in a few words: 1. Isolation and protection of all sources of water supply. 2. Introduction of filtration systems. This has been strongly and ably urged by Dr. Thomas Darlington, President of the Department of Health, in a report to Mayor McClellan. 3. Education of the medical profession and public, outside of as well as in the city, as to the necessity of the immediate sterilization of all typhoid excreta.

Could an efficient system of filtration of the water supply of New York City be introduced, there is every reason to feel confident that the death rate of typhoid fever of Greater New York would sink from 16 per 100,000 to 5 per 100,000 at the most. Such has been the result of the introduction of filtration in the large continental cities—Berlin, London, etc.,—and it is due to such filtration and to nothing else.

Typhoid fever is a preventable disease. The mode of prevention seems clear; it is little short of criminal if the community fails to act.

REMARKS ON THE INFECTIONS OF JOINTS.*

BY ROBERT W. LOVETT, M.D., BOSTON.

IN no department of joint disease has so great a disturbance occurred in the last few years as in the views regarding infections in their relation to joint affections.

For many years it has been recognized¹ that joint inflammation of a more or less severe type occurred in connection with certain forms of general sepsis, as in pyemia; in certain forms of specific infection, such as gonorrhea, scarlet fever, typhoid fever and pneumonia, and after trifling local infections. But that acute articular rheumatism was an infectious disease and possibly only "an attenuated pyemia," and that the joint manifestations accompanying rheumatism were possibly very closely allied to those septic joints mentioned above, is a point of view that has dawned on the medical community only of late years, and is at present the most interesting question before one interested in the diseases of joints. A quotation from a paper by Poynton, however, points out better than I could do the difficulties of the situation:² "It is no easy task to grapple with the subject of arthritis . . . for around gout, rheumatism and rheumatoid arthritis, theory has cast her bright and attractive mantle, beneath which gray and sober fact is liable to be stifled."

In general, acute joint infections fall into four groups:

(a) Acute osteomyelitis of the articular end of one of the long bones involving the joint secondarily.

(b) Acute suppurative synovitis or joint abscess.

(c) Acute plastic synovitis leading to joint obliteration.

(d) Acute serous synovitis.

* Read before the Boston Society for Medical Improvement, March 12, 1906.

¹ Howard Marsh: Infective Arthritis, *Lancet*, Dec. 13, 1902, p. 1603.

² Poynton: *Arthritis, Practitioner*, 1903, 71, 128.

Tuberculous and chronic joint disease are not considered in this classification.

I shall pass briefly over the first three divisions, which are treated at length in all modern textbooks, and call your attention particularly to the relation of infection to serous synovitis.

(a) *Acute osteomyelitis*³ involving the joints needs only to be mentioned. It is an acute suppurative inflammation of bone, due to infection of the bone marrow by pyogenic organisms. The staphylococcus is the most frequent, the other organisms found being the streptococcus, the pneumococcus and the typhoid bacillus. The bacillus fetidus, the colon bacillus, the pyocyaneus and anthrax have been reported. The condition can be produced in animals by the introduction of pyogenic organisms into the nutrient arteries of the bones and by trauma to animals infected through the general circulation. The bone infection occurs in infectious diseases, such as measles, typhoid fever and the like. It appears following some slight local supuration such as paronychia or furuncle. It is to be found as the result of direct infection, as in septic compound fractures, and in many cases no source of infection can be found. It must be remembered, however, that what is to be said of the throat as a port of entry for pyogenic organisms in connection with rheumatism, applies here as well. A typical instance is as follows:

A healthy boy of six blistered his heel from his boot, and this blister was opened by his mother with a pin. That night a chill occurred, followed by great pain in the left tibia. This tibia was operated upon and opened a few days later and found to be stripped of periosteum over much of its surface. The left hip then became extremely swelled and painful, and was flexed and abducted. Sleep was impossible and general sepsis was present; the temperature was 103 and the child's condition most serious. The hip was incised and a collection of pus found in the joint. The neck of the femur was drained, the hip a few days later dislocated spontaneously, but was replaced and held by a plaster bandage, and the boy made a good recovery.

The treatment of such cases consists in the earliest possible free drainage of the joint, with a careful search for the infected area of bone, which should be opened.

(b) Acute suppurative synovitis, or joint abscess, occurs as the result of various infective organisms and appears in infectious diseases of a wide range, and also as the result of direct infections of the joints, as in wounds.

The infectious diseases in which joint infections have been reported are as follows:

Cerebrospinal meningitis, diphtheria, dysentery, erysipelas, epidemic parotitis, glanders, gonorrhœa, epidemic influenza, measles, pneumonia, pertussis, puerperal fever, pyemia, septicæmia, scarlet fever, smallpox, tonsillitis, typhus fever, typhoid fever, after the use of sounds and catheters, and possibly in malaria.⁴

The treatment consists in free drainage of the

³ Edward H. Nichols: *Acute Infectious Osteomyelitis*, *Jour. A. Med. Ass'n*, 1904.

⁴ For literature, see Bradford and Lovett: *Orth. Surgery*, 2d ed., p. 194.

joint as soon as the evidence of suppuration can be established by aspiration or other means.

(c) Plastic or ankylosing synovitis, in which an acute and severe joint inflammation is followed by partial or complete obliteration of the joint without suppuration, is most familiar as an accompaniment of gonorrhea. It occurs in connection with other infections and is seen following attacks of general febrile disturbance, often diagnosticated as "grippe," in which very acute joint inflammation is present. Beyond the fact that it is at times clearly of infectious origin, and presumably always of this sort, but little can be said of its etiology. No satisfactory treatment has been formulated.

(d) *Acute Serous Synovitis*. That acute serous synovitis occurs in connection with general infectious diseases has long been recognized. It is familiar under the names of "scarlatinal rheumatism," "rheumatic typhoid arthritis,"⁵ "gonorrheal rheumatism," etc., and the occasional occurrence of simple joint effusions in most of the general infections is a matter of common information. Aside from these demonstrably infectious cases are the cases of single, or more often multiple, acute serous synovitis without obvious infectious cause, which are universally classed under the name of "articular rheumatism." Of course to the laity and to some of the profession, every joint pain is rheumatism until it is demonstrated to be something else, but taking the cases of legitimate acute articular rheumatism, it may be properly asked, What is the evidence for and against their infectious character?

(1) In the first place they do not differ essentially in type from cases of synovitis obviously due to infection, except that "articular rheumatism" is assumed not to suppurate; if it suppurates it must be considered infectious. An acute polyarticular or monoarticular serous synovitis occurring in the course of scarlet fever, for example, is recognized as infectious; if a similar synovitis occurs without obvious source of infection, it is generally classified as "articular rheumatism."

(2) The bacteriology of acute rheumatism has been carefully studied. Cocci in general resembling the streptococcus have been found in the joint effusion, in the endocardium and in other structures in acute articular rheumatism by many observers.⁶ This organism injected into animals has produced synovitis and endocarditis,⁷ and even choreiform movements were observed in some rabbits thus inoculated.⁸

The identity of this coccus has been disputed and its relation to the streptococcus is not clear. Three names have been given to it,—micrococcus rheumaticus (Walker),⁹ diplococcus rheumaticus (Poynton),¹⁰ streptococcus aus chorea (Wasserman). Menzer, who has been one of the pioneers in this subject, sees no reason to assume that this

is a specific organism any more than is the coccus of erysipelas, and would regard it merely as a modified streptococcus.¹¹ Cole¹² has produced, by the injection of streptococci from various sources into animals, synovitis and endocarditis. In spite of the weight of English opinion,¹³ it cannot therefore be regarded that the existence of a specific micrococcus has been established.¹⁴ As against the infectious theory of rheumatism, Cole and Jochmann failed to find any microbe in the joints or blood in cases of typical articular rheumatism, and McCrae,¹⁵ in two hundred and seventy cases of acute articular rheumatism, reported the bacterial results as "practically negative." It must be remembered, however, that the bacillus of tuberculosis is found with difficulty in tuberculous joint abscess, and that the organisms of typhoid fever and gonorrhea are frequently absent from the joint effusions of those affections. Moreover, positive evidence is always more valuable than negative, and the identification of the bacteria found has been made by perfectly competent observers.

It is, of course, possible that the joint inflammation may be due to toxins produced by the bacteria rather than by the bacteria themselves. Charrin¹⁶ produced suppurative arthritis in joints by the injection of a sterilized culture of the bacillus pyocyaneus.

(3) *Source of Infection*: The existence of a primary focus of infection is wanting in many if not most cases of acute articular rheumatism; that is to say, no obvious source of pyogenic infection is found. The difficulty that such cases may offer is shown in the following cases:

A healthy young woman, the relative of a physician and under his observation, was confined and delivered of a healthy child. Her convalescence was absolutely normal and free from all suspicion of sepsis. On the tenth day, before sitting up, she developed an effusion in the left knee, accompanied by little or no pain. Traumatism in the ordinary sense was not in the question and her health and temperature were normal. No source of infection could be found, and a diagnosis of infectious synovitis of unknown source was made. The affection pursued a sluggish course and completely recovered. Some weeks later she had a doubtful attack of appendicitis which brought out the fact of former pains in the region of the appendix. Some six months after the synovitis the appendix was removed and was found to be full of pus and showing chronic inflammation of long standing. Here was a source of infection at least competent to have produced the joint symptoms.

A long step toward explaining a possible source of pyogenic infection in articular rheumatism was made some years ago in the recognition of the existence in the tonsils of organisms capable of producing the symptoms of rheumatism when injected into animals.¹⁷ The tonsils act as a filter and are more effective as such when they

⁵ Keen: *Surgical Complications of Typhoid Fever*.

⁶ Thelberg: *Med. Record*, Dec. 14, 1901, and Menzer: *Der Aetiologie der Akuten Gelenkrheumatismus*, Berlin, 1905.

⁷ Meyer: *Deutsch. Med. Wchsft.*, Feb. 7, 1901.

⁸ Triboulet: *Revue de med.*, xvii, 189.

⁹ Practitioner, February, 1903, 185.

¹⁰ *Ibid.*, 1903, lxxi, 128.

¹¹ Wiener *Kl. Wchsft.*, 1901, 20.

¹² *Jour. of Infect. Diseases*, 1904, i, 114.

¹³ Symes: *The Rheumatic Diseases*, London, 1905, p. 394.

¹⁴ *Jour. Am. Med. Assn.*, Feb. 4, 1905,

¹⁵ *Ibid.*, Jan. 3, 1903.

¹⁶ Quoted by Cave: *Lancet*, Jan. 12, 1901, p. 83.

¹⁷ Menzer & Meyer: *Loc. cit.* *Allaria Rev. Crit. de Clin. Med.*, Nov. 23, 1901.

are normal than when inflamed. When their cells are injured or diseased it is obviously easier for the pyogenic organisms of the locality to enter the circulation, and the frequent association of sore throat with the early stages of rheumatism suggests a lesion of the tonsils, impaired efficiency of filtration and consequent easier entrance into the circulation for the pyogenic organisms. This, to a certain extent, explains the more frequent occurrence of rheumatism during the cold and damp weather most likely to attack the throat. Chronic tonsillitis is, therefore, to be regarded as a most likely source of rheumatic infection, either in the acute cases or in the chronic joint affections to be mentioned.

A healthy boy of twelve was attacked by a sore throat followed by inflammation of the middle ear, accompanied by the usual high temperature, and the ear drum was punctured. A few days later a throat infection was made evident by a rise of temperature and sore throat. A second increase of throat symptoms followed again in three or four days. About two weeks after the onset of the original trouble, the left ankle swelled, and in a few hours became exquisitely sensitive and extremely painful to any movement and suggested the beginning of a serious purulent joint infection, but the temperature was normal and leucocytosis was absent. It was fixed in a millboard and sheet wadding dressing and two days later was free from pain. On the third day after the involvement of the ankle an effusion of less acute character appeared in the left knee. This was treated in the same way, and in a week from the onset in the ankle both joints were practically well.

The case then stands as follows: The synovitis of acute rheumatism does not differ clinically or pathologically from the synovitis of infectious diseases, except that it is more benignant. It can be produced by the injection of cultures of pyogenic organisms, and pyogenic organisms have been found in the joint effusions. The specific character of these has not been accepted. No satisfactory explanation of rheumatism has ever been offered, and the history of late years shows a steadily larger number of "rheumatic" cases constantly being classed among the infections. It cannot as yet be regarded as a specific infectious disease, but the evidence rather tends to identify it as an "attenuated pyemia," as it has been called. The evidence is not yet conclusive and the fact that the symptoms are so readily controlled by salicylic acid in many instances is not in accord with what we know of infections.

With regard to the relations to each other of chronic rheumatism, arthritis deformans, chronic infectious arthritis and the like, and with regard to their infectious origin, there is little convincing evidence. Their frequent outcome from what appears to have been an acute rheumatic attack, the almost universal failure to recognize arthritis deformans as such when it begins acutely, and the close clinical resemblance of all the affections to each other suggests the possibility of a common cause.

The bacterial evidence for the infectious

nature of these conditions is suggestive, but nothing more.¹⁸ Organisms have been found in a comparatively few cases. These organisms have in some cases given negative results on inoculation, and in others have caused lesions similar to those of arthritis deformans, but in most cases organisms have not been found. The clinical evidence in many cases suggests an infectious origin for those processes, some of them especially in children originating during some general infection and pursuing a chronic course.¹⁹

Summary. — Acute infections of the joints of undoubted bacterial origin occur in all grades of severity in connection with many infections. As a rule, any one of two or three types of joint inflammation may occur in connection with any one infection, no one type of joint disease constantly accompanying any one infection. In many cases the source of infection cannot be established, and in such cases the importance of remembering the function of the tonsils and the presence of pyogenic bacteria in the mouth as a ready source of infection is of importance. That acute articular rheumatism is an infection seems probable from bacterial and especially from clinical evidence, but this cannot yet be regarded as definitely proved.

THE TEACHING OF HEMATOLOGY.

BY RALPH C. LARRABEE, M.D., BOSTON.

Instructor in Hematology and Medicine, Tufts College Medical School; Physician to Out-Patients, Boston City Hospital; Physician for the Children's Department, Boston Dispensary.

THIS paper is based upon the writer's experience in teaching hematology at Tufts College Medical School. As our methods differ somewhat from those commonly employed, it has seemed worth while to present this brief account of them.

Our course is in charge of an instructor, who, with two assistants, is responsible to the Department of Clinical Medicine. It is given in the first part of the third year. The student has then finished his courses in anatomy, histology, pathology and other fundamental subjects, including the elementary study of the blood and its pathology. He is familiar with the use of the microscope and has just begun the clinical work in which his technical knowledge of laboratory methods will be needed and applied.

Laboratory work occupies the student two hours a week for ten weeks. The class is divided into sections of about twenty. This is a convenient number for two assistants to handle and saves needless duplication of costly apparatus — an important item when it is considered that each student must have at his disposal a counter and a microscope with an immersion lens and a No. 5 Leitz objective. The first four laboratory exercises are occupied in teaching technique pure

¹⁸ Poynton & Paine: *Path. Soc. Trans.*, 1902, i; White: *Guy's Hosp. Rep.*, lvii; Gask: *Lancet*, May 9, 1903; Schüller: *Berl. Klin. Wchsft.*, 1893, 865; Blaxall & Bannatyne: *Lancet*, 1896, i, 1120; Dor: *Comptes rend. Soc. de Biol.*, 1893, 899; Stengel: *Am. Jour. of Med. Sci.*, March, 1903.

¹⁹ Goldthwaite: *BOSTON MEDICAL AND SURGICAL JOURNAL*, April 7, 1904; Harrington: *BOSTON MEDICAL AND SURGICAL JOURNAL*, Jan. 28, 1904.