

XLII.

THE RELATION OF ENLARGED TONSILS TO ENDOCARDITIS.

BY ALBERT C. GETCHELL, M. D.,

WORCESTER, MASS.

In the Transactions of the Association of American Physicians for the year 1899 is a paper entitled "Endocarditis Occurring in the Course of Tonsillitis," by Dr. F. A. Packard. While the writer was guarded as to his conclusions, yet the paper stands as a definite exposition of the relationship of tonsillitis and endocarditis, and has been so quoted by other writers from time to time. The question Dr. Packard discussed was a pathologic one, but with the growth of the operation for the complete removal of the tonsil it has become also a practical one. For if so serious a malady as endocarditis depends to any considerable degree upon the presence in tonsils of a causative agent, any operation, however difficult or dangerous, is not only proper but necessary.

There is no question that among throat specialists in the United States there has of late years been a growing feeling of the importance of the tonsils as the portals of entry for the infection of many serious diseases, and one finds this idea gaining ground among the profession at large.

Looking over the literature of the subject among writers on diseases of the heart and diseases of children, I find this view rather cautiously stated by some, while others make no mention of it whatever. Thus, not to attempt an exhaustive list but by way of illustration, Whittaker in "The Twentieth Century of Practice," 1895, Vol. IV, p. 156, says: "The tendency of modern belief is to regard the throat as the avenue of entrance for the microorganisms of rheumatism and endocarditis." Haig-Brown found conclusive evidence of endocarditis in 8 out of 345 cases of tonsillitis. These and other quotations may be

found in Packard's article. Babcock in his book on the heart, page 156, says: "It (endocarditis) has been known to follow tonsillitis and so apparently a local process as furuncle." Holt says (edition of 1910, p. 1140): "Acute tonsillitis often ushers in an attack of rheumatic arthritis, and occasionally acute endocarditis."

The most recent book on diseases of the heart that I have consulted, James Mackenzie, makes no mention of the matter. And also Edmund Cautley, in his "Diseases of Infants and Children," just published, makes no mention of the relationship.

On the other hand, Osler in the article on endocarditis, which he writes in his recent "System of Medicine," speaks in no uncertain terms. He says (Vol. IV, p. 138):

"The tonsils, the mycotic hot beds, are responsible for a great many cases, and if as is now commonly believed the infection of acute rheumatic fever is here nurtured, they take the first rank as sources of infection."

Again, page 139:

"The so-called endocarditis from cold is probably always rheumatic, and of tonsillar origin, and it may occur in the febrile attacks of children as the result of slight or even overlooked tonsillitis."

And again, page 149:

"Much could be done to lessen the number of cases of rheumatic fever, of chorea, and of endocarditis if we attacked more vigorously and more systematically the enlarged tonsils of children. Here is the point toward which our efforts should be directed. A child subject to recurring attacks of tonsillitis or with marked adenoids should have the tonsils or adenoids thoroughly removed. Other measures of local treatment simply trifle with that what is always a very dangerous condition."

Here the question with which this inquiry started is answered, positively, almost dogmatically, by an eminent authority. The tonsils of a child subject to recurring attacks of tonsillitis are dangerous structures, whose complete and thorough removal is demanded to save the child from so serious a disease as endocarditis. It seems to me, however, that even this does not settle the question, but that further study of the problem is needed. I used to think that the relation of tonsillitis—or rather, to be more specific, the relation of chronically en-

larged tonsils to endocarditis was a very definite one. For the last four or five years, however, I have been studying more carefully the cases of enlarged tonsils and adenoids that have come to me for operation, and I do not feel that the causative relation is so definite as I once did.

I have had continuous charge of the throat department at the Memorial Hospital in Worcester for nineteen years. In that time there have been somewhat over 2,000 tonsil operations. Until recently I have done all the operating myself. For the last few years my assistants have done a good deal, but I still exercise close supervision over the work, operating myself one day in the week. For safety of anesthesia it has always been our custom to examine with bare chest the heart and lungs of patients before operation, and if the house physician finds anything abnormal, the matter is brought to the attention of the operating surgeon. These cases include boys and girls up to the age of 16 and adult women. Since I have begun to pay especial attention to the matter, I have been struck with the extreme rarity of heart lesions.

I can recall but one case of undoubted cardiac disease. Of this case I shall speak later. And so of all the cases that have come under my observation in this hospital, in private practice and in other hospitals, while I have found definite cardiac lesions in persons who came for the removal of enlarged tonsils, yet these cases are so few that the complication in my experience is one of great rarity. If, then, enlarged tonsils, which have been subject to recurring attacks of inflammation, are so definite a cause of endocarditis, why should evidence of endocardial trouble in these patients be not only not the rule but the rare exception?

I desire now to cite the case I referred to a few moments ago. It was a boy of 14, with well marked heart disease—enlargement, thrill, apex systolic murmur transmitted to the axilla and heard plainly in the back. He had very large tonsils, and was referred to me for removal of them on account of his heart trouble. At first sight it seemed to be a case where there was a direct relation between enlarged tonsils as such and endocarditis, and so the doctor thought who referred the case. On careful inquiry, however, I found that this was not so. This boy, so far as he and his mother knew, had never had sore throats, and up to two years ago was always well. Then

he was ill with diphtheria, and was at the Isolation Hospital. Since then he has not been so well, and has had to call a doctor from time to time. When he was at Isolation Hospital no note was made of any heart trouble. It is hardly possible that a heart like his could have escaped detection in this well conducted hospital. It is, therefore, plain that his endocardial trouble developed after his diphtheria, and probably as a result of it. His tonsils were removed and examined by a pathologist. They were simply hypertrophied tonsils.

Before much can be gained in settlement of this question, we must have definite ideas of what we mean by endocarditis. Is the pathologic process limited to the lining membrane of the heart, or does it involve to a greater or less extent the heart muscle? If the former, the lesion is much more serious than the latter, for the mechanism of the heart is permanently damaged, by alteration in the structure of the valves. On the other hand, if the lesion which causes the murmur is due to myocardial change and dilatation of the orifices, this condition may be temporary and the heart may later recover its perfect power.

Osler says (op. cit., p. 134): "Infective endocarditis is a valvular, rarely a mural lesion." But later on (p. 136): "In addition to changes in the endocardium there are usually alterations in the myocardium," and (p. 145) "Sturgis very correctly insisted that a majority of the cases are best described as carditis, so frequently are the epicardium and the substance of the heart involved."

Mackenzie (p. 215) says: "One must consider carefully the murmurs arising in the course of a febrile attack, even in rheumatic fever, for the presence of a murmur may not necessarily mean the invasion of the mitral valves by the inflammatory process, but may be due to the tonicidity of the poisoned heart muscle failing and giving rise to incompetence of the mitral orifice, due therefore not to endocardial, but to a myocardial affection. Endocarditis and pericarditis, both acute and chronic, bulk so largely in medical literature, only because an abnormal sound invariably impresses the mind more than an abnormal sign perceptible by other senses, and the easy recognition of a valvular murmur and friction sound has led to the associated symptoms being ascribed to the same lesion."

(See also Thayer "On the Commoner Types of Functional

Cardiac Murmurs," Transactions of the Association of American Physicians, Vol. XXV, p. 75.)

Will not these considerations account for the fact that if children are carefully examined a murmur may be found and a diagnosis of endocarditis made, while later in life this same person may apply for removal of the tonsils and no cardiac lesion be found? This may happen peculiarly in children, in whom a cardiac abnormality may develop without being accompanied by evident symptoms.

This is a case in point: A young girl upon whom I operated last summer for the removal of tonsils. About once a year she had had attacks of tonsillitis. Her parents were people who never hesitated to call the family physician, a careful and competent man. A cardiac murmur was accidentally found in the examination of the gymnasium physician, its existence or heart trouble not having been suspected.

Tonsillitis is common in childhood. Osler says: "Tonsillar infection is universal in childhood." Is acute endocarditis proportionally common then? Gibson, "Diseases of the Heart and Aorta," 1898, page 393, says: "Acute endocarditis is essentially a disease of the active period of adult life, its occurrence being most frequent between the age of twenty and forty." To finish Osler's remark just quoted: "Tonsillar infection is universal in childhood, while rheumatic infection although common only occurs in a comparatively small number of children" (p. 139). Julius Derschfeld says (Allbult's System, Vol. 1, p. 630: "Infective endocarditis occurs between 20 and 40. It is rare in children according to Osler, although he (Derschfeld) thinks it is not so rare."

The diseases which are supposed to cause endocarditis are scarlet fever, measles, smallpox, influenza, erysipelas, pneumonia, pyemia, gonorrhea, tuberculosis, rheumatism, otitis, diseases of the vagina and uterus, empyemia (Gibson).

Researches demonstrate conclusively the invasion of the heart by the specific organisms of rheumatic fever, pneumonia, typhoid fever, diphtheria, erysipelas, influenza and various septic infections. (Mackenzie, page 214.)

Whittaker ("Twentieth Century Practice," page 156) gives in addition to those already mentioned, osteomyelitis and periostitis, furunculosis, dysentery, malaria, and says: "It is universally admitted that even the most trivial infection may be

followed by endocarditis. Thus endocarditis has been observed after infections as inconsiderable as quinsy and mumps." That is to say, many, probably all the general diseases which are due to a microorganism, may produce heart disease. Of these, rheumatism, in all probability a germ disease, is more liable to cause heart infection, which locates on the valves. It is true also that other infections, more likely to be local than general, may produce the same result, such as septic infections. The mouth, being the opening of the body most accessible, it seems that the point of entrance of the infecting agent would be there, and it is undoubtedly true that it is in many instances. Moreover, if the structures in the mouth are inflamed or diseased, resistance is lowered, and infection is more liable to take place. In this way chronically enlarged and inflamed tonsils would, and undoubtedly do, make the throat more liable to infection. But it does not follow that the tonsils themselves are the necessary and sole foci of infection. Thus, a careful reading of Packard's five cases shows that in only one of them were there swollen and inflamed tonsils, that is, a typically acute tonsillitis. One of the cases had a slightly inflamed tonsil with an enlarged cervical lymph node of the same side. The second had a mild angina with catarrhal inflammation of the pharynx. The third had greatly enlarged and acutely inflamed tonsils. The fourth had pain in swallowing, and on examination the mucous membrane of the fauces and pharynx was found to be swollen and reddened, and the submaxillary lymph nodes were plainly enlarged. The fifth had a slight sore throat, and Dr. Packard's services were called because of a glandular swelling on the neck a few days later. I quote these cases thus fully because this paper has been so often referred to. It is evident that there was in all these cases an infection of the throat, but it is not evident that the tonsils were in all of them either the cause or the avenue of the infection.

Sore throats,—anginas as they used to be called,—even in children, are not all due to inflammation of the tonsils. They are very often due to an inflammation of the glandular tissue of the pharynx, even an infection with a deposit simulating diphtheria starting here.

I have under observation now an adult, a nurse in a hospital, whose tonsils I removed, with the main intent to put a stop to infective sore throats. The tonsils were thoroughly removed,

but she has had the sore throats which excite suspicion of diphtheria as she did before, and she tells me of a friend who has a similar experience.

In adults a not uncommon cause of a sore throat is an inflammation of the lateral columns of the pharynx. The pain and other symptoms in this variety of sore throat simulate tonsillitis, and might very well be called tonsillitis by practitioners not accustomed to carefully inspect the throat with a good light. So that we should be cautious in concluding from the mere fact of sequence of sore throat and endocardial symptoms that the initial disease was tonsillitis. But, if in a certain number of cases the sore throat is due to tonsillitis, and endocarditis follows this sore throat, the question still remains, were the tonsils the accidental or the essential foci of infection?

The question here is not, does endocarditis follow in a considerable number of cases attacks of tonsillitis, especially in persons who have rheumatism, which clinically seems well established; but does the enlarged tonsil, by and of itself, contain the infecting agent likely to produce the disease? If this is so, endocarditis associated with enlarged tonsils should occur most frequently in the period of life when tonsillar inflammation is most common, which it does not, and persons having enlarged tonsils should show not rarely but frequently signs of endocarditis, which in my experience is not true.

An answer to this question should be found in the study of the bacteria of infective endocarditis and of the bacteria of the tonsils. It would seem from the studies of Libman and Cellar ("Observations on the Etiology of Subacute Infective Endocarditis," Transactions of Association of American Physicians, Vol. 25, p. 5) that we are getting to a definite knowledge on this subject. The next step will be to study the bacteria of the tonsils, and see if they correspond to those found in the cardiac lesions, and if they may be made to produce endocarditis experimentally. This Dr. David J. Davis of Chicago has done. He has reported upon this work in a short communication in the *Journal of the American Medical Association* for July 2, 1910, page 26. This report and a personal letter from him indicate that our knowledge of the bacteriology of the tonsils is very imperfect and inadequate; that the whole subject must be gone over again and newer methods employed. He finds his own work far from conclusive, and at present states only facts,

refraining from any general conclusion. He finds that the bacteria of the crypts are of one kind, and those of the surface another. The bacteria of the crypts injected into rabbits intravenously, almost invariably localized in the joints and tendon sheaths, but they in no case produced endocarditis. On the other hand, in one instance he succeeded in producing endocarditis with an injection of the surface bacteria. These bacteria he cautiously concludes are the same that have been found in the blood in cases of chronic infective endocarditis by Rosenow and others.

To make a general statement, so far as one can be made at this time: the tonsils, lying as they do in the outer portals or vestibule of the digestive and respiratory tracts, are organs upon which certain bacteria lodge, and the infection of the tonsils with the bacteria cause an inflammation of the tonsils, and later within the system these same bacteria may cause an inflammation of the heart. Hence the sequence in time of tonsillitis of endocarditis. But the tonsils are the accidental and not the essential agents in this infection.

Early in this paper I said that its object is a practical one. It is this: So far as enlarged tonsils have a relation to endocarditis, when is the operation for their removal indicated, and what kind of an operation should be done? Before answering this question it may be well to review briefly the present status of opinion on these two points in reference to the general question of tonsil operations.

In a recent paper by Guy L. Hunner, on "Chronic Urethritis and Chronic Uteritis Caused by Tonsillitis" (*Journal of the American Medical Association*, April 1, 1911, p. 937) the following paragraph may be found:

"In a comparatively recent paper Rosenheimer has reviewed the literature and found the following list of ailments ascribed in certain cases to tonsillar infection: aneurysm, appendicitis, erysipelas, and a number of other skin manifestations, meningitis, iritis, pleuritis, pericarditis, pneumonia, paraplegia and strabismus, parotitis, nephritis, osteomyelitis, phlegmon of the lower extremities, oophoritis and orchitis, septicemia, typhoid beginning with an angina, and tuberculosis."

We are told by reputable men that at the base of innocent looking tonsils lie foci of disease, and that every particle of

tonsillar tissue should be removed to prevent serious systemic disease.

The character and type of operation generally practiced now may be inferred from the instruments used. I counted in a recent instrument catalogue 64 varieties of tonsil knives, many sharp and pointed.

At one of our best medical schools recently I saw a child brought before the class for operation. The child was profoundly etherized and put in the upright position, and the instructor told the class that the present method of operation required the complete etherization, and that nothing else would do. The tonsils were then removed by the dissection method.

In a recent paper (*Boston Medical and Surgical Journal*, Vol. 164, No. 12, p. 415) Dr. E. A. Crockett says: "Twelve deaths have occurred in and about Boston in the last two years following the removal of tonsils, besides a large number of considerable hemorrhages that would have been fatal unless checked by experienced hands." Is it not time to pause and ask if the operation for the removal of tonsils as at present practiced is necessary, and if this toll of mortality is justified?

So far as endocarditis is concerned, I am convinced that a simply hypertrophied tonsil has little to do with it as a causative factor; that when it happens to have been inflamed before the onset of endocarditis, the infection has been on the surface of the tonsil, and has not started deep in the substance of the organ, and that the removal of the tonsil has little effect on the incidence or recurrence of the disease, except, if it be subject to recurring attacks of inflammation, it in so far makes the tissues about the pharynx, including the tonsil itself, more susceptible to infection.

It is beyond the province of this paper to discuss the relation of the tonsil to other diseases. But I infer it is the same. An uninflamed simply hypertrophied tonsil has slight causal relation to systemic disease. An inflamed and diseased tonsil may have such relation, and undoubtedly does in a limited number of cases.

The indications for removal, so far as this question is concerned then, are, is the tonsil subject to recurring attacks of inflammation, or is it evidently diseased? If the tonsil is simply hypertrophied, such removal, either in whole or in part, as will prevent future attacks of inflammation is sufficient. If it is

evidently diseased, and especially if buried and bound down by inflammatory tissue, it must be thoroughly dissected out.

In my experience, an operation with the tonsillotome is adequate for the first class. The instrument I have used now many years is a modification of the Mackenzie tonsillotome. Since the operation for the complete removal of tonsils has been practiced, I have slightly modified my method of operating, and have found that a complete removal can be accomplished by this instrument.

This operation in experienced hands, in most cases, removes the tonsil as thoroughly as it can be removed by any operation, and in my opinion is as safe as a tonsil operation can be. In inexperienced hands, while the operation may not be as complete, the risks of serious accidents are slight. The operation for the removal of tonsils of the second class by dissection methods is a difficult, and in inexperienced hands a dangerous one. It should be undertaken only by operators of mature experience and skill, and as Dr. Crockett has truly said, by those able and ready to meet dangerous complications that may arise.