That a gland could make such an impression on the trachea as I have described is evident to me from a case which I attended this year. I was called one night to see

CASE XIV.—YOUTH. Slight tonsillar congestion; glands enlarged three days; stridulous breathing two days; intermittent irregular beat.

a boy, aged 15, with urgent dyspnoea. No air could be heard entering the right lung; the heart was labouring heavily, and the right side so distended that it could be detected by palpation and percussion 2½ in. to the right of the mid-sternal line. There were no other signs or symptoms. Hypodermic injections of strychnine, the inhalation of oxygen, and the application of poultices relieved him. Twenty-four hours later the dyspnoea suddenly came on more urgently than before, and artificial respiration only staved off the end for an hour. Dr. Kirkland saw the boy with me during life, and also attended the *post-mortem* examination next day. We found the right heart extremely engorged; the middle and lower lobes of the right lung were quite black with engorgement, and a fragment sank in water. There were some enlarged glands pressing on the right bronchus, which was flattened, contracted, and for the space of 1½ in. below the branch to the upper lobe intensely congested inside. Dr. Collins examined the engorged lung, and found it sterile. Whether this was another instance of the glandular infection we have been considering I do not know; but if W. M.'s gland on his trachea had been twice as swollen as it actually was I think he must have died, and that his end would have been even more speedy than was this boy's.

Pain in the occiput and back of the neck, and even tenderness on deep pressure in this region, were frequent, and as the retropharyngeal glands are so easily injected experimentally from any portion of the naso-pharynx and tonsillar region, I am inclined to think that the enlargement of these glands must be a constant accompaniment of this disease. If they were much involved the trouble would easily spread through the suboccipital triangle, and account for the symptoms described. The tonsil drains both directly into the upper cervical glands, especially the jugulo-facial, and indirectly into them through the submaxillary glands. The tonsillar inflammation, even when quite slight and superficial, extends over the pillars into the mouth and pharynx both very freely and very early, and the wide-spreading nature of this primary trouble is no doubt responsible for the wide-spread glandular infection which follows the onset of the disease. But the still more widely-spread glandular infection which is apt to follow later is no doubt due to the virulence of the poison spreading rapidly from gland to gland. This was shown by many of my cases, but by none quite so conspicuously as by that of W. M., whose glands were evidently infected through one another, for there was quite obviously no mischief in any of the areas, for instance, drained by the lymphatics of the humeral glands.

I have seen letters describing extensive epidemics of this disease in two or three towns in England. I have myself had cases who arrived in Cheltenham with the disease on them from three other towns in England, and medical friends, nurses, and laymen have told me of, or written to me about, epidemics or groups of cases occurring in fifteen

other towns of Great Britain and Ireland. I also know of cases in Belgium, France, Saxony, and America. Some of my correspondents may be mistaken, but it is highly improbable that all are.

A nurse wrote to me from a town in North America to say that there had been an epidemic in the boarding house in which she lived, and that thirteen patients had been ill with it. They had inflammation of the tonsils and adjacent structures, and swollen necks. They had been attended by two physicians, who called in a specialist, and one patient had her tonsil lanced seven times. There has evidently been an epidemic of a similar but much more serious nature in the United States, but I will not refer to that as Dr. Kirkland is describing it in detail.

The incubation period in my cases seemed to be three or four days.

The feature of this disease that most strikes an observer is the unusually large, unusually tender, and unusually numerous glands, so large and so tender that on more than one occasion both medical men and laymen have asked me the question, "Are you quite sure that this is not a case of oriental plague?"

But, though the swollen and tender glands are the features that at once arrest the observer's attention, yet the importance of this new disease lies in the insidious onset of cardiac lesions, which are in most cases slight; the dilatation, under rest, is temporary, but a murmur may persist. So devoid of premonitory symptoms is this complication, and so very widespread the disease, that one cannot help feeling that there must be at the present time a large number of unsuspected cases with cardiac lesions who are walking about and, by doing so, endangering the integrity of their hearts. We have to deal with a "silent endocarditis," not as a clinical curiosity, but as a prevalent disease.

I am not prepared to determine the nosology of this epidemic disorder. It is not mumps or rheumatism, nor septic tonsillitis; nor, as I was at first disposed to call it, for want of a better term, influenza; nor is it glandular fever, from which it differs by the absence of nephritis, except in some cases, and of enlargement of the spleen, liver, mesenteric, inguinal, and axillary glands, and by the presence in almost all cases of tonsillitis and pharyngitis, and in many of mild carditis or endocarditis. Moreover, adults are more frequently attacked than adolescents, and adolescents than children. I am disposed to think it is a new or hitherto unrecognized infectious disorder, and that it is caused by a streptococcus, probably a new variety.

EPIDEMIC CERVICAL ADENITIS WITH CARDIAC COMPLICATIONS.

By ROBERT KIRKLAND, M.B.

CONSULTING PHYSICIAN, CHELTENHAM GENERAL HOSPITAL.

In October, 1912, I saw, in consultation with Mr. Buckell, a boy aged about 14, with cardiac mischief quite unlike anything I had previously seen.

Some time before he had consulted Mr. Buckell on account of not feeling very fit. Mr. Buckell examined his chest and found nothing amiss. He told him to rest in bed; the temperature, taken morning and evening, was normal, and the pulse was quiet and regular. But in a few days a murmur appeared at the mitral orifice. It was then I saw him. The boy was healthy looking, there was no pallor, the tongue was clean, there was no definite evidence of throat trouble, no glandular swelling in the neck, no rash on body or limbs, no joint pains or swelling; no history of growing pains or past chorea. The pulse was 72, regular in volume and rhythm, the temperature was normal, there was no sweating, and there was nothing abnormal in the urine. The apex beat was in the normal position, but there was a soft systolic bruit at the apex conducted towards the axilla, and a slightly accentuated second pulmonary sound.

Sixteen days later Mr. Buckell again asked me to see the lad. The mischief now had extended from the mitral to the aortic valve, and we had the see-saw murmur of aortic obstruction and regurgitation. The slow smouldering endocardial fire had done this great damage without the boy feeling or looking ill, for his temperature all this time had reached 99° F. on one occasion only, and his pulse was never quick and always regular; there was no joint pain or swelling, and no sweats; there was no enlarged painful spleen or haematuria or emboliform phenomena on skin or in the lungs. We could call it an infective endocarditis, and there our knowledge ended, for Dr. Collins examined the blood and found it sterile.

Sir William Osler was consulted, who confirmed our diagnosis as far as it went. We thought the portal of entry for the infective agent must be the tonsils. It was not like a rheumatic infection; to call it so was simple begging the question.

On February 28th, 1913, I was asked by Dr. Longridge to see a little boy he thought was suffering from endocarditis. He had had a sore throat, with swollen and painful glands in the neck, and a slight temperature. The heart's apex was in the nipple line—the normal position in a boy of his age—but there was a systolic murmur at the apex, heard round towards the axilla, with a slightly accentuated pulmonary second sound.

Bearing these cases in mind I began to be on the outlook for a new infection, or for a new behaviour of an old infection, and in March I think I found the former. It seems to me to be a definite entity, for it breeds true.

The malady begins with malaise, sore throat, pain and stiffness in the neck, sometimes on one side sometimes on both, occasionally aching of the back and limbs. The glands of the neck are early affected.

In one of my early cases, within three days of the onset the glands on the right side of the neck were much swollen. These subsided, only to be followed by enormous swelling of the same glands on the left side. This became brawny, painful, and very tender, and in about a fortnight I detected fluctuation. On cutting down, under an anaesthetic, deep-seated pus was evacuated.

The first gland to be affected was usually that behind the posterior belly of the digastric muscle, then the glands in front, beneath, and behind the sterno-mastoid muscle. The appearance of the neck in some cases resembled mumps, but in no case was the parotid involved, nor was there severe epigastric pain and vomiting to indicate affection of the pancreas. In a few cases the swelling of the neck resembled bubonic plague, but never were the groin glands enlarged, always the neck. In three cases I know of suppuration of the glands occurred. The temperature ranged from 99° F. in slight cases to, in severe cases, 103° F., and on one occasion I believe to 106.6° F. The throat was sometimes red, glazy, and erysipelatous-looking; in other cases it looked like follicular tonsillitis, while yet in others there was a membrane indistinguishable from that of diphtheria except that it was perhaps whiter.

The period of incubation seemed to be about four days. It affected all ages, from 2 upwards. I should say that in about 10 per cent. of the cases I saw the myocardium or endocardium was affected, and this began usually about the end of the first week. As Dr. Pruen has pointed out, the heart was affected chiefly in those cases with slight glandular swelling.

In two undoubted cases of this illness the inflammatory mischief spread quietly, as in Mr. Buckell's case, from the mitral valve. And in one doubtful case we had a primary aortic affection, a systolic murmur being heard over the aorta, and low down on the left side of the sternum a diastolic bruit, with a reduplicated second sound, the bruit following the second of the two sounds. With Dr. Longridge I saw a lady suffering from this disease, whose heart and general condition pointed to malignant endocarditis, but there was no splenic enlargement or embolic phenomena. She ultimately recovered.

In my experience primary acute nephritis never followed; the infection, perhaps, in one or two cases lit up a fresh attack in old damaged kidneys. The prognosis as to life was good, except when there was high temperature, bronchopneumonia, or granular kidneys. None of the heart cases I saw died, but some still bear the effects of the myocardial or endocardial trouble.

Swabs taken from the throat revealed a streptococcus, and the blood examination, when it was positive, showed also a streptococcus, haemolytic in one case.

It may be said that all this sounds like scarlet or rheumatic fever, both considered by many to be also due to a streptococcus. But this new illness never began, in my experience, with initial vomiting; the tongue was never strawberry-looking, and afterwards raspberry-like when peeling was over, and no rash, as far as I know, occurred except in one fatal case, when an erysipelatous rash appeared on forearm and leg. There was no infra-clavicular formation of pinholes towards the end of the first week, and, as I have already said, nephritis was not a sequela. I was told it was most likely aberrant scarlet fever, but every case cannot be aberrant in a totality of hundreds.

It was not acute rheumatism, similar as the cardiac conditions were, for we never had in grown-up people the intensely painful blush-red swelling, flitting about from joint to joint, or the profuse acid, sour-smelling sweats. Moreover, in rheumatism we have not the typical neck glandular swelling, and it is not an infectious disease as we understand it.

It was not diphtheria, for in every case I had knowledge of, when a swab was taken from the throat, the bacteriologist's report was: "Streptococci present, no Klebs-Loeffler bacilli." The membrane in the throat in a few cases looked suspiciously like that of diphtheria, but repeated examinations in one such case brought back the same report—"No Klebs-Loeffler present." In cases in which suppuration occurred in the glands some may suspect diphtheria because the *Streptococcus pyogenes* is a frequent attendant of the Klebs-Loeffler bacillus. But the latter was never found, and in my own case, where suppuration occurred, there was no membrane on the throat. We had no characteristic ozaena, no palatal paralysis, strabismus, ptosis, or loss of power of accommodation; never was the larynx affected. The heart condition in diphtheria, with its very slow or very quick arrhythmic pulse and the physical signs of gallop rhythm, or embryocardia, probably due to a neuritis of the cardiac nerves, was quite unlike that of this disorder.

I have received the following letter from a friend—one of the ablest physicians in the south-west of England:

Many thanks for your interesting letter. I recognize the accuracy of your clinical description of these cases. I have seen a number of them and have been at a loss what to call them; in fact, one of my own relatives has just had a mild attack. The posterior wall of the pharynx was very red, glazed and painful, neck glands swollen, and aching in back and legs. No Klebs-Loeffler bacilli, but streptococci. I have also seen at least two cases of endocarditis of a subacute character following on. I think there is no doubt you have spotted a new infection.

At the International Medical Congress in August I made the acquaintance of Doctors Libman and Baehr, physicians to the Mount Sinai Hospital, New York, who had brought over with them about 50 specimens of subacute infective endocarditis, due to a streptococcal infection. The vegetations appeared first on the left auricular surface where the blood stream impinges, then on the mitral valve and chordae tendineae. In two cases aneurysm of the mitral valve resulted, which was well shown in the specimens, as was also the site of the vegetations. In other cases, from embolic streptococci being carried to the vasa vasorum or from infarcts lodging and then causing softening of the arterial walls, aneurysms, especially of the femoral, popliteal and superior mesenteric arteries, resulted. The kidney lesions in these cases were remarkable. The kidney resembled the large white kidney, without much roughening of the capsule, but with interstitial changes. Microscopically, several glomeruli could be seen to be embolized with adhesion of the glomerulus to Bowman's capsule through cell proliferation—a glomerular nephritis.

The symptoms were hectic temperature, anaemia, splenic enlargement and pains in the bones, especially over the sternum on percussion; this they assured me was a striking and well-marked phenomenon. When the auricular surface only was affected there were no bruits, but as the mitral or aortic valves became involved, the corresponding murmurs appeared.

Dr. Baehr told me that many of his cases were sent into hospital with the diagnosis of Banti's disease, the anaemia, cardiac bruits, and splenic enlargement suggesting this. But the liver was never enlarged to any extent. The symptoms of the kidney lesion were haematuria, then anasarca, later uraemia. The urine was albuminous, excessive in quantity, but of high specific gravity, never of a low specific gravity. This is remarkable and was distinctive of the illness. In some cases painful nodules occurred on the sides of the extremities of the fingers and on the thenar and hypothenar eminences. They lasted for from twelve to twenty-four hours. In some of the cases the bacteria disappeared and the vegetations on the auricular surface and valves smoothed and healed.

The illness lasted for from four to eighteen months. The majority of cases ended fatally; there were a few spontaneous recoveries, and a few recoveries were attributed to various forms of treatment. When death

occurred it was sometimes from profound anaemia, sometimes from embolism, but mostly from uraemia.

The microbe causing the endocarditis, isolated in 73 out of 75 cases, was almost always a streptococcus of a peculiar type, intermediate between the pneumococcus and the ordinary streptococci, to which Dr. Libman gave the name *Streptococcus mitis*. It was not the same as the rheumatic diplococcus of Poynton and Paine.

The streptococcal infection which I have tried to describe, and which I consider possesses definite features, does not, of course, resemble that of Dr. Libman and Dr. Baelr. But it is suggestive that in New York there should have been cases of subacute streptococcal endocarditis, and that in Cheltenham we should have many cases of streptococcal throat, a certain number of which developed endocarditis and myocarditis.

Within the last few months some observers who have had cases of sore throat followed by cardiac disease in children, have isolated a streptococcus which they call the *Streptococcus rheumaticus*. I have a shrewd suspicion that a goodly proportion of these cases come under our category. Our epidemic was certainly not acute rheumatism, for although in children it is possible to have cardiac trouble without muscular pains or joint manifestations, in adults this is rare.

The heart conditions in our cases were unlike malignant endocarditis. The heart became affected about the end of the first week, the signs being those of myocarditis or endocarditis. The latter was of a slow, smouldering type, followed sometimes by permanent damage to the valves; but in other cases, as far as we know at present, by recovery. We never saw embolic phenomena. In fact, what astonished me was to see patients developing cardiac disease under one's very eyes, placidly lying, not feeling at all ill, not anaemic, with a quiet pulse, and usually normal temperature. The pulse, however, shot up on the patient changing from the recumbent to the sitting posture. I feel sure that if the heart had not been examined no one would have suspected heart mischief.

The streptococcus responsible for this malady is certainly not the *Streptococcus mitis* of Dr. Libman. In a report of the Clinical Research Association on one of Dr. Pruen's cases the *Streptococcus salivarius* and *Streptococcus pyogenes* were found. The latter, of course, were met with in the cases where suppuration occurred in the neck gland.

Some of my colleagues suspected the milk supply as being the probable source of the outbreak. I cannot be sure of this, because my patients had milk from different supplies, and my first definite case of the illness had his own cows on his own estate, carefully tended and looked after, and the only other person to contract the disease was his agent, who had paid him a flying visit to discuss business matters with him when ill. Other patients had it, too, who drank only milk that had been boiled. Two patients also developed the illness immediately on their arrival here from Woolwich and Sheffield. Unquestionably milk is an excellent medium for the multiplication and growth of streptococci which might gain access to it from mastitis in cows, or from those handling the milk, suffering from the disease, as in some cases of scarlet fever epidemics.

THE tenth Congress of the German Roentgen Society will be held in Berlin in April (19th to 21st), under the presidency of Dr. Max Levy-Dorn. The secretary is Dr. Immelmann, Berlin W. 35, Lützowstrasse, 72. There will be an exhibition in connexion with the congress.

THE Board of Trade has addressed a letter to the Secretary of the Liverpool School of Tropical Medicine thanking it for arranging an exhibit illustrating the prevention and cure of tropical diseases at the International Exhibition at Ghent last year. The Board expresses its thanks to Professor J. W. W. Stephens, who represented the school on the committee of organization, and Professor R. Newstead, who collaborated with him in the preparation of the exhibits relating to malaria, sleeping sickness, yellow fever, and ankylostomiasis. The collections illustrating these diseases, the first of their kind to be contributed by this country to an international exhibition abroad, were among the most important features of the tropical diseases section of the exhibition. The Board hopes that it may be found possible to demonstrate even more fully on a future occasion the activities of British medical institutions in the field of tropical medicine.

CHRONICALLY ENLARGED TONSILS AND THEIR TREATMENT.

By W. J. HARRISON, M.B.,
NEWCASTLE-ON-TYNE.

Is the question whether these tonsils should be completely or partially removed, or treated by one of the many methods which are advocated, certain points require consideration.

Anatomy and Situation.

The tonsillar crypts, with rare exceptions, extend to the capsule, and when they become infected it is impossible to say to what depth the invasion extends. On removing tonsils apparently only enlarged, it is a common thing to squeeze out septic matter or to find one or more septic foci lying beneath the capsule. This occurs not only in definitely septic tonsils, but also in those which only appear enlarged. Partial removal is of little value in such a condition. The plicae semilunaris and triangularis play an important part in the retention of foreign substances and prevention of drainage of the crypts which they cover. They do not embrace a small tonsil very closely, but as it enlarges they become more closely applied to its surface and offer increasing obstruction to drainage. The crypts which are most frequently and severely diseased are those covered by the semilunar fold, and these are generally left when the tonsil is partially removed.

The situation of the tonsil renders it very liable to infection, being constantly bathed in septic fluid and exposed to injury, especially when enlarged, by hard particles of food. When enlarged it pushes the palatoglossus and palato-pharyngeus muscles apart and impedes their action. The movement of the soft palate being interfered with, the character and tone of the voice suffer. The posterior pillar of the fauces being pushed backwards narrows the sides of the oro-pharynx, and in some cases causes obstruction to the Eustachian orifice. Partial removal of the tonsil does little to relieve these conditions.

Function.

As far as is known, the principal function of the tonsil is to act as a barrier to the entrance of bacteria into the system. The healthy tonsil easily disposes of bacteria, and the viscid secretion which it manufactures retains bacteria which come in contact with it and destroys them by its bacteriolytic action. When, however, its resistance is lost, the tonsil becomes an ideal culture bed for bacteria.

The secretion (the first line of defence) is often dried up in mouth-breathers, and they are the class most frequently affected by tonsillar troubles. Another factor is their lowered vitality, in which the tonsil shares, due to imperfect preparation and assimilation of air for respiration and the lack of nasal stimulus to deep breathing.

As in other parts of the body, the lymphoid tissue of the tonsil produces lymphocytes, but if it is diseased or weakened it is doubtful if it can perform this function to any appreciable extent.

Masini advanced the theory that the tonsil has an internal secretion after the nature of the suprarenal gland, but this has not been confirmed.

Chronic Enlargement of the Tonsil.

Many theories have been advanced to account for this, and while in some enlargements other causes may be at work, the most reasonable explanation in most cases appears to be some local irritation. As mentioned above, the form and situation of the tonsil favour the entrance of bacteria, and it is not surprising to find enlargement in a gland so constantly exposed to infection, when one considers that enlargement in other glands is most commonly brought about by chronic irritation.

The course of events is briefly as follows: The tonsil becomes infected, and three things may happen: (1) It recovers completely; (2) it recovers, but with weakened resistance, which allows it to be from time to time reinfected; (3) some chronic irritation remains. In the two last named conditions the resulting hyperaemia brings about hypertrophy of the tonsil and later the formation of fibrous tissue. Examination of a number of