formed are nearly always fatal, in spite of the relief of the laryngeal obstruction. Every effort should be made to avoid tracheotomy, but delay is sometimes dangerous.

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THE HAEMOLYTIC STREPTOCOCCUS: WITH SPECIAL REFERENCE TO SCARLET FEVER*

BY

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I appreciate with real sincerity the honour of your invitation to open this important discussion, for you know that I have nothing new to tell you; I can claim to know nothing of the subject which is not better known to many of you, and very little which is not known to all. So the fruitfulness of this meeting will rest not upon what I say but upon what you say, and my task is the easy one of getting you going.

We are about to discuss one of the most important subjects in medicine, for to the European the streptococcus is public enemy No. 1, and deep down in the minds of all is the conviction that we ought to be able to circumvent it. We have given up the idea of exterminating, or incarcerating it, but we believe, with much reason for our belief, that we can manage to make it live amongst us as a respectable citizen.

Forty years ago I gained a prize for an essay on the streptococcus which covered a full sheet of foolscap! Since then knowledge of the varieties and attributes of this organism has grown so much that in the *Annals of the Pickett-Thomson Laboratory* it commands eight large volumes; the literature dealing with it and what it does to man would fill a respectable library. But recently those streptococci which interest us have been found to be of more manageable proportions, and from Dr. Dora Colebrook's report, which is the latest word on the subject, we can reduce the varieties of streptococcus important to human pathology to some dozen or so.

Although during the past fifty years there has been a dramatic decline in the prevalence and fatality of the acute diseases, the ravages of the streptococcus have increased. Alone of all the important causes of death, fatal streptococcal infection shows an increase which is exalted on standardization. But it is not only as a lethal agent that the streptococcus is important to us; it is the commonest of all causes of temporary inconvenience, and also of permanent defection from normal health. Probably nobody in temperate climes passes through life without paying some tribute to the great enemy; none of us knows when he may next be called upon for a contribution, the amount of which cannot be foreseen. We in this room can have some assurance that we shall not suffer from most of the diseases caused by parasites, but from those due to the streptococcus we have none, yet it is in the minds of all of you, as it is in mine, that this ought not to be; that the

enemy is not so elusive that it is beyond our powers to keep it at bay. So far we have failed, but our failure has been by no means complete. In many ways what has disappointed us opens up new hopes of an eventual success greater than any we have visualized in our limited endeavours of the past.

Varieties of Infection

The diseases caused by the streptococcus fall into two groups: (1) the three notifiable diseases, scarlet fever, erysipelas, and puerperal fever; and (2) a miscellaneous group embracing almost every form of sickness from which we suffer. But multifarious as are the manifestations of streptococcal infection, they are all so similar fundamentally that the clinicians preceded the bacteriologists in separating them from sicknesses caused by other parasites. Any and all of the forms of disease dependent upon streptococcal infection can be caused by any or all of the varieties of the parasite known to be pathogenic to man, though in certain types of lesion certain varieties of parasite are more frequent than others. From this it would seem that our attack upon the streptococcus must be generalized, though without doubt we must employ many different arms to win the battle.

The reservoir of the haemolytic streptococci pathogenic to man is the human nasopharynx; elsewhere it is found only in lesions caused by it. Epiphytic streptococci found on other parts of our surface are not pathogenic, nor are streptococci from extra-human sources, unless these have been recently distributed from a human carrier. The pathogenic varieties are found in from 1 per cent. to 100 per cent. of healthy pharynges according to season, period, and locality. It is questionable whether they are more frequent in unhealthy or convalescent throats than in healthy throats. They can, of course, always be found in the throats of scarlet fever and streptococcal tonsillitis patients, and for a short time after the reaction has subsided, but I doubt if true carriers are more common in those who have reacted than in those who have not. By a healthy throat I mean one which functions sufficiently to enable its possessor to get about his business without knowing that he possesses a throat, unless he thinks about it. In the prevention of massive infection, three things stand out clearly—the use of masks in surgical operations, even of a trivial character, and in midwifery; the relative isolation of those likely to be discharging the parasites profusely; and the adequate spacing of all sick persons, especially of those suffering from sicknesses in which an added streptococcal infection is most to be feared. These are old measures, sound in theory, and of proved efficiency in experience.

The virulence of the pathogenic streptococci appears to be exalted by passage through a host for other hosts similarly circumstanced. This is most obvious in localized outbreaks of puerperal fever and erysipelas, but is seen in all streptococcal diseases—for example, in peritonitis following clean operations. This exalted virulence seems to be specific. It is evidenced by shortening of the incubation period, rapid invasion of the tissues, and heightened fatality. In outbreaks of puerperal fever the virulence is enhanced mainly, if not exclusively, for puerperal women, not for their attendants.

During the last few years I have had the opportunity of studying the interaction of scarlet fever and the puerperium and have formed the following conclusions—at present held tentatively.

1. Parturient women are just as likely to suffer from scarlet fever as are non-parturient women.

2. If the implantation is at the normal site—the throat or nose—both scarlet fever and puerperium run their normal courses.

* Read in opening a discussion in the Section of Public Medicine at the Annual Meeting of the British Medical Association. Oxford, 1936.

3. If the site of implantation is the genital canal the result is serious, for metritis, pelvic inflammation and/or septicaemia may ensue. The explanation of this is simple. If foreign strains of streptococci are implanted on the throat, they meet with structures with an inherited potential to react to them; if they are implanted on a site to which normally they do not wander, there is no such protection. Septic metritis of the delivered uterus is a much more serious matter than tonsillitis, and pelvic cellulitis than cervical adenitis, although pathologically they are essentially similar. Moreover, during or immediately after delivery there is recent trauma, allowing direct entry into the blood stream of any organisms implanted on the surface. Yet surgical scarlet fever due to implantation of the streptococcus upon wounds and burns is generally trivial.

Scarlet Fever

Of the diseases of man caused by streptococci, the most frequent, but the least serious, is scarlet fever. During recent years the notifications of this disease have been about one-fifth as numerous as the registered live births. Since many cases are not notified because they are not detected, we shall probably not be in error in stating that one person in four goes through an attack of clinical scarlet fever. The damage done by scarlet fever is great, but, considering that about 100,000 cases of scarlet fever are reported yearly in England and Wales, it is relatively surprisingly small. It is fourteen years since 1,000 deaths attributable directly to scarlet fever occurred in one year; in 1934, the year of highest mortality since 1923, the total deaths were 963. A fatality rate above 1 per cent. is now quite uncommon, but this does not include fatality from otitis, nephritis, and carditis which kill remotely but more frequently than does the parent disease in its acute stage.

So far as we can judge from our records, the incidence of scarlet fever, though it varies much from year to year, has not varied from period to period, but its fatality has altered almost sensationally during the lifetime of our more elderly members. At the end of the last century, fatalities of 10 per cent. were not uncommon, and a generation before that, the disease was even more fatal. We should be inclined to look upon the reported fatalities of former times as biased from failure of notification of mild cases were it not that a fatality rate of 10 per cent. is still found in South-Eastern Europe, and the septic and toxic cases which were frequent in the past are now seen but seldom. What has caused the fall in scarlet fever fatality we do not know. Charles Chapin of Providence believes that it has come about from hospitalization of the severe cases producing a kind of eugenic breeding out of streptococci unfavourable to man; but if this were so, why should the loss of fatality be limited to scarlet fever? It is not shared by other streptococcal diseases.

The geographical distribution of scarlet fever is interesting. It becomes progressively rarer as we approach the equator. At Nairobi, in Kenya, there have been but three notifications of scarlet fever in the history of the municipality, and elsewhere in the tropics the disease is negligible. It is almost, but not quite, limited to Europeans, a phenomenon the reverse of what we should expect in a white man's disease introduced into native populations. This comparative immunity of the dark-skinned races is not limited to scarlet fever, but extends to all streptococcal diseases. Puerperal septicaemia is hardly more common in India, or Africa, than it is in England, though if English women were confined as are their Indian sisters we should expect a very high fever rate.

Since scarlet fever may be caused by any type of pathogenic streptococcus, and these types produce different antigens which call forth different antibodies, we should

expect scarlet fever to be a recurrent disease. As you know, it is not, although of all the diseases which were formerly called zymotic, it is the most likely to recur, and the relapses which we see not infrequently are suspiciously like new infections. It is, however, only in the rash phenomenon that scarlet fever is not recurrent. Streptococcal tonsillitis, liable to be followed by complications identical with those of scarlet fever itself, is recurrent, and the diagnosis of this from scarlet fever without eruption is to make a distinction without a difference. Although scarlet fever has fallen greatly in fatality and, in spite of some local threats, shows no sign of becoming more lethal, it has not altered in type; there is no complication which occurred in the old days which is not seen to-day; there is nothing seen to-day which was not seen by our fathers.

Does an attack of scarlet fever confer any immunity against other streptococcal diseases? Presumably it should give fairly good protection from future assaults of the type of streptococcus which caused it; those who believe in the existence of non-specific antibodies would surely expect complete immunity to one type of streptococcus to give something valuable in resistance to other types nearly akin? This is a point not for philosophical speculation, but for deduction from clinical experience. For many years I have been building up life histories of all citizens in the town in which I work, one object of which is to obtain evidence of the influence of past disease upon the future life of the recoverer. Obviously I can never hope to answer the questions at issue, but they may be answered in course of time. The evidence so far available suggests that an attack of definite scarlet fever does do something to protect against future invasions of streptococci.

Though we do not regard scarlet fever as a specific disease, it has many of the clinical features of specific infections, and superficially appears to have their epidemiological characteristics also, for scarlet fever appears in periodic epidemics much resembling those of measles, small-pox, or typhoid fever. But if one has the opportunity of watching a population through an epidemic period, one finds that notified scarlet fever is not the whole, but a part of a more extensive biological event. It is well known that epidemics of scarlet fever are frequently accompanied by an excessive prevalence of tonsillitis, but evidence of exalted activity of the streptococcus can be found in other directions, although, since other streptococcal diseases are rare in comparison with sore throats, their unusual prevalence will not be obvious unless it is sought, and in few districts will the medical officer of health be in a position to estimate the local and topical variations in the prevalence of non-notifiable streptococcal diseases.

Whilst I have observed that in times of epidemic prevalence of scarlet fever other streptococcal diseases are dominant also, I have observed the converse, namely, that when the epidemic ceases a period of comparative freedom from streptococcal diseases of all varieties follows. I admit that, even if the observation is accurate, its explanation may be not a temporary exaltation of immunity, but a temporary scarcity of streptococci. For we know that the supply of the parasites in their natural reservoir—the human throat—is subject to periodical variations.

What is the connexion between scarlet fever and acute rheumatism? I put forward the proposition that acute rheumatism—which is badly named, for it is seldom acute and rarely rheumatism—cannot be separated from scarlet fever; they are both different manifestations of the same biological process. So I expect it to be found eventually that strains of haemolytic streptococci identical with those which cause scarlet fever are the causal parasites of acute rheumatism.

Erysipelas

Erysipelas is one of the few diseases that we can recognize in the Hippocratic collection, and it appears to have varied little in historic times. It was made notifiable in England and Wales by the Act of 1889, and since then has been notified as accurately as any disease. Its incidence is extraordinarily even; in recent years it has varied from 13,000 to 20,000 cases per annum, giving an incidence of about 0.35 per 1,000. But its fatality has been increasing, the deaths in 1934 being nearly double those in 1924. In 1926 and 1933 there were 14,581 and 14,515 notifications, respectively, but the deaths in the former year were 807, in the latter 1,191. Part of this increased fatality disappears on standardization, but not all of it, so we must acknowledge that improvement in treatment has lost in the struggle with increasing malignancy.

The same may be true of the much-discussed puerperal fever. This again can be recognized in the Hippocratic collection, and appears also never to have been different from what it is to-day. Though compulsorily notifiable since 1889, notification until quite recently was totally unreliable, but the mortality from the disease has remained constant. Speaking with great reserve from my own experience, I think its incidence has increased during the past forty years, but its fatality has declined, leaving the mortality constant. We seldom see epidemics of this disease to-day, but I think the sporadic cases are more numerous than they were.

Other Streptococcal Lesions

Of the less well-defined forms of streptococcal disease we cannot speak very definitely, for until recently infections of wounds and surgical operations and acute inflammations of the throat and respiratory tract were rarely bacteriologically differentiated, so we do not know if these septic processes were in the past similar in parasitic causation to what they are to-day. Streptococci can be found in most lesions, for there are numerous types which are universally epiphytic on man, but, since only a few types are pathogenic, the mere presence of streptococci tells us little or nothing. Streptococci which are avirulent can be recovered from any catarrhal and most healthy surfaces. They may wander into wounds and sinuses, and towards the end of life even into the blood stream; so we must not call all disease processes streptococcal because streptococci can be recovered from them. In this, as in so much else in medicine, the greatest misleader of the physician is slipshod clinical pathology.

Immunization and Treatment

Faced with a disease which is not only common and formidable but which appears to be winning the race against our efforts to stem it, we naturally look about for some new or improved means of attack. In accordance with the trend of medicine during the past fifty years, we first look to the laboratory for help. Recently the bacteriologists have reduced the numbers of varieties of the streptococcus important to human pathology to about a dozen. The feasibility of conferring artificial immunity to this group is therefore promising. But, unfortunately, immunity to streptococcal disease is of a very different order from that to toxæmias, to virus diseases, and to those dependent upon bacillary invasion.

It seems that immunity to the streptococcus is largely local, and strains innocuous at a site where the host has previously reacted to them may be dangerous at a fresh site. This makes things awkward. Immunization against scarlet fever has been practised for many years with some success, but the immunity conferred appears to be limited

to the toxæmia of scarlet fever, and lacks proof of influence upon the liability to other streptococcal diseases. Passive immunity to toxæmia and also the abolition of toxæmia should it occur can be obtained by anti-scarlet serum, but evidence that this passive immunity protects against streptococcal invasion and septicaemia, which are the real dangers, is not encouraging.

Anti-scarlet-fever serum is used as a prophylactic against puerperal fever, but most of us are sceptical of its value. In treatment, anti-scarlet serum has superseded other streptococcal antitoxins, a point of some significance. Its use is now fairly general in scarlet fever, though there is much variation in its exhibition. In erysipelas and puerperal fever it is not so popular, but many of us try it with high hopes which are not always disappointed. In surgery and general medicine its use is irregular, but it might be used more for tonsillitis, in which disease its action is often striking. How far it has been employed by others for acute rheumatism I do not know, but recently I have used it for rheumatic carditis and other complications of child rheumatism with results that have decided me to persevere. Generally this serum seems to counteract toxæmia, but it is without influence upon invasion, which is the chief danger of streptococcal infection. In uncomplicated scarlet fever its action is quite dramatic, but there is much doubt whether it does, or does not, influence the complication rates. It undoubtedly cures all streptococcal diseases which will get well of themselves, but whether it can avert fatality is uncertain, though we believe that it does so occasionally.

From what I have said you will not accuse me of gross optimism, but I believe that if we use our wits and our energy we shall conquer the streptococcus, although the conquest will neither be speedy nor dramatic. The matter calls for much patient inquiry and intelligent and honest trial in the field of the products of the laboratory. May I recall to those of my own age what happened rather more than forty years ago when the two greatest remedies of modern medicine—diphtheria antitoxin and tuberculin—were discovered? Did we not all believe that what was given to us against diphtheria was the beginning of the end of the dangers of parasitic disease? Let us avoid this mistake in future, if we can. When anti-streptococcal serum was introduced, many believed that it would do for scarlet fever what diphtheria antitoxin did for diphtheria; but surely such hope was illogical. For pure toxæmic diseases, such as diphtheria and tetanus, an antitoxin may be expected to be curative, if given early, but against a parasite which invades the tissues no such action can be expected; for although we hear much of biological agents alleged to have anti-bacterial powers, their use in the field, if it does not discourage us, tells us that we are still a long way from the possession of such remedies. Active immunization is not very promising, owing to the local character of streptococcal immunity; but something may come of it, for if it is the invading property of the streptococcus which makes it so grave a menace, it is its toxins which kill, and immunity to these toxins is within hope.

The body possesses great powers to rid itself of streptococci which invade it. Recovery from septicaemia is not rare, and one form of post-scarlatinal haematuria in which masses of streptococci are discharged by the kidney without setting up disease in that situation is suggestive of great powers of riddance. Attempts to kill streptococci in the tissues, or in the blood, by chemical agents are not very hopeful. The most promising were the arsenical compounds, but the damage these do to the leucocytes overbalances any direct lethal action they have on the cocci; so they have fallen from favour. Personally I believe that these and similar remedies have no future, but I hope I am mistaken.

Prophylaxis

About the prevention of streptococcal implantation little can be said which is not common knowledge. The prevention of scarlet fever by isolation of the sufferers is the most magnificent failure of preventive medicine, but the isolation of the non-infected has been an equally magnificent success, for this is nothing but Listerian surgery carried to its logical conclusion. The surgeon sometimes fails, not through his fault or that of his dressers or nurses, but from the peculiar character of his enemy, which can lie dormant in places immune to its action, but from which places it wanders to new sites which are damaged and cannot resist its attentions.

The removal of septic foci seems a logical remedy for this danger, but doubts have arisen of its soundness and there are probably better ways to be discovered of overcoming this persistent menace to all of us. That we shall ever prevent pathogenic streptococcus from living parasitically upon us is, I think, unlikely, but that we shall

discover means of rendering this biological union harmless can be looked for with some confidence. The key to it will probably be found in the study of scarlet fever.

Before I close, I would make two remarks to stimulate the bacteriologists present to take part in the discussion. The question of the type of the streptococcus is of profound importance, but there seems to be much difficulty in its determination. Formerly, when desiring the opinion of a bacteriologist upon types of streptococcus, I sent cultures on similar media taken at the same time, in similar conditions, to four bacteriologists of unimpeachable veracity. No two reports were ever in complete agreement.

I mentioned a belief that the passage of a streptococcus through a host exalted its virulence for similar hosts. I instanced the progress of epidemic puerperal fever. The fact—for I have been told that it is a fact—that these streptococci alleged to be exalted in virulence for puerperal women do not show exaltation of virulence for male guinea-pigs has nothing to do with the case.

TRAUMATIC OEDEMA

A PITFALL IN PHYSICAL MEDICINE

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Physiotherapy, largely on account of its unique flexibility of application, has at last received tardy recognition, and now takes its place as an important branch of medical treatment. There are, however, certain disease processes which at first sight would appear of the type likely to benefit from such methods but which fail to respond as expected. I have recently encountered in hospital and private practice such a group, and after floundering in a state of considerable uncertainty for some time I arrived at certain conclusions regarding their appropriate treatment as a result of the experience thus obtained.

To those whose work brings them into regular contact with industrial compensation cases the development of oedema after injury to an extremity offers great difficulty, and it is important that the peculiar problems of true traumatic oedema in regard to its treatment, expected course, and prognosis should be fully understood. My main object has been to discuss the various methods of treatment and their influence upon the development and prognosis of the disease. A summary of present knowledge of the aetiology and pathology has also been included, and an attempt has been made to review the various theories of causation in the light of such knowledge. The following cases will serve as illustrations.

Case I

A quarryman, aged 32, on April 16th, 1934, sustained a severe crushing injury to the left foot from a fall of stone. There was moderate shock, bruising of the lumbo-sacral region, pulping of the great toe with protrusion of bone, and there were two fractures of the terminal phalanx.

For seven weeks before admission the foot was immobilized in splints, but the patient was allowed to sit up for short periods daily after the third day. The oedema diminished but did not disappear. After a further fifteen weeks on crutches the oedema gradually increased and pain became severe.

He was seen in the out-patient department of this hospital on October 3rd, five months after the original injury. The limb was grossly oedematous almost to the knee, and the skin was cyanosed, glossy, and tender to pressure; he was unable to put his foot to the ground.

X-ray findings: "Patchy areas of osteoporosis contrast with intense sclerosis, particularly in the metatarsal heads."

TREATMENT

He was treated for one month with local whirlpool baths, alone and with massage, contrast baths, and underwater douches. The pain was intensified after each treatment, especially after massage, and the oedema increased until the patient was unwilling to submit to further treatment along these lines. On November 3rd the limb was immobilized in plaster-of-Paris from toe to knee for one month. The patient was advised to rest with the limb elevated.

On November 30th, when the plaster was removed, the oedema had diminished considerably and there was no pain. The x-ray report was: "Decalcification proceeding; areas of porosis now predominating; sclerosis disappearing." Massage was instituted and was followed by the immediate return of pain and oedema. After three days plaster was reapplied for three weeks. On December 22nd the plaster was removed again, the oedema and pain being greatly improved; elastoplast was substituted and rest and elevation advised, but the patient was permitted to move about the house. The x-ray report was: "Decalcification now excessive; mottled appearance still evident."

Very slow progress was maintained for the next twelve months. With the object of speeding up recovery he was referred for operation, but the surgeon considered progress satisfactory under the conservative treatment and operation therefore unjustifiable. All attempts to hasten progress by physiotherapy invariably ended in failure and temporary relapse, the patient finally refusing further experiments in this direction.

The foot is now shrunken, mobility is reduced at all joints, and there is severe pain on attempting to increase forcibly the range of movement. The skin is still cyanosed and cold. He complains, too, that the pain is worse in cold weather. Several efforts to find him a light job have ended in failure. Deterioration in his morale is now obvious, the unfortunate result of prolonged absence from regular work and the limitations imposed by living in a small village.

The report on an x-ray taken in October, 1935, is: "General decalcification, mottled appearance disappearing; some increase of density in the shadow cast by peri-articular soft structures."

Case II

The lady superintendent of a rest home, aged 58, on July 29th, 1934, sustained a Colles fracture of the left wrist from a fall while climbing; there was little shock but much deformity. The wrist was immediately immersed in cold water for half an hour. It was then reduced without anaesthesia, and splints were applied. The position was modified the next day under gas. X-ray showed the fragments in satisfactory alignment after the second reduction. Thereafter she resumed her normal duties with the wrist in splints and a sling. After a week daily passive movements were started with massage. The swelling, after diminishing during the first week, rapidly increased and the pain became worse.