this disease. Since then the conception that acute rheumatic fever is a systemic disease has gradually found more acceptance, but this has been greatly hampered by the perpetuation of the nomenclature acute articular rheumatism, although we may have acute rheumatic fever without joint lesions.

The history of the etiology of acute rheumatic fever shows many different theories. First, it was thought that it was due to cold, dampness and changes of temperature. Then, in 1831, Dr. J. K. Mitchell advocated the theory that it was due to a lesion of the central nervous system, and this theory even advanced so far as to localize the site of disease in a hypothetical joint centre. Following this the chemical theory held sway, chiefly advanced by the English writers, in which lactic and uric acids were given pre-eminent places. Next the toxic theory was advanced, which ascribed the disease to intestinal putrefaction and the absorption of the toxins so formed. In 1888, however, a new conception of acute rheumatic fever was realized when Cheadle conclusively demonstrated that the arthritis was but one manifestation of a general disease. With the advance of bacteriology the closé resemblance between acute rheumatic fever and other infectious diseases was observed. Achalme[1] described a bacillus which he isolated from the joints of a case of acute arthritis. Singer[2], Sahli[3], Maragliano[4], and others reported Staphylococcus aureus as the cause.

Then Dana[5], Charrin[6], and Lubarsch[7] isolated a diplococcus or streptococcus from acute rheumatic fever cases. In 1899 Wassermann and Westphal[8] published an exhaustive experimental study of a streptococcus which they isolated from a case of chorea following acute rheumatic fever, and with which they produced polyarthritis in rabbits.

Since 1900 a most important and exhaustive study of acute rheumatic fever has been carried on by British investigators, chief among whom are Poynton and Paine[9], Beattie[10], and Shaw[11]. They have repeatedly isolated a diplococcus from the fluids and tissues of patients suffering from acute rheumatic fever, both antimortem and post-mortem. This organism they have named "Diplococcus Rheumaticus."

They have grown it from the blood, urine, and pleural exudate of acute rheumatic fever patients during life, and from the heart valves and the pericardial fluid after death. They have also demonstrated it in sections of the heart valves and pericardium, and of a rheumatic nodule in a fatal case of acute rheumatic fever.

It seems from the rapidly accumulating facts that acute rheumatic fever is a bacterial infection, and that the organism is a streptococcus. That it is a distinct variety of the streptococci has yet to be proven. Bacteriologists are not ready at the present time to classify streptococci as Streptococcus erysipelatis, pyogenes