

# The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. LXI, No. 10

CHICAGO, ILLINOIS

SEPTEMBER 6, 1913

## EPIDEMIC STREPTOCOCCUS SORE THROAT —ITS SYMPTOMS, ORIGIN AND TRANSMISSION\*

JOSEPH A. CAPPS, M.D.

CHICAGO

Epidemics of streptococcus sore throat have recently sprung up as a scourge, so new that they receive no mention even in the latest editions of our text-books.

Within two years three extensive and alarming epidemics have occurred, which have no counterpart in American medical history. In some respects they call to mind the first severe invasion of influenza, but the latter disease spread everywhere throughout the country, whereas each of the streptococcus sore throat outbreaks was confined to a single community.

It is quite probable, however, that many of our local tonsillitis epidemics, loosely designated as "grip," have in reality been due to streptococcus.

In England comparatively small outbreaks of "septic sore throat" have been described<sup>1</sup> in seventeen localities, and a probable relationship to the milk-supply has been recognized; but in few instances were careful bacteriologic investigations carried out. The streptococcus was first demonstrated in cultures obtained from cases of sore throat in Finchley (1904), where 550 persons were attacked. The *Streptococcus pyogenes* was first detected in the suspected milk in the Guildford outbreak (1903). The following year in the Colchester epidemics, claiming 600 victims, the streptococcus was obtained both in the throat exudate and in the milk. This organism was likewise recovered from the throat and the milk in the Christiania epidemic of 1908, in which 548 persons fell a prey to the disease.

The existence of mastitis in the cows was noted in all four of these foreign outbreaks.

The American epidemics of streptococcus sore throat were carefully studied from a clinical and bacteriologic point of view, and many new facts bearing on the etiology were brought to light. A brief summary of the salient features of these outbreaks may be profitable.

**Extent of Epidemic.**—About 1,400 persons were stricken in the Boston outbreak, and of this number 1,043 were investigated. In Baltimore about 1,000 were affected and 602 recorded. The total in Chicago was estimated to be over 10,000, though only 1,271 were studied.

**The Organism.**—In all three cities a peculiar type of hemolytic capsulated streptococcus was secured from

throat cultures or from the peritoneal exudate of fatal cases. Davis compared cultures from each outbreak and found them practically identical.

**Season.**—The cases appeared in the winter and spring months, occurring in Chicago during the last of December and in January, in Baltimore during March, and in Boston during May.

**Explosive Outbreak.**—A sudden appearance of a large number of cases was characteristic of each epidemic. Often several members of a household were taken ill almost at the same time, suggesting a common source of infection rather than dissemination by personal contact.

**Clinical Symptoms.**—The sore throat manifestations were similar in all epidemics. Intense hyperemia with or without a grayish exudate was the usual picture.

Enlargement of the cervical lymph-nodes was one of the most characteristic symptoms, occasionally resulting in suppuration. Extreme prostration and a tendency to relapse were emphasized by most observers.

The three epidemics reveal a striking similarity in the complications. Otitis media, peritonsillar abscess, erysipelas or some other form of skin eruptions and nephritis were common sequels. Arthritis, endocarditis and myocarditis occurred in many cases. Pleurisy and pneumonia often ushered in a fatal septicemia. But the most dangerous and remarkable complication was peritonitis, which was responsible for a great number of deaths.

**Relationship to Milk.**—The infection was traced definitely to a single milk-supply in every city. The sore throat cases traced to one dairy made up 70 per cent. of the total in Boston, 65 per cent. in Baltimore and 72 per cent. in Chicago. As the suspected dairy in each instance supplied only a small proportion of the city consumption, these percentages become highly significant. There is no doubt that secondary cases developed by means of contact, but it quite overturns our accepted theories of transmission to observe that the secondary contact cases were few compared with the number of cases directly infected by the use of milk.

As this fact was established in all the epidemics we must in the future revise our notions concerning the importance of contact in the dissemination of streptococcus infections.

**Ultimate Sources of Contamination.**—The indictment in each of the epidemics against a single dairy as the carrier of the germs was so strongly supported by clinical facts as to be final and conclusive.

The evidence submitted to show when and how the milk became infected may be considered under the following classification:

1. Streptococcus in the mixed milk at the collecting plant or at the farm.
2. Mastitis in cows, from whose udders streptococcus is directly obtained.
3. Streptococcus sore throat in the milkers.

\*Read in the Section on Pathology and Physiology of the American Medical Association, at the Sixty-Fourth Annual Session held at Minneapolis, June, 1913.

1. Savage, W. G.: Milk and the Public Health, Macmillan & Co., London, 1912

4. Streptococcus sore throat in other employees who handle the milk.

In his study of the Boston epidemic, Winslow<sup>2</sup> found no evidence of cattle disease and no well-defined cases of sore throat in a milker. He did discover, however, cases of sore throat in employees at the farm and dairy. The actual infection he attributed to a human carrier.

Stokes<sup>3</sup> succeeded in obtaining more definite information in the Baltimore outbreak. He found the streptococcus epidemic in the raw mixed milk of one herd but could not trace the infection to the cow. There was no history of septic sore throat cases among the farmers or employees. Although final proof was lacking, Stokes expressed the belief that the infection originated in the cows.

Investigations in the Chicago epidemic revealed the *Streptococcus pyogenes* in the milk obtained directly from the inflamed udder of the cow and also in cultures taken from a milker with sore throat. A history of septic sore throat among employees was also established.

Direct contamination of the milk by human carriers does not offer a satisfactory explanation of these epidemics, because such a contamination is likely to be casual and occasional, whereas the outbreaks indicate a continuous infection for a few weeks. Furthermore, the amount of infectious material introduced by a human carrier in coughing, etc., is relatively small to produce such appalling results.

A mastitis infection with streptococcus, on the other hand, would entirely explain the continuous stream of milk contaminated with enormous numbers of organisms.

A comparison of the American epidemics does not indicate at first glance that the source of infection was the same, because mastitis was not found at all in the Boston herds, and only a non-pathogenic form of garget was discovered in Baltimore.

These apparent discrepancies, however, may be cleared up by recent investigations. Davis<sup>4</sup> has succeeded in producing in the udder of a healthy cow an infection with a human strain of *Streptococcus hemolyticus*. The germs, introduced through a slight abrasion of the teat by the contaminated hand in the act of milking, caused an ascending infection of the teat and udder, characterized by the presence of pus and myriads of streptococci that persisted for two or three weeks. Strange to say, there was no caking of the bag. We cannot therefore place any dependence on caked bag as a necessary accompaniment of mastitis. Streptococcus mastitis without caking may well have existed in some of the cows in the Boston or Baltimore herds and yet have escaped detection.

In the future, it will be necessary to examine the milk from each cow in a suspected herd for pus and streptococci.

I believe that streptococcus mastitis will be recognized as the most important source of extensive epidemics of septic sore throat. That the mastitis may originate by transmission of human streptococcus through the medium of the milker seems to be established. Whether or not mastitis due to a specific bovine streptococcus may become virulent to man is a problem yet to be determined. When we come to a consideration of prophylaxis

all other measures and precautions sink into insignificance when compared with thorough pasteurization. A general enforcement of pasteurization would put an end to a malady that otherwise promises to be one of the most formidable and wide-spread of the infectious diseases.

122 South Michigan Boulevard.

## CHRONIC STREPTOCOCCUS ARTHRITIS\*

DAVID J. DAVIS, M.D.  
CHICAGO

The relation of streptococci to arthritis is a very broad one. The highly acute purulent inflammation of joints which may be simply part of a general streptococcus pyemia or septicemia is well known and fairly well understood. It occurs in connection with severe streptococcus infections, such as puerperal fever, infected wounds, erysipelas and common infectious diseases. Of the latter I shall mention especially septic sore throat as it has been observed by Capps and others in connection with milk-borne epidemics. In all of these there is a tendency, at times not very marked, but at other times striking indeed, for the streptococci to produce arthritis.

The tendency on the part of streptococci to localize in joints of experimental animals seems to concern the several varieties, though to an unequal extent. The common hemolytic streptococci and the *Streptococcus mucosus* readily attack joint cavities; the *Streptococcus viridans*, on the other hand, rarely does so. The *Diplococcus rheumaticus* is also very prone to attack joints as shown by Poynton and Payne, Beattie, and especially by Rosenow's recent work. The streptococcus found in septic sore throat, which appears to be a highly virulent variety of the hemolytic streptococcus, readily produces multiple arthritis in animals with often other lesions such as endocarditis, pericarditis, myocarditis, peritonitis, etc. This agrees with the occurrence of the serious complication in human beings observed during these milk-borne epidemics.

The subacute or chronic types of arthritis leading toward some permanent change in the joint or periarticular structures form a large and miscellaneous group of joint disorders, and their classification is as yet imperfect and unsatisfactory. "Arthritis deformans" is, perhaps, the term most commonly applied to them. Their etiology is undoubtedly manifold and they probably furnish an illustration in which similar and often indistinguishable pathologic changes and clinical symptoms are brought about by widely different agents. From this large group in recent years, however, certain smaller groups have been separated; for example, traumatic arthritis deformans and certain cases of gonococcal arthritis. In other words, as soon as the true etiology of a case, or group of cases is discovered it is removed from this large group of arthritis deformans to a class or group designated by its etiologic agent.

In this paper I wish to discuss chiefly from an etiologic point of view a certain group of these cases of chronic arthritis or arthritis deformans whose cause can with fair definiteness be attributed to a streptococcus infection, usually though not always originating somewhere about the upper respiratory passages. I believe that it is often possible to separate these cases clinically and

2. Winslow, C. E. A.: An Outbreak of Tonsillitis or Septic Sore Throat in Massachusetts, and Its Relation to an Infected Milk-Supply, Jour. Infect. Dis., 1912, x, 111.

3. Stokes, W. R., and Hachtel, F. W.: Septic Sore Throat, a Milk-Borne Outbreak in Baltimore, Pub. Health Rep., 1912, No. 103, p. 44.

4. Davis, D. J.: Communication read at the American Society for Clinical Investigation, Washington, D. C., May 5, 1913.

\* Read in the Section on Pathology and Physiology of the American Medical Association, at the Sixty-Fourth Annual Session held at Minneapolis, June, 1913.

\* From the Pathological Laboratory of St. Luke's Hospital, Chicago.