CONCLUSION.

The whole subject of syphilis has reached a stage of great interest and importance. For years the extraordinary clinical manifestations of the disease and the difficulty of obtaining a definite clue to its exact pathology engrossed the attention of observers of the highest powers of observation. Increasing attention was also given to the national importance of the disease as a factor in the production of degenerative processes resulting in serious individual inefficiency and in national physical deterioration. But until the pathology of the disease was placed on a definite and certain basis, all thought in connexion with it was more or less incoherent and action vacillating. To the genius of the lamented F. Schaudinn, in the view of practically every authority on the subject, are we indebted, by the discovery of the *Spirochaeta pallida* and of its rôle in the production of the disease, for an "open sesame" which will undoubtedly result in a coherent and definite conception of syphilis in all its aspects, biological, pathological, and clinical, and will thus clear the way for organized efforts, founded on definite knowledge of the etiology of the disease, for neutralizing its effects, and in some measure at least, minimizing its incidence. The great discovery of Wassermann—a discovery which, whatever the ultimate theoretical explanation of it may be, was undoubtedly based on the conception of syphilis as an infective disease—has opened the way for investigations into which very many workers in the field of biological research have seen sufficient inducement to enter, and new light is almost daily being thrown on the nature of the disease and of its manifestations. The method of complement deviation upon which this most important and far-reaching means for extending our knowledge of the nature of syphilis depends, is one which has proved highly fruitful in results in other fields of investigation, and engrosses a large amount of attention in both its theoretical and practical aspects. The conditions which lead to active or passive anticomplement action and the explanation of the action, the nature of complement, antibody, antigen, etc., and the meaning of their variations under different conditions are being studied with such vigour that our knowledge of them will undoubtedly soon be much more definite and certain. Our conception, indeed, of infective disease in general, and probably of many diseases not at present so regarded, will, in the not remote future, be so altered and enlarged that our present knowledge will seem rudimentary and crude. It is more and more becoming apparent that every man aspiring to practise medicine must in the future, if he wish to keep abreast of the advances, not only in the science, but in the art of medicine and surgery, have a very much fuller and more practical acquaintance than is at present considered necessary with the knowledge now available, as the result of an enormous amount of research and thought, of the mechanism and conditions of infective disease.

We desire to express our thanks to Dr. Hiller (Melbourne), Dr. McKelney, Dr. Margaret Harpur, Dr. Chapman, Dr. McMurray, Dr. Ludowici, and many others for the gift of material in connexion with the paper.

REFERENCES.

- ¹ Bordet: *Annales de l'Institut Pasteur*, 1900, vol. xiv.
- ² Bordet et Gengou: *Annales de l'Institut Pasteur*, 1901, vol. xv.
- ³ Gengou: *Annales de l'Institut Pasteur*, 1902, vol. xvi.
- ⁴ Malvoz: *Annales de Soc. Médico-Chir. de Liège*, 1901, vol. xl.
- ⁵ Moreschi: *Berl. klin. Woch.*, 1905.
- ⁶ Neisser and Sachs: *Berl. klin. Woch.*, 1905, p. 1388.
- ⁷ Wassermann and Bruck: *Medizin. Klinik*, 1905, No. 1; *Deut. med. Woch.*, 1906, No. 12.
- ⁸ Wassermann, Neisser, and Bruck: *Deut. med. Woch.*, 1906, No. 19.
- ⁹ Wassermann and Plaut: *Deut. med. Woch.*, 1906, No. 44.
- ¹⁰ Wassermann: *Deut. Zeitschrift f. Nervenheilk.*, December, 1908.
- ¹¹ Neisser, Bruck, and Schucht: *Deut. med. Woch.*, 1906, No. 48.
- ¹² Levaditi et Yamanouchi: *Comptes rend. Soc. de Biol., Paris*, 1907, T. lxiii, p. 740.
- ¹³ Levaditi et Yamanouchi: *Bull. de la Soc. de Path. Anat.*, T. i, 1908, No. 1.
- ¹⁴ Levaditi et Marie: *Annales de l'Institut Pasteur*, 1907, vol. xxi.
- ¹⁵ Levaditi et Marie: *Comptes rend. de Biol.*, 1907, No. 42; *Semaine médicale*, 1907, No. 21.
- ¹⁶ Sachs and Altmann: *Berl. klin. Woch.*, 1908, Nos. 10 and 14.
- ¹⁷ Weil: *Wiener klin. Woch.*, 1907, No. 18.
- ¹⁸ Bruck and Stern: *Deut. med. Woch.*, 1908, No. 12.
- ¹⁹ Muir and Martin: *Journ. of Hygiene*, 1906, vol. vi.
- ²⁰ Theobald Smith: Rockefeller Reprints, 1905.
- ²¹ Citron: Report XIV, Internat. Cong. Hygiene and Demography, Bd. iv, p. 101.
- ²² Preston Keyes: Rockefeller Reprints.
- ²³ Fornet: *Muench. med. Woch.*, 1907, No. 30.
- ²⁴ Bauer: *Deut. med. Woch.*, 1908 and 1909.
- ²⁵ Bauer.
- ²⁶ Blaschko: *Deut. med. Woch.*, 1909, No. 9.
- ²⁷ Lessar: *Deut. med. Woch.*, 1909, No. 9.
- ²⁸ Bauer and Meier: *Wien. klin. Woch.*, 1908, p. 1765.
- ²⁹ Fischer and Meier: *Deut. med. Woch.*, 1907, No. 52.
- ³⁰ Porges and Meier: *Berl. klin. Woch.*, 1907; *Berl. klin. Woch.*, 1908; *Wien. klin. Woch.*, 1908.
- ³¹ Noguchi: *Journ. Experim. Med.*, January, 1909.
- ³² Noguchi: *Journ. Experim. Med.*, March, 1909.
- ³³ Nonne: *Deutsch. Zeitsch. f. Nervenheilk.*, Bd. 36, Heft 1-2, 1908.
- ³⁴ Elias, Neubauer, Porges, and Salomon: *Wien. klin. Woch.*, 1908.
- ³⁵ McKenzie: *Journ. of Pathol.*, January, 1909.
- ³⁶ Henderson Smith: *Proc. Roy. Soc.*, July, 1907.
- ³⁷ Hiller: *Proc. Austral. Med. Con.*, 1908.
- ³⁸ Mainwaring: Rockefeller Reprints.
- ³⁹ Dreyer: *Journ. of Path.*, January, 1909.
- ⁴⁰ Kolle and Schatilloff: *Deut. med. Woch.*, 1908, No. 27.
- ⁴¹ Mellanby: *Journ. of Physiol.*, vol. xxxvi, Nos. 4 and 5.
- ⁴² Ehrlich and Morgenroth: *Berl. klin. Woch.*, 1899.
- ⁴³ Klausner: *Muench. med. Woch.*, 1908, No. 46.
- ⁴⁴ Michaelis.
- ⁴⁵ Tschernogubow: *Deut. med. Woch.*, 1909, No. 15.
- ⁴⁶ Hugo Hecht: *Wien. klin. Woch.*, 1908, No. 50.
- ⁴⁷ Mott: *BRITISH MEDICAL JOURNAL*, February, 1909.
- ⁴⁸ Maslokowitz and Liebermann: Quoted by Tschernogubow, loc. cit.
- ⁴⁹ Landsteiner, Müller, and Pötzl: *Wien. klin. Woch.*, 1907, No. 46.
- ⁵⁰ Browning and McKenzie: *Lancet*, May, 1909.
- ⁵¹ Browning and McKenzie: *Journ. of Path.*, January, 1909.
- ⁵² McIntosh, *Lancet*, May, 1909.
- ⁵³ Fleischmann: *Berl. klin. Woch.*, 1908, No. 10.
- ⁵⁴ Selemew: Cited by McIntosh, loc. cit.
- ⁵⁵ Schilling, Claus, and v. Hoesslin: *Deut. med. Woch.*, 1908.
- ⁵⁶ Harloch and Yakimoff: *Wien. klin. Woch.*, 1908, No. 21 and 40.
- ⁵⁷ Manteufel: *Arbeit. aus dem K. Gesundheitsamt, Berl.*, Bd. 28, 1908.
- ⁵⁸ Manteufel and Woithe: *Arbeit. aus dem K. Gesundheitsamt, Berl.*, Bd. 29, 1908.
- ⁵⁹ Butler: *New York Med. Journ.*, 1909.
- ⁶⁰ Peritz: *Deut. Zeit. f. Nervenheilk.*, 1908, Bd. 36.
- ⁶¹ Blumenthal: *Semaine médicale*, 1908, No. 12.
- ⁶² Kurt Meyer: *Deut. med. Woch.*, 1908, No. 20.
- ⁶³ Wechselmann: *Deut. med. Woch.*, 1909, No. 15.
- ⁶⁴ Benda: Quoted by Mott, loc. cit.
- ⁶⁵ Reuter and Schmorl: Quoted by Mott, loc. cit.
- ⁶⁶ Katzenstein: Quoted by Wechselmann, loc. cit.
- ⁶⁷ Murero: Quoted by Wechselmann, loc. cit.
- ⁶⁸ Furecz: *Med. Klin.*, 1907, No. 35.
- ⁶⁹ Ross and Jones: *BRITISH MEDICAL JOURNAL*, May, 1909.
- ⁷⁰ Fleming: *Lancet*, May, 1909.
- ⁷¹ Muir and Ritchie: *Bacteriology*, Fourth edition, p. 108.
- ⁷² See References 12 14 16 24 30 31 45 46 49 70.
- ⁷³ See References 23 30 32 33 34 43 49.
- ⁷⁴ Levaditi: *Comptes rendus de Biol.*, December, 1907.

NOTE ON ALCOHOL IN RELATION TO
MULTIPLE NEURITIS.

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NEARLY nine years have elapsed since the epidemic of multiple neuritis which occurred in Manchester and the surrounding district. It will be remembered that during the latter part of the year 1900 a great increase in the number of cases of multiple neuritis was noticed, and especially in persons who were drinking beer. The idea occurred to many medical men that there must be some unusual toxic substance present in the beer, but it is to my colleague, Dr. Reynolds, that the credit is due for first thinking of arsenic, and for first detecting its presence in the beer. His discovery was soon confirmed by others, and the matter was thoroughly investigated.

Before this epidemic it was assumed that the development of neuritis in an alcoholic subject was due to the action of alcohol on the peripheral nerves. Doubts as to the correctness of this assumption were raised by the discovery of arsenic in the beer, and by the views expressed by Dr. Reynolds. The epidemic drew attention to the importance of considering not only whether neuritis in a drinker might be due to the presence in his beverage of some poison other than alcohol, but also as to whether alcohol by itself could cause neuritis.

In his account of the epidemic Dr. Reynolds stated:

I have for many years doubted whether ethylic alcohol *per se* does cause peripheral neuritis at all, and I have personally felt more and more confirmed in this opinion each succeeding year.

Again, in responding to the discussion on his paper read at the Medico-Chirurgical Society he said:

I am willing to admit that alcoholic neuritis exists, but I am not convinced thereof.

He further threw out the suggestion that all cases of so-called alcoholic neuritis might be really due not to alcohol but to arsenic. More recently he has stated that he never meant to deny the existence of alcoholic neuritis, but his belief is that it is of rare occurrence and usually slight in degree.

Such statements were opposed to the generally accepted views on the subject, and it was obvious that subsequent experience was necessary before their correctness could be

estimated. Now, if arsenic were the chief cause of so-called alcoholic neuritis, we should expect to find a great diminution in the number of cases occurring after the epidemic, that is, when the beer was free from arsenic. On the contrary, the percentage of cases admitted to the Manchester Royal Infirmary since the epidemic is almost identical with that of cases admitted before the epidemic. Thus, if we take the seven years included by the years 1892-1898, there were 109 cases of peripheral neuritis out of 9,300 medical in-patients, that is, 1.01 per cent.; whilst in the seven years, 1901-1907, there were 116 cases out of 10,630, that is, 1.09 per cent. But in the year of the epidemic, namely, 1900, there were 62 cases out of 1,343, or 4.6 per cent.

Hence it is clear that, before and after the epidemic, arsenic, if present at all in the alcohol consumed, was in very small quantities. Further, it is to be noted that pigmentation, keratosis, and other skin lesions, so prevalent in the epidemic, were not mentioned in the reports of earlier cases by such acute observers as Dr. Ross and Dr. Dreschfeld. On the other hand, we must admit that the majority of Manchester cases occurred in beer drinkers, and that a small amount of arsenic in the beer may have been a contributory factor.

It is therefore desirable to consider the question of neuritis in drinkers of spirits. In this connexion we may note the valuable statistics of Dr. F. Buzzard. In the ten years included by the years 1891-1900, out of 6,458 cases admitted to the National Hospital there were 108 cases of alcoholic neuritis—that is, 1.6 per cent. Of these 108 cases, 25 were due to excess in spirits only, and only 1 case due to beer only. Dr. Buchan, also, in 19 cases of alcoholic neuritis found that whisky was the chief beverage in 18, and the only one in 6.

These results are in accordance with the records of the earliest observers of peripheral neuritis in alcoholic subjects, as witness the graphic account by Dr. Lettsom of the disease as occurring in those who drank brandy and gin in excess, and that by Dr. James Jackson, who attributed the disease to ardent spirits. They are also borne out by the statements of modern authorities on the subject. Thus Dr. T. Buzzard says his experience has been that neuritis is due especially to spirit drinking. The late Dr. Ross, in an analysis of 90 cases, found that the form of alcoholic beverage was as follows: In 22 spirits, in 3 absinthe and vermouth, in 13 beer and spirits, in 5 beer only, whilst in 47 the kind of alcohol was not given. Moreover, of recent years many cases of peripheral neuritis from spirit drinking have been observed in which beer could be definitely excluded. Dr. Williamson has recorded a case from excessive whisky drinking, no beer having been taken, in which the diagnosis was verified by a microscopical examination of the peripheral nerves. In this case three samples of the same whisky gave no reaction for arsenic with Reinsch's test.

Reverting to the question of beer drinking, I may mention that several cases of *well-marked* and *severe* multiple neuritis, which have come under my care since the epidemic, have been carefully investigated, and arsenic as an etiological factor definitely excluded, no trace of this poison being found in the hair or the urine of the affected patients, nor in samples of the beer that had been taken.

It is open to any one to say that it is not the ethylic alcohol but some accidental impurity that is responsible for the neuritis, but until this toxic agent is discovered, which is certainly not arsenic, it is impossible to resist the conviction that alcohol, whether in the form of beer, wine, or spirits, is a common cause of multiple neuritis.

But this statement does not necessarily imply that multiple neuritis is of frequent occurrence in alcoholic subjects, nor that the disease is a direct result of alcohol. Indeed the proportion of alcoholics who suffer from neuritis is a small one, and in such sufferers other factors may play a contributory part. The action of alcohol may be an indirect one in the production not only of neuritis but of hepatic cirrhosis and of changes in the muscle of the heart. It is a reasonable assumption that the lowering of tissue resistance by alcohol permits the successful attack of toxins, derived from micro-organisms or from the changes associated with chronic inflammation, upon the nerves, liver, heart or other structure.

A similar view is held by Dr. F. Parkes Weber, who

suggests that the true causes of multiple neuritis of alcoholic type are infectious, alcohol being merely a predisposing agent.

But whether the neuritis is a direct or an indirect result of the poison there is ample evidence to justify the view that well-marked cases of multiple neuritis (although not common) are produced more frequently by alcohol than by other poisons.

PAROXYSMAL TACHYCARDIA DISAPPEARING AFTER AN ATTACK OF HERPES ZOSTER.

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THE following case presents features of sufficient interest to make it worth recording. The history is briefly as follows:

L. S., aged 15 years, has been under my care for some time suffering from recurrent attacks of tachycardia.

She is rather short, somewhat anaemic, and has never been very strong. She easily takes cold, and has had repeated attacks of influenza. The first attack of tachycardia occurred when she was 5 years old. She awoke suddenly about midnight, feeling "as though she were going to die." She was very pale, and the heart beat very fast. After a short time she vomited a small quantity of liquid; the heart at once became quiet, all symptoms quickly passed away, and she soon fell asleep. This attack was attributed by her friends to a strong cup of tea she had taken the evening before. From that time onwards she had been subject at least every two months to similar attacks. She is obliged to sit still whilst the attack is on, and, however

long it lasts, is unable to take any solid food, but can sip a little liquid. An attack always continues until she vomits, after which all the symptoms quickly disappear. Latterly, she has been given warm salt and water to produce vomiting, and this has cut short the attacks. The vomit consists of one or two tablespoonfuls of sour fluid. The attacks have been attributed at various times to sudden exertion, such as running, to excitement, or to irregularities in diet, but no common etiological factor could be found. In other respects she seems fairly well, the bowels act regularly, and the urine is normal.

The first time I saw her, about five years ago, she was suffering from an attack. She was propped up in bed, very pale, complaining of tightness across the chest and of rapid heart-beat. The pulse was about 160; the impulse was in the nipple line, and no murmurs were audible. Since then I have seen her in several similar attacks; the pulse-rate has varied from 120 to a rate faster than could be counted. They have lasted for various periods, from less than one hour to nearly three days. No drug among the many that have been tried has had any effect in cutting short the attacks, nor has attempted pressure on the vagus been more successful. The attacks have always lasted until she has vomited, when, within fifteen to thirty minutes after the pulse would fall to normal.

On October 10th, 1906, she had severe pain in the left wrist which continued to the next day, when it affected the inner side of the left arm, the front of the left shoulder, and the back. On the morning of October 12th a rash appeared about the left shoulder and arm, the left ear became tender, and she had also some pain in the right wrist. Dr. Arthur Hall saw her at that time in consultation. She had typical herpes zoster affecting the area of the second left dorsal segment—namely, the under and inner part of the left arm, the left breast in the region of the axillary fold, and over the left scapula in the upper part (see diagram).

The eruption ran the usual course, and from the cardiac attack which preceded the eruption by about a month up to the present date—that is, for three years—she has had no recurrence of the tachycardiac attacks, her heart is quite normal, and she has never felt in better health than she does now.