Medical Sociology

The importance of nutrition in medical sociology is evidenced by the programme of work before that Section at the present Annual Meeting, wherein it is given a prominent place; it will, then, speak for itself. But I am constrained in this connexion to refer again to the remarkable experiences of the late Miss Margaret McMillan mentioned in her book—*The Nursery School* (1930). Here she has described the weakly, ill-nourished, and ill-conditioned children of the slums who come for admission to the nursery school at Deptford: children who are rachitic and bronchitic; children with inflammatory states of the nose, ear and throat; children who have adenoids and dental caries. After they have been nurtured and properly fed for a few years they are, she says, almost all cured of any ailments from which they may have suffered on their entrance to the school. They are all straight and well grown, the average child is well made with a clean skin, is alert, sociable, and eager for life and new experience . . . "He does not need to see the doctor or the dentist, and he has none of the minor ailments which affect the children of the slums." Surely, we have in experiences such as this the root of the whole matter. We now know that large numbers of our people have not the means wherewith to provide themselves with proper food even though they had the knowledge to enable them to judge of what is proper; a state of affairs damnable of our civilization. But it would be wrong to suppose that poverty is the only cause of faulty nutrition. "Access to abundance of food does not necessarily protect from the effects of food deficiency, since a number of factors—penury, prejudice, ignorance, habit—often prevent the proper use and choice of health-giving foods."⁴ To these impediments to normal nutrition I would add indifference, neglect, and the weak indulgence of their children by many well-to-do parents which fosters tastes and habits detrimental to normal growth, nutrition, and health. Education of the people, the better education of ourselves: these are urgent necessities. It may be that the medical curriculum is already overgrown. If so, let it be pruned, let us teach the student less about "disease" and drugs and more about "health," more about nutrition, which is the very basis of health.

Medical History

I come now to the last of the Sections—that of the history of medicine. In this history no chapter is more romantic, more inspiring, more replete with hope for the future of our calling and for the well-being of mankind, than that dealing with the newer knowledge of nutrition. Its opening pages tell of the dawning idea of the relation of certain diseases, notably scurvy and beri-beri, to defects in food. These are succeeded by others describing the discovery of vitamins. As the story unfolds the numbers of these and of other elements and complexes, found to be necessary for normal nutrition, increase, and the conception of disease as a manifestation of disturbed structure or functions of the body consequent on faulty feeding and on the faulty nutrition resulting therefrom comes into being. Patient research, the discovery of facts, generalizations from facts, the prediction of fresh facts—its pages tell of these. These are the ways of science; these the ways in which the science of nutrition has been built up.

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ACUTE STREPTOCOCCAL INFECTIONS OF THE MIDDLE EAR*

BY

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In my endeavour to find inspiration for the writing of this paper I turned to a discussion held in 1932 at a meeting of the Ear and Throat Sections of the Royal Society of Medicine. I doubt if I can add much of interest to what was stated then, and so I crave your indulgence. The wording of this discussion really includes all acute infection of the ear, because the streptococcus is the infecting organism in the vast majority of cases. The pneumococcus comes a poor second, and Pfeiffer's bacillus and the staphylococcus are bacterial curiosities.

Aetiological Factors

How does the streptococcus arrive in the middle ear? We shall hear from the subsequent speaker the frequency with which the streptococcus is found in the nasopharynx. Mr. Barwell stated in the 1932 discussion that

"spread of inflammation by the lymphatics or the blood vessels is so rare that it need not be considered in this connexion although it is possible for the ear to be involved in the course of a septicaemia of nasal as of any other origin, but ordinarily the inflammation reaches the ear, either by continuity along the mucous membrane to and along the Eustachian tube, or by discharges from a septic focus in the nose being blown through the Eustachian tube into the tympanum, without involvement of the intermediate tract of mucous membrane. The latter is, I think, by far the commoner method."

What Mr. Barwell said in 1932, laryngologists and otologists might well agree with in 1936. For my own part I am not convinced of the truth of the last statement in his remarks. I have had, however, one striking example of infection being blown to the ear. A doctor said "Good night" to a friend at midnight. He blew his nose forcefully, and immediately felt something go to his ear; he had a night of pain, and at 10 o'clock the next morning I opened the membrane and found pus. This is in my experience a rare occurrence; for such an accident to occur must there not be active inflammation in the nasopharyngeal membrane?

Further, I would like to mention the fact that the passage of the Eustachian catheter, and inflation of the tube through it, do not produce infection in the middle ear. Among the thousands of times I have used the catheter, only once am I aware of having started an acute otitis media, and I refuse to believe that my catheter has failed to encounter at least one streptococcus in its passage through the nose and nasopharynx. The case in which I did cause an acute otitis media was that of a child whose tubes I was inflating three or four times for catarrhal deafness; inflation was performed at 9.30 a.m., and at 9 p.m. incision of the membrane released pus. The child fell at a nursing home on a visit to a sick relation, and thus absolved me from blame for the otitis media!

By far the commonest cause of acute ear infection is an acute infection of the whole upper respiratory tract, such as the common cold, so-called influenza, tonsillitis, and streptococcal infections after removal of tonsils; should the antrum become infected, ear infection is more likely. When measles and scarlet fever are the cause of the mucous membrane infection the ear is very often involved. In the summer bathing in fresh-water baths is the common cause of acute otitis media.

* Read in opening a discussion in the Section of Oto-rhino-laryngology at the Annual Meeting of the British Medical Association, Oxford, 1936.

Dr. Lowndes Yates pointed out a long time ago that mucus was an antiseptic, and that if the mucus was removed or diluted it failed to keep the organisms in check. Fresh water is one of the worst offenders in diluting the mucus, and the two important ways by which the nasopharynx mucus is so diluted is by bathing in fresh-water baths and by using nasal douches, another cause of acute otitis media.

Lowered resistance due to other factors doubtless paves the way to streptococcal infection of the ears. Some American observers during the war undertook some entertaining experiments to find out what forms of exposure gave rise to "colds." Batches of recruits were placed in different conditions of discomfort; some were subjected to cold by inadequate clothing, some were made to go naked to the waist, and some had their feet kept constantly damp and cold. It was found that the incidence of infection was greatest among the last group.

In the same discussion Mr. Layton made a suggestion which attracts me very much. He thought that the virulence of the streptococcus might be enhanced by the presence of another micro-organism, or by the virus of the common cold or influenza. The following case may perhaps support this suggestion:

A young man suffered from staphylococcal septicaemia, under the care of a physician in Guy's Hospital. On three separate occasions *Staphylococcus aureus* had been recovered from the blood. He was extremely ill with a high fluctuating temperature rising to 103° F. To complicate matters, he developed double acute otitis media, both ears discharging pus; he became very deaf and his temperature rose higher still, to 104° F. It seemed that he would die unless something was done, and it was thought that the extra strain of pus in the ears was a factor that could be removed. Both mastoids were opened under gas and oxygen; both contained pus throughout. Cultivations from the bone on both sides gave a pure growth of the streptococcus. The man recovered with normal hearing.

It might have reasonably been expected that here at last was a blood-borne staphylococcal infection of the mastoids. This case may also be used as a strong argument against blood-borne otitis.

Perhaps another factor enters the picture: the anatomical arrangement of the Eustachian tube and middle ear. It is always stated that in children the tube is relatively large and short, but it may also be that in some the mucous membrane is more easily overcome—perhaps through some endocrine failure. Certain it is that some children and adolescents get a recurrent otitis media many times whenever they catch a cold or are exposed to cold and over-exertion. We have all known such patients, and I feel that when they get past a certain age they cease to be so affected. They "grow out of it," a good phrase often on the lips—perchance wise lips—of our forebears.

Prophylaxis

Prophylaxis as a logical outcome of aetiology therefore consists in keeping the nasopharynx reasonably clean by removing infected adenoids as well as septic foci in the nose and paranasal sinuses. Avoid bathing in fresh-water baths, and nasal douches; both are important in the case of subjects prone to colds or having a nasal or post-nasal catarrh. To avoid colds is impossible, but patients known to be susceptible to acute ear infections would do well to have a course of anti-catarrh vaccine treatment.

It is of much interest to note that persons who are found to carry haemolytic streptococci in the throat or nasopharynx are less susceptible to streptococcal infections. A Dick test will show whether a patient is susceptible to the haemolytic streptococcus, and these

patients can be immunized against this organism. This is a commendable prophylactic procedure if patients, often children, are going to live in a community in which scarlet fever has appeared or "influenza" is prevalent.

Treatment

It is a truism that the severity of the illness of patients with acute streptococcal infections varies enormously. Mr. Layton, whose experience of acute haemolytic streptococcal infections in the specific fevers is unrivalled, states that he is unable to recognize clinically the cases that are not due to haemolytic streptococci. The patient may suffer from an acute otitis media which clears up in ten days, or may suddenly become acutely ill with what must be an acute toxæmia, or even septicaemia, of which the acute otitis and mastoiditis is only one manifestation.

During epidemics of influenza one has seen patients die in two or three days from meningitis secondary to ear disease. On one occasion a nurse was attacked by acute streptococcal infection, with acute otitis media and mastoiditis as the early sign; she then developed acute pericarditis, thrombosis of a mesenteric artery, and died in less than a fortnight. At the other end of the scale the patient is scarcely ill. A mild acute otitis media develops, the mastoid antrum and cells become infected without acute symptoms and, when operation is subsequently performed, recovery follows without causing the surgeon a moment's anxiety.

I do not intend to bore you by discussing in detail the treatment of acute otitis media and its accompanying mastoiditis. In spite of the tendency to leave alone the red membrane of otitis media, I am still in favour of incision in all cases of pain and obvious general bulging of the membrane if the temperature is high. In cases of general bulging there would seem to be a tendency for the occurrence of those very large perforations often accompanied by considerable deafness. Many cases of apparently mild otitis media under expectant treatment subside so far as the acute inflammation is concerned, but sow seeds of deafness which only becomes evident some years later.

I am not going to discuss when to operate on the mastoid cells, except to suggest that no uncomplicated case of acute mastoiditis ever required a midnight operation, and that this particularly applies to those acute toxæmic (? influenzal epidemic) cases. It is far better in these cases to tackle the patient instead of the ear, the procedure being to take cultures of the serosanguineous fluid, to give the appropriate serum, to take a blood count, and even to group the blood with a view to possible blood transfusion. The public has at last begun to realize that blood transfusion is not a last hope.

In the following case serum injection was given with advantage in a very acute streptococcal otitis media.

A boy was sent home from school because he had had a severe sore throat. He arrived at 3 p.m. feeling well, but half an hour later had acute earache. Two to three hours subsequently the boy was suffering from very severe pain, and the membrane was already red and bulging markedly. Incision released serosanguineous fluid under much pressure; there was already marked tenderness over the mastoid, and the temperature was 103° F. The same evening severe epistaxis occurred, necessitating plugging, and the boy felt very ill. The amount of discharge from the ear was so profuse that large pads of wool were soaked through quickly, and the pads had to be changed frequently. Professor Eyre saw the boy, and gave anti-scarlatiniiform serum. This profuse discharge continued with high temperature and much malaise; on the third day the discharge began to be slightly purulent, and the mastoid operation was performed. Recovery was slow but uneventful.

Sometimes a blood transfusion may cure the patient without operation, even though tenderness is present. The following case is an example:

A boy, aged 14, had a so-called "Brighton" throat in 1931, with severe epistaxis, and headache over the right eye; he was very pale and the temperature rose to 102°. Pain in the right ear followed. The membrane was red and bulging, and already there was tenderness over the mastoid. On incision of the membrane a serosanguineous discharge escaped. In view of the boy's poor general condition and considerable loss of blood, a blood transfusion was given. Recovery was dramatic; in two days all tenderness disappeared.

Blood transfusions and pentnucleotide injections mark a great advance in the treatment of severely ill cases of infection; I have seen recovery accelerated and life saved by both procedures. Actually I am wondering whether pentnucleotide alone would not raise some patients' resistance so much as to avoid the transfusion.

Case 1.—A young man suffered from a mucopurulent discharge from the right ear for three months. A simple mastoid operation was performed, and pus was found throughout. The wound healed, but the ear continued to discharge. A further operation was performed, and small cells were found in the root of the zygoma; A few days later the

temperature rose, and the patient became ill. Cultures from the blood produced a profuse growth of streptococci. A blood transfusion was given. Two days later there were streptococci in the blood, and after another two days streptococci were still recovered from the blood, but they were much fewer. The third culture of the blood produced no streptococci, but the white cells were only 4,000 per c.mm. There followed a third blood transfusion and a dose of pentnucleotide; and thereafter there was slow but steady recovery.

Case 2.—A boy, aged 11, had an acute mastoiditis for which operation was performed. Although the wound appeared satisfactory, and the ear discharge ceased, the boy complained of severe pain over the homolateral eye and temple. This headache continued at intervals for four weeks; it was so severe that he lay with his hand pressed on the forehead and temple and begged his mother to keep her hand pressed there too. Dr. Riddoch saw the boy and transferred him to London, where I was asked to see him. After discussion it was suggested that a condition of petrositis was the cause of the severe pain. The previous wound was opened, and pus was found in a cell at the apex of the petrous, from which a considerable amount of creamy pus flowed. The boy was still ill, and the blood count showed only white cells. Blood transfusion was performed by Dr. Knott, and a small dose of pentnucleotide was given. The improvement in twenty-four hours was dramatic, and a perfectly smooth recovery followed.

ACUTE STREPTOCOCCAL INFECTIONS OF THE THROAT*

BY

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Acute streptococcal infection of the pharynx and larynx was known forty years ago as a number of different conditions. It was called acute septic inflammation of the pharynx and larynx; phlegmon of the pharynx and larynx; acute inflammatory oedema; erysipelas of these structures; and finally, phlegmonous cellulitis of the neck—that is, Ludwig's angina.¹ In 1895 Felix Semon read a paper at the Royal Medical and Chirurgical Society in which he argued that all these various forms of acute septic inflammation of the throat should be considered as varying degrees of virulence of one and the same pathological process. There is a great variation in the clinical signs and symptoms depending upon the severity and type of the infection, the resistance of the patient, and the situation of the lesion.

For clinical purposes two chief degrees of inflammation are recognized:

1. Tonsillitis, with considerable variation in the severity and in the extension of the inflammation and oedema to surrounding tissues.
2. A gangrenous inflammation, with a sloughing membranous exudate and toxæmic and septicaemic symptoms which often begins with acute tonsillitis, and is a later stage of the more severe types of the first group.

Symptomatology

There are many cases between these two somewhat extreme types. Tonsillitis is more common in children and young subjects. The septicaemic and virulent infection of the pharynx occurs in elderly patients, often as an intercurrent disease in such conditions as nephritis or chronic alcoholism. In the fulminating virulent cases the oedema spreads from the tonsils to the fauces and uvula, then down the lateral wall of the pharynx to the epiglottis and arytenoids. When the epiglottis or arytenoids are involved the pain and difficulty in swallowing

are markedly increased, so much so that there is almost complete inability to swallow even saliva. In a very large proportion of cases the oedema is localized to the tonsils. A primary streptococcal laryngitis is rare, and is generally part of a pneumonia or is a direct extension from the pharynx. The epiglottis and arytenoids and soft tissues of the larynx become oedematous, and acute laryngeal obstruction may be expected. Alcoholic patients appear to be susceptible to this type of infection. Cervical adenitis is always present, and varies with the virulence of the infection. The more virulent cases show considerable swelling of the neck, Ludwig's angina, and may go on to suppuration. Though extensive adenitis means a severe infection, it also indicates a localization of the inflammation, and a general septicaemia is not so likely to arise. It has been noticed that when the tonsils have been enucleated, even some years before, the inflammation of the pharynx and the adenitis are more marked. Suppuration appears to be more frequent, but extensive adenitis occurs when the tonsils are present or absent.

Aetiology

The most common causal organism is the *Streptococcus haemolyticus*, which is found in the swab from the throat and in the severe septicaemic cases can be cultured from the blood. Streptococci and pneumococci are often found together in the normal throat, and both these organisms will often be present in septic conditions of the throat, so that it may be impossible to say which is the causal organism and which is the secondary invader, or there may be a mixed infection. R. G. Henderson has given a very useful account and summary of eleven cases of acute pneumococcal pharyngitis. I cannot do better than quote extensively from his paper published in the *Lancet*.³ The pneumococcal case can be recognized clinically, but it requires the bacteriologist to prove the infection, and the co-operation of the bacteriologist is essential when serum is to be given. The typical pneumococcal case is of rapid and sudden onset, sometimes with a rigor. The patient looks and feels very ill, and has a high temperature and rapid pulse. The early oedema and redness of the throat is followed by a greyish-yellow membranous exudate extending on to the pharyngeal wall and uvula. Pain and the resulting dysphagia are very marked; the cervical lymphatic glands are tender and swollen. In the

* Read in opening a discussion in the Section of Oto-rhino-laryngology at the Annual Meeting of the British Medical Association, Oxford, 1936.