender Laboratory perform an autopsy. Dr. J. A. Egan, secretary of the Illinois State Board of Health, in the monthly bulletin, March, 1908 (*Record*, pp. 3706-3707), says: "40. to 60 per cent of all milk-producing cows are tuberculous." (*Reprint of Vanished Official Document*, pp. 17—18—60, 61).

All evidence points to the universality and omnipresence of bacteria.

*Omnipresence:* The omnipresence of disease germs presents a problem which follows closely on the carrier question. For the purposes of this discussion, germs of the air, soil, water, and food may be ignored (except where man has spread pollution), only those on or in the body being of importance. Many varieties of pathogenic germs are to be found in various situations in the organism, some tissues

literally swarming with them. The French pathologist Lumiere states that bacteria or their spores are inherent in every tissue. Although ubiquity is one of their outstanding characteristics, the organism is not ordinarily hampered by their presence. The omnipresence of the germ in the body and in organic matter mitigates against the practicability of serum therapy. Furthermore, there is very little to support the theory of air-borne infection, and even if this theory

could be proved it would constitute an argument for quarantine rather than inoculation. Not even quarantine would be very effective against pathogenic microbes carried by the air.

Maignen states: "There is absolutely no danger from air germs; they do not thrive in the presence of the active germs which hold the field."

Lord Lister declared: "The floating particles of the air may be disregarded in our surgical work, and if so, we may dispense with antiseptic washing and irritation, provided always that we can trust ourselves and our assistants to avoid the introduction into the wound of septic defilement from *other than atmospheric sources.*"

Béchamp, in the *Contemporary Review*, August and November, 1909, wrote: "If the simple or evolved microzymas, which may be found in certain humours of the body, came from the air and penetrated so easily the cells of the human body, there is one humour, in ceaseless contact with the air we breathe, which we should find always the same in all animals. This is the saliva of the mouth. It is found, however, that the properties of human saliva and that of other animals are different. The epithelial cells, and microzymas, and the bacteria of the tongue of man have a special chemical action personal to themselves, and *altogether different* from those of the tongue of the cow or the pig, the horse or the dog. Now, if the germs of the air do not operate to modify the function of a humour which is so unceasingly, so largely, and so directly in contact with the common air, it is difficult to understand how they operate to modify the functions of the inner tissues and humours protected by insurmountable barriers."

Concerning the artificial production of disease, Béchamp says (*Les Microzymas*, p. 819): "In all the experiments of recent years it has been the microzyma proper to an animal and not a germ of the air that has been found to be the seat of the virulence. No one has ever been able to produce with germs obtained from atmosphere any of the so-called parasitic diseases. Whenever by inoculation a typical known malady has been reproduced, it has been necessary to go and take the pretended parasite from a sick animal; thus to inoculate tuberculosis, the tubercle has been taken from a subject already affected."

Metchnikoff tells us that he has found the bacilli of Asiatic cholera widely diffused in the waters of many localities, while these same localities were practically free from cholera. Again: "The bacilli of typhoid fever have been found in inhabited localities in which typhoid fever has never been known to occur."

*Contagion*: The word "contagion," although becoming obsolete, expresses the idea that a person suffering with the disease and coming in contact with another individual may communicate the germ to him. In this latter phenomenon it is conceivable that the germ, conditioned by its recent location with virulence and an accumulation of toxic material from the infected person, may contact an environment favorable to its continued activity or even a further exaltation of the virulence. Sir Richard Douglas Powell states that if tetanus and gas gangrene germs are washed clean and freed from their environment, they are quite harmless. Note

that environment conditions the microbe and thus has more to do with the initiation of specific infection than the microbe itself.

### *Serum Therapy as Sciolism*

Even though an organism may perhaps be conditioned according to strict scientific procedure, so that bacterial activity of a certain specific nature may be prevented, slowed down, or terminated, such scientific conditioning may be an irrational act, in spite of the fact that it is scientific. A practice may be scientific and yet irrational to the  $n$ -th degree. We are dealing with the most important point in this book; perhaps one of the most important points in the mental life of every individual—a pivot on which the career of every person depends, that of clear, precise thinking. Here is the opportunity for application of the scientific habit of thought—the most important function of civilized man. When premises are untenable, and a practice is founded upon such premises, it becomes pseudo-knowledge, pseudo-science, or, more properly, sciolism. Even where the immediate result is scientifically predictable, the system remains a half-truth, for the premises are untenable and the ultimate result may be positively detrimental.

Some of the knowledge in bacteriology has been scientifically arrived at, and some of its practices can be demonstrated to work out as predicted. It is therefore assumed that serum therapy is a science. Granting this, however, does not preclude that damage may follow the practice, nor that there may be better ways of arriving at the desired objective. The uncertainty of serum therapy is everywhere conceded; some of its dangers and failures have been presented. Others will now be pointed out.

Scrum therapy is a sciolism; it is a practice which may at one and the same time be both scientific and irrational. A chemist might prepare the most complex and highly lethal gas ever conceived of, and he might employ an airplane to disseminate it in the most efficient manner ever devised, and snuff out the lives of the entire population in a fifty-mile radius. This would be a scientific, but an irrational act. Science is not the only standard by which we must judge. And this is not a criticism of science. We insist on employing science where health is concerned, but we must also be rational. Deranged or misguided scientists are a menace—and the cult of immunology is a case in point.

Let us discuss the philosophy underlying the practice. The introduction of any material which prevents, slows down, or terminates bacterial activity is in the nature of a paralyzing agent, an embalming fluid, or preservative. To admit that the agent acts on the tissues as a cell-conditioner which regulates the phenomenon is merely to move our point of attack a step further; the procedure still remains vulnerable to this argument. There can be no controversy as to whether the agent prevents, slows down, or terminates the activity; this is conceded. But there may be some quibbling as to how such result is accomplished. The important point is that under ordinary circumstances bacterial metabolic processes develop when material suitable to such activity has sufficiently accumulated in the organism, and enervation or other abnormal conditions are present. The simplification or disintegration of this material will in all likelihood be to the ultimate benefit of the organism, and the completion of the process will result in the elimination of the microbe and the pathogenic substances which supported it.

There is no questioning the fact that certain infectious diseases of childhood are self-limited. What constitutes the factor of self-limitation if it is not the *conversion* of those substances on which bacteria flourish or from which bacteria evolve? What

subsequent recurrences of the disease are possible without a replenishment of those substances? This is in line with Pasteur's theory. As Stedman gives it, Pasteur held that "Immunity produced by an attack of a disease or vaccination is due to exhaustion of the soil necessary for the growth of the specific microorganism."

We do not, however, concur with Pasteur's inclusion of vaccination.

The artificial prevention, termination, or slowing down of the bacterial process, while it may be scientifically accomplished, may also be an irrational act. There is a vast difference between the so-called immunity naturally attained and that which is artificially provided. It would appear that artificial immunity preserves the material, kills, embalms, or paralyzes the microbe, or changes the nature of the cell, while natural immunity occurs when the material has been "converted" or simplified—quite a different phenomenon. That these two immunities are not the same is proved by the facts of anaphylaxis and hypersensitiveness to serum after artificial immunization, which *never* occur with natural immunity. If anaphylaxis or hypersensitiveness occurred in every case in a group, it could be said that foreign substances or poor technic were the cause, but there are many instances in which the inoculation of a standard batch of serum was uneventful except in one victim, a circumstance which is definite proof of the dissimilarity of the body's reaction to a disease and to inoculation.

Furthermore, the tissues of an artificially-immunized person are different from those of a naturally-immunized person. "The blood of an inoculated person has greater immunizing power than the blood of a person who has merely had the infection." (*Chemistry in Medicine*, pp. 565-566) Specific bacterial activity is interfered with by inoculation; bacterial conversion of certain substances is suppressed by artificial immunization. Thus we have two different subjects: one in whom bacterial metabolic activities have been permitted to convert and simplify complex substances and follow through to a spontaneous termination; another in whom such bacterial activities have been suppressed and the complex substances allowed to remain for a different form of bacterial activity to gain ascendancy and perhaps precipitate a more devastating process upon the human organism. J. W. Hodge, M.D., says: "Serum therapy, the outgrowth of the germ theory, is regarded by many eminent pathologists as the principal factor in the increased prevalence of cancer

and consumption."

A. F. Coca (*The Newer Knowledge of Bacteriology and Immunology*, p. 1011) says: "Hay fever or asthma cannot be induced in a normal human being by injection of horse serum. However, the re-injection of horse serum after the incubation period of serum disease is sometimes followed by an accelerated or immediate reaction of serum disease, which is so severe that it may even endanger life or actually end it." Serum therapy changes the tissues, and the burden of proof is upon its practitioners to demonstrate without equivocation that widespread damage is not being caused by the practice. That the body is definitely altered by inoculation is attested to by Jousset in *The Pathogenic Microbes* (p. 140): "The cellular theory wished to explain the natural immunity, like the acquired immunity, by phagocytosis and the property which the phagocytes should have of neutralizing the microbial poisons; but that explanation only increased the difficulty, for it was necessary to explain why the leucocytes have become refractory to the microbial poisons. What is most grave, is that the facts demonstrate that phagocytosis exercises no influence in the natural immunity, since the hen, into which one injects a pure culture of tetanus, far from

destroying the microbe by phagocytosis, transforms it, elaborates it and makes of it a vaccine.”

Again, on page 159: “Roux first asserted that the anti-toxines are much more abundant in the blood of animals as they have received the more toxins. He added, that as one ceased to inject the toxine, and as they frequently bled the immunized animals, the anti-toxine was rapidly exhausted and disappeared.

“These arguments are absolutely contradicted by the experiments which we have already reported, and from which it results that the immunizing and curative property of the serums persists, even after the renewing of the entire mass of blood through bleeding.”

Let it be emphasized, then, that, although toxin-antitoxin may confer a very brief “immunity,” it permanently alters the tissues of the body—a point more fully discussed under hypersensitiveness. Note, also, that if all the blood is withdrawn and the subject is still “immune,” the antibody theory must be revised or discarded. Obviously Roux was guessing—on a matter where scientific experiment of the simplest kind would have given him facts. It is with such men and their methods that we take issue. Searching inquiry rather than dogmatism should be the ambition of the scientist. Many questions arise on this greatest of all problems—human welfare and health—and guessing and dogmatism must not be substituted for research.

*Substitution of Disease:* What proof is there that degenerative diseases such as cancer and the cardio-renal-vascular combination are not increasing because of errors of commission rather than of omission? Why is it always assumed that something must be done to the body, and never suggested that the body be left alone? How are we to know whether or not one or more of the genes of the germ cells are being tampered with by the immunologist—thus inducing hereditary defects? The cancer cell is an altered cell. How are we to know whether or not benign germs are so modified by the immunologist that they lose their function of symbiosis? Where are the body of evidence and the statistics by which one can be definitely convinced of the value of chemical versus biological methods in preventive work, and the comparative value of combative versus cooperative methods in therapy? Where is the evidence to prove definitely that the vast majority of people, given fair hygienic, sanitary, and nutritional conditions, would be handicapped or doomed if deprived of the present-day medical practices which we hold are scientific? This is not cavil; it is a demand that those on the affirmative present proof in support of their position. We revolt against physical and mental coercion; we don’t want the torch of reason replaced by a policeman’s baton. Bear in mind the foregoing questions when reading the reports which follow presently on the failures and dangers of serum therapy. In regard to vaccination against smallpox, for instance, we read: “In Holland it has been tabu ever since they learned from statistics that during ‘the period from 1920 to 1928 531 persons were killed through the use of it. The dreaded disease known as vaccination encephalitis was a common fatal complication.” (*Plain Talk Magazine*, October 1933)

In the *Journal of the American Medical Association* (March 15, 1930), R. A. Perritt, M.D. and R. C. Carrell, M.D., Chicago, writing of postvaccinal myelitis, say: “Cases of postvaccinal disseminated encephalomyelitis in which both the brain and the spinal cord are involved have become of late, as has been said, not unusual, especially in Holland and England. Clinically they give a picture of diffuse involvement of the central nervous system and generally have a grave prognosis

(death in about 57%). ... In Perdran's opinion the histologic changes are similar to those that occur in cases of encephalomyelitis caused by smallpox. In Spiller's case . . . the patient was a man, aged 38, who exhibited a flaccid paraplegia eight days after the eruption; the upper extremities were not involved; there was no pain, but the sensation was somewhat impaired; the bladder was distended up to the umbilicus; the mental condition was good. Death occurred on the thirty-eighth day of the disease. In another patient, a girl, aged 19, a severe smallpox progressed well up to the third week of the disease, when she became unable to move her legs in bed. Sensibility was somewhat impaired; and there was incontinence of bladder and rectum. Death followed at the end of about 8 or 9 weeks."

The foregoing occurs in comment on the author's case of a girl of 18 who developed anterior poliomyelitis after vaccination and remained paralyzed in the lower extremities.

Such reports make imperative the formulation of certain postulates which we will presently introduce. Here are still others:

"In 1933, 172 children were taken seriously ill, with one death in eastern France, following injections of anti-diphtheric serum. The injections caused abscesses locally and some of the children 'had to be hurriedly operated on,' and 'scores narrowly escaped death' . . .

"The tragedy at Luebeck took the lives of 76 babies as the result of vaccination with a tuberculin serum of the Pasteur Institute— Calmette's—and 100 more were made seriously ill. It was said at the time that it was feared that none of the 253 vaccinated could escape death.

"Another wholesale poisoning with the same vaccine came to light during the Luebeck trial for manslaughter of the doctors and nurse specially implicated—that of 757 infants of the Madrid Children's Home. In this, 164 died and 333 became ill. Rumors also of another big tragedy of the same kind, in eastern Europe, were afloat at the time.

"These things are on a big scale, but what of the minor 'accidents'? What of individual deaths, and illnesses, and health impaired for life? What archives tabulate these?"

Inability or unwillingness of the courts to convict does not alter the facts. Death or injury cannot be tempered or minimized by judicial findings. Wholesale disaster calls for rigid scientific inquiry. Failure to convict in a court of law does not relieve the principals of criminal responsibility, nor does it establish their innocence. A court of unbiased scientists would find otherwise. The following criteria should form the basis of such an investigation:

To prove the superiority of serum or vaccine therapy over all other systems, it must be shown that

1. Death or serious illness is inevitable in every case in which serum or vaccine is not used.
2. Recovery is inevitable in every case in which serum is used.
3. The serum cannot so condition the body as to render it susceptible later to anaphylactic shock.
4. The serum will make no other major change in the tissues. (A guarantee against allergy.)
5. Serum therapy is absolutely the only method worth considering; that is, no other method has either rational or empirical justification as an alternative.

In connection with these postulates it will be necessary to insist that close follow-up records be kept of all individuals inoculated, as well as of the controls. No one knows what diseases follow the use of toxin-antitoxin or other biological products. In this connection we can paraphrase the complaint from “the prevention of blindness committee of the Union of Counties Associations for the Blind in England which has issued an important report on hereditary blindness,” according to the London correspondent of *The Journal of the American Medical Association*:

“No authentic knowledge has been obtained of the extent to which diseases of genetic origin occur sporadically and apparently devoid of genetic potentiality. Cases can be pointed to in which one man or woman suffering from, say, retinitis pigmentosa has resulted in some forty cases of blindness in subsequent generations. But no one knows the proportion of the total number of persons suffering from such disease who have married without any such ill effects. . . . Complete statistics compiled (1) on a uniform basis, (2) on the evidence of properly qualified persons, and (3) including all persons affected by the diseases and not only those selected from certain pedigrees would assist in determining the total amount of blindness that can be prevented by the control of parentage. At present such statistics do not exist.”

This complaint is paralleled in immunology. No one is able to say what deferred sequelae follow serum therapy. Until statistical information is ‘available, all conclusions must be held in abeyance. We require negative evidence as well as the glowing half-truths pictured to us by immunologists. We demand that they prove the effects of what they *don't* do, as well as what they do. We want assurance that their work is confined to the one specific effect which they claim. We require them not merely to affirm, but also, conclusively to demonstrate the converse of their proposition. For example, it is not sufficient to recommend a cathartic, cholagogue, or contraceptive which actually does as predicted; it is further necessary to guarantee that the agent will cause no injury. We impose the same kind of scientific requirement upon the proponents of artificial immunology. Until they meet this requirement we shall class their practice as a sciolism.

It is popularly assumed that disease processes are something to be fought; that disease is unnatural, and injurious. Such assumptions are without foundation. Disease is a natural process, and when properly managed generally terminates in a manner compatible with the continued survival of the organism. Infection occurs only where the field is primed for it. This is not an appeal for bigger and better diseases, but a plea for a better understanding of the principles underlying the phenomenon and a more rational therapy which will cooperate with bacterial activity rather than suppress or combat it. Serum therapy constitutes an unwarranted interference with a greatly misunderstood phenomenon. No better proof of this contention could be advanced than the following well-authenticated records of its failures and dangers (which include paralysis and death). Diphtheria and tetanus are selected because it is the particular delight of certain enthusiasts to boast proudly that these diseases have been mastered by serum therapy.

*Dangers of Serum Therapy*: In the *Journal of the American Medical Association* (April 18, 1931), Dr. Ernest E. Irons, of Chicago, in giving the results of a questionnaire, says:

“Excluding hearsay reports, we obtained records of 140 instances of harmful results, including a number of cases in which death was considered to have been



caused by the use of vaccines subcutaneously injected. Seventeen cases of asthma were reported to have followed courses of bacterial vaccines, administered to patients who previously were not known to have suffered from asthma.”

In an editorial in the *Journal of the American Medical Association* (June 6, 1931), it is said:

“Everyone who deals extensively with immune serums realizes that their practical use is attended extensively with certain menaces. . . . anaphylactic shock occasionally occurs. . . . The most common symptom is a skin eruption, which is usually urticarial but may vary considerably in type. Edema may appear in various parts of the body, notably the face. Multiple joint pains, albuminuria, leukocytosis and general malaise are sometimes encountered.

“The possibility of these occurrences may indicate why the use of therapeutic scrums awakens hesitancy in a physician faced with the possibility of discomfort, harm and even death in a patient despite all care to avert any untoward effects.”

In the *Journal of the American Medical Association* (April 2, 1927), Drs. W. E. Gatewood, and C. W. Baldrige say that: “A multiplicity of untoward sequelae have been observed in patients treated with immune serum.”

The *Journal of the American Medical Association* (December 6, 1919), reports forty severe reactions and five deaths in Dallas, Texas from toxin-antitoxin. Damages ranging from \$100 to \$1,000 were awarded in each of fifty cases.

Owing to its dangers some states have now abandoned toxin-antitoxin for toxoid, but in the *Southern Medical Journal* (August, 1931), in an article, “Diphtheria Immunization in Private Practice,” W. W. Anderson states:

“The reactions following toxoid are a little greater than those following toxin-antitoxin.”

Understand that the product which preceded toxoid was touted as perfect by some “authorities,” but toxoid was produced because it was an “improvement” on what the gullible had been told could not be improved. Be informed, in passing, that toxoid contains formaldehyde. The commercial ballyhoo of manufacturers of biological-pathological products has led the medical and scientific world astray. It has certainly paid them to advertise, but it has been hard on the public, and has played havoc with medical ethics and reputation.

In the *Journal of the American Medical Association* (July 9, 1932), a doctor asks if a child has been exposed to diphtheria, would it be better to give that child “an immunizing dose of toxin-antitoxin or wait and give a large dose of antitoxin if the child developed diphtheria.”

Notwithstanding the advocacy of toxin-antitoxin by the American Medical Association, the editor replied: “Formerly it was general practice to give an immunizing dose of antitoxin to persons who had been exposed to diphtheria with the idea that it was harmless. It is now known that even a small primary dose of serum may produce alarming and serious reactions. It may also render the individual sensitive to serum administered subsequently. With the multiplication of immunizing and therapeutic serums and their more general use, it has become apparent that hypersensitiveness to subsequent injections of scrums should not be established when it can be avoided.”

In the *Journal of the American Medical Association* (April 10, 1926, p. 1169), there is a description of a case of anaphylactic shock from anti-tetanus serum in which it is said: “This induced in less than one minute a very severe anaphylactic shock, only temporarily relieved by epinephrine injections. It did not subside

completely until late the next day. Phenomena of serum sickness and urticaria kept the patient in bed for two weeks longer; complete recovery not occurring until after two months. Inquiry revealed that the woman had been treated with diphtheria antitoxin at the age of two. The interval between the sensitizing injection and the injection unleashing the anaphylaxis was thus nineteen years.”

In the *Journal of the American Medical Association* (April 2, 1932, p. 1139), a report of twenty-one cases of paralysis “associated with the use of the tetanus antitoxin,” is referred to by Dr. Forrest Young. “The cases in the group showing signs of involvement of the central nervous system are on the whole much more serious as regards life. The usual clinical courses seem to be as follows: The patient receives serum, generally intraspinally, in repeated doses. After a time, varying from three days to a month, and without warning, another injection is followed by a severe reaction. This is manifest usually by a generalized urticarial eruption, which is followed in a few hours by convulsions, opisthotonus, coma, high irregular pulse, irregularity in respirations, and possibly death.”

In the *Journal of the American Medical Association* (October 31, 1931, p. 1339), we read: “The physician who administers anti-tetanus serum as a routine in cases of insignificant abrasions does not do so without risk.”

In the *Journal of the American Medical Association* (May 7, 1932, p. 1625), Dr. Alfred Gordon of Philadelphia says: “Since the introduction of anti-microbial serums, a fairly large number of complications have been observed, motor and sensory phenomena occupying the most important place among them. Anti-tetanic serum is the one frequently given as a preventive measure, and in this capacity it is incriminated as the direct cause of the paralytic phenomena that occasionally follow its administration.

“To avoid all possible litigation, it is suggested to have the patients sign a certificate relieving the physician from all responsibility and consequence of immunization work.”

What a science! Imagine an architect, engineer, attorney, or any other highly trained specialist hedging and forcing his client to assume great risks in fields wherein the expert’s services had been retained for the specific reason that the client was totally ignorant of the risks! Is it any wonder that fear and suspicion grip those patients who are able to think in terms of their own welfare?

If greater publicity were given the dissenting opinions, the controversies, and the discussions originating within the medical profession itself; if the opinions of the more competent and candid members of the profession (those who are honestly and seriously seeking progress and knowledge) were readily available to the laity—to the people who buy medical services, there is little doubt that the public attitude toward the profession and its accomplishments would be quite different from what it is. The publicity, the propaganda, the accounts of success and progress—these for the most part emanate from biased sources, sources subservient to and governed by men of mediocre professional standing, incompetent local “health officers” (who are frequently uninformed laymen), politico-doctors, staffs of medical and surgical supply houses, publicity mongers in the employ of biological laboratories, and similar financially interested go-getters.

There is no widespread publicity of controversial issues, the doubts, and the failures attending their practices. In certain instances the layman is led to believe that he may purchase certain of these professional services in very much the same manner as he purchases commodities, that is, with a reasonable degree of assurance of the

value thereof. To the contrary, the doctor in too many cases accepts fees from his patient having only a remote idea that the “services” for which he has accepted compensation will be of any value. How different would be the patient’s attitude if he were fully aware of this! How essential that the publicity and propaganda be of the nature to command confidence! That fears and suspicions are well founded is attested to by what follows hereafter. Even though strict censorship keeps this sort of “news” out of the press, yet the thinking public in every community is cognizant of the mystery and silence which shroud the practice of serum therapy.

*Failures of Serum Therapy:* In *Medical Notes from the Front*, Dr. A. Lumiere, the well-known scientist of Lyons, in the *New York Medical Journal* (August 4, 1917, p. 224), tells of fifty-four patients “who had received a preventive injection and in whom, nevertheless, the disease developed. . . .” Trismus (lockjaw) developed in twenty-six of the fifty-four cases “with quite the same intensity as in a case in which no prophylactic injections had been administered.”

In the *Journal of the American Medical Association* (August 12, 1922, p. 569), cases of Dr. Lop of Marseilles are described, and it is said: “Lop does not use, at the present time, the preventive injections of anti-tetanic serum, nor has he done so since 1906, in which year he observed two fatal cases of tetanus in patients who had received\* preventive injections of anti-tetanic serum that had come directly from the Pasteur Institute. Among the 15,000 injured persons that he has treated since that time, there were some who presented serious bruises and lacerations. Nearly all the wounds were contaminated with grease and dirt.”

Dr. George Wilson, LL.D. (Edinburgh), F.R.S., D.P.H. (Cambridge), Medical Officer of Health for Warwick, president of the State Medicine Section of the British Medical Association, “probably the leading authority in Great Britain upon preventive medicine,” and a member of the Royal Commission on Vivisection, says, in his minority report of that commission, that large quantities of the anti-tetanus serum were shipped out to Africa during the late war, but Dr. Martin admitted that “clean surgery prevented the occurrence of tetanus, notwithstanding the enormous number of wounded, and I therefore contend that the prompt cleansing or washing of all wounds is the only natural prophylactic. It does not follow that because either a man or a horse, when suffering from a wound or abrasion, escapes an attack of tetanus, after being injected with anti-tetanus serum, he would suffer from tetanus if he were not injected. As it failed as a remedy, I contend that on the same grounds it is valueless as a prophylactic.”

Speransky has shown the limited beneficial effects of tetanus antitoxin to be due to its action as a specific nerve stimulus. He has shown that novocaine injections are even more effective than the supposedly specific antitoxin in controlling tetanus. Tissot shows that the use of tetanus antitoxin is not only erroneous and ineffective but also is harmful and dangerous.

It is constantly claimed that children are rendered immune from diphtheria by toxin-antitoxin. Medical literature confirms the contrary. Schwartz and Janney say in the *American Journal of Diseases of Children* (March, 1930) that toxin-antitoxin “has two distinct disadvantages, the first is its variable protective value—about twenty-five per cent of the patients remaining insufficiently immunized after three doses of toxin-antitoxin; the second is the danger of serum sensitization following its use.”

They quote the Gary, Indiana schools as having “30 per cent of the patients remaining susceptible to diphtheria.” At Whitefish Bay, Wisconsin, “33.7 per cent remained susceptible to diphtheria.” The Cook County Hospital, Chicago, during an epidemic, found that “67.2 per cent” of the immunized nurses “gave a positive reaction to the Schick test,” and out of “29 cases of diphtheria among the nurses, 9 had had toxin-antitoxin.”

The *United States Public Health Report* (July 8, 1932) says of diphtheria: “Among the geographical sections, however, the West, North Central, South Central, and Mountain and Pacific areas showed excesses over last year of 8 per cent, 18 per cent, and 10 per cent, respectively. In the South Central and Mountain Pacific groups the incidence was not only higher than it was last year, but it was the highest for this period for four years.”

The *United States Public Health Report* (October 28, 1932) says: “Each geographic area reported an increase over the preceding four-week period. . . .” “For the four weeks ended October 8, there were reported 5,695 cases, more than double the number of cases reported in the July 8 issue.”

Speransky has shown that the characteristic pathology of diphtheria is produced by abnormal nerve impulses. Tissot has consistently produced the characteristic diphtheria pathology with products of both wheat meal and barley meal. He cites numerous statistics showing that diphtheria “immunization” increases both the incidence and the mortality of diphtheria. (Summarized elsewhere in this book.)

In the *Journal of the Medical Society of New Jersey* (January, 1930, p. 16), John McK. Mitchell, M.D., says: “During the period covered by the increased use of toxin-antitoxin and therapeutic serums, the incidence of serum disease has also increased. . . .

“It is well known that one dose of therapeutic serum will frequently sensitize to a second dose. If in addition toxin-antitoxin is capable of sensitizing, we have in the more frequent use of therapeutic serums, and in the wide use of toxin-antitoxin, the probable explanation of this recent increase in serum disease.

“Serum disease following scarlet fever antitoxin is more frequent and more severe than that following diphtheria and erysipelas anti-toxins. A possible explanation of this lies in the greater percentage of scarlet fever patients who have previously received toxin-antitoxin. Among 701 patients in Gordon and Creswell’s series (table IV) who received scarlet fever antitoxin, 37.6% had previously received toxin-antitoxin while 18% of 1556 diphtheria patients and only 4.5% of 200 erysipelas patients had received it. Serum disease among these three groups occurred in 43.6%, 30.2% and 7.5% respectively

“That 18% of diphtheria patients should have previously received toxin-antitoxin may come as an unpleasant surprise. However, Silverman has recently reported that of 1000 children in Syracuse who received a full course of toxin-antitoxin, 31% gave a positive Schick test six months later. In a clinic with which the writer is personally connected, of 219 children who received a full course of toxin-antitoxin and a Schick test within one year, 31% gave a positive reaction. These children were all under six years of age, at which time immunization is supposed to be most effective. [Are you sure your child is “safe”?-J.R.V.]

“Reactions following the use of therapeutic serums are, on the whole, mild. Gordon and Creswell report only two severe reactions out of 917 cases. On this ground the physician working in institutions may with reason feel that he can ignore

the sensitivity induced by toxin-antitoxin. To the physician who encounters these reactions in the home, however, the matter presents itself in a different light.”

Of course, it is all right to have “two severe reactions out of 917 cases” in an institution. There are no irate parents to demand explanations. In private practice it is different. Why risk immediate and remote dangers in quest of a doubtful and temporary “immunity” which you may never secure? If, perchance, you wish to risk being one of the two severe cases in 917, or one of the more numerous “mild” ones, well and good, but do not place a helpless child in jeopardy. This is the worst kind of lottery.

C. D. Mercer, M.D., F.A.C.P., of West Union, Iowa, reports that out of 125 children between the ages of six and twenty who appeared for immunization 27, or 20%, had albuminuria after the third injection of toxin-antitoxin, whereas only 13, or 10%, had albuminuria prior to inoculation. A stock serum manufactured by a well-known firm and supplied by the State Board of Health was used and the regulation dose of 1 cc. at seven-day intervals was administered.

Speculation as to the cause of the albuminuria in the 10% prior to inoculation raises the question as to whether this group of children had been Schick tested. If the third inoculation left the albuminuria cases more than doubled, is it not fair to ask whether Schick testing may not cause at least some such cases—assuming, of course, that the Schick test was applied? This is a fair question to put—a just speculation; that it cannot be answered is an excellent demonstration of the paucity of medical facts from which to draw scientific conclusions.

As a further illustration of this point we quote from The New York Times (October 5, 1933): “The resources of the State have been marshalled against an epidemic of brain fever among horses of three Northern Utah Counties, which has resulted in the deaths of more than 1,000 and continues unabated.

“The total loss from the disease, estimated at \$100,000, will not be known until after the fall round-ups have determined the extent of the epidemic among horses on the range.”

We should like to know if these horses were at any time mallein tested. We view with suspicion such preventives and such therapies and the humbugs who try to mystify us with them.

Let us once more examine Koch’s postulates for the purpose of getting more generalized evidence concerning the practice of serum therapy. Here they are in their most rigid form—the only form in which they are of any scientific value: The specific microbe should be

1. Found in every case of the disease.
2. Never found apart from the disease.
3. Capable of isolation and culture outside the body.
4. Capable of producing by injection the same disease as that undergone by the body from which it was taken, and again recovered.

*Exception to postulate no. 1*

“There have been many cases of diphtheria in which for one reason or another no bacilli were found in the cultures by the examiner. ... In many of these cases later cultures revealed them.” (Stedman’s Reference Handbook of the Medical Sciences, Vol. III, p. 609)

*Exception to postulates nos. 1 and 2*

“One of the original props of Pasteurian orthodoxy, the Klebs-Loeffler bacillus, arraigned as the fell agent of diphtheria, was by Loeffler himself found wanting in 25 per cent of the cases; while, on the other hand, it is constantly revealed in the throats of healthy subjects.” Diphtheria again has our special attention.

*Exception to postulate no. 3*

Rosenow, Manwaring, Pasteur, Roux, and numberless other investigators have demonstrated conclusively that what the microorganism is in behavior and morphology depends upon what they want it to be. They condition the environment and the germ changes its behavior and form in keeping therewith.

*Exception to postulate no. 4*

A report from the Milwaukee County Hospital for Mental Diseases regarding the inoculation of malaria germs as a treatment for paresis states that eleven out of ninety-nine patients proved immune to malaria.

*Spontaneous Appearance of Infectious Disease:* Additional evidence of exceptions to Koch’s postulates appear elsewhere throughout this text, but the fact that infectious disease may appear spontaneously deserves special notice. Horace P. Homes, M.D., says: “It is a well-known fact that where a body of men, as soldiers, miners, etc., are quartered in a new country, they are liable to an epidemic of typhoid fever. This is the case even where the atmosphere is the purest, the waters absolutely uncontaminated, and the drainage perfect, as in mountain camps. Another fruitful source is the turning up of new soil, as in the breaking of the wild prairies, excavations for railroads, ditches, street grades, sewers, etc. Even where there has never been a case of typhoid, an epidemic will most likely follow these new country excavations. It is not the case in the old farming regions.”

It is a well known clinical observation that the early symptoms of many so-called germ diseases appear some time before it is possible to detect the germ in the body fluids of the patient. This is especially the case in pulmonary tuberculosis, diphtheria, and typhoid fever. If the germ were the cause, one would certainly expect it to precede the manifestations of the disease. The opposite is the case—a fact which lends support to the idea that the germ is a product, rather than the cause, of the pathology.

In *Plain Talk Magazine* (May, 1930), W. A. Gills, M.D., U.S.N. (Retired), Navy Medical Service, writes: “On the 33-year-old-ship that carried 1100 persons to Guam—a 24-day journey—and on to the Philippines—five days longer—800 enlisted men were herded in unsanitary quarters. Since the sailors’ quarters were almost on a level with the sea, which was far from calm, portholes had to be kept closed despite the intense heat of the tropics. At night when the men came up for air, they slept on the decks which were scrubbed daily and which because of the porous condition of the venerable boards, were never completely dried. Since obviously there was not room on the decks for 800 men, many of the sailors had to remain in their stifling holes. Adequate bathing facilities were not provided and there was an insufferable odor from toilets too long in use to be adequately cleansed. Before the ship reached Guam measles *appeared*.” (Emphasis by J.R.V.) “Appeared” is the correct word. Measles, in this case, developed spontaneously; no extraneous germ was necessary.

In *Abstracts of War Surgery* (p. 115), a publication of the Surgeon-General's Office, we read that ". . . infection occurs more or less spontaneously in individuals possessed with lower resistance." "Spontaneously" is the correct word, even though it may have been employed here unintentionally.

Florence Nightingale, whose practical experience qualifies her to speak with authority on the spontaneity of disease, asks on page 19, of her *Notes on Nursing*: "Is it not living in a continual mistake, to look upon diseases, as we do now, as separate entities, which must exist, like cats and dogs, instead of looking upon them as conditions, like a dirty and clean condition, and just as much under our own control; or rather, as the *reactions . . . against the conditions in which we have placed ourselves?* I was brought up by scientific men and ignorant women distinctly to believe that smallpox was a thing of which there was once a specimen in the world, which went on propagating itself in a perpetual chain of descent, just as much as that there was a first dog (or a first pair of dogs) and that smallpox would not begin itself any more than a new dog would begin without there having been a parent dog. Since then I have seen with my eyes and smelt with my nose smallpox growing up in first specimens, either in close rooms or in overcrowded wards, where it would not by any possibility have been 'caught,' but must have begun. Nay, more, I have seen diseases begin, grow up and pass into one another. Now dogs do not pass into cats. I have seen, for instance, with a little overcrowding, continued fever grow up, and with a little more, typhoid fever, and with a little more, typhus, and all in the same ward or hut. For diseases, as all experience shows, are adjectives, not noun substantives. . . . The specific disease doctrine is the grand refuge of weak, uncultured, unstable minds, such as now rule in the medical profession. There are no specific diseases; there are specific disease conditions." Very recent substantiation of the merging of one disease into another is presented by Major Joseph A. Mendelson. The citation and our analysis will be found in the chapter on immunity, under the discussion of smallpox.

An example of how the specific-disease doctrine, spoken of by Miss Nightingale, leads investigators astray is evident in the "five postulates predicted of pollen in relation to its causation of hay fever," *Hay Fever and Asthma* (p. 546), by Coca and others:

1. The pollen must contain an excitant of hay fever.
2. The pollen must be anemophilus or wind borne, as regards its mode of pollination.
3. The pollen must be produced in sufficiently large quantities.
4. The pollen must be sufficiently buoyant to be earned considerable distances.
5. The plant producing the pollen must be widely and abundantly distributed.

If, following the example of Koch, the investigators had included the postulate: "Hay fever must in no case arise from any other cause," their method would have been scientific. A set of postulates must overlook no possibility. The five conditions they have enumerated are not postulates in the accepted sense, but merely a superficial attempt to justify an *a priori* hypothesis.

*Unapparent Infections*: A revolutionary concept in epidemiology was advanced in the journal called *Scientia* (March and April, 1933) by Professor Charles Nicolle, of the College de France, Paris, and the Pasteur Institute in Tunis. Nicolle had been injecting guinea-pigs with the virus of typhus fever and waiting for them to generate the desired antibodies. Guinea-pigs are chosen for the purpose because they are

much more mildly affected by typhus than are human beings, a transient rise in body temperature being practically the only clinical manifestation. The most satisfactory method of transporting the serum from the laboratory to the place where it is to be used (from Tunis to Paris, for example) is to ship the live animals that have been actively immunized in this way.

In the course of such work Nicolle discovered that a certain percentage of the animals failed to develop any fever. He at first suspected that he had been guilty of some fault in technique and was annoyed for fear that he was losing some of his precious material. Further investigation revealed that the blood of these afebrile, symptom-free animals was, for a time, just as capable of transmitting the disease to healthy animals as was the blood of animals visibly affected by the injections of the virus. There was no question but that they had been successfully inoculated and were harboring the virus. Yet *they had no symptoms*. Furthermore, a little later, the blood of these atypical cases had all the immune properties of animals that had manifested the objective evidences of infection.

In other words, *a subject may become infected and acquire immunity without manifesting a single symptom or disturbance of function*. Call this infection, if you will, but it is certainly not disease. Nicolle calls it “unapparent infection,” and he makes use of the concept to explain such facts as the following: Children who have come into contact with diphtheria sometimes acquire immunity to diphtheria without *apparently* getting the disease. Very few of the children exposed to poliomyelitis become patients, a fact which he explains by saying that most cases of poliomyelitis are unapparent infections. It is often impossible to reproduce experimentally a particular infection in human beings. This is because the subject has acquired immunity by reason of a previous unapparent, and hence unsuspected, infection. The supposed immunity of certain animal species to particular diseases may in reality constitute general susceptibility to the disease in the realm of unapparent infection. It is also suggested that man may constantly be acquiring some unapparent infection from domestic animals which he carries from one animal to another, the animal suffering from the disease in its apparent form. Domestic animals may likewise serve as vehicles in the transmission of manifest disease from one human being to another.

It is obvious that all this implies a tremendously complicated sanitary science, unless the emphasis is shifted from germs and viruses to the fortifying of the body. If Nicolle’s suggested applications to sanitation and prophylaxis were to be carried out, probably the greater part of the human race and of all domestic animals would be constantly in quarantine. After endless serum tests and serumization we would, if we still survived, be declared sufficiently immunized to be allowed at large.

To us Nicolle’s discovery is convincing proof of the contention that there are factors within the human body which can be relied upon, when properly understood and controlled, to maintain our safety in a world of germs without recourse to artificial immunization.

Speransky has shown that man need not be “victimized” by the environment; man can overcome most environmental hazards by rational and scientific means.

*Allergy and Anaphylaxis*: How much of hay fever and asthma and other allergic conditions has been caused by earlier tampering with and conditioning of the organism? How many of the million children with defective hearts can attribute their handicap to vaccines and serums? What are the possibilities that serum or vaccine may condition the endocardium for bacterial development? Is it possible that some



of the numerous and increasing cardiac failures may be due to delayed anaphylaxis? Maignen says: "If heart disease is due to microorganisms and toxins from lesions in some part or other of the anatomy, is it unreasonable to suppose that the serums prepared with the bacteria or toxins of such diseases possess characters which are apt to affect the heart in the same way as the original infective matter?"

Does this sound speculative? If so, these are matters for the proponents of serum therapy to reflect upon and publish their conclusions. The burden of proof is upon their school of practice to present conclusive evidence that vaccination and serumization are not causing widespread damage—proof of the same rigorous nature which is demanded in all fields of science. Again: *Primo non nocere*.

"The same mixture which is indifferent for a certain number of guinea pigs," writes Jousset in *The Pathogenic Microbes* (p. 162), "becomes toxic for others that are absolutely healthy; and it will always kill guinea pigs of the same weight, being *apparently* in health, but which have some time previously served for other experiments." Remember that death is only one of the unfavorable possibilities.

"If one injects some blood from a rabbit under the skin of a guinea pig, the serum of that animal, which naturally possessed no property harmful for the organism of the rabbit, becomes, after two or three injections of three to five cubic centimeters, globulicidal to such a point that it destroys the blood globules of the rabbit in several hours and causes its death." (*ibid.*, p. 56)

Remote or latent allergy or anaphylaxis are phenomena about which too little is known to encourage wholesale serum therapy. It would seem that enough is already known of the tragic possibilities forever to condemn the practice.

*Laboratory Difficulties in Connection with Sera and Vaccines:* The problem of the laboratory is fraught with great difficulties and risks:

1. The task of securing immunizing material from the  
horse (diphtheria)  
rabbit (rabies)  
man (infantile paralysis)  
calf (smallpox)
2. The guaranteeing of the laboratory animals' health
3. The standardizing of potency
4. The maintenance of proper temperature during storage and transportation
5. The assurance that the supply is sufficiently fresh
6. The guarding against contamination

*Manwaring Passes Judgment:* W. H. Manwaring, professor of bacteriology and experimental pathology at Stanford University, well sums up the history of immunology in the following language:

"Our first half-century of modern immunology has been characterized by recurring waves of clinical hope and clinical disappointment. A hundred theoretically logical monovalent, polyvalent, prophylactic, and curative antisera proposed, clinically tested, and commercially exploited during the transitional years of the twentieth century. Ninety-five per cent of them thrown into the clinical discard. A scant five per cent of them of more than historic interest. A thousand frantic clinical trials with theoretically logical opsonic index and leucocytic extract. All shelved with the miasmas and phlebotomies of our Revolutionary ancestors.

“This overwhelming clinical disappointment has served one useful purpose. It has graphically dramatized the errors and inadequacies of the immunological and consistent deductions. No immunological hypothesis of the past half-century has had a clinical verification probability of more than five per cent.

“A clinician whose collateral reading leads him into the field of primitive anthropology and comparative religion will be surprised to find that theoretical immunology, which he has previously considered the newest branch of scientific medicine, is in reality the oldest clinical science. The medicine-men of the Congo and the jungle doctors of the Orinoco have today an immunological theory that is more detailed and wider of clinical application than the boasted immunoscience of Nordic medicine. There is not a fundamental deduction from present-day infectious theory that was not known, predicted or parodied by the predynastic Osiers, fifty centuries before the nineteenth century renaissance of the same deductions.”

Similar testimony bearing on the historic aspect of vaccination elicited great guffaws from vaccination enthusiasts in the past. Sanitation did what the vaccine got credit for, and smallpox is not very common now. But error dies hard.

*Non-Medical Therapy:* It will be argued that, in view of the benefits claimed for antitoxin, the ratio of damage cannot possibly offset the good it accomplishes. To which we reply that this argument might be tenable, were there no other means available, but when it is pointed out that safer, more rational and more efficient methods can be employed, the argument is shown to be specious.

There are several schools of rational therapy—some dietetic, some physical or manipulative, some suggestive, some an eclectic combination of these,, as well as a “do-nothing” school, the latter prescribing nothing but strict mental, physical, and physiological rest. It is interesting to recall that Hippocrates, the Father of Medicine, said: “The more you feed a sick person the more you harm him. Your food should be your remedies and your remedies your food.” He placed a strong reliance on the recuperative powers of the body to eliminate or overcome disorders, even of the serious kind, if aided by the proper regimen and improved environment; with this was coupled an equally strong disinclination to interfere with the normal functions of the organism by the administration of drugs. He said: “Give me fever and I can cure every disease.” To prevent or suppress the acute fevers of childhood and later to produce artificial (“therapeutic”) fever to kill the germs of chronic disease is thinking backward.

Hippocrates employed some rational practices which many of his professional descendants have seen fit to ridicule. There are several rational schools of healing founded on the good common-sense methods of Hippocrates; serum therapy is not one of them. In spite of legislative obstacles and the opposition of press, pulpit, politics, and philanthropy, all of these systems are in demand and their practitioners supremely confident. The outstanding success of every one of these rational systems warrants investigation by competent observers. “There is a principle which is a bar against all information, which is proof against all argument, and which cannot fail to keep a man in everlasting ignorance! That principle is condemnation before investigation,” declared Herbert Spencer. Investigate these methods!

It should be remembered that disease, being a process, must depend upon certain conditions for its initiation, continuance, acceleration, decline, or cessation. Bacteria, since they are living organisms, must depend upon the condition of their environment for their metabolic activities. Infectious disease must terminate when

the pathogenic material in the organism has been “converted” into simpler compounds and eliminated. Thus infections are self-limited when managed in a rational way. Osier says the spontaneous healing of tuberculosis is an every-day affair. Almost every person, according to autopsy, has had tuberculosis, but few have ever been aware of it. Cooperation with this tendency to spontaneous recovery is the keynote of all rational, non-medical systems.

The attempt to define non-medical therapy and differentiate it from medicine is one which may lead into much controversy unless the fundamental differences which characterize the two practices are made clear. The two schools approach the problem of disease in radically different ways. The medical school has always considered disease (particularly in the infections) as an attack; has always looked upon the body as having defenders or a defense, and as resisting attack. Practitioners of the medical school behave as if disease were an entity; they try to fight it, to combat it; they declare war on germs and other manifestations of disease. The non-medical or drugless school, on the other hand, looks upon disease as a manifestation of deranged physiology or anatomy, rather than as an attack or invasion; as something that must be understood from that point of view and treated accordingly. If it be held that the medical school considers disease from the same standpoint, the fact is that their therapy is of an antagonistic nature; the dominant note of their work is to cure by contraries (or by similarities—a form of cajolery).

The drugless school holds that the body is able to rise to the occasion of its own accord, when spared any such obtrusive treatment. This school demands that the body shall bring about its own cure: “Only nature cures.” It does not fight or combat disease. It does not employ force, coercion, compulsion, or heroic measures. It does not suppress symptoms or functions. Rather, it cooperates with the various organic activities of the body and aids and assists in those functions which are conducive to the more rapid development and ultimate termination of the disease processes. It looks upon bacterial manifestations not as an attack, but as the activities of microorganisms which will cease as soon as the material upon which they feed is eliminated, or the parts involved properly innervated.

Knowledge of this distinction between the two schools is tantamount to the understanding of their work. Once this concept is clear, the reasons for all details readily suggest themselves. The drug-less school regards the body as a sanative and autonomous entity, and cooperates with its inherent healing powers to re-establish health. It considers disease as endogenous. It does not substitute one disease for another. It does not suppress symptoms and restrain functions. It very definitely draws a distinction between substitution and cure; between suppression and elimination; between combat and cooperation; between what is natural (or spontaneous) and what is artificial; between palliation and correction. Its practitioners do not look upon the body as a battlefield but as a “colony” of living units, normally so organized and integrated that it is sufficient unto itself for all acute emergencies. Chronic disease results only from mismanaged acute disease.

The non-medical practitioner makes no attempt to supply artificial antibodies, synthetic hormones, artificial vitamins, and artificial enzymes to the body, but requires rather that the body fulfill all these functions itself—as it did before symptoms developed, or as it does in other subjects, presumably healthy. (Obviously, he makes no attempt to include in the scope of this practice congenital or inherited defects, or hopeless traumatic lesions; he deals with disease which was preceded by health.)

So we see that the theories, doctrines, and philosophies underlying the practices of the two schools are genuinely different. There is no subterfuge or subtlety in the distinction; it is not a distinction without a difference.

We reiterate, then, that infectious disease is not an attack; it is an expression of bacterial activity. The body does not “defend” itself. It reacts to stimuli—physical and chemical. “Attack” is an animistic obscurity; “defense,” a teleological sophism. H. Gideon Wells, in *Chemistry in Medicine* (p. 564), speaks of the “soluble poisons of the bacteria which are their chemical weapons of attack.” But, on page 551 of the same volume, E. O. Jordan says: “In the majority of the best studied diseases, however, the toxic substances appear to be mere incidental products of the attempt to satisfy hunger, and are not weapons of warfare.” Dr. Jordan’s explanation is the more rational one. The organism is not a battlefield, and such animistic analogies are productive of serious errors in reasoning.

When the organism is unfavorably conditioned for bacterial activity, such activity is impossible—a truism which must be emphasized. Granting that both schools of therapy can so condition the organism as to influence the initiation, progress, or termination of this activity, it then becomes a matter of personal choice. Shall the therapy be combative or cooperative?

That the laboratory leads us astray is attested to by Jousset: “If, in effect, one considers that the bacilli endowed with pathogenic properties as incontestable and as special as Eberth’s bacillus, the comma bacillus, the bacillus coli, the pneumococcus, streptococcus, Koch’s bacillus and others still, may exist indefinitely, and exist in effect in the latent or saprophytic state in the organism, one reaches the conclusion that *in the clinic* the pathogenic microbe has not the power within itself sufficient to produce disease.

“We say in the clinic, because in the laboratory the features occur differently; the virulence of the cultures, the relatively considerable quantity of the cultures injected, the choice of place for the injection, all the circumstances which we repeat are not met with in the clinic, give to the pathogenic microbe an artificial power which permits it to develop the disease every time it is not found in the presence of an absolute immunity.”

F. M. Alexander reaches similar conclusions from quite a different route:

“Are we to be inoculated against every known disease till our bodies become depressed and enervated sterilities, incapable of action on their own account? I pray not, for such a physical condition would imply a mental condition even more pitiable. The science of bacteriology has its uses, but they are the uses of research rather than of application. Bacteriology reveals a few of the agents active in disease, but it says nothing about the conditions which permit these agents to become active. Therefore, I look to that wonderful instrument, the human body, for the true solution of our difficulty, an instrument so inimitably adaptable, so full of marvelous potentialities of resistance and recuperation, that it is able, when properly used, to overcome all the forces of disease which may be arrayed against it.” (*Man’s Supreme Inheritance*, p. ix)

*Summary:* In this chapter we have shown the practice of artificial immunization to be invalidated by:

1. Unsound theory, even in the case of diphtheria
2. An etiology applicable only under artificial conditions

3. The non-specific character of bacteria and serums
4. The discovery that a filterable virus may have an intracellular origin
5. The superiority of sanitation over serums
6. The admission that immunology is no more than a cult
7. Such factors as hypersensitiveness, adaptation, and a conditioned organism, which may outweigh the germ in the infectious process
8. The fact that virulence may appear spontaneously
9. The existence of carriers and the omnipresence of bacteria
10. The dependence of contagion on filth
11. The sciolistic character of the practice
12. The possibility of substituting one disease for another
13. The proof that serum therapy is often dangerous and fails to accomplish its purpose
14. The inability to satisfy Koch's postulates
15. The discovery that infection need not be accompanied by symptoms
16. Allergy and anaphylaxis as concomitants
17. Laboratory risks
18. The condemnation of one of America's foremost bacteriologists
19. The existence of better methods

## 67 BECHAMP'S THEORY OF THE MICROZYMA AND HOW IT WOULD CLARIFY THE PRESENT CONFUSION

To delete from history's pages the record of Béchamp's work is on a par with the crime of those physicians who prepared the hemlock cup for Socrates.

Some of the phenomena presented in the preceding chapters have not been adequately accounted for. Certain present-day bacteriological problems are possible of solution if approached from a slightly different angle. Up to this point we have dealt with the interrelations of bacteriology and pathology. The inquiry will now digress into a very brief outline of normal cytology in order to point out certain biological facts which, when properly evaluated, will revolutionize our interpretation of the place of bacteria in nature.

The cell is nourished, drained, and innervated as shown in the accompanying figure. Arterial blood enters the capillary network from the left. There is an exchange, of an osmotic or diffusive nature, as oxygen and nutriment pass through the wall of the vessel, mix with lymph in the intercellular spaces, and pass through the cell wall according to the needs of the cell, while the cell at the same time eliminates waste and carbon dioxide which the venule and the lymph capillary carry away. The calibre of the vessels is controlled by the nerve fibers which accompany them.

Avoiding the artifacts and debris which have been misleading investigators for seventy-five years, Antoine Béchamp biologist and pioneer bacteriologist, employed vital methods which yielded biological discoveries of profound importance.

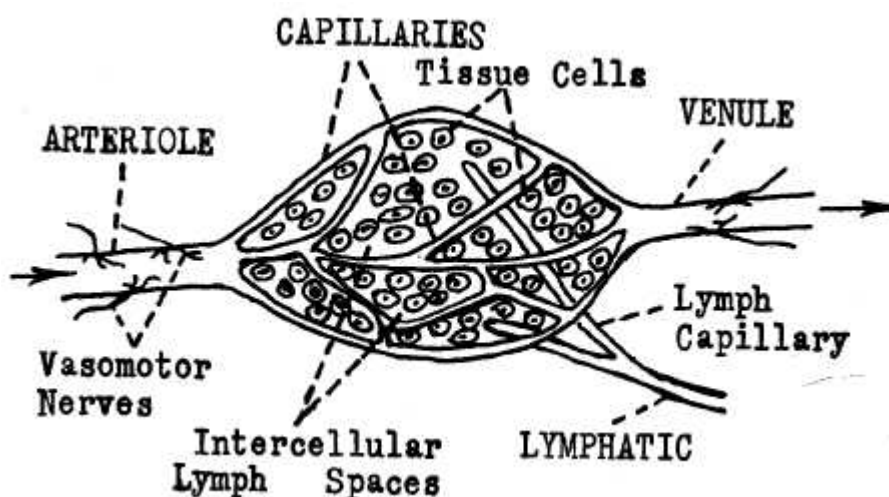


FIG. 26 Diagram showing innervation, blood supply, and drainage of tissue

Within the cell are the nucleus and the cytoplasm, both of which consist of a stroma of many living criss-cross fibers which are extensions of living microzymas and which have an arrangement similar to a three-dimensional spider web. There is tissue fluid in the interspaces, and within the fluid there are many small granular units of living entities. These intracellular primal bionts produce the chemical changes in each cell. The latter have been called by Wilson in *The Cell in Development and Heredity* chromidia or chromatin granules. When cell division

occurs, some of the granules in the nucleus form a spireme and then divide into chromosomes. It has been, and still is, held by some authorities that these granules are autonomous living entities, capable of reproduction. Whatever the terminology, we refer to subcellular units, capable of autonomous existence and reproduction.

When circulation is defective or drainage impeded, or the cell enervated, these intracellular units deteriorate proportionately, the specific type of change being governed by the particular type of tissue, the temperature, the nature of the factor or agent initiating the process, the condition of the organism prior to the inception of the change, and, doubtless, other factors as yet undetermined. Thus an enervated, improperly nourished, poorly drained cell will change with its environment. The chromidia are now without nourishment and oxygen, and are obliged to live in cellular waste products. The cell, as an entity, dies, breaks down, or degenerates, while the living units in the cell undergo metamorphosis into whatever forms the prevailing conditions may induce. Catabolic changes ensue, giving rise to increased heat—hence, inflammation. The period of heat production in the case described would be conducive to a spontaneous devolution of chromidia into more primitive forms. Spontaneous is used to mean arising from internal causes without external excitation. It cannot mean “without cause” because internal causes are known.

This conception has nothing in common with the doctrine of spontaneous generation. We merely call attention to the likelihood of spontaneous evolution, devolution, mutation, transmutation, variation, metamorphosis (or whatever term may be appropriate), of complex cell organisms into simpler forms in keeping with their environment. That these forms may be pathogenic microorganisms is supported by the statement that “Ryan and Arnold of the University of Illinois were able to report the experimental evolution of certain granules in apparently sterile body fluids which passed through two or three intermediary stages to their final ‘transformation’ into conventional bacteria or yeasts.” (*Journal of the American Medical Association*, August 27, 1932) These stages may be beyond the range of microscopic visibility.

Jennings, in *The Biological Basis of Human Nature* (p. 270), says that “a part of the chromosomes *are seen to fade away* in the egg; others remain and influence the offspring.” Fading and re-appearance of microorganisms and ultra-microorganisms are matters of common knowledge, although the implications of such phenomena have rarely been considered.

Consider what light all this sheds upon the spontaneous appearance and spontaneous termination of many infectious diseases, animal cells producing vegetable enzymes, the problem of bacterial species, why animal experimentation is likely to lead to erroneous conclusions, why the virus survives (or “reproduces”) in living tissue only, why bacteria seem to have no nucleus (because they are all nucleus, according to Crile and others, or because the nucleus and the cytoplasm are intermingled), the mystery of the origin of bacteria, the omnipresence and universality of pathogenic bacteria, the seeming inconsistency of bacterial symbiosis and predacity, carriers, immunity, latent or active virulence, filterability, fluctuations from large size to submicroscopic size in response to environmental conditions, why “invaders” do not need to “migrate” (because they have the ability to initiate remote metamorphosis into forms identical, with their own form and “function”), parasitology, the bacteriophage phenomenon, and many problems of cytology and genetics. All such problems are simplified by an understanding of the life cycle of these primal bionts. Degenerated bacterial forms in ageing cultures, versatility,

involution, and anaphylaxis all add to the confusion which is bacteriology, and all impeach the major premise of infection, condemning as sciolistic the practice founded upon it.

Just as the theory of phlogiston impeded scientific progress for a century, so the blighting dogma of Pasteur that “A germ is a disease and a disease is a germ” has been a millstone about the neck of humanity, for the issue is of far greater import than phlogiston. Irrational practices have been founded on the one, but not on the other—and statutes made to enforce the acceptance of such practices. Each new discovery and development is rationalized to fit into the theory, all of which would be unnecessary if the phenomenon briefly outlined in this chapter were clearly understood. And there is nothing difficult or involved in the matter.

In the article, “The Bacteria,” in *The Nature of the World and of Man* (p. 203), E. O. Jordan says: “It is possible that bacteria—or some of them—may be the degenerated descendants of higher forms of life, but this does not appear so probable as that bacteria represent a very primitive type of living thing. How then did bacteria originate?” Jordan’s concern with this “first cause” problem obscures his vision. It is possible to be so close to the truth that it may be overlooked. Why speculate on the origin of bacteria? Why not consider the possibility of metamorphosis?

Wilson, on page 719, of *The Cell in Development and Heredity*, in summing up various cell theories, says: “None of these has been found adequate, and in point of accuracy, simplicity and fruitfulness of method the formulas employed in modern genetic analysis based on the particulate hypothesis almost rank with the atomic and molecular formulas of the physicist and chemist.” But, whether a matter is simple or complex is not very important. The validity of the premises and the rationality of the practice based thereon are what matter.

Jennings, in *The Biological Basis of Human Nature*, in dealing with chromosomes and genes, on page 271, speaks of chromosomes as “surviving” under certain circumstances which usually result in their “death.” On page 273, he speaks of “unmated” chromosomes and talks of them as “dying,” as “living” and “uniting.” On page 274, he refers to some of them as “incompatible”; he speaks of their “work”; he says they “destroy” and “poison” others. On page 291, he says that genes are immortal; on page 270, genes “resist”; on page 316, “the altered genes continue to *live and multiply*.” On page 330, he cites the difficulty of producing differences without “killing” genes; also “genes must be so ‘altered’ that, as they *grow and divide . . .*” Although Jennings does not credit these units with being autonomous entities, his language clearly implies such a hypothesis.

Professor Béchamp, of the University of Montpellier, a contemporary of Pasteur, observed and described in detail the life-cycle of these intracellular units and called them microzymas. His work has never been seriously questioned; refutation has never been attempted. Pasteur, according to Vallery-Radot (*Life of Pasteur*, Vol. II, p. 25), referred to the work of Béchamp and other distinguished contemporaries in the following words:

“They are far behind us now; they are now relegated to the rank of chimeras, those theories of fermentation imagined by Berzelius, Mitscherlich, and Liebig, and re-edited with an accompaniment of new hypotheses by Messrs. Pouchet, Fremy, Trecul, and Béchamp. Who would now dare to affirm that fermentations are contact phenomena, phenomena of motion, communicated by an altering albuminoid matter, or phenomena produced by semi-organized materia, transforming themselves into



this or into that? All those creations of fancy fall to pieces before this simple and decisive experiment.”

In view of the fact that Béchamp did not affirm any of the “chimeras” enumerated by Pasteur, there is but one of two possibilities: Either Pasteur misunderstood Béchamp’s theory or he deliberately misrepresented Béchamp. There is no other possibility. And, of all things, for Pasteur to claim or even insinuate that he had disproved spontaneous generation is absurd. His own reports to the French Academy conclusively prove him a champion of abiogenesis, changing his position only when he finally grasped the import of Béchamp’s discoveries. On pp. 413 and 415, *Annales de Chimie et de Pharmacie*, 3e serie, 52, he states that lactic ferment or lactic yeast “takes birth spontaneously.” He later added a footnote, but the subterfuge does not save his face, except for his biographer and other credulous persons. Credit is due Béchamp for this work, as documentary reports conclusively prove. How an academy of science, of all institutions, could be taken in by such unfair tactics and made to believe otherwise is amazing. His lack of understanding of the phenomenon is exposed by his remark that no change occurred in his experiment except the development of an odor. His friends missed that remark. Furthermore, the contemptuous manner in which Pasteur dismisses reputable scientists reveals his intolerant nature. Quite in contrast to the attitude of Pasteur and his biographer (the French Boswell, Vallery-Radot) is the appreciative obituary notice of Béchamp, in *Nature* (May 7, 1908, p. 13):

“It is mainly in connection with the early history of what is called coal-tar chemistry, and more especially in connection with the fields of investigation with which the name of Pasteur is preeminently associated, that Béchamp’s services will be recalled. The method of manufacturing aniline ultimately made use of by Perkin in England, and by the brothers Renard in France, was due to Béchamp. . . .

“But it was to the domain of biological chemistry that Béchamp’s energies were principally directed, and he took an active part in the inquiries and ‘controversies which ultimately led to the triumph of Pasteur and his immediate followers. Although much of Béchamp’s work on fermentation, on the production of moulds, on the silkworm disease, and on zymases, ran parallel with Pasteur’s inquiries, his interpretation of the phenomena was generally opposed to that of Pasteur, and the two investigators were frequently in acute controversy on these subjects [which always ended with Pasteur purloining Béchamp’s discovery and getting credit therefor—in spite of the fact that the papers are on record at the Academy, *and dated*—J.R.V.]. Béchamp’s fame has probably suffered in consequence. We must, however, do him the justice to admit that his main contention, that unorganized ferments play a larger and more important part in the phenomena of metabolism than the immediate followers of Pasteur were willing to concede, is intrinsically sound. Béchamp developed his view into a general theory. . . . Whatever may be the ultimate fate of his theoretical conceptions, his experimental work on blood, fibrin, milk, proteins, and his position in regard to the great and fruitful controversies of half a century ago, out of which modern bacteriological doctrine has sprung, will ensure him an honourable place among the founders of biological chemistry. . . . He occupied himself to the end in searching for support for the comprehensive generalization of organic change on which his fame will ultimately rest.”

“Béchamp’s fame has probably suffered” as a consequence of his acute controversy with Pasteur. And the scientific world remains mute while injustice is visited upon a noble character. Darwin personally shared his honors with Wallace;

the theories of Ptolemy, Copernicus, Lamarek, Laplace, Spencer, and Aristotle are included in all scientific curricula. But Béchamp, who, according to Loevenhart and Stratman-Thomas (*Chemistry in Medicine*, p. 673), preceded Ehrlich in the development of arsenicals, is almost never mentioned. It is time that overdue credit was given to this nineteenth century investigator. The results of his labors are on record at the French Academy of Science and in the library of the British Museum. There is an English translation of one of his works, *The Blood*, at the New York Public Library and at the Library of Congress in Washington. It was Béchamp who established the fact that the blood is a tissue. He anticipated present-day geneticists. It might be said of him that he was to biology what Lavoisier was to chemistry.

Béchamp discovered that “the ‘molecular granulations’ of the cells assist in fermentation, that they are autonomous entities, the living principle, vegetable and animal, the originators of bodily processes, the factors of pathological conditions, the agents of decomposition, while, incidentally, he believed them to be capable of evolving into bacteria.” (Béchamp or Pasteur? by E. Douglas Hume, p. 8)

We quote the editorial entitled “*Béchamp or Pasteur?*” from *The Chemical Trade Journal and Chemical Engineer*, London, England, March, 1928, so that you will know the opinion of a reputable and qualified editor in this matter:

“Readers of this journal will probably remember that just over four years ago we drew attention to a book of Miss E. Douglas Hume in which the relative claims of Pasteur and Antoine Béchamp as pioneers in biochemical investigation were discussed, much to the discredit of the former. The facts therein detailed, which have never been questioned, are too precise to admit of more than one opinion, and that is Louis Pasteur was no more than a plagiarist and one who did not even understand what he plagiarized. We are impelled to draw attention again to this subject as a result of hearing a talk on the wireless this week on Pasteur in which he was given credit amongst other things, for being the first to explain the mystery of fermentation. We think that anyone setting out to instruct the public on such matters should be aware that the honour of this discovery was Béchamp’s and his alone.”

This little digression into biography may not be strictly apropos of the matter in hand, but it is necessary for someone to call a halt when scientists resort to the methods of the Inquisition. Béchamp’s name has been kept out of the *Encyclopedia Britannica*; his works have been excluded from the *New York Academy of Medicine*. Is there an Index Expurgatorius in science? The findings of Béchamp were perfectly rational and scientific, and there is no reason why they should not be made known, at least to students of science. A knowledge of Béchamp’s work would have made unnecessary much recent duplication.

According to Dr. Boycott, of the University of London, in the *London Lancet* (Vol. 207, p. 998), Béchamp is compared with Rutherford by d’Herelle. What Rutherford has done in the field of atomic structure, Béchamp had done for the cell.

On the matter of d’Herelle’s discoveries, an item in *The New York Times* of December 28, 1931, is interesting:

“Disease germs, like dogs, have tiny fleas whose bites sometimes turn the bacteria into raging destroyers and sometimes drug them into harmlessness.

“Discovery of certain profound effects of these bacterial fleas was reported to the Society of American Bacteriologists today by Dr. F. d’Herelle of the School of Medicine of Yale University.

“The bacterial pests do not merely drive disease germs berserk, but somehow alter heredity so that a different species of bacteria develops. The new species may be either more dangerous or less so than were its parents.

“Dr. d’Herelle’s discovery suggests the possibility of controlling bacteria by breeding, provided it can be ascertained what kind of flea bites will rob disease germs of their virulence.

“Dr. d’Herelle’s phage fleas have already been used with good results in checking isolated cholera outbreaks in India. . . .

“Disease bacteria, it was shown, have the power to change their forms, to break up into tiny bits, which are all but invisible even in microscopes. In this dust-fine scattering they retain life and an as yet little understood ability to ‘come back, that is, to grow again.

“These ‘come-backs’ explain the source of some epidemics, and possibly also why epidemics sometimes rather suddenly die down, according to Dr. C. E. A. Winslow of the department of public health of Yale University.”

Of course, Dr. d’Herelle would disclaim responsibility for this romancing, but it must be pointed out that he is the one who coined the term “bacteriophage”—very evidently a misnomer. Further, he is responsible for the conception that health, like disease, can be “caught.” In all his discoveries he was anticipated by Béchamp, who gave rational and consistent explanations of the phenomena.

At first glance, Béchamp’s theory clashes with three important doctrines which are presented here with their refutations:

*Doctrine:* The cell is the smallest unit of living matter.

*Refutation:* A bundle of genes carries life and characteristics from the male. This bundle of genes is not a cell and it is a collection of living units—some of which can be killed and their characteristics lost. Stating that they are dependent upon their environment, that they are inseparable, and that they cannot exist as units and, therefore, cannot be regarded as entities is merely evading the issue, for neither can the cell of multicellular organisms exist as an independent unit outside its environment. Further, it is being taught that certain colloids are living. The cell theory must be revised to fit the facts.

In *Medical Record* (January 16, 1935, p. 71), Ferdinand Herb, discussing the chemical changes in cells, writes:

“These chemical changes, whether anabolic or catabolic, are not performed by the cell as such, but by some constituent part of it that is not at all dependent upon the integrity of the cell. It has been, and still is, the general belief that the protoplasm itself is the seat of this activity. This is not the case. Experiments performed by Büchner of Germany, prove this to the fullest satisfaction. He broke up yeast cells by trituration and found that the pulp, containing the protoplasm in a thoroughly disintegrated state, functioned chemically just as well as the cell itself, as the formation of alcohol and carbonic acid went on unimpeded. More than that. The chemical processes going on in the pulp were influenced in their activity by physical agents in the same way as in the integral cell: they were increased by light and decreased by darkness.

“These plain and simple deductions based upon Büchner’s experiments, namely, that the functioning of this unicellular organism is not dependent upon the integrity of the cell, or of its protoplasm, but upon bodies that are contained within and perform their work in an absolutely independent way—cannot be denied.”

2. *Doctrine:* Transmutation of species is impossible.

*Refutation:* This need not be called a transmutation; there can be a compromise on metamorphosis. The phenomenon must not be denied to satisfy an a priori theory.

3. *Doctrine:* Infectious disease cannot originate spontaneously.

*Refutation:* Adequately presented in this text.

In an article on page 146 of the *Scientific Monthly* (August, 1933), entitled "The Use of the Experimental Method in the Study of Human Parasitic Infections," Prof. Ernest Carroll Faust, of the Tulane University School of Medicine, mentions "problems arising from the apparently spontaneous development of newly recognized diseases, such as Brill's disease, and endemic typhus, and Rocky Mountain spotted fever in the Eastern United States."

What Morgan did with the fruit fly, drosophila, can be done much more readily with bacteria. This is one explanation for the appearance of new diseases. What can be done in conditioning genes to cause mutations in organisms like drosophila as described by Morgan and in bacteria as described by Jordan, can be done in transmuting intracellular bionts into bacteria. Morgan, Jordan, and Béchamp are all dealing with the same fundamental phenomenon. Morgan's work in genetics lies outside the scope of this text, but let us consider some of Jordan's observations (*General Bacteriology*, p. 65, et seq.), as follows: [Emphasis by J.R.V.]

*"Involution and Degeneration Forms.* Under constant and favorable conditions of life each kind of bacterium generally exhibits a true constancy of form. Long-continued growth in artificial culture-media, however, appears to have an injurious effect upon certain varieties of bacteria. In old cultures or in cultures kept under relatively unsuitable conditions many bacteria pass into unusual forms which are plainly the result of degeneration, and indicate that the cell has received some damage from untoward physical and chemical influences. These degenerative or involution forms often depart very widely from the typical form, and sometimes give to a pure culture the appearance of being contaminated by a foreign organism. Certain bacteria are especially prone to produce involution forms, and in at least one case, that of the plague bacillus, the occurrence of involution forms upon a particular culture-medium (nutrient agar, containing 2.5 to 3.5 per cent NaCl) has been thought to be characteristic and to serve as a valuable aid to the differential diagnosis of the organism.

"Monstrosities and abnormalities are sometimes observed in bacteria contained in animal tissues or fluids (plague bacillus, cholera spirillum), and although these may bear little resemblance to the typical form of the species, such involution forms are known to be alive and capable of development. The branching filaments that are occasionally observed in cultures of the tubercle bacillus, diphtheria bacillus, and other forms are believed by many to be degenerative rather than truly developmental. Loeb has shown the influence of osmosis upon the production of branching in the typhoid and tubercle bacilli. Probably in some cases the so-called involution forms are degenerative, in others simply teratological."

On page 128, he adds: "There is now no doubt that true mutations occur among bacteria as among the higher forms of life. Barber, for example, has described an instance in a strain of the dysentery bacillus in which the variation occurred suddenly and fully formed; it appeared in a relatively small number of individuals, was not adaptive, and the new characteristics were transmissible to offspring through many generations. A similar instance has been observed in *B. coli*, in which cells with the power of fermenting saccharose and raffinose suddenly appeared in a culture (from a

single cell) that had not previously possessed this power. The parent strains and the mutating strains have each maintained their separate characteristics for over four years and during some hundreds of test-tube transfers.”

There is much other experimental evidence to support Béchamp’s theory. It is not a great jump from these bacterial mutations to a mutation of virus into bacteria, evidence of which is supplied by Drs. Dochez, Kneeland, and Mills when they say that swine influenza requires more than one etiological agent to produce the typical disease. “It has been demonstrated that in swine influenza there exists a filterable virus which by itself produces only the mildest of diseases; if administered together with a culture of certain bacterium it gives rise to typical, highly contagious swine influenza.”

Dr. R. L. Kahn, of the Michigan Department of Health, has reported finding that the tubercle bacillus reproduces itself by dividing into several small oval pieces. *These turn into granules*, which grow into small rod-like objects. These rods then develop to the size and shape of the parent bacillus, and are capable of producing tuberculosis in guinea pigs.

When the bacillus is in its *granule and rod stages*, it apparently loses its protective coating of wax, or its “acid-fastness” (a property so-called because the wax gives the bacillus the power to resist acids).

Kahn’s findings also seem to disprove the belief that the virulent and mild types of tuberculosis are caused by two different types of bacillus. He found that the germ capable of producing the virulent type may have offspring of the mild variety and vice versa. The solution of this difficulty lies in the work of Béchamp. This is a case of mutation.

The most comprehensive and rational solution of present-day bacteriological problems is given by Béchamp, and the sooner his interpretation of bacterial metabolic activities is understood in its entirety the better for all concerned.

There is a very simple experiment which would quickly and definitely settle the validity of Béchamp’s theory. According to Béchamp, the cell undergoes devolution and the units which he claimed composed that cell evolve into other forms—which we generally term bacterial. All that would be necessary to confirm this mutation would be to allow some sterile tissues to degenerate in a sterile medium. This is a very simple matter. The chicken heart fibers of Alexis Carrel are guaranteed to be germ-free or sterile. This tissue is kept in a sterile medium bathed and nourished under very precise conditions. At frequent intervals the growing specimen is trimmed to keep its bulk within reasonable size. It would be a very simple matter to place some of these trimmings under the same conditions, except to deprive them of new nourishment, and allow the specimen to take its course without further interference. If the theory of Béchamp is correct, the tissue would degenerate in its own waste products. The cell excretions and lack of new nourishment would initiate a devolution of the units composing the cell into bacterial or similar mutations. This is a simple experiment which would settle the question definitely.

Of course, it would be pointed out that autolysis accounts for this. But that is exactly the issue: The mechanism of autolysis was described in detail by Béchamp. Examine the record! Since his time, the word *autolysis* has been substituted for the phenomenon. He said that all things are the prey of life—and autolysis is a biological phenomenon—not simply lysis.

That the virus possesses the ability to initiate the same disease with which it had been associated—even after the virus has been crystallized—indicates how close the connection is between living and nonliving agents.

Béchamp proved that the primal biont carried with it (for a while) the ability to initiate, in a new environment, the same type of infectious process with which it had been recently associated. This is what happens in vaccination, or smallpox, or rabies, or in any similar experience where the agent causes the disease: under certain conditions, the pathogenic agent initiates a type of metamorphosis among intracellular bionts which closely resembles its own recent experiences.

As stated on a previous page, some agent in the environment damages the cell. The “cell” may mean the nerve system. The nerve system now may take on “maleficent” characteristics and inflict damage and “supervise” destructive processes. Under the aegis of the nerve system, a metamorphosis of intracellular bionts may proceed. This coordinates the discoveries of Béchamp and Speransky. That the nerve impulse is competent to do this is not specifically stated by Speransky, but he has shown very clearly that the nerve impulse carries a quality of excitation which is competent to institute an intracellular metamorphosis which is identical (or sufficiently similar) to the specific agent which is responsible for the irritation of the nerve. This clears up such mysteries as the intracellular behavior of the virus.

Dr. R. R. Mellon, of the Western Pennsylvania Hospital Institute of Pathology, described seven stages of the tuberculosis germ’s life-cycle. “The life-cycle of the tubercle bacillus consists of four stabilized stages which are vegetative and three transition stages between them which are reproductive. Of these reproductive stages two are probably asexual, while evidence for the possible sexual nature of the third was obtained.” (*The New York Times*)

Howard B. Cross, of the Department of Bacteriology, Johns Hopkins University, in the *Johns Hopkins Hospital Bulletin* (July, 1921), asserts: “One of the disturbing surprises at autopsy is the occasional presence of leucocytes containing bacteria in transudates that have repeatedly been reported as sterile. . . . In many of the recorded cases, the clinical examinations and cultures have been made so recently before death that the remaining time is altogether inadequate to account for the extensive phagocytosis observed.” Familiarity with Béchamp’s findings and discoveries would eliminate the “disturbing surprises at autopsy”—possibly make autopsy unnecessary in that particular case at that particular time. Besides an inquiry into the possibility of spontaneous bacterial evolution, this report calls for a thorough investigation of the premises of the theory of phagocytosis.

According to Dr. Alice C. Evans, research worker of the Public Health Service’s National Institute of Health, the same germ may take on three different forms. “It may appear as a streptococcus or chain of cells such, as causes pneumonia; a rod-like, sausage-shaped object like the diphtheria bacillus, or it may be a filterable virus like that which causes infantile paralysis, in which case it is invisible. The form that it takes depends on conditions under which it is grown, the kind of nourishment available and other unknown influences.

“The discovery suggests,” said a statement from the service, “that the development of disease is influenced not only by germs but by the kind of environment in which the germs develop. It raises the question in the belief of some scientists, whether the same germ may cause two different diseases, or two different forms of the same disease, depending on the part of the body in which it develops.

“Dr. Evans’ experiments confirm work of other scientists who already had discovered that germs apparently have the power to change from one form to another.

“She worked with a streptococcus, or chain-like germ taken from the brain of a man who died of encephalitis, a form of sleeping sickness. It was not known whether the germ was the cause of the disease. When grown on different kinds of media, or germ-food, the streptococcus changed from its chain shape to the sausage shape and then to the form of a filterable virus.

“The next step will be to determine whether the different forms of the germ cause different diseases or perhaps different forms of the same disease. If that is the case, the whole science of bacteriology, which deals with germs, may have to be revised.”

“The whole science of bacteriology” is undergoing revision at the present moment; it is in the process of a revolution. And this has been made necessary because Louis Pasteur led the world astray, and succeeded in alienating the scientific world from Béchamp—the investigator who did all the experiments of the present-day workers, and who formulated rational explanations for the mysteries which are baffling his blundering professional descendants.

Béchamp’s theory places “lack of resistance,” “predisposition,” “idiosyncrasy” in a different light. It explains why it is possible to inoculate 99 paresis patients with malaria and have only 88 take malaria. It explains the problem presented by W. D. Hubbard, of Ibamba Ranch, Namwala, Northern Rhodesia, concerning the disease-resistant qualities of wild animals. He says:

“Wild creatures exhibit apparent immunity to all the tick-borne stock diseases, to anthrax, contagious abortion and the deadly nagana . . .

“In India, the Far East and the Philippines a disease known as surra attacks domestic animals and is lethal to many. In South America there is the disease called mal de caderas. Both of these are closely allied to the widespread disease found throughout Africa called nagana, which is carried by the tsetse fly.

“Ticks carry East Coast and Texas fever, anaplasmosis, piroplasmosis and spirochaetosis. These pests are found universally throughout tropical countries and in the Southern United States.

“To each and all, wild animals are, so far as a close study can reveal, immune. It is our hope that by study and experimentation we may devise means or methods by which this immunity may be transferred or artificially developed in domestic animals.”

Domestication changes the tissues of animals. The cell undergoes a devolution, and cell units undergo metamorphosis into bacteria. This microbiosis may be initiated by an agent external to the subject, but need not be; generally, it is spontaneous—the cause being inherent in the subject. Domestic animals are susceptible because they have been so conditioned. Environmental changes precipitate organic adjustment. Is this a question of breeding or feeding? Is it a problem of genetics or one of nutrition? Why not study the vitamins of domestic stock before considering the genes of wild cattle? A comprehension of Béchamp’s theory makes this all quite clear. Béchamp was never deceived by a “first-cause” hoax, nor by belief in single, isolated causes. That was, and is, an error of the Pasteurian school. Béchamp’s theory postulated a multiple cause of infection.

At the thirteenth triennial session of the International Medical Congress, held in Paris in 1903, Dr. Rudolph Virchow said: “Microbes are always found where there

is disease. They are also found where there is no appreciable disease, and may be the result and not the cause of disease.”

That the scientific world is gradually approaching a change in its explanation of the phenomenon of infection may be inferred from the nature of much of the recent literature. In a very notable article on “Homeopathy and Modern Medicine” in *Revue Générale des Sciences Pures et Appliquées* (May 31, 1933), Dr. Leon Vannier says:

“The microbe is an apparent, but not a real, cause. When the curtains which still obscure the understanding of our biologists shall have fallen, microbes will be considered as symptomatic microscopic elements of variable form according to the soil in which they evolve. Then numerous filterable viruses will be described and behind these viruses, still insufficient to explain the true cause of diseases, will soon appear mysterious emanations with formidable properties as yet unsuspected. Thus therapeutics will remain always uncertain and insufficient if medicine persists in following the Pasteurian path which leads inevitably to an impasse.”

“The curtains which still obscure the understanding of our biologists” should never have been woven. Béchamp described, a generation ago, the behavior of microorganisms and ultra-microorganisms, and their connection with infection. In *The Blood* (p. 376), he says: “We now know that the microzymas are functionally different in the various anatomical systems of the same animal, and that they may be functionally different also in the same organs of the same structure in man and animals. *It thence results that it is not always permissible in experimenting to draw conclusions from one animal to another and least of all to man.* So that if we could admit with Bichat that life is a property of tissue, this property is not the same in all the tissues of the same structure and in their microzymas.”

He criticized the practice of inoculation as follows: “All is danger in this kind of experimentation, for the reason that it is not anything inert that is acted upon but that there is a modification more or less injurious, of the microzymas of the inoculated.”

He criticized the theory of air-borne infection as follows: “If virulent germs were normal to the atmosphere, how numerous would be the occasions for their penetration independently of those by the way of the lungs and intestinal mucous! There would not be a wound, however slight, the prick even of a pin, that would not be the occasion for inoculating us with smallpox, typhus, syphilis, gonorrhoea.”

A brief but thorough summary of Béchamp’s work is given by E. Douglas Hume on page 176 of *Béchamp or Pasteur?*: “In place of the modern system of treating that phantom shape, a disease-entity, and trying to quell it by every form of injection, scientific procedure on Béchamp’s lines will be to treat the patient, studying his personal idiosyncrasies. For these depend upon his anatomical elements, the microzymas, which, according to Béchamp, build up his bodily frame, preserve it in health, disrupt it in disease, and finally when the corporate association is ended by death, these, with or without extraneous help, demolish their former habitat, themselves being set free to continue an independent existence in the earth, the air, or the water in which they happen to find themselves. Any morbidity, which may be in them or in their evolutionary bacterial forms, is quickly dispelled by fresh air. And since the microzymas of different animals, different plants and different organs, lungs, kidneys, colon, as the case may be, are themselves all different, so will there be variation in their bacterial development, and so the innumerable forms of bacteria perceived everywhere are readily accounted for. As the British Empire, or the United States of America, or the Republic of France, are composed of innumerable



varying individuals, so the corporate body of plant or animal is an association of living entities; and as the work of myriad individuals composes the life-processes of the nation, so the action of the microzymas constitutes the life-processes of all corporate beings.

“What might not the new outlook on life and disease have been had Béchamp’s belief been developed instead of stifled under the jealousy of a rival!”

An understanding of the fundamental principles which he laid down, and the bringing up to date of his basic concepts, as has been done with the theories of Darwin and Newton, might save therapeutics from “remaining always uncertain and insufficient,” as Dr. Vannier describes it.

## 68 STATISTICAL PROOF OF THE INEFFICACY AND DANGER OF CURRENT PRACTICES

“The language of figures, otherwise the science of statistics, is not one which he who runs may read. It is full of pitfalls for the unwary, and requires either special aptitude or special training to avoid these pitfalls and deduce from the mass of figures at our command what they really teach.” —Alfred Russel Wallace

*The Problem:* Doctors in the past have used the declining death rate from smallpox, typhoid fever, and diphtheria as proof of the efficacy of serum therapy. In order for such statistics to furnish valid proof of their theories it would be necessary for them to prove: (1) that the decline in the death rate is not due to other causes; (2) that the statistics used in the periods compared are identical; (3) that inoculations actually prevent the disease as claimed. After the preceding points have been proved, it still behooves the medical profession to prove that such serum therapy causes no other serious change or sequela.

As we shall point out in this chapter, not only is the medical profession unable to show a satisfactory basis for their statistical argument, but there is a great deal of evidence indicating that the statistics as used are the result of faulty mental processes. We shall point out, also, that there is considerable evidence that serum therapy may result in severe sequelae.

*Interest in Statistics:* In the United States it has been extremely difficult to stimulate lay interest in vaccination by the use of figures or facts.

The ridiculously inconsistent demand that vaccination be a compulsory school requirement as a protection against smallpox for pupils already adequately “protected” by the “blessed vaccine” is still a popular one in some cities. Although claiming immunity through vaccination, advocates force opponents to submit on the illogical ground that the unvaccinated are a menace to the vaccinated. They do not understand that their requirement invalidates their argument. If the facts were given an open and unprejudiced hearing, the people, in their own interest, would prevent compulsory vaccination. They have been so thoroughly propagandized to believe that the scientific world is on the side of vaccination that it has been almost impossible to get them to deal with the matter on a factual, rational basis; to them the case is closed. The verdict of science, they believe, has been rendered.

*Political Interest in Statistics:* So far as the politician is concerned, it may be said that this is not the proper place for a discourse on human liberty, but one cannot help but remark the powerful medical lobbies that “guide” the laymen legislators into enacting pro-medical statutes. The “tribune” of the people is used to protect and enhance the economic status of the physician. When we stop to think that many of the present legislators were elected on a platform of personal liberty (in the matter of liquor), it is astounding to contemplate their lack of understanding in the matter of personal health liberty. Trust in a popular fallacy is responsible for their attitude; but it is difficult to explain this trust when it is realized that they must be fully aware of the fact that medical news is censored, and statistics altered.

*Lay Lethargy in the Matter of Statistics:* Even when statistics discredit vaccination, as in our opinion they do, there is a mental reservation held by the trusting and lethargic majority. The medical “head-fixing” department is influential because it so glibly appropriates such terms as “science,” “scientific,” “newest discoveries,” “latest developments,” “great progress.” So we find the paradox of minds closed to facts which require a little reasoning, and at the same time open to the lying ballyhoo of interested exploiters of public health.

*Dishonest Statistical Reports:* There is abundant evidence that figures prepared by interested parties show a bias. Questionable practices in the preparation of health and vital statistics render conclusions drawn from such figures invalid. The editor of *Medical Journal and Record* (May 3, 1933), in an editorial on “Forensic Health,” severely criticizes the publication of such biased statements.

“During the past administration in Washington we were constantly reassured that the economic depression would do no harm to the physical well being of the people of this country,” he writes. “We felt at the time that their statements were quite asinine and probably prompted by political expediency, but it is difficult to refute the asseverations of men in high places, especially when they are known as great engineers and efficient medical men. We took issue with them at the time in spite of the lovely statistics which they presented.

“We now see that tuberculosis has increased in Westchester County thirty-four per cent in the last year. The people who are in touch with actual conditions definitely state that this increase is due to malnutrition, which is in turn caused by economic pressure. This increase in tuberculosis in one of the richest counties in the United States, if not the richest, is a distinct refutation of the statements which have emanated from Washington just prior to the election of our presidential candidate and made by the parties in power. These statistics are a definite proof that our contention was correct, that the present economic depression would lead to an increase in this type of ailment.

“We feel that those who were in power at the time and made the various statements which they did, trying to reassure us about our general health conditions and a number of other things, are definitely of a vicious and, from a forensic point of view, criminal nature. When office seekers try to reassure us about a thing so vital as our national and individual health, in order that they may be re-elected, it may be classed among the lowest forms of human endeavor. To sacrifice others in order to aggrandize oneself strikes us as a rather low procedure. The least that we could have expected from these people was honesty and a statement of fact, even though the facts be unpleasant and certain responsibilities must be taken by those who present these facts. We have no respect for those who offered soothing and comforting misstatements from Washington during the last administration.”

That was on May 3, 1933. Then, on July 31, 1933, *The New York Times* printed the following in an editorial, still repeating the lying ballyhoo and propaganda of some political clique. This, despite the fact that the source of information open to the *Medical Journal and Guide* was also open to *The Times*:

“Public Health Maintained—Health conditions during the first six months of 1933 reflect the better economic situation, so say the vital statisticians. . . . The statement that business improvement has brought health improvement must not be taken to mean that business depression brought a serious impairment of the public health. Nothing of the kind occurred. The first six months of 1932 established an all-

time low for the general death rate in this country, and that in the third year of the depression. . . .”

*Incompetence of Those who Compile Statistics:* Some remarks of Alfred Russel Wallace in *The Wonderful Century* (article on “Vaccination a Delusion—Its Penal Enforcement a Crime”) deal particularly with statisticians, and are quite pertinent to the present discussion: [Emphasis by J.R.V.]

“One misconception is that, as vaccination is a surgical operation to guard against a special disease, medical men can alone judge of its value. But the fact is the very reverse, for several reasons. In the first place, they” are interested parties, not merely in a pecuniary sense, but as affecting the prestige of the whole profession. In no other case should we allow interested persons to decide an important matter. . . . In the administration of medicine or any other remedy for a disease, the conditions are different. The doctor applies the remedy and watches the result, and if he has a large practice he thereby obtains knowledge and experience which no other persons possess. But in the case of vaccination, and especially in the case of public vaccinators, the doctor does not see the result except by accident. Those who get smallpox go to the hospitals, or are treated by other medical men, or may have left the district; and the relation between the vaccination and the attack of smallpox can only be discovered by the accurate registration of all the cases and deaths, with the facts as to vaccination or re-vaccination. When these facts are accurately registered, to determine what they teach is not the business of a doctor but of a statistician, and there is much evidence to show that doctors are bad statisticians, and have a special faculty for misstating figures. . . . The facts and figures of the medical profession, and of government officials, in regard to the question of vaccination, *must never be accepted without verification*. . . . Jenner’s biographer tells us that he had a horror of arithmetical calculations, due to a natural incapacity, which quality appears to be a special characteristic of those who advocate vaccination, as the examples I have given sufficiently prove.”

Wallace, the great scientist, attacked the problem of vaccination against smallpox as a statistician. It is now considered smart in some circles to belittle him for matters which have no bearing on his standing as a scientist or mathematician and thus attempt to embarrass opponents who are skeptical about vaccination’s reputed benefits. In spite of this *argumentum ad hominem*, it is generally conceded that his interpretation of statistics influenced the British Parliament to insert a conscience clause into the vaccination law.

*Faking Statistics:* Recent testimony supporting the charge by Wallace that doctors make bad statisticians is contained in this letter to the editor of *The New York Times* (June 10, 1933):

“There was published this morning another of the long series of reports of the Department of Health of New York City to the effect that the death rate has been dropping during the depression. The latest report is that for the week ending June 3, when the death rate in the city of was 9.76 per 1,000 population, which is taken to represent a decline from the rate of 10.90 for the corresponding week of the last six years.

“Similar reports issued by the Department of Health of other cities appear to suggest that the only effect of malnutrition, overcrowding and starvation during the depression has been, by some magic, a general improvement in health conditions.

“Lest anyone fall into the delusion, I wish to call attention to the fact that the statistical basis upon which the Department of Health makes these estimates is utterly fallacious.

“The Department of Health still works on the assumption that the population of New York has increased every year since 1930 at the same rate as in the decade from 1920 to 1930. In other words, it is assumed that the population of Greater New York increased from the census figure of 6,930,446 on April 1, 1930, to 7,325,000 on June 1, 1933. This is obviously wrong. It is self-evident that the population has not increased during the last three years of depression at the same rate as during the decade of prosperity. While no one knows how many thousands of persons have left the city in the last few years, we do know, from the reports of the Department of Agriculture, that the farm population has increased by about 3,000,000 since the depression, contrary to the traditional trend to the cities. This increase has obviously come largely from city populations.

“The Statistical Bureau of the Department of Health, upon inquiry, admits that there is some evidence that their calculation over-estimates the city population by about 200,000. It is just as good a guess, however, to say that it is over-estimated by 500,000.

“If we assume that the New York City population is over-estimated by only 200,000, the death rate for the week ended June 3 would be 10.01 rather than 9.76. On the other hand, if we assume that the population is over-estimated by 500,000, the death rate becomes 10.46 instead of 9.76.

“So long as no reliable figures are available as to the actual population of the city today, death rates given out by the Department of Health are meaningless as an indication of the effects of the depression on health.”

It may be added that a lower birth rate would also tend toward a lower death rate and that a week is too short a period for any proper basis of comparison. It all indicates how skeptical one must be of figures supplied by interested persons. They base them upon whatever suits their purpose and they make much about what really amounts to very little. The trifling gain which is claimed in this case should certainly have the support of very accurate census figures. Under the circumstances the Department of Health has very little to ballyhoo about. Their methods are questionable, to say the least. Thus the serious charges of tampering with and censoring news on health matters and faking statistics are substantiated.

Next it will be interesting and instructive to see how different opinions in the popular interpretation of case statistics can be arrived at. Some feel that case statistics complicate the problem and impress but few; while others are confident that publishing facts and figures will indicate that the popular therapy of the present day is irrational.

*Conflicting Interpretations of Statistics:* From what follows with reference to diphtheria we see how the same tables of case statistics lead different students to diverse conclusions. Dr. Walter R. Hadwen, of England, in commenting on a lay article, in 1921, said:

“The death rate from diphtheria to the living population is today higher than it was before antitoxin was introduced. The Registrar-General’s statistics of my country show that antitoxin, instead of decreasing has increased the death rate, and the only way by which the statistics can be made to appear lower is by juggling with the case mortality figures, calling common sore throats ‘diphtheria’ and so ‘curing’

harmless cases by antitoxin, cases that would have got well without any treatment at all. If, for instance, you have a death rate of twenty-four per cent from genuine diphtheria, and you add two harmless sore throats to every one of diphtheria you reduce the death rate to 8 per cent. That is how the trick has been done and the public deceived. The cases have been trebled and the death rate fictitiously brought down. They cry: 'Look what antitoxin has done!' I reply, 'Look what your statistical jugglery has accomplished!'

"The 'first day cases' are nothing more than common sore throats; they don't wait to see if the child will have diphtheria, they are satisfied by finding a fictitious germ. The other figures are all arbitrary. In the so-called fourth day cases, he says the death rate is sixteen and one-half per cent. Those are genuine diphtherias from which they die (unless killed by antitoxin), and when they die from the disease itself they die chiefly from suffocation, which this reckless writer says no longer exists."

Dr. Hadwen challenges case statistics; his opponents place faith in them. An impasse develops in the matter of case statistics and the layman is hopelessly bewildered.

*Mortality Statistics Better than Case Statistics from which to Draw Conclusions:* On the other hand, there is no controversy about morbidity and mortality statistics and for this reason we shall deal principally with such statistics in an appeal to the open mind with a demonstration that perfectly rational, scientific, and logical conclusions can be drawn by the rational school from the very same evidence presented by their opponents.

It is asserted in Louis Reed's *Healing Cults* (abstract, p. 10) that there are "hundreds of thousands at any one time whom medicine has not helped." Reed, it should be noted, uses "medicine" here in the broadest possible sense. This is a sweeping indictment. The fact that it comes from the newest apologist for medicine adds to its force.

On page 11, *ibid*: "Eighty to ninety per cent get well under any or no treatment." This is a very damaging insinuation as to the honesty and integrity of the medical profession. Have they taken money under false pretenses? We do not make the accusation, but it is the logical inference to be drawn from the author's statement. Further, since statistically all recoveries are attributed by them to their form of treatment, the value of such statistics is questioned. If 80% to 90% would get well without any treatment, how can they claim as much as they do? He further admits that they have neglected massage, and that there are elements of value in the cults which might well be utilized by the medical fraternity, thereby placing that fraternity in the serious dilemma of being challenged to accept what they have already publicly condemned. If his book falls into the hands of people who think for themselves, his medical clients will have a lot of explaining to do, and what he wrote in their defense will act as a boomerang. How they must wish to be saved from such friends!

*Gross Morbidity as Great as Ever:* The termination of the war saw a series of influenza epidemics, "which took a swifter and greater toll of life than any pestilence which our history has known since the black death of the middle ages," says another author. This is an indictment which does not require detailing and for which there is no excuse; a substitution of diseases cannot be called an advance.

The Congressional Record (December 21, 1937) gives numerous statistics on the inefficacy and danger of immunizations.

*War Typhoid Not Reduced by Serum:* World-war (1914-18) sickness and death were as great as in any other war, except in so far as sanitation and hygiene have been conceded to be responsible for their reduction. Deducting whatever percentage the statisticians wish for these measures leaves the total in sickness and death no better than in former conflicts.

War typhoid was reduced in the camps by sanitary and hygienic measures, yet according to the British Royal Society of Medicine, there were, up to October, 1916, in the French Army alone, 113,465 cases of typhoid fever with 12,380 deaths, despite the fact that all these soldiers had been inoculated against typhoid. Such inoculation had been made compulsory in March, 1914, five months before the war broke out. In reference to these statistics: We are not concerned with any implications which might be obtained by expressing them as per centum items; we are not interested in data giving death and case rates, nor should we be in the least impressed by a comparison of these with typhoid statistics for any other group subjected to different environment. It is the absolute, not the relative number of cases that matters here. That in any group which has complied with the requirements of compulsory inoculation this so-called preventive should fail in so many instances is sufficient reason to question the value thereof or even to discredit the agent.

In *Revue de la Presse Médicale Polonaise* (Warsaw, Vol. I, No. 2, p. 154) Dr. J. Zurkowski reports "An Epidemic of Typhoid Fever in a Group Vaccinated Against Typhoid Fever." This report is valuable:

"In spite of vaccinations against typhoid fever there have been observed three successive epidemics in the regiments. Let us note that half of the regiment were vaccinated by mouth, the other half by the subcutaneous route. The number of cases of typhoid was 43, of which 11.5% were fatal.

"Among those vaccinated subcutaneously there were 17 cases, among those vaccinated by mouth, 25.

"The fact that there were fewer patients among the soldiers vaccinated subcutaneously does not authorize affirming that vaccination by the subcutaneous route is more efficacious than that practiced by mouth.

"It is the unsanitary conditions under which certain companies of the regiment found themselves that was the cause of the greater number of patients in certain companies. The principal cause of the epidemic resides in the fact that the number of germs in the drinking water surpass the theoretical number called 'critical.'

"Preventive vaccinations do not confer absolutely certain immunity because the latter may be conquered by certain agents favoring the outbreak of the epidemic. . .

"Preventive vaccination is efficient enough against endemic typhoid fever, but powerless against epidemics that break out suddenly.

"All that we have said on the subject of vaccinations against typhoid fever is equally applicable to prophylaxis against other infections. It cannot be expected that these vaccinations will always confer immunity."

*Statisticians Claim Infallibility:* And why not, we ask. The layman has been led to believe that certain diseases could be definitely and specifically immunized against. We are not speaking of a merely proportionate decrease, but of the absolute elimination or stamping out of the disease. Infallibility has been claimed. At the

present writing New York City subway trains carry the health department advertisement that diphtheria can be exterminated—not merely reduced, but made extinct.

“The campaign to rid the Bronx and Queens of diphtheria is progressing satisfactorily, Health Commissioner Wynne announced yesterday.” (*The New York Times*, Sept. 20, 1933) We submit that the word “rid” means one hundred per cent; it means extermination. ‘Twas ever thus; for example, in *The Lancet* of January 16, 1892: “No one need die of smallpox; indeed, no one need have it unless he likes—that is to say, he can be absolutely protected by vaccination once repeated.”

According to Rollo H. Britten, statistician of the Public Health Service: “Scarlet fever, measles and smallpox are about as widespread as ever but much less fatal. The latter three diseases were important causes of death a century ago.” (*The New York Times*, March 5, 1933) Again “The necessity for making the district free from diphtheria has been stressed.” (*The New York Times*, October 12, 1933)

Ample proof has been provided in this text from the most dependable sources to indicate how much in error *The Lancet* was. And so with the advertisement in the subway; consider its origin, and exercise extreme caution before being psychologized and indoctrinated. Not only do public officials claim infallibility in these “scientific” wonders, but H. G. Wells (not the English H. G. Wells, who writes so well about history and so poorly about vivisection), in *Chemistry in Medicine* (p. 566), proclaims: “. . . as we now can control diphtheria and tetanus, concerning which it is not too much to say that all deaths are avoidable and unnecessary.” There is no room for equivocation in that positive statement; “all” means one hundred per cent. In the presence of infallibility we humbly ask for proof. And so with other infections. If anyone expects that vaccination will always and unfailingly confer immunity, such expectation has been and is being fostered by the proponents of the theory, but unless very definite benefits can be demonstrated at this time, the sooner the theory is revised to fit the facts, the better for all concerned. On the statements of many of its own proponents we deny its touted efficacy.

If you don’t get the disease, the vaccination gets the credit. If you do, it’s just too bad. What a science! The inoculation is useless against the severe type—epidemic—typhoid; but is effective against the mild type—endemic—typhoid, the kind which sanitation and hygiene reduces to a minimum. We fail to see any justification for compulsion after such reports. In the trenches typhoid was rampant—but was not always so diagnosed or reported. In the trenches it still must be reckoned with. Typhoid is not a problem in modern sanitary camps.

*Statistics of a Sanitary Engineer:* On page 654 of *Chemistry in Medicine*, Ashford, in an article on hookworm, states that the mere building of latrines in the South was responsible for 65% reduction in typhoid. This fact alone challenges any statistics which might be presented to prove the efficacy of vaccines and sera. Why vaccinate and suffer vaccination’s admitted injurious after-effects when it can be shown that vaccines and sera do not guarantee protection, while elementary sanitation does? Why burn down a good building to get rid of rats, when there are other means of accomplishing the same result?

*Disease Increasing:* Insanity, cancer, renal, and cardio-vascular diseases are admittedly all on the increase. The totality of sickness is the important fact—not the itemizing of it. Dr. Oliver T. Osborne, professor emeritus in the Yale School of



Medicine, has written a sensational article in *The Medical Mentor* entitled "The Patient Pays." From it *The Literary Digest* of February 25, 1933, quotes as follows:

"A patient cares little what you call his disease; he is interested only in what the physician is going to do to cure him, or at least, to make him comfortable.

"This highest object of medicine, the object for which medical men are created, is now forgotten by the first class medical schools. The patient pays the price of such neglect. Were it not for the great advances in the science of public health, which teaches how to prevent disease, and especially epidemics, the criticism of medical education today would be far more severe than it is.

"The number of persons who are ailing is increasing. This is due to the speed of our era. Men and women do not rest. Even children suffer from this speedy and restless age; too little sleep, too many side issues in school, too much competition, regulated exercises and games, noises, bright lights, dust, radios, etc., not only make children restless, but impair their health.

"Children have many infections in spite of preventive measures, and are ailing in spite of sunbaths, cod liver oil, spinach, raw carrots, tomato juice, etc. Adults read the health journals, do their 'dailies,' count their calories, take vitamins, and go on diets, also to no avail.

"The medical schools of today are producing 'doctors,' but are they producing physicians? The recent M.D.'s do not know how to evaluate the symptoms of incipient disease or to cure the symptoms of functional troubles.

"Students see in the hospitals only the terminal stages of chronic disorders, a few of the acute diseases, some unusual diseases, and the disturbances that may occur in post-operative cases. In the dispensaries they see an ever-moving picture of ailments of all types but rarely ascertain the termination of really ill cases."

*Medical Causes of Increase in Mortality and Morbidity Statistics:* At this point *The Literary Digest* adds: "It will surprise many to learn that frequently medical students are not taught the symptoms and signs of incipient diseases and the treatment of ordinary ailments, but Dr. Osborne assures us that such is the case. In fact, full-time instructors cannot give such information unless they have the experience of private practices themselves." Dr. Osborne is then further quoted as follows:

"Few chairs of therapeutics are still extant. The treatment of disease is supposed to be taught at the bedside. Generally such instruction is given very meager consideration by the instructor.

"The medical graduate knows how to give transfusions, antitoxins, vaccines, and subcutaneous injections of arsenic and iron; he knows how to give cathartics, morphine, hypodermics in large doses. He knows about the administration of several hypnotics, how to manage shocked conditions, and, perhaps, many of the emergencies; how to treat syphilis, typhoid fever, pneumonia, some of the contagious diseases, diabetes, Bright's disease, ulcer of the stomach, and many of the terminal conditions of the serious diseases.

"Some graduates do not know how to write an ordinary simple prescription. They order ready-prepared mixtures which have the doses stated on the bottles.

"Without a course in materia medica, without a course in applied therapeutics, without a course in prescription-writing, and without any knowledge of the pharmacopeia, the young doctor begins private practice with handicaps to which older physicians were not subjected.

“The recent graduate in medicine does not know how to increase an appetite. He knows very little of the milder laxatives. He knows little about modifying the diet to suit the idiosyncrasies of the private patient. He may know how to use some of the physical therapeutic measures, but he rarely knows the harm that many of these measures may cause, even the harm of too much ‘sun-bathing’ . . .

“Many of the prescriptions that reach a drug store for compounding are from thoughtless physicians who write for four or five ingredients, each one representing some patented pharmaceutical preparation, when equally efficient preparations could be found in the pharmacopeia. Druggists must stock all of these mixtures in order to be ready to fill the orders of the physicians. These preparations are always high-priced.

“These are defects in therapeutic education of medical students for which the patients pay.”

*Medical Confusion:* “The number of persons who are ailing is increasing . . . due to the speed of our era.” Give us proof, doctor, please, on the question of speed as the cause of the increase of disease. We do not doubt that it is increasing, but are you right about the reason for this increase in illness? Kindly show us that the medical graduate’s knowledge of giving “antitoxins, vaccines, and subcutaneous injections of arsenic and iron; carthartics, morphine, hypodermics in large doses, hypnotics,” etc., has nothing to do with “the increase of the number of persons who are ailing.” Medical “science” must also prove that it, itself, is not guilty of increasing the number of persons who are ailing—as it was formerly guilty of causing the high infant and maternal mortality rates. We cannot quite reconcile the doctor’s remark concerning “the great advances in the science of public health, which teaches how to prevent disease, and especially epidemics,” with “The number of persons who are ailing is increasing. . . . Children have many infections . . . and are ailing.” We agree with his criticism and appreciate his candor and honesty, but fail to find any plan suggested in this article whereby to correct or ameliorate the serious situation outlined by him.

“One can be drunk on noise,” says Dr. J. L. Pomeroy. “Noise causes injury to the ear drums, disturbs digestion and gives high blood pressure. It is one of the causes of gangsters. Records show that most automobile accidents occur at noisy intersections.” Osborne’s remedies should include peace and quiet, we suppose. We submit that the greatest offenders against public health are the doctors with their theories. They are driving people mad.

Note the ads on constipation, tuberculosis, cancer, deliberately designed to worry people.

Haggard, in *Devils, Drugs, and Doctors* (p. 7), says: “The primitive woman led a life of active work; in consequence, her child was small. By her exertions, carried on to the day of her delivery, the child was literally shaken into the normal head-down position for the easiest and safest birth. Even in urban communities today hard work and some privation have their effects in making childbirth easier.” Osborne says our troubles are due to hustle, bustle, stress, and struggle. Is Haggard’s theory consistent with Osborne’s? “You puts up your money and you takes your choice,” says the cockney. This is the same profession which has taught that infants should average at least eight pounds at birth, and strongly counselled against all physical activity, including bathing, during pregnancy. Where diametrically opposite positions may be taken consecutively, there is no science. When statistics are supplied, there is

at least an opportunity to agree definitely or to take specific exception to the figures or conclusions drawn therefrom.

*Totality of Disease versus Specific Case Reports:* As an illustration of this we quote from Herbert Spencer, *Facts and Comments* (p. 270):

“A Parliamentary Return issued in 1880 (No. 392) shows that comparing the quinquennial periods 1847-1851 and 1874-1878 there was in the latter a diminution in the deaths from all causes of infants under one year old of 6,600 per million births per annum; while the mortality caused by eight specified diseases, either directly communicable or exacerbated by the effects of vaccination, increased from 20,-524 to 41,353 per million births per annum—more than double. It is clear that far more were killed by these other diseases than were saved from smallpox. . . . This was in the days of arm-to-arm vaccination, when medical men were certain that other diseases (syphilis, for instance) could not be communicated through the vaccine virus. Any one who looks into the *Transactions of the Epidemiological Society* of some thirty years ago, will find that they were suddenly convinced to the contrary by a dreadful case of wholesale syphilization. In these days of calf-lymph vaccination such dangers are excluded; not that of bovine tuberculosis however. But I name the fact as showing what amount of faith is to be placed in medical opinion.” It is evident, then, that true science is not a two-faced Janus which can hold first one opinion and then its contradictory.

*Trickery in Selecting Statistics; Lack of Control:* Concerning the figures discussed by Spencer observe how obviously they indicate that those who originally compiled them had a point to make—had the will-to-believe attitude; and how completely Spencer disposes of the case. It is evident that interpretations of figures can be controlled—if the compiling of the figures is controlled. The public is exposed to this possibility by allowing an interested profession” to prepare its own statistics, interpret them according to its whim or fancy, and secure legislation to enforce its edict on the strength of its favored position. In short, the members of this profession decide what caused death; they select from total mortality or vital statistics what suits their purpose; they put their own interpretation upon the figures; they request and secure power from the legislature to compel compliance with their program. There is no check or control at any point. Those who question or challenge are anathematized; they are condemned and vilified as ignorant obstructionists of scientific progress.

Read pages 95 to 105 of *Medical Biometry and Statistics*, the standard text of the colleges, by Raymond Pearl, M.D., and you will be amazed at the “selectivity” of causes of death allowed the physician who writes the death certificate. And then realize that on the whim of the physician, vital “statistics” are compiled, and legislatures pass laws to enforce medical wish on the basis of such a house of cards. Pearl’s text (unconsciously) exposes the chicanery of medical statistics.

*The Will-to-Believe in Preparing Statistics:* Just as the will-to-believe attitude is evident in much of the statistical data, so it is also present in the mental processes of the suffering public. And this likewise influences statistics. Just as an astrologer prepares the mind of the dupe to anticipate a predicted event, so the astute “scientist” prepares patients wholesale to expect a popular disease. No less an authority than Sir Thomas Horder has recently said, “Concern about high blood

pressure is one of the chief factors in maintaining it, and this holds good whether the concern be on the patient's part or upon the part of the doctor. . . .”

“Another instance is the propaganda for early diagnosis of cancer,” says the editor of the *American Journal of Public Health*. “Many people, especially women, spend their lives dreading cancer. Here there seems little doubt that the propagandists have exaggerated somewhat for the purpose of putting the point over, as well as enlisting the sympathy of the philanthropists in order to raise money for a purpose which unquestionably has a sound foundation.”

We contend that this attitude influences the compilation of vital statistics, and since the *compilation* of vital statistics may be affected by the tactics of the compiler, so the *conclusions* drawn therefrom may be highly questionable.

Dr. Logan Clendening, in *The New York Post* (October 21, 1933), writes: “It is not impossible that other great diseases, which we think we have conquered by the means of preventive medicine, are only in a resting state. Smallpox is an example. We think we drove it off with vaccination, and the evidence for that belief is very good. But taking a long historical view, we see that it was very prevalent in the eighteenth century. It was not particularly prevalent in the seventeenth and sixteenth centuries. It died down at the end of the eighteenth century. We ascribe this to the introduction of vaccination, but perhaps the cause was that it had come to the end of one of its cycles. Perhaps it is only sleeping again.”

Dr. Clendening directs attention to an important controversy of the past century which is still an issue. We wonder if he would extend this same line of reasoning to diphtheria inoculations—as we do. From what he points out, it is quite evident that medical statisticians have been opportunists. They make the most of fortuity.

*Re-classification of Specific Diseases Statistically Dishonest:* The will-to-believe attitude evident in the statistician and in the sick public in the preparation, interpretation, and acceptance of statistical data is not the only deceptive practice which enters the field. The dishonest procedure of re-classifying disease so as to prove a point is testified to in an article in a New York paper:

“Washington, April, 4—Charges of gross carelessness and negligence in preventing and controlling the spread of typhoid and paratyphoid fevers in the army are made against many medical officers serving with the forces overseas, in a circular published by the chief surgeon of the American Expeditionary Forces and made public today by the Public Health Service in connection with a warning that vaccination does not give complete immunity from typhoid. The chief surgeon cites many instances where epidemics prevailed among troops. In a brief review of the occurrence of typhoid fever in the Expeditionary Forces, the chief surgeon says that from June, 1918, the disease spread through many units. During the Chateau-Thierry offensive, the circular discloses, 75 per cent of the troops engaged were afflicted with diarrhoeal disease, such as simple diarrhoea, bacillary dysentery, typhoid and paratyphoid. According to the chief surgeon the high incidence of intestinal disease in this section was due to entire disregard of the rules of sanitation. . . .”

Note also the report of Joseph D. Harrigan, M.D.: “On the peninsula of Gallipoli there were 96,000 cases of medical illness during the short time the British troops were engaged there. Among these 96,000 cases of disease there were enteric cases as follows:

Dysentery	17,837 cases
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Pyrotoxia	1,490	“
Typhoid	425	“
Paratyphoid	8,103	“

“Now, very conveniently for their own ends, the inoculationists have seen fit to make distinction between the different ‘phases’ of the one disease condition; that is, the ‘typhoid’ condition was made to almost disappear, and paratyphoid A, B, C, D, . . . X, Y, Z, loomed large before the dust-filled eyes of a befooled laity.

“Absolutely ignoring the fact that there are ‘stages’ or ‘phases’ in disease conditions of non-epidemic nature—for example, syphilis, tuberculosis, scrofula, scorbutus, etc.—they had the audacity and crass unfairness to draw sharp lines of demarcation between certain of these ‘phases,’ as though they were, pathologically, entirely distinct, when no such distinction could by any manner of means be truthfully said to exist. If this wild method of procedure were to prevail throughout all medicine we would be calling the secondary manifestations of syphilis not syphilis at all, but entirely new diseases; tuberculosis of bone would be an entirely different disease from tuberculosis of the lungs; diabetes, with coma, would be a distinct disease from diabetes without coma, etc.”

Thus, from every angle, medical statistics are questionable.

*Statistical Innuendo:* Insinuation and innuendo are employed to imply statistics which do not exist. Until recently, great stress was laid upon the incurability of cancer. Suddenly, at one of the 1932 conventions great publicity was given to a complete reversal of that position. The 1933 convention continued the ballyhoo. In a letter to the editor of *The New York Times* (October 20, 1933), Bernard Rosenberg says:

“In your report of the second annual cancer symposium, held at Chicago, we are again reminded of the necessity for the early examination of persons who may have cancer. The American College of Surgeons asserts, as have other interested groups from time to time, that early diagnosis largely increases the chances of cure either through operation or the application of radium or Roentgen rays.

“It seems to me that the propaganda of the medical profession in this direction would be greatly strengthened by figures which they must possess but which they have never given to the public. It would be of immense interest to know: first, what is the incidence of cancer among medical men and their immediate families, and, second, what is the mortality among this group from this cause. These figures could be placed alongside those which apply to the general public and should strongly emphasize the claim urged for early examination and detection of cancer.” We can hardly blame Mr. Rosenberg for his scepticism. From obituary reports one is led to assume that physicians who worked with x-rays and factory employes who used radium were aware of cancer, but died in spite of the approved treatment.

*Statistics on Life Expectancy:* Let us now examine the most recent statistics on life expectancy and consider comments made thereon by the most prominent medical statistician of today. On February 19, 1933, *The New York Times* printed a curve and Doctor Louis I. Dublin asked: “Have these added years been spread over the entire span of life? Or were they concentrated in any one period of it? Is there any possibility that there has been an actual loss in expectation at any point in life, and to

what causes shall we attribute such gains or losses? These questions have been carefully studied in recent years, and we are in a position to answer them.

“In the first place, a comparison of the table of life expectation at the beginning of the century with the most recent one for 1930 shows that the greatest gain is at the beginning of life—at birth. The gain at each successive ten years of age is smaller until at age 50 it is almost negligible. ... At 60 there was no gain at all registered for males and only a very slight one for females. In the last decade there were a few minor losses in the expectation of life among males at the older ages. It is clear, therefore, that the story of what has happened will be found mainly in an examination of facts among infants and children, and to a lesser degree among adolescents and younger adults.”

Dr. Dublin asks some pointed questions and says, “we are in a position to answer them.” A close reading of the article fails to disclose a complete and satisfactory answer to the last question: “Is there any possibility that there has been an actual loss in expectancy at any point in life, and to what causes shall we attribute such gains or losses?” In reply to the first part of this question Dr. Dublin frankly acknowledges that adults actually have a shorter life expectancy than formerly; the second part of the question he ignores. What is it that the statisticians are so jubilant about? All schools concede that the life expectancy for infants has been increased, but the history of its incline might be said to resemble the incline gained when the Spanish inquisitors ceased their slaughter. The improvement in mortality rates in both was due to the fact that murderous practices were discontinued. On the strength of the figures it is claimed that life is being “saved” at birth. “Saved” from what? “Saved” from the murderous methods once employed. The descendants of the inquisitors make no claim that they at present are “saving” lives merely because they have failed to destroy them. Yet they would be equally justified in so doing.

Now this kind of reasoning may have certain objectionable features, but it is precisely the method employed by Dr. Haggard when he says that “Homeopathy did not save more but killed less.” If this is fair for him, it is fair for us to say that doctors are not saving more babies but killing *fewer*.

However, although the logic may be a bit strained, we are merely drawing attention to the fact that when figures are based upon the comparison of present practices with the malpractices of the past, the curve of life expectancy is going to ascend in a truly remarkable fashion, and great credit is going to be extended to the professional descendants of very incompetent accoucheurs for the apparently miraculous gain shown by the statistics. The fact of the matter is, however, that censure and condemnation should be meted out for the blunders of the past, rather than praise for improvements of today. As a matter of fact, criticism is still the order of the day. At the June, 1933 convention of the Medical Society of the State of New York, Dr. George W. Kosmak submitted an analysis of “351 deaths in childbirth in New York State, 40% of which were due to faulty management by hospital or doctor. About 20% were caused by personal negligence by the patient or her family. Among the 351 cases, Dr. Kosmak estimated only 33% were non-preventable. The figures do not apply to New York City.”

Does this sound as if credit should be given for “saving” life? With more hospitals and more physicians per capita than any other country in the world, the United States is eighth among civilized nations in infant mortality and twenty-second and last in maternal mortality—a scandalous situation.

When Dr. Dublin says that “in the last decade there were a few minor losses in the expectation of life among males at the older ages” he assumes that you will allow him to pile the gain at infancy into your calculation. To quote an editorial from *The New York Post*:

“Dr. Lewellyn F. Barker of Johns Hopkins University, made a pertinent point regarding longevity. There is little if any proof, he says, that the maximum span of life has been increased by twenty years since 1880, but that average is achieved by decreasing the number of deaths in infancy and youth. Apparently, the span of adult life is so closely bound up with heredity that medical science can do little to alter it except over periods of time.” “Heredity” is proved to be a rationalization by the fact that adult longevity is decreasing and the phrase “periods of time” is therefore meaningless.

On this point we are upheld by Professor H. C. Sherman of Columbia University, who is reported by William L. Laurence in *The New York Times* (September 17, 1933) as stating:

“As there has been so strong a tendency to attribute longevity entirely to hereditary factors, it may be worth while to emphasize the fact that here, in parallel groups of exactly the same heredity, the influence of food on longevity is demonstrated with such degree of mathematical certainty as is represented by ‘chances’ much better than 10,000 to 1, or with a hundred-fold greater certainty than is usually considered necessary for the conclusive establishment of such scientific observations.”

It is a rationalization to say that cancer and other degenerative diseases are increasing because the individual was saved from an earlier fatal disease. If this reasoning were correct it would be possible to show a corresponding decrease with respect to something else in vital statistics. As this cannot be done, the fallacy of such reasoning is patent. To attribute the increase of cancer to the excellence of early and improved diagnosis merely diverts the attention of the student. A gross increase of disease is the reality. The adult individual’s life span is shorter and not longer than formerly.

Thus we see that this much-touted “gain” is, in reality, no more than an approach to what is normal to the infancy of all animal life; that the use of this fact and the figures which express it as indicative of positive value in medical methods is unfair and misleading; that there is a definite and marked loss in adult life expectancy; and that evasion and ignorance of these facts are very common. If Dr. Dublin is in a position to answer the questions he claims he can, he is withholding some very vital information, for he has given the public nothing but a very superficial treatment of the entire subject.

What has happened during these twenty-nine years—the period designated by Dr. Morris Fishbein as that of “scientific medicine”—that there should have been an actual, graphically-demonstrable loss in the adult’s life expectancy? Was this the period of “modern medical science,” or was it merely an age of ballyhoo during which an impotent profession lulled the public to sleep while it fiddled with non-essential sciolistic capers? *If*, in the graph, the 1901 and 1930 lines remained parallel to the end, it could be said that the gain made in infant mortality was a real one, but, as these lines converge, actually a *loss* is indicated.

To account for this loss by saying that, as infant mortality rates improve, those “saved” are likely to die before attaining the peak of the life span, is to imply that defectives are being “saved.” This is specious reasoning, for, at birth, this group is

just as fit as any other group (since survival has been attributed to what the doctor did not do, rather than to what he did). Because a given number of soldiers are killed in battle one year and none killed the following year, it does not follow that the latter are fated for untimely death because of *defects*. Defect does not enter into the problem of reduced infant mortality to any appreciable extent. It is introduced as an excuse.

An analysis of the life expectancy table to show distribution of the gain in infancy demonstrates that almost one-half the gain is lost before age twenty, and sixty-five per cent lost before age thirty—a truly scandalous situation—and those who seek praise for the “gain” must shoulder blame for the “loss.”

To sum up, no real increase can be shown in individual adult life expectancy. No decrease can be shown in totality of disease. All that can be shown is a substitution of disease or re-classification of disease—nosological humbuggery. Decrease in smallpox or diphtheria is accompanied by increase in chickenpox, influenza, renal-cardio-vascular diseases, etc.

*Statistics Should be Prepared by Disinterested Persons:* The preparation of statistical data together with the development of such conclusions as may be derived therefrom should be undertaken with the purpose of establishing certain definite facts. That such information may merit the consideration of those to whom it is made available requires, first, that the sources from which the data are obtained be representative, accurate, and reliable; and, second, that the logic of the derived conclusions be clearly demonstrated. No argument, however clear the processes of reasoning may be, can establish a definite conclusion, unless the statistical data upon which the argument is based have first been shown to be reliable and accurate. Where information of this kind is intended for the lay public, these features should be emphasized, rather than minimized. Considerable responsibility is assumed by persons disseminating information of the nature to which we have referred; they may directly or by implication induce the public to accept as factual conclusions which are controversial or even decidedly erroneous. The attitude that the layman is incapable of appreciating and following a well-developed argument which purposes to sustain a particular conclusion is not tenable, nor does it warrant a superficial treatment of the material submitted for his consideration.

The use of questionable statistics and debatable conclusions drawn therefrom for propaganda is a most reprehensible practice. And this is just why such efforts as Dr. Dublin's offend. There is so much about these statistics which could be criticized that an adequate criticism becomes extremely complex and at times takes on the aspect of quibbling, which, however, it certainly is not. We must insist that statistics be prepared and presented by disinterested persons and that the conclusions drawn therefrom be subjected to the same rigorous and comprehensive examination before acceptance that is customary in other departments of science. They who have an axe to grind should have their statistics examined with extreme care. They who claim responsibility for one aspect of human progress must also accept responsibility for retrogression, when it distinctly comes within their province. Credit and blame are inseparable, when a profession presumes to take entire charge of a problem, and the claim for credit must be examined very closely.

*Pneumonia Still Leads:* According to Wells in *Chemistry in Medicine* (p; 575) [emphasis by J.R.V.]: “Pneumonia still leads in the causes of death. Peritonitis and



other acute infections still kill myriads. An influenza epidemic coming next month would find us no better prepared to prevent or to cure an attack than did the last one, and so on. Our efforts to immunize accomplish at the best but partial success, and often they achieve nothing. Our few curative serums, of which *only* diphtheria has a high proportion of success, are at most but feeble, dilute solutions of the protective agents, gleaned with difficulty from other animals and used in an unnatural environment. Our prospects of any great advances by means of improvements of methods we now know and use seem but slight.”

*Recent Decline in Smallpox:* According to statistics, there has been a decline of 72% in smallpox cases in the United States and Canada in three years. There were 46,654 cases in 1930, 29,493 in 1931, and 13,121 in 1932. Is it not peculiar that the decline should be so abrupt following a long period of compulsory vaccination? Why did the vaccination take so many years to get in its work? Cotton Mather, the zealot, gave it its impetus, but the time involved raises our suspicions. Has there been an equal increase in health—a decline in gross morbidity as smallpox has declined? Obviously not. Apparently, moreover, there has been no increase in vaccination to which this decline in smallpox can be attributed. Something other than the “great gift of Jenner” will have to be credited with the decline.

*Exaggeration and Misuse of Statistics:* Consider snake bite, which has a fatality record as low as two to seven per cent, according to some authorities; less than fifty per cent, according to others. “When Colonel Crimmins returned from France to take command of Camp Bullis, Texas, he was astonished at the toll of life taken by poisonous snakes. *Almost half* the bites of *poisonous* snakes in 1922 were fatal. Colonel Crimmins tabulated the number—more than 600—of fatalities recorded by newspapers and became convinced that the potassium permanganate treatment then in use was not a success. He realized that these figures did not represent the total number of persons bitten by venomous snakes.” (*New York Times*) Contrast this with the fact that popular fancy has snake bite mortality fixed at one hundred per cent. Yet a specialist places the figure below fifty per cent, and other authorities make a much lower estimate. What a remarkable reputation anti-venom will achieve in the days to come when it will be credited with one hundred per cent effectiveness. This statistical method was used by vaccinationists to further their objective in the pre-vaccination era.

Conditioning the cell with either biological or chemical agents fails to impress one as being a rational procedure. Even though it may be scientific, it may at the same time be irrational. Science and rationality are not synonyms. This is a tremendously important point. Dr. Nikola Tesla said: “The scientists, from Franklin to Morse, were clear thinkers and did not produce erroneous theories. The scientists of today think deeply instead of clearly. One must be sane to think clearly, but one can think deeply and be quite insane.” His charge is very applicable to the field of therapeutics. In no other branch of learning, perhaps, has there been more profundity coupled with insanity.

*Substitution of Disease a Statistical Trick:* It avails us nothing to be “saved” from a specific infection only to be rendered susceptible or weakened in another way—particularly so when there is a rational therapy in the first place—yet one can come to no other conclusion than that we are being deceived by disease substitution

after reading the following remarks on diabetes—a disease which propaganda led the public to believe was mastered by insulin. Specifically, diabetes is not the province of bacteriology, but substituting diabetes for infectious disease brings it within our field.

“Statistics show, Dr. Mosenthal said, that the use of insulin has resulted in a drop during the period from 1900 to 1950 of about 50 per cent in the diabetes deaths due to coma. On the other hand, diabetics are still susceptible to bacterial infections, gangrene, cardiovascular conditions and renal diseases, and deaths attributed to these causes have shown a rise of 35 per cent in the period from 1900 to 1930.

“At the present time about 2,000 diabetes deaths are registered annually in New York City. The diabetes death rate is about 50 per cent higher than it was twenty years ago. These facts call for some remedial effort.

“A distinct rise in the number of diabetes deaths is occurring, not only in New York City, but throughout the United States, as shown by the mortality statistics from other cities.” (*The New York Times*)

For immediately utilitarian purposes, the germ and serum and vaccine therapy theories may work out “as if” they were true. But for the ultimate outcome of orthodox therapy the application of the “as if” philosophy is irrational and devastating—and the patient “pays.”

*Damaging Statistics Suppressed:* The citing of 47,369 cases of smallpox in the Philippines with 16,447 fatalities, and 1326 cases with 869 fatalities in Manila in 1918 after about ten years of the biggest and most vigorous vaccination campaign ever conducted anywhere in the world, reported by the *Philippine Health Service* for 1918, is generally received with a shrug of the shoulders, as something about which there are two sides, with science in the directing role and the skeptic blocking progress. In fact, there is even much evidence to show that this report with its appalling figures on smallpox in the Philippines was suppressed by officials. It is small wonder, then, that specific figures and their implications are frequently minimized through prejudice. General morbidity and vitality statistics, however, are not so lightly considered. That is the reason why we place small reliance upon tables of figures of specific diseases to convince the laity.

*Diagnostic Quackery which Influences Statistics:* It is so easy to decide that a case is not smallpox if the patient has a vaccination scar; and so simple to call chickenpox, smallpox; or diphtheria, tonsillitis, and vice versa—the physician being guided by wish fancy just often enough to make statistics based on such practices worthless. In the March, 1927 issue of *The Journal of the Michigan State Medical Society*, in an article entitled “Smallpox,” on the page devoted to the Michigan Department of Health (p. 181), Guy L. Kiefer, M.D., Commissioner, states:  
[Emphasis by J.R.V.]

“The following points are important aids in the *diagnosis* of smallpox:

“The absence of any history of having had smallpox or a *successful vaccination* within the past five years.”

Obviously, statistics compiled on such practices are unreliable and dishonest.

And so the words of the defendant convict him. He stands indicted for tampering with case statistics. The unquestioned statistics of total morbidity or

mortality cannot so easily be tampered with, and they prove him guilty again, this time on the count of commercially exploiting human life and health.

*A Guessing Game:* With one author blaming diminishing longevity on heredity, and another on speed, while still others, with voice and pen, poke fun at civilized man's efforts to prolong his life by "sunbaths, codliver oil, spinach, raw carrots, tomato juice," reading health journals, doing "dailies," counting calories, taking vitamins, dieting, etc., the problem of the shortening of the span of adult human life takes on the appearance of a guessing contest. Are we listening to sorcerer-humorists or scientific humanists? Is this science or popular entertainment? Has the trouping Dr. Fishbein qualified himself as a scientist or as a comedian? He certainly is not one to inspire public confidence in the declining art of medicine. When these men, with years of training and experience behind them, busy themselves with the scientific solution of this serious problem, we shall be glad to study their findings. In the meantime, it falls to our lot to debunk and censure them and their sciolistic methods and practices—and their own statistics serve us very well for the purpose.

*Appendicitis Vital Statistics not Widely Circulated:* "Many persons are exposed needlessly to the risk of death by appendicitis operations, according to Dr. Frederick L. Hoffman, consultant statistician. In an article in *The Spectator*, August 3, 1933, he charges that many surgeons undertake appendicitis operations although they are incompetent to perform them.

"Although the death rate from appendicitis in this country for the last two years has shown a slight decrease, he asserts that it is far higher than need be. For 1932 the rate was 15.7 for each 100,000 of population.

"He quotes Sir James Berry, British surgeon, as holding that in many cases medical treatment is preferable to an operation.

"Dr. Hoffman calls attention to wide variations in the death rate between various cities. Bethlehem, Pa., Cicero, Ill., and Fresno, Cal., had no deaths from appendicitis in 1932. New York had a rate of 14.8. Salt Lake City had a rate of 46.9, the highest recorded.

"'No satisfactory explanation can be given for these extraordinary differences, which, however, are maintained from year to year with approximate consistency,' he writes. 'No thorough study of the geographical incidence of appendicitis has ever been made for this country. Regardless of its practical importance, appendicitis still continues to be neglected as a public health problem.'" (*The New York Times*) See Pearl for very simple and correct explanation.

What was the death rate in appendicitis before the advent of surgery? What is the death rate in appendicitis where modern surgery and local manipulative examinations are not resorted to? How many appendicitis deaths occur annually without a doctor? Is the doctor's presence an additional hazard? Again, how can any reliance be placed upon statistics which are so variable in different parts of the country? Does the local Chamber of Commerce influence the physician in his diagnosis? What faith can be placed in a practice which is so inefficient?

Dr. Hoffman refers in this article to the "you-must-do-something" school. As there were approximately 20,000 deaths in appendicitis in 1931 in the continental United States, and as there were large districts reporting a death rate so ridiculously small as to call into question the accuracy of the figures, it is quite apparent that the

“you-must-do-something” school did that something very effectively. Torquemada has modern rivals—in the name of “science,” instead of religion.

## 69 CONCLUSION

“Medicine is the spoiled child of philanthropy.”—Nicholas Murray Butter

We have presented facts and figures from authoritative sources to show the need of a new and revolutionary point of view in considering the relation of bacteria to disease. We have examined the evidence for serum therapy in the light of history, biography, science, law, statistics, and cold reason and found this evidence wanting. More than merely negative evidence for serum therapy, it is positive evidence on which to justify a new and rational practice and condemn the old.

In our discussions we have emphasized realism versus animism in the interpretation of disease; spontaneous versus inoculable disease in consideration of etiology; elimination versus suppression, cooperation versus combat, and correction versus palliation, in treatment; genuine normalizing versus substitution in evaluating results; science versus sciolism in both prophylaxis and therapy; and, finally, reason versus scientific superficiality in the application of the latter.

A recent issue of *Popular Mechanics* announces that 2,000 British physicians have banded together as scientists to oppose the germ theory. It is not sufficient, however, merely to challenge certain aspects of a specific theory. Small progress will have been made, if these physicians do no more than substitute chemotherapy for serum therapy. We hold that chemotherapy is likewise an irrational practice—even though scientific. The world still flounders in the confusion created by Pasteur, champion go-getter of the nineteenth century. When we shall have finally uprooted the dogmas with which he enchained us, and acquired a broad, well integrated, philosophic view of the entire matter, then we may look for progress.

Dr. Wood, the cancer specialist, when he declared that we “face an absolutely blank wall unless we get further along and face cancer from a different point of view,” might well have been talking of bacteriology. The promise of added progress in cancer research, he said, lies in the discovery of some marked and constant difference between the cancer cell and the normal cell, rather than in “vague experimentation and the injection of everything that can be thought of into the unfortunate patient.” (We quote from *The New York Times*, October 21, 1932.)

“Dr. Wood gave much of his time to a classification of the qualifications needed in a cancer research worker. He must combine a knowledge of ‘philosophy, biology, chemistry and statistics with an enormous amount of common sense, brains and the ability to withstand the shock of disappointment,’ he declared.

“He must work with large numbers of animals and must have a large number of case histories to draw upon before he can even commence to formulate any of his less important theories.

“ ‘The more we experiment the more difficult the whole problem appears,’ he admitted.

“Dr. Wood estimated that there were not more than fifty competent cancer research workers in the world, and because of the paucity of trained brains to undertake this work he would not recommend, he said, large endowments for cancer research laboratories scattered about generously over the country.

“Dr. Sigerist said that he felt that ‘cancer to a certain extent is a philosophical problem,’ and that research workers should approach the solution of this problem

with a thorough background of speculative philosophy behind them, instead of bringing to their laboratory researches the somewhat imaginative attitudes too prevalent today.

“Malignant tumors, he said, were as old as history and we were not much further advanced today in the fundamental solution of the problem than were the ancients. We have the advantage of better tools and our therapeutics are more efficient although they follow the same general principles of Greek therapeutics. The decline of speculative philosophy in the research laboratories of cancer experts may account to some extent, he believed, for the frequent sterility of present-day research.”

We feel that this comment applied to the field of bacteriology and immunology would be equally fitting, and that it so perfectly summarizes our position in the matter that it belongs rightfully in this chapter.

In concluding our thesis, there is one vital aspect of the subject which we wish to leave in your mind. It is the aspect in which the subject ceases to be a problem of science and becomes a problem of social ethics. We have touched on it before, but nowhere is it better put than in the words of Dr. J. H. Greer, as quoted in Charles Sprading’s *Liberty and Libertarians*:

“The acquisition of authority and the exclusive privilege of controlling the bodies of others for mercenary purposes appears to be the chief aim of the medical fraternity. To aid in accomplishing their designs, by deception and wily subterfuge they have secured the enactment of unconstitutional laws and the appropriation of state funds to be placed at their disposal. Thus have they established and do they maintain one of the most gigantic trusts that ever cursed a free-born people. Medical monopoly is the last remnant of mercenary priestcraft to thrive upon mankind’s afflictions and misfortunes. But its chains, forged centuries ago by ignorance and superstition, have gradually weakened by the continuous strain put upon them by education and enlightenment. Tighter and tighter does it seek to draw those weakened chains, and greater and greater grows the resistance afforded by increased public knowledge. Before long the fetters must give way, and humanity will be free to enjoy the liberty of striving to know all things and of exercising the right of self-preservation;”