

**Remarks on the relations of pulmonary tuberculosis to other diseases / by F. Parkes Weber.**

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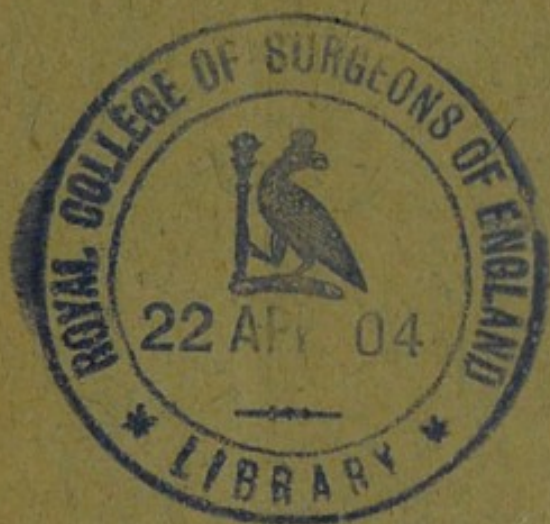
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REMARKS ON THE RELATIONS  
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PULMONARY TUBERCULOSIS  
TO OTHER DISEASES

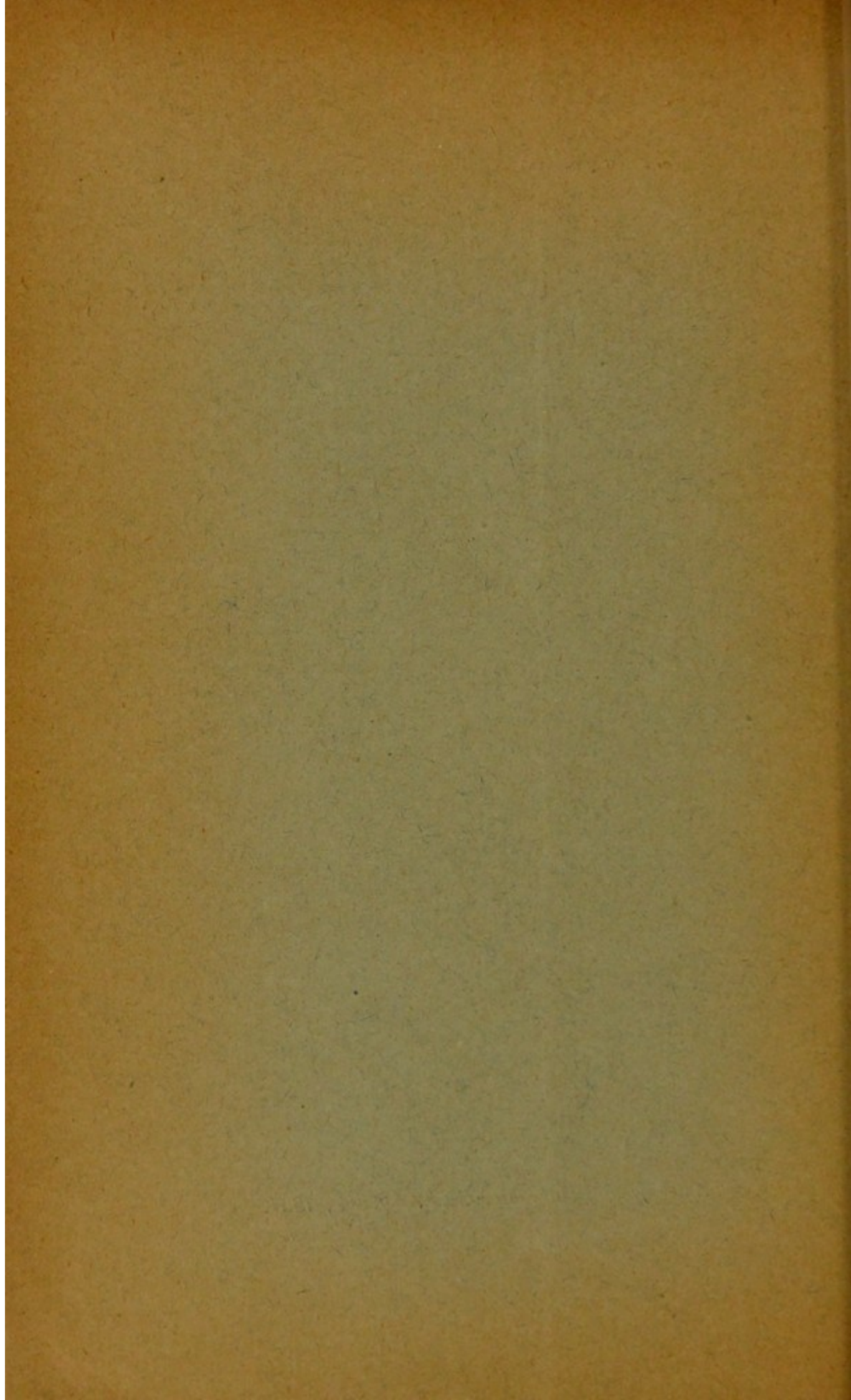
BY

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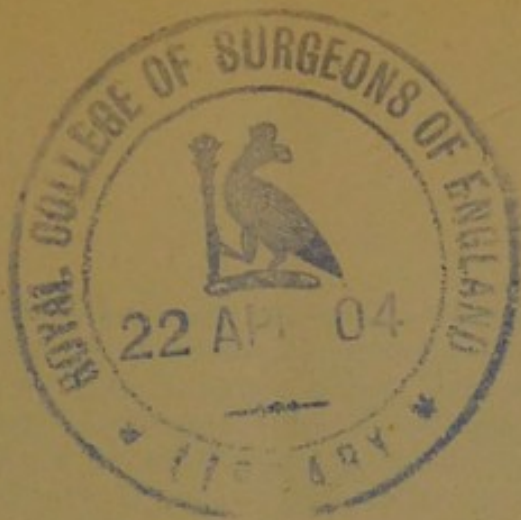
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REMARKS ON THE RELATIONS OF  
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TO OTHER DISEASES.<sup>1</sup>

PULMONARY tuberculosis is so widely disseminated that it is not surprising to find it occasionally associated with nearly every other disease. It is partly on this account no easy matter to decide whether a given disease has any causal relationship whatever to pulmonary tuberculosis. The following questions arise: Is one disease favourable or antagonistic to the onset or spread of the other, or is there some more indirect relationship between the two diseases—for instance, that a certain state of the body which is opposed to one of the diseases is likewise either opposed or favourable to the other, or lastly, is the association of the two diseases merely accidental? These questions are often exceedingly difficult to answer. Statistics are mostly meagre or untrustworthy and in many cases we are driven back to general impressions of the various observers on the subject. I shall not have time to consider tuberculosis of the lungs in all its associations with other diseases but must limit myself to the question of its relations to certain constitutional defects and to certain diseases of the heart, liver, and kidneys. I shall not discuss its relations to other diseases of the lungs and air passages.

*Relations of pulmonary tuberculosis to diseases of the heart.*—  
Louis and Rokitsansky<sup>2</sup> held that there was an antagonism

<sup>1</sup> A post-graduate lecture delivered at the Mount Vernon Hospital for Consumption on Feb. 11th, 1904.

<sup>2</sup> See the remarks on this subject in Wilson Fox's "Diseases of the Lungs," edited by Sidney Coupland, London, 1891, p. 547.

between heart diseases and phthisis, but Traube<sup>3</sup> thought that there was no antagonism between aortic reflux and phthisis, whilst Peacock,<sup>4</sup> Traube,<sup>5</sup> and nearly all modern authorities find<sup>6</sup> that patients with congenital pulmonary stenosis, if they live to an age of about from 15 to 20 years, frequently die from tuberculosis of the lungs. On the whole it seems that conditions of the circulation which tend to passive hyperæmia of the lungs (not merely increased vensity of blood) have a hindering action on the growth of tubercle, whilst anæmia of the lungs favours tuberculosis. Thus it is maintained that active pulmonary tuberculosis rarely arises in the subjects of mitral reflux or of mitral stenosis, and cases have been observed in which the signs of pulmonary tuberculosis became much less marked and the disease became decidedly less active after the development of rheumatic mitral disease. It is said that tubercle bacilli have been found in vegetations from endocarditis, but even if we grant that their presence has once or twice been demonstrated Teissier's paper<sup>7</sup> as to the tuberculous origin of cases of mitral stenosis does not carry conviction with it.<sup>8</sup> It may here be mentioned that some medical men, influenced by Bier's writings on his method of treating tuberculosis of the joints<sup>9</sup> by artificially induced hyperæmia, have advocated the adoption of certain positions of the body and certain local thermal methods as part of the treatment of pulmonary tuberculosis,<sup>10</sup> with the object of increasing the amount of blood in the vessels of the lungs. In regard to the association of congenital pulmonary stenosis with tuberculosis of the lungs it must be remembered that this may be attributed not only to defective supply of blood

<sup>3</sup> L. Traube: *Allgemeine Medicinische Central-Zeitung*, 1864, No. 100.

<sup>4</sup> *Malformation of the Heart*, second edition, London, 1866, p. 189.

<sup>5</sup> Traube: *loc. cit.*

<sup>6</sup> Vide Meissenburg: *Zeitschrift für Tuberkulose*, Leipzig, vol. iii., Part 5; and C. Fischer: *ibid.*, vol. v., Part 3.

<sup>7</sup> Pierre Teissier: *Clinique Médicale de la Charité*, Paris, 1894, pp. 913-1009.

<sup>8</sup> See the criticism by Hugh Walsham, *Brit. Med. Jour.*, 1899, vol. ii., p. 1170.

<sup>9</sup> August Bier: *Behandlung Chirurgischer Tuberkulose der Gliedmaßen mit Stauungshyperämie*, 1893; also *Behandlung der Gelenktuberkulose mit Stauungshyperämie*, 1895. At first Bier confined himself to passive hyperæmia.

<sup>10</sup> E. Jacoby: *Verhandlungen des XIV. Congresses für innere Medizin*, Wiesbaden, 1896, p. 576, and *Münchener Medicinische Wochenschrift*, 1899, Nos. 19 and 20. On the "Postural Treatment" of Pulmonary Tuberculosis see also Schenk, *Wiener Medicinische Wochenschrift*, July 6th and 13th, 1901.

to the lungs but likewise to the general defective development of such patients, especially to a tendency to "infantilism" (see later section). Percy Kidd<sup>11</sup> says: "Such persons are born with imperfect lungs and it is not to be wondered at that these organs should be prone to so common a disease as tuberculosis."

*Relations of pulmonary tuberculosis to diseases of the liver.*  
—*Amyloid disease of the liver* is well known as occasionally due to suppuration connected with tuberculous disease in various parts of the body, but it is really only a very rare complication of tuberculosis of the lungs and, owing to modern surgical and other methods of treatment, it is doubtless becoming a much less frequent complication of tuberculosis altogether, even of hip disease and spinal caries. Enlargement of the liver from *fatty infiltration* is so frequently found in consumptive patients dying after prolonged hectic fever that a causal connexion between the two conditions is universally admitted. Most probably the glandular cells of the liver become so damaged by the febrile condition, or, rather, by the toxæmic cause of the pyrexia, that their metabolic functions become seriously impaired. Their feeble power of combustion is then shown by the accumulation of the fatty droplets. I should here state that probably there is no sharp demarcation line between fatty infiltration and fatty degeneration of the liver; the latter process is probably really a fatty infiltration in an atrophic liver when the hepatic cells have degenerated not only in functional power but in the amount of their living cell-substance also.<sup>12</sup> *Cirrhosis of the liver* is not very rarely found associated with peritoneal or pulmonary tuberculosis or both, and there are good grounds for believing that in a few cases hepatic cirrhosis may be due to a local tuberculous process.<sup>13</sup> In regard to the association of an ascitic form of tuberculous peritonitis with cirrhosis of the liver Dr. S. Gee<sup>14</sup> says: "Perhaps the tuberculous poison sets up cirrhosis, like alcohol, and one is reminded of a similar association of ovarian tumours with cirrhosis of the liver." There is, however, another and more generally acknowledged connexion between

<sup>11</sup> St. Bartholomew's Hospital Reports, London, 1887, vol. xxiii., p. 252.

<sup>12</sup> Cf. Ribbert: Die Morphologie und Chemie der fettigen Degeneration, Deutsche Medicinische Wochenschrift, 1903, No. 44, p. 793.

<sup>13</sup> Vide A. Gilbert and J. Castaigne's case at the Société de Biologie, Paris, June 3rd, 1899.

<sup>14</sup> St. Bartholomew's Hospital Journal, London, May, 1900, p. 116.

pulmonary tuberculosis and hepatic cirrhosis—namely, the fact that alcoholism may be a factor in the production of the pulmonary as of the hepatic disease. Osler<sup>15</sup> writes: "A remarkable feature is the association of acute tuberculosis with cirrhosis. In seven cases of my series the patients died with either acute tuberculous peritonitis or acute tuberculous pleurisy. Pitt states 22½ per cent. of the cases of cirrhosis dying in Guy's Hospital during 12 years had acute tuberculosis. Of 121 autopsies at the Manchester Royal Infirmary<sup>16</sup> in cirrhosis about 23 per cent. gave evidence of tuberculous infection. 12 of these had tuberculosis of the peritoneum and 12 died directly from the tuberculous infection." This naturally brings me to the following subject.

*Relations of pulmonary tuberculosis to alcoholism.*—Much has been written on the effects of alcohol in regard to consumption and there is still much difference of opinion as to the value of alcohol in the treatment of the disease. Yet one can well understand that if alcohol in moderate doses increases the patient's appetite, favours digestion, and makes him more cheerful, it may, by promoting the general nutrition of the body, indirectly help to check the progress of the tuberculous disease. On the other hand, abuse of alcohol, at least, chronic alcoholism, is now almost universally admitted to be a great predisposing cause of pulmonary tuberculosis. That this should be the case is not very surprising if one remembers how readily the subjects of chronic alcoholism fall victims to other diseases of microbic origin, such as acute pneumonia and septic inflammations. Chronic alcoholism favours degenerative processes all over the body and diminishes the resistant power of the tissues to microbic infection. The cells of the different parts of the body in alcoholism seem to become sluggish in regard to their metabolic functions so that the vital reactions of the tissues become less prompt and less efficient. This supposition explains the tendency to accumulation of fat in the liver and subcutaneous tissues and explains why pyrexia is often less marked and why alcoholics succumb more readily than others on very prolonged exposure to cold. Probably just as the tissues of alcoholics respond less readily than those of

<sup>15</sup> Quoted by J. M. Cowie: Tuberculosis, London, January, 1904, p. 27.

<sup>16</sup> T. N. Kelynack: Medical Chronicle, Manchester, 1897, vol. vi., p. 262.

ordinary persons to demands for increased heat production, so in infective diseases their antitoxin-forming and other microbe-resisting powers are less efficient. The bloated type of alcoholic—for instance, the old style of brewer's drayman—has always been notorious for extreme liability to wound infection and acute septic gangrene following injuries and in such a person one can imagine that not only have the anti-microbic and antitoxin-forming functions of the cells of his body been dulled by the habitual action of alcohol, but even that, owing to his "bloated" condition, the really living portions of his body—namely, the cells—are abnormally separated from each other and therefore less able to cope with the intruding microbes. Be this as it may, French inquiries<sup>17</sup> into the subject as well as English observations<sup>18</sup> leave no doubt about the reality of the connexion between alcoholism and pulmonary tuberculosis. Moreover, the children of alcoholics are often born with deficient resistant power and thus fall an easy prey to tuberculosis. I must here state, however, that I believe it is going too far to regard alcoholism, as some writers on the subject have done, as the only connecting link between pulmonary tuberculosis on the one hand and peripheral neuritis and hepatic cirrhosis on the other. Cirrhosis of the liver may occasionally, as I have already mentioned, be of local tuberculous origin and polyneuritis may in some cases be due as much to the pulmonary tuberculosis itself as to any associated alcoholism, but to this I shall return later.

*Relations of pulmonary tuberculosis to diseases of the kidneys.*—Typical amyloid disease of the kidneys (I do not here refer to slight amyloid changes accompanying parenchymatous nephritis) is occasionally met with in cases of pulmonary tuberculosis but I believe it to be exceedingly rare. Parenchymatous nephritis<sup>19</sup> and forms of mixed parenchymatous and interstitial nephritis are, on the other hand, much more frequently found. They occur especially in patients with advanced pulmonary tuberculosis and in these cases the kidney disease is, I have no doubt, usually a result of the lung disease, of which it constitutes a most serious complication. I regard

<sup>17</sup> See F. Imbault's Paris thesis, 1901, for French work on the subject.

<sup>18</sup> See quotations and figures in T. N. Kelynack's "The Relation of Alcoholism to Tuberculosis," *THE LANCET*, August 3rd, 1901, p. 277.

<sup>19</sup> Cf. C. von Kahliden: *Ueber Nephritis bei Phthisikern*, *Centralblatt für Allgemeine Pathologie*, Jena, vol. ii., p. 97.

the nephritis in most of these cases as caused by irritation due to toxic substances absorbed into the circulation from the tuberculous and suppurating foci in the lungs, but it must be remembered that the nephritis may occasionally be associated with local tuberculosis and that the presence of tubercle bacilli has sometimes been detected in the kidneys, even in the absence of regular tubercles.<sup>20</sup> Here I would mention that slight amyloid changes, easily to be detected by examining microscopic sections stained with methyl violet, frequently accompany the changes of chronic parenchymatous nephritis, both in the case of consumptive patients and others. This is not surprising when one remembers the experience of N. P. Krawkow<sup>21</sup> and others regarding the artificial production of amyloid disease. Krawkow seems to show that amyloid disease is usually the result of absorption into the blood of products of the metabolism of parasitic microbes, and he actually succeeded in inducing amyloid changes in the tissues of a rabbit by repeated injections of the mere filtrate of a culture of the bacillus pyocyaneus.

Recurrent attacks of gout with typical chronic interstitial nephritis are relatively rarely<sup>22</sup> associated with pulmonary tuberculosis. A good example is a case I described in 1892.<sup>23</sup> The patient was a sallow, thin man, aged 46 years, who died with uræmic symptoms in the Brompton Hospital in 1890. He gave a family history of consumption but said that his

<sup>20</sup> Cf. Arthur Heyn: Ueber Disseminirte Nephritis Bacillaris Tuberculöser ohne Nieren-Tuberkel, Virchow's Archiv, Berlin, 1901, vol. clxv., p. 42; also Hugh Walsham, Excretion Tuberculosis of the Kidney, Transactions of the British Congress on Tuberculosis (1901), London, 1902, vol. iii., p. 583; and likewise the authors quoted by Walsham.

<sup>21</sup> Centralblatt für Allgemeine Pathologie, Jena, 1895, vol. vi., p. 337; Archives de Médecine Expérimentale, Paris, 1896, vol. viii., p. 106; Archiv für Experimentelle Pathologie, Leipzig, 1898, vol. xl., p. 194.

<sup>22</sup> In regard, however, to the kidney disease, without taking gout into consideration W. H. Dickinson (Diseases of the Kidney, London, 1875, p. 378) found tubercle in 20·8 per cent. of 250 persons dying from granular degeneration of kidneys, which, according to statistics, was less than the average frequency of tubercle in all post-mortem examinations. In a paper on the Association of Chronic Interstitial Nephritis with Pulmonary Tuberculosis (1892) I made out that in a series of 145 necropsies on patients over 40 years old with pulmonary tuberculosis (nearly all of whom had actually died from their lung disease) there was evidence of some degree of chronic interstitial nephritis in 40 per cent., whereas in a series of 44 necropsies on patients over 40 years old with healed or quiescent pulmonary tuberculosis 61·3 per cent. showed evidence of chronic interstitial nephritis.

<sup>23</sup> On the Association of Chronic Interstitial Nephritis with Pulmonary Tuberculosis, 1892, p. 3.

father had suffered from gout. He himself had been discharged from the Royal Navy at the age of 18 years on account of pulmonary phthisis. Afterwards his lung trouble improved and he enjoyed good health excepting for an acute attack of gout at the age of 31 years and four other attacks since then. In his forty-first year, however, he was troubled with cough and occasionally spat up some blackish blood. In the hospital he was found to have impaired resonance and signs of local disease over the upper parts of both lungs. Gouty swellings were noted in the fingers and toes. His urine was of low specific gravity and contained a good deal of albumin and a few granular casts. He suffered from uræmic vomiting and finally coma came on. Just before the end a uræmic eruption resembling eczema appeared all over his body and, though apparently unconscious, he was observed to be scratching himself continually. At the post-mortem examination the lungs showed no recent tuberculosis but extreme fibrosis around the caseous remains of former tuberculosis. Urate of sodium was seen deposited in the articular cartilage of one of the small joints which was examined. The heart was somewhat hypertrophied and the kidneys showed chronic interstitial nephritis. In this case and similar ones, although the kidneys may have been damaged at the time of (and as a result of) the previous active tuberculous process in the lungs, the chronic interstitial nephritis was doubtless intimately connected with the gout. This brings me to a consideration of the following subject.

*The relations of pulmonary tuberculosis to gout and the effect of meat in the preventive and curative treatment of tuberculosis.*<sup>24</sup>—Dr. J. E. Pollock<sup>25</sup> believes that "gout, like rheumatism, when the specific attack of the disease is developed in a case of tubercle, retards the latter." Sir Dyce Duckworth<sup>26</sup> supposes that gout or the gouty diathesis is antagonistic to phthisis. Sir Hermann Weber<sup>27</sup> knows of many persons who recovered from pulmonary tuberculosis but suffered from distinct gout in some form. In fact, many observers have held that there is a certain antagonism

<sup>24</sup> Cf. F. Parkes Weber: *Zeitschrift für Tuberkulose*, 1900, vol. i., p. 93.

<sup>25</sup> *The Elements of Prognosis in Consumption*, London, 1865, p. 273.

<sup>26</sup> See *A Treatise on Gout*, London, 1890.

<sup>27</sup> Discussion at the British Congress on Tuberculosis, *Brit. Med. Jour.*, August 3rd, 1901, p. 317.

between gout and tuberculosis, and although there are great exceptions to the rule (notably in regard to gouty persons addicted to alcohol and gouty persons who have become generally decrepit) I believe that this antagonism does to some extent exist. This resistance of gouty persons towards tuberculosis is probably partly due to the meaty foods (butcher's meat, eggs, and all animal proteid foods) which most persons with acquired goutiness have been accustomed to indulge in freely during much of their lives. I referred to the subject in 1892 in a paper on the Association of Chronic Interstitial Nephritis with Pulmonary Tuberculosis, and I then suggested that there might be some substance circulating in the blood in gouty persons in minute quantities yet sufficient to have an antagonistic action towards the growth of tubercle and that perhaps this was likewise the case in persons taking an unusual amount of food, which "might partly account for the good result following the extra feeding of phthisical patients when duly assisted by hygienic influences."<sup>28</sup>

The wealthy classes on the whole are inclined to indulge excessively in animal food and by this means, in some countries at least, favour the development of gouty diseases. On the other hand, their mortality from pulmonary tuberculosis is relatively slight when compared to that of the poorer classes. In the case of large cities<sup>29</sup> in which the streets are classified first in regard to the wealth of the inhabitants, and, secondly, in regard to the mortality from consumption, this can be conclusively shown. Doubtless overcrowding and all kinds of bad hygiene play an immense part in determining the relatively great incidence of tuberculosis amongst the poor, but the insufficiency of meaty food is, I believe, likewise partly responsible. The poorest classes, though they often obtain abundance of carbohydrate food, can generally obtain very little of the more expensive meaty foods. Great meat eaters, if not alcoholic, rarely, even in the most unhygienic surroundings, become phthisical, and it may likewise be noted that in all charitable hospitals and sanatoriums for tuberculosis more animal food is provided than the patients, owing to their poverty,

<sup>28</sup> On the Association of Chronic Interstitial Nephritis with Pulmonary Tuberculosis, London, 1892, p. 11.

<sup>29</sup> In regard to Paris compare Dr. E. P. Léon-Petit, "*Le Phthisique et son Traitement Hygiénique*," Paris, 1895; and in regard to London see Sir Hugh Beevor's oration on the Declension of Phthisis, at the Hunterian Society of London, 1899.

can generally obtain in their own homes, whereas many of them have been already accustomed at home to as much carbohydrate food as they care for. The want of resistance to disease shown by many children who have been fed with abundant carbohydrates and with too little animal proteids has been pointed out by various authors<sup>30</sup> Charles Richet's<sup>31</sup> experimental comparison (a) of the effects of a mixed carbohydrate and meat diet with (b) the effects of an exclusive raw meat diet in dogs previously inoculated with tubercle is not without interest in this respect, but his results have not been entirely confirmed.<sup>32</sup> On the whole, I believe that deficiency of meat in the diet acts not rarely, especially in young persons, as a predisposing cause of the onset of pulmonary tuberculosis, and in such cases a change to a diet containing more meat may often have a most beneficial effect, especially, of course, if combined, as it always should be, with suitable general hygienic treatment. It is on the foregoing considerations that I base my opinion and not on the argument that because carnivorous animals generally show good resistance to tuberculosis therefore a meat diet must be the proper treatment for tuberculosis. To the latter argument it might be objected, as Woods Hutchinson<sup>33</sup> has suggested, that the great relative resistant power of carnivorous animals might depend on the survival of those of their kind in past generations who have not succumbed to infection from eating tuberculous animals, the families having died out the members of which succumbed easily to infection from eating tuberculous material.

Various explanations have been suggested for the beneficial action of meat in cases of tuberculosis. Richet and Héricourt<sup>34</sup> attach the greatest importance to the meat being raw and think that it is only the muscle plasma which is active, the parts of the meat which are soluble in water containing the active substance. Hence they term their method of treatment "zomotherapy"—that is to say, "meat

<sup>30</sup> Cf. J. Dvorak's remarks, Bericht über den Kongress zur Bekämpfung der Tuberkulose als Volkskrankheit, Berlin, 1899, p. 619.

<sup>31</sup> Académie du Médecine, Paris, Nov. 29th, 1899.

<sup>32</sup> See C. Fränkel and Sobernheim's paper, Zur Frage der Zomotherapie, Berliner Klinische Wochenschrift, July 15th, 1901; also Lawrason Brown's criticisms in the American Journal of the Medical Sciences, June, 1903 vol. cxxv., p. 1071. Brown agrees, however, that "much meat, with a judicious admixture of carbohydrates, fats, &c., is essential to the treatment of pulmonary tuberculosis."

<sup>33</sup> Quoted by Dr. Harry Campbell, THE LANCET, Feb. 16th, 1901, p. 496.

<sup>34</sup> Académie des Sciences, Paris, Feb. 26th, 1900.

juice therapy." Though it is admitted that myosin albumin<sup>35</sup> is easily digested by most patients and can be of great value, it is scarcely likely that the value of meat in the diet of consumptives or of persons predisposed to consumption depends entirely on the meat being raw or only very slightly cooked. It must be remembered also that a certain amount of cooked meat often powerfully stimulates the appetite and the digestion, and in some persons much more so than meat juice and raw meat do. Dr. H. Harper<sup>36</sup> taking into consideration the antagonism between tuberculosis, on the one hand, and gout and meat-eating habits on the other, introduced the treatment of consumption by pure urea, and according to his views much of the effect of meat eating on tuberculosis would be attributed to increased formation of urea. He says that from an extensive experience of urea in tuberculosis it is the only thing that he can call a remedy.<sup>37</sup> Though doubtless not in the largest possible doses, I for a time gave urea almost as a routine to my patients with consumption both at the Mount Vernon Hospital for Consumption and at the German Hospital. I thought I obtained some good results at first but afterwards I felt uncertain, and my later observations failed to convince me that the good results obtained in the early cases were really due to the urea.<sup>38</sup> Moreover, there are theoretical difficulties in the way of accepting the claims made for urea.<sup>39</sup>

There still remain other points to be considered in regard to the effect of meat diets in tuberculosis. Striped muscle,<sup>40</sup> whether as part of a living body or as butcher's meat, is not a good soil for the growth of tubercle bacilli. Jacob and Pannwitz<sup>41</sup> say that all observers regard muscle as a bad

<sup>35</sup> See Lawrason Brown, loc. cit.; and F. W. Forbes Ross, *Meat Preparations: the Possibilities of Myosin Albumin*, THE LANCET, June 22nd, 1901, p. 1757.

<sup>36</sup> See his papers in THE LANCET, March 9th (p. 694), June 15th (p. 1672), and Dec. 7th (p. 1567), 1901; also in Brit. Med. Jour., Oct. 18th, 1902, p. 1235.

<sup>37</sup> THE LANCET, March 9th, 1901, p. 697.

<sup>38</sup> Cf. the adverse conclusions arrived at by S. Vere Pearson in his paper on Pure Urea in the Treatment of Chronic Pulmonary Tuberculosis, THE LANCET, Nov. 22nd, 1902, p. 1383.

<sup>39</sup> See the remarks on this subject by A. W. Gilchrist, "The Low Phosphates and Urea in the Urine of the Tuberculous," THE LANCET, Nov. 29th, 1902, p. 1456.

<sup>40</sup> See Part III. of the Report of the Royal Commission to inquire into the Effect of Food derived from Tuberculous Animals (London, 1895).

<sup>41</sup> "Entstehung und Bekämpfung der Lungentuberkulose," Leipsic, 1901, vol. i., p. 262.

culture medium for the bacilli and they give Röckl as an authority for the statement that the muscles are involved only in eight out of 1000 cases of tuberculosis. This may have a bearing on the meat diet question and likewise on several other questions. Thus it is a generally acknowledged fact that, on the whole, persons with well-developed muscular systems are more resistant to tuberculosis than persons with relatively small muscles. An active life is believed to diminish the chances of tuberculosis and it seems probable that this result is obtained not merely owing to the necessarily increased exercise of the lungs but likewise owing to the striped muscular system throughout the body being kept in good condition by muscular activity. Men with extreme *congenital* deficiency of the generative organs (absent or rudimentary testes with infantilism) seem to be more liable to pulmonary tuberculosis than the average. This has, I believe, been noted by several physicians. Recently I had a man, aged 29 years, under my care at the German Hospital suffering from a rather bad type of pulmonary tuberculosis. His generative organs were very imperfect (the right testis was very minute and the left one was absent) and the infantile voice, the feeble general development, and the absence of hair from the chin, &c., all corresponded to the extreme hypoplasia of the generative organs. Perhaps the poor muscular and general development in such cases is connected with the increased liability to tuberculosis, which may also, perhaps, be compared to the liability of other very imperfectly developed persons (with or without actual infantilism)—for instance, the subjects of congenital stenosis of the pulmonary orifice of the heart. (See previous section on this subject.) In this connexion it may be remarked that patients with idiopathic muscular atrophy (primary progressive myopathy) seem specially liable to be attacked by pulmonary tuberculosis, but in these cases the weakening of the respiratory muscles must likewise be taken into consideration.

Whilst still on the subject of meat in tuberculosis I must allude to a recent paper by Dr. J. J. Galbraith<sup>42</sup> who strongly advocates a diet rich in animal nitrogen. He finds that by such a diet together with the regular open-air method of

<sup>42</sup> The Dietetic Treatment of Pulmonary Tuberculosis from the Point of View of its Hæmatology and Histopathology, Brit. Med. Jour., March 14th, 1903, p. 600.

treatment a certain change in the blood is effected. The characteristic features of the change, according to him, are (1) a moderate constant leucocytosis; (2) a large absorptive lymphocytosis; and (3) an almost constant eosinophilia (the eosinophile cells varying from 4 to 5 per cent. of the total leucocytes). Dr. Galbraith likewise maintains that from some cause or other the bodies of consumptive patients are relatively and absolutely deficient in nitrogenous constituents and that though there is wasting of subcutaneous fat there is extensive storage of fat in the viscera which would be drawn upon were this material mainly needed. He concludes that in pulmonary tuberculosis a meat diet supplies a direct physiological want. In connexion with Dr. Galbraith's remarks on fat I will add that obese persons, especially those of the ruddy and plethoric type of obesity, who generally possess a strongly developed muscular system and who are often great meat eaters, are, as one would expect, unless they have become victims of alcoholism, rarely or hardly ever the subjects of phthisis. I have, however, during recent years frequently seen a woman, now aged 43 years, of the flabby-fat type, who suffers from pulmonary tuberculosis. In her case the fatness apparently followed oöphorectomy; at all events, she underwent an abdominal operation eight years ago which was followed by permanent amenorrhœa and great increase in weight. I know also of one man of the full-blooded stout type with a genial ruddy face and fond of good dinners who ultimately died from pulmonary tuberculosis but only after suffering from diabetes mellitus with great wasting. On the other hand, in a fat man at present under my care a fear of consumption seems to have been the starting point of the obesity. This patient when ~~fifteen~~ years old was told that he was threatened with consumption and either of his own accord or under advice he began to eat as much as he could and more than he felt inclined to. He is now 24 years of age and I can find no signs of pulmonary tuberculosis, but he has become obese and inactive, his aspect is plethoric, his pulse too frequent, his lips are livid, and his legs slightly œdematous.

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*The relations of pulmonary tuberculosis to cancer.*—It has been maintained that a certain antagonism exists between cancer and tuberculosis. If that is the rule there are certainly a good many apparent exceptions. Quite recently I had a patient at Mount Vernon Hospital, a young woman,

aged 31 years, suffering from well-marked pulmonary tuberculosis, with cavity formation in the upper lobe of the right lung. She had also a rapidly growing carcinoma involving the lowest portion of the pharynx—that is to say, the entrance of the œsophagus. Moreover, in the post-mortem room one occasionally meets with evidence in the lungs of active tuberculosis (recent miliary tubercles) in cases of fatal carcinomatous stricture of the œsophagus. In such cases I am inclined to think that the chronic starvation due to the œsophageal stricture by lowering the general vitality of the patient induces the quiescent remains of some previous tuberculous trouble to break out afresh. If there is really any antagonism, as many have believed, between cancer and pulmonary tuberculosis I believe it must be an indirect one. Indeed, in the first place, patients suffering from cancer are mostly of an age not specially liable to tuberculosis; and in the second place, I think one may say that, on the whole, cancer has a tendency to attack robust persons, “good livers,” and those who have always had plenty to eat—in fact, persons drawn from a class relatively resistant to pulmonary tuberculosis.

*The relations of tuberculosis to lymphadenoma and leukaemia.*—Lymphadenoma (pseudo-leukæmia, Hodgkin's disease), unlike cancer, tends to become associated with tuberculosis. A typical example of this association is the following case.

The patient, a girl, had some enlarged glands removed from the right side of the neck in March, 1898. In March, 1901, when 14 years old, she was an in-patient under my care at the German Hospital with anæmia, an enlarged spleen, and moderate pyrexia. She improved considerably under arsenic treatment and after the middle of April was practically free from fever. Whilst under treatment I may mention she had a slight attack of arsenical herpes zoster. In April, 1902, the patient was again in the hospital and in June I noticed that the spleen reached to within half an inch of the anterior superior spine of the ilium. The liver could be felt below the costal margin. In the latter part of June the patient suffered from a short acute attack of pneumonia at the base of the left lung. She left the hospital in July, but was troubled with a good deal of cough and occasionally a little mucous expectoration, sometimes streaked with blood. She was readmitted on Nov. 7th, 1902, with moderate ascites and œdema. Some crepitation

could be heard over the left lung. No tubercle bacilli were found in the sputum. The blood showed extreme anæmia with relative leucocytosis; the hæmoglobin was only 30 per cent. of the normal. The urine contained a trace of albumin. There was very little fever, but the general weakness increased and death occurred on Dec. 1st. The necropsy showed enlarged glands in the neck, posterior mediastinum, and abdomen. A few of the glands seemed slightly caseous, especially some not very large ones at the root of the lungs. A hard fibrous mass situated at the back of the upper part of the abdomen and displacing the stomach and liver forwards had microscopically the appearance of chronic hard lymphadenoma. The spleen weighed 26 ounces and contained some whitish masses ("hardbake" type), one of them approaching caseation. There was a considerable serous effusion in the peritoneum with some matting together of the abdominal viscera (probably due to peritoneal tubercle). The lungs showed scattered tubercles and by microscopic examination minute tubercles were detected in the liver and spleen. Tubercle bacilli were seen in specially stained sections of the liver and (what is much more remarkable) in the bone marrow from the shaft of the left humerus.

The association of lymphadenoma with tuberculosis has been repeatedly described in medical journals and has constituted a subject for discussion at medical societies. Some observers have gone so far as to suggest that all the cases in question are tuberculous—in fact, that lymphadenoma is a variety of glandular tuberculosis. In the discussion on Lymphadenoma in its Relation to Tuberculosis at the Pathological Society of London, in December, 1901, Dr. F. W. Andrewes<sup>43</sup> concluded that lymphadenoma is a distinct disease, not due to the action of the tubercle bacillus; that there is a form of tuberculosis of the lymphatic glands clinically indistinguishable from lymphadenoma; that a fair number of cases of lymphadenoma occur in which secondary infection with tubercle has taken place; and that this secondary infection may be local and unimportant, or the patient may die from generalised tuberculosis with a mixture of lesions of the most puzzling kind. It was such a mixture of lesions that the case to which I have just alluded showed. Cases of this kind should be separated from cases of multiple

<sup>43</sup> Transactions of the Pathological Society, London, vol. liii., p. 314.

tuberculosis of lymphatic glands. There are doubtless, as Dr. Andrewes says, some cases of the latter class which cannot be distinguished clinically from lymphadenoma (Hodgkin's disease), but there are also cases which can be recognised during life (without "bioscopic" help, including exploratory operations, of any kind and without even the use of tuberculin), as examples of chronic multiple tuberculosis of lymphatic glands. They may be diagnosed (1) by there being no, or only very slight, enlargement of the spleen or liver; and (2) by there being evidence of associated pulmonary tuberculosis.<sup>44</sup> Such a case I have at present under observation at the German Hospital.

The patient, a married woman, aged 32 years, is a fairly "well-nourished" woman with a rather florid complexion. The lymphatic glands of each side of the neck and of the right axilla are enlarged. The inguinal glands are enlarged but not more so than they are in many healthy persons. The spleen and liver do not seem abnormally large. The patient first came under observation in October, 1903. She knew that the glands in the right axilla were affected two and a half years previously. But those in the neck had only been enlarged during the last six months. She had lost weight and presented physical signs of slight tuberculosis of one lung. The blood showed that there was no great anæmia: hæmoglobin, 90-95 per cent.; red cells 4,600,000, and white cells 12,000, per cubic millimetre. The urine was free from albumin and sugar. In the hospital her general condition has remained about the same.

Several cases have been published illustrating the association of tuberculosis with leukæmia (mostly cases of lymphatic leukæmia<sup>45</sup>). It has been observed that after tuberculous infection there are often retrogression in the size of the spleen and lymphatic glands and diminution in the number of the white cells in the blood; for this reason Professor H. Quincke of Kiel,<sup>46</sup> has even suggested the employment of tuberculin in leukæmia. Retrogression of leukæmic

<sup>44</sup> Cf. Professor Bäumlér's paper, *Multiple Lymphdrüsentuberkulose*, at the recent "Oberrheinischer Aertzetag" on July 16th, 1903. (*Münchener Medicinische Wochenschrift*, 1904, No. 1.)

<sup>45</sup> See W. J. Susmann's table of cases, *Practitioner*, London, October, 1903, p. 536; W. Neutra, *Zeitschrift für Heilkunde*, Vienna, 1903, vol. xxiv., p. 349 (with bibliography); H. Quincke, *Deutsches Archiv für Klinische Medizin*, Leipsic, 1902, vol. 74, p. 445; and G. Carpenter, *Medical Press*, Jan. 28th, 1903, p. 79.

<sup>46</sup> Quincke: *Loc. cit.*

symptoms has also been observed as a result of erysipelas<sup>47</sup> and other microbic invasions. Susmann<sup>48</sup> suggests that the excess of nucleo-albumin and the increased phagocytic power of the blood in leukæmic patients may exercise an antagonistic action in regard to tubercle. It is not certain, however, that leukæmia is really antagonistic to tuberculosis, though tuberculosis seems to check the progress and even to cause retrogression of leukæmia.

*The relations of pulmonary tuberculosis to diabetes mellitus.*—Pulmonary tuberculosis is well known to attack not very rarely patients suffering from grave diabetes, and Sir Lauder Brunton<sup>49</sup> has suggested that the presence of sugar in the tissues facilitates the growth of microbes and renders diabetics peculiarly liable to certain bacterial affections, pulmonary tuberculosis amongst others. Though tuberculosis is a most unfavourable complication of diabetes a diminution in the sugar, or even its complete disappearance from the urine, is occasionally observed to follow the onset of the pulmonary disease. The cautious employment of tuberculin might, as far as I can see, be proposed for diabetes as it has been for leukæmia. And probably with as little likelihood of success, for one must remember that the diminished excretion of sugar in diabetics when they become consumptive may, as has been suggested, be due merely to the pyrexia or the mixed bacterial toxins causing pyrexia; moreover, disappearance or diminution of the sugar may be a temporary result of other microbic invasions, accompanied by pyrexia, besides tuberculosis.

*The relations of pulmonary tuberculosis to syphilis.*—A certain amount of antagonism between syphilis and tuberculosis has been supposed to exist by some medical observers, but many authorities, especially the majority of French writers, think that syphilis by lowering the general powers of resistance to disease predisposes persons to tuberculosis. It has even been suggested, I believe, that a reason why syphilitic gummata are so rarely seen in the lungs is that a counter-infection with tubercle bacilli generally occurs, so that a case of pulmonary syphilis is generally transformed

<sup>47</sup> See H. Zinkeisen's second case, *Deutsches Archiv für Klinische Medicin*, Leipzig, 1903, vol. 75, p. 505.

<sup>48</sup> Susmann: *Loc. cit.*

<sup>49</sup> "Disorders of Assimilation, Digestion, &c.." London, 1901, pp. 53, 54.

into one of pulmonary tuberculosis.<sup>50</sup> Whether persons who have had syphilis are more easily, or less easily, infected with tubercle than persons who have never had syphilis is a question which for obvious reasons cannot easily be answered. It has been supposed by some that syphilis, like alcoholism, in the parents renders the children peculiarly liable to pulmonary tuberculosis.<sup>51</sup> I formerly repeatedly saw an intelligent man who was suffering from pulmonary tuberculosis, and also from fibrous stenosis of the larynx for which tracheotomy was performed. In 1899, when I first saw him, he was 41 years old, and had had a form of lupus verrucosus of the right hand for 18 years. He was dumb and almost completely deaf. The presence of pulmonary tuberculosis was made certain by a marked general reaction following the injection of half a milligramme of Koch's old tuberculin and afterwards by the discovery of tubercle bacilli in the sputum. Mr. R. Lake, who operated on the patient's larynx in the Mount Vernon Hospital, believed the great amount of old cicatricial tissue he met with to be of syphilitic origin and the supposition of congenital syphilis would probably account for both the laryngeal and aural conditions present. In spite of his unfavourable circumstances he seemed rather resistant to the progress of the tuberculous disease. He died at his own house, I heard, in February, 1903, but I am uncertain of the exact cause of his death.

*The relations of pulmonary tuberculosis to multiple neuritis.*—It is generally recognised that some causal relationship exists to account for the relatively frequent association of multiple neuritis of the alcoholic type with pulmonary tuberculosis. Dr. H. W. G. Mackenzie<sup>52</sup> says that it is almost invariable to find tubercle present in the lungs in patients dying in the course of alcoholic paralysis. The frequency of this association has been used as an argument to prove that alcoholism creates a predisposition to tuberculosis. It doubtless can do so, and I have already considered that subject, but it is no longer supposed as, I

<sup>50</sup> Compare J. Patoir's observations in *Presse Médicale*, Paris, Jan. 16th, 1901, p. 25.

<sup>51</sup> See J. Goldschmid: *Münchener Medicinische Wochenschrift*, 1901, No. 9, p. 344.

<sup>52</sup> In G. A. Gibson's *Text-book of Medicine*, 1901, vol. i., p. 35. See also T. N. Kelynack, *Medical Chronicle*, Manchester, 1896, vol. iv., p. 180.

believe, it formerly was that any case of multiple neuritis affecting the lower extremities is necessarily due to alcohol. It is notorious that those most addicted to alcohol are not always the ones that suffer from neuritis. Given exciting cause or causes of polyneuritis it is evident that all persons are not equally affected by them, some being more liable than others (for instance, women more so than men). In those who are specially predisposed it is probable that tuberculosis (or rather a toxæmic condition arising from pulmonary consumption), like several other microbic diseases, may act as an exciting cause in association with alcohol or even independently.

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