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Traumatic Tuberculosis,
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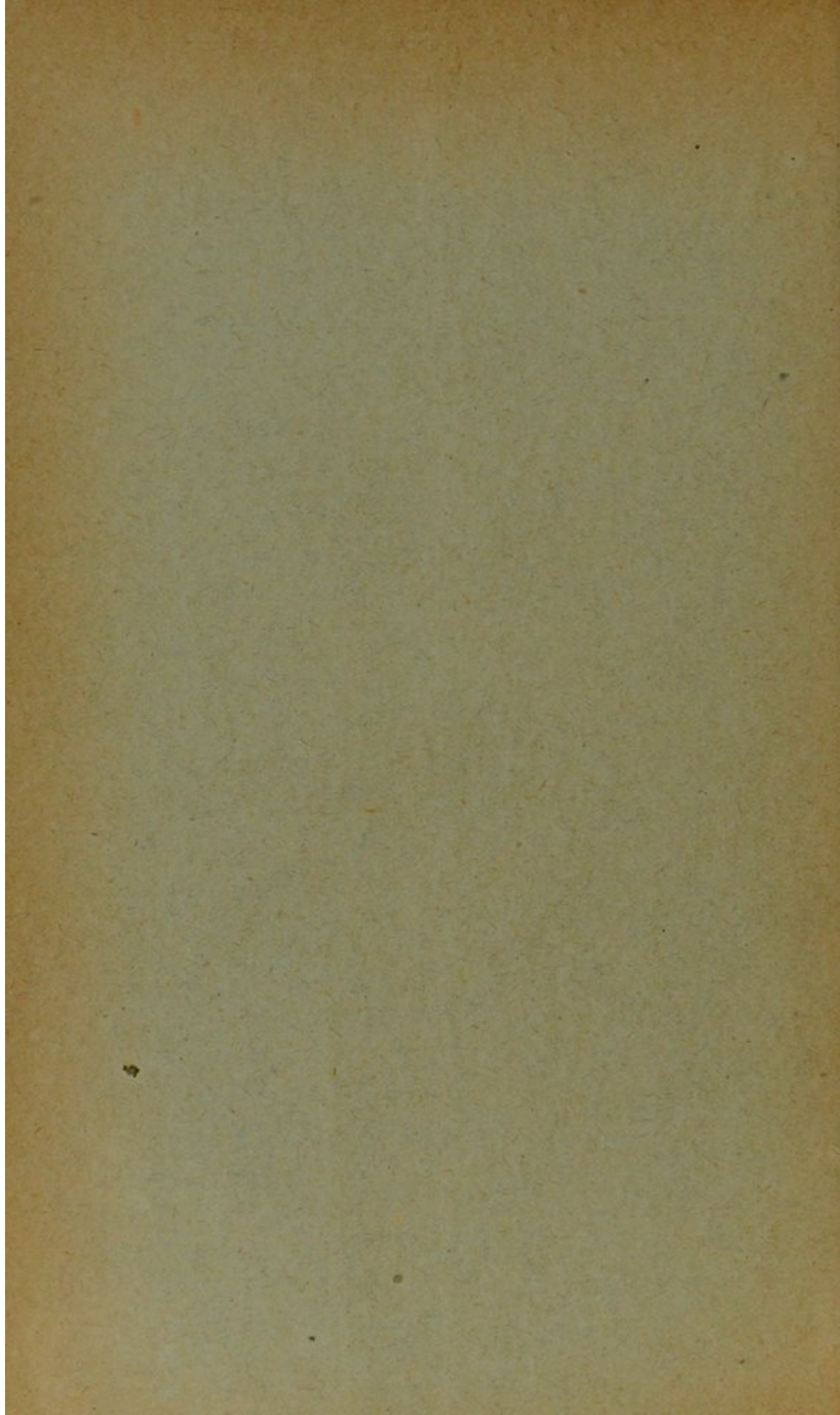
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TRAUMATIC TUBERCULOSIS, TRAUMATIC PNEUMONIA AND TRAUMATIC PLEURISY.

By **F. PARKES WEBER**, M.A., M.D., F.R.C.P.

(Physician to the German Hospital, London; Physician to the Mount Vernon Hospital for Chest Diseases, Hampstead; and Medical Officer to the North British and Mercantile Insurance Company).

PART I.

TRAUMATIC TUBERCULOSIS.

Pathologically, it would doubtless be correct to assert that many cases of local or disseminated tuberculosis in the development of which traumatism has been alleged to play a more or less important part have not been examples of "traumatic tuberculosis" at all (the evolution of the disease having been quite independent of, and uninfluenced by, the trauma in question), and that all real "traumatic tuberculosis" (excepting that due to direct inoculation, see later) may be explained as tuberculosis which as the result of a trauma has been converted from a "latent" case—that is to say, one in which symptoms either did not exist or were not noticed—into a case with sufficient symptoms to render a diagnosis possible.*

* Cf. R. Stern, *Ueber die traumatische Entstehung innerer Krankheiten*, Jena, 1896 (with analysis of published cases); also A. Ascarelli, *Policlinico*, Rome, 1907, Vol. 14 (Sezione pratica), p. 1025, and the papers of Honsell and Jordan to which I shall refer later on.

Clinically, cases of alleged traumatic tuberculosis may be divided into those due to direct inoculation from cuts, etc., and those due to injuries by which tubercle bacilli could not have been introduced from without.

Cases of *traumatic tuberculosis from direct inoculation* are nearly all superficial and include tuberculous warts (*lupus verrucosus*), "necropsy tubercles," and other forms of superficial tuberculosis, such as may be acquired by pathologists and *post-mortem* room assistants in making necropsies on tuberculous subjects (as some of us doubtless have had the opportunity of observing in our own persons), and butchers, &c., from tuberculous cattle. Similarly tuberculosis may be inoculated by the accidental breaking of a glass or porcelain spittoon containing tuberculous sputum, if the latter contaminates the wounds made by the sharp fragments of the broken vessel. Some rare cases may be explained as an infection from the mouth of a tuberculous individual, who has either applied his lips to the wound or to the instruments or ligatures used at operations (tattooing, circumcision). Such inoculation of wounds is, however, not what is generally meant by the term "traumatic tuberculosis" and I do not propose to consider it further in this place.

Cases of alleged *traumatic tuberculosis due to injuries by which tubercle bacilli could not have been introduced from without* may be clinically divided into the following three groups:—

- (A) Cases in which a decided traumatism of some kind is followed by signs of either acute disseminated miliary tuberculosis or acute metastatic localized tuberculosis.
- (B) Cases in which signs of pulmonary tuberculosis follow, or are first noticed, after a supposed injury to the lungs.
- (C) Cases in which an injury to bones, or joints, or parts of the body other than the lungs, is followed by signs of tuberculosis more or less localized to the region of the trauma.

(A) *Miliary Tuberculosis.*

The first group includes cases, fortunately rather rare, which run an acute and often very "dramatic" course. A person

(usually a young person), apparently in excellent health, may receive a chance blow or contusion of some kind and die in four or five weeks of acute miliary tuberculosis.

A well-built man, a German, aged 33 years, was admitted under my care at the German Hospital, on April 5th, 1897. He had fever (temperature 103.6° F.), vomiting, great dyspnoea and cyanosis, intense dryness of the throat, and scanty expectoration, in which a trace of altered blood pigment was at one time visible. On examining the lungs no definite signs of consolidation could be made out, but a certain amount of fine inspiratory crepitation was heard over both sides, and the case seemed to be one of acute double pneumonia before the outer parts of either lung had become hepatised. The history was that ten days before admission, when on board ship on his way back from the Transvaal, he received a blow on the left testis, which was followed by local and general symptoms. After five days his condition improved somewhat, but two days previously to admission he had a rigor and pains over the whole of the body. Except for some slight improvement soon after admission, due to rest and cessation of vomiting, his condition underwent but little change. The dyspnoea and orthopnoea increased, and the fever remained continuous till he died, practically suffocated, on April 20th, about 25 days after receiving the blow on the testicle. In the meantime, owing to the absence of any crisis, and since there were no signs of definite hepatisation of the lungs, we had come to suspect that the pulmonary disease was one of acute miliary tuberculosis. The crepitations were variable; on one occasion distinct inspiratory crepitations could be heard below both clavicles. The sputum was examined for tubercle bacilli with negative results. The urine contained a little albumin. At the necropsy both lungs were found to be engorged with blood and absolutely stuffed with miliary tubercles. There was some fibrosis from earlier disease at both pulmonary apices. The bronchial lymph glands were enlarged, and besides miliary tubercles in the pleuræ there was evidence of older pleurisy, especially over one lung. The left epididymis (*i.e.*, where he had received the blow 25 days before his death) contained a softened, caseous

nodule, smaller than a small cherry. The cerebral meninges contained no tubercles, nor were tubercles found in any other organs, except a few minute ones in the renal cortex below the capsule. The liver might, of course, have contained microscopic tubercles, though no macroscopic ones were found. The heart was distended with blood, but showed no evidence of disease. The spleen was somewhat enlarged and pulpy; it weighed nine ounces and contained one small white infarct.

In this case apparently the tubercle bacilli were by the injury set free from the caseous focus in the epididymis, and were carried by the venous blood to the right heart. Many of the bacilli were then caught up in their passage through the lungs, and thus gave rise to the miliary pulmonary tuberculosis. This theory explains why the pulmonary tubercles were practically all of about the same size, why they were uniformly distributed throughout every part of the lungs, and why there was not evidence of general miliary tuberculosis in other parts of the body. A few of the microbes had, however, passed through the pulmonary circulation, otherwise we should have found no miliary tubercles in the kidney. Moreover, by careful microscopic examination, we might have found evidence of commencing tubercles in other organs. Tubercle bacilli are supposed not to produce anatomical changes until 9 to 16 days after injection or after entering the blood-stream. This would correspond, I think, to what was found in the lungs at the necropsy, but in order to understand the whole case one must, I think, assume that the patient's condition at the time of the injury was abnormal—namely, that there was something peculiar about his blood and tissues, at all events, about his lungs—which favoured the rapid multiplication of the tubercle bacilli set free from the caseous focus in the epididymis.*

Similar cases were observed prior to the discovery of the tubercle bacillus and gave rise to various inquiries as to the possible relation between the injury and the lung disease. A remarkable

* Cf. O. R. Wild, "Ueber die Entstehung der Miliartuberculose," *Virchow's Archiv*, 1897, Vol. 149, p. 65; and H. Ribbert, "Ueber die Miliartuberculose," *Deut. Med. Wochenschrift*, 1906, Vol. 32, p. 5.

example was the following, which I have from my father, and which occurred in Germany long ago.

An apparently healthy student, about 21 years of age, received some injury to one of his ankle-joints. Shortly afterwards he fell ill with an acute pulmonary affection, characterised by frightful dyspnœa and cyanosis, and, like the previously-mentioned patient, died practically from suffocation about 25 days after the injury to his ankle. At the necropsy both the lungs were shown to be completely stuffed with miliary tubercles,* and in the ankle some caseous material was found. It was known that during childhood the patient had suffered from a disease of his ankle.

Other examples, though not all quite so typical as the two foregoing, cannot, I think, be excessively rare, *i.e.*, cases in which acute miliary tuberculosis of the lungs is set up by injury to some part of the body which happens to be the site of previous tuberculous disease. The wonder is that such cases are not more often observed. I have heard of a case of miliary tuberculosis of the lungs, as rapidly fatal as the case just referred to, following an attempt to break down adhesions in an old tuberculous joint, and I have been told also of a child about nine years of age, who died with general disseminated tuberculosis three or four weeks after an operation on a tuberculous joint. J. Orth † refers to surgeons losing patients from fatal acute tuberculosis after operations on chronic localized tuberculous lesions (in bones) in which it might have been difficult to detect the presence of any bacilli. A striking example of the kind is related by Urban ‡ in the case of a healthy-looking girl, aged 19 years, who on account of adduction from old hip disease of the right side, wished to have a "bloodless" manipulation performed to improve the position of the hip joint. The surgeon who was consulted yielded, against his own inclinations, to the importunity of the patient and her parents. By manipulation under general

* It is possible that the reason why this cramming of the lungs with tubercles is better seen in adults than in very early life is a mechanical one, and that it has to do with more elaborate development of the capillary network in adult age.

† Orth, *Berliner Klin. Wochenschrift*, 1904, Vol. 41, p. 337.

‡ Urban, *Muenchener Med. Wochenschrift*, 1899, Vol. 46, p. 346.

anæsthesia, the faulty adduction was corrected, but on the following day the patient's temperature rose to 104°F., and in five weeks she died of acute miliary tuberculosis. No old tuberculous lesions were discovered at the necropsy excepting those of the right hip joint. To explain this case, Urban mentions that of a boy aged 12 years, whose knee joint was ankylosed in a flexed position as a result of old local tuberculosis, supposed to have been cured five or six years ago. The joint was resected and was found to contain a single encapsuled caseous focus of the size of a pea; a guinea-pig, inoculated with this, died of tuberculosis in 3½ weeks after the inoculation. Had a bloodless manipulation (or wrenching) been carried out instead of the "open" operation (joint-resection) the boy might have fallen a victim to acute miliary tuberculosis as a result of traumatism to the minute caseous focus in question.

From the pathological point of view, certain cases of death from acute miliary tuberculosis (especially of the lungs) a few weeks after childbirth or abortion, may be compared with the preceding cases. F. Weil* described two such cases in young women who died almost exactly four weeks after abortion. In both cases the miliary tuberculosis affected the lungs chiefly, and the necropsy showed the presence of earlier tuberculous caseous foci in the pelvic organs. Weil thinks that in both cases the uterine tuberculosis was the cause of the abortion and the open blood vessels of the uterus constitutes the channel by which the rest of the body, especially the lungs, was "flooded" with tubercle bacilli. He refers to similar cases described by Fischel (1883), Schelling (1885), Hünemann (1893), Davidson (1899), and Westenhoeffer (1903). An analogous case is recorded by Hochhaus† in which the patient, a young woman aged 22 years, died 47 days after abortion. The necropsy showed the presence of a caseous focus in the uterus, and disseminated miliary tuberculosis of the lungs, peritoneum, &c.

Dr. C. T. Williams ‡ refers to the case of a gentleman who was

* F. Weil, *Muenchener Med. Wochenschrift*, 1910, Vol. 57, p. 359.

† Hochhaus, *Allgemeiner Aerztlicher Verein zu Köln*, April 20th, 1903.

‡ C. T. Williams, *Polyclinic*, London, April, 1900, p. 235.

thrown in the hunting field and injured several of his ribs. An acute miliary tuberculosis followed, and proved rapidly fatal. The cases in which a thoracic injury is followed at first by local pulmonary signs and much later by miliary tuberculosis really belong to Group B.

In this connection, also, mention may be made of the case of an unmarried woman, aged 31 years, who had a small tuberculous gland removed from the neck. Some months later she appeared to be suffering from pneumonia, but very soon this was found to be an acute attack of pulmonary tuberculosis, from which she died after an illness of three weeks.* Two girls, one aged 5 years and the other aged 14 months, died within a month of undergoing laryngeal intubation for diphtheria, and at the *post-mortem* examination in both cases a caseous mass was found in the larynx, together with the lesions of acute miliary tuberculosis.† In a man aged 62 years, according to the account of Aufrecht‡, a tuberculous pneumonia, supposed to be of embolic origin, supervened after massage of some old tuberculous lymphatic glands in the neck. In another case an injury to the calf muscles led to local venous thrombosis and pulmonary embolism, followed by pulmonary tuberculosis.§

Benda,|| in three cases of miliary tuberculosis following operation or injuries to tuberculous joints and bones, found that tuberculosis of blood-vessels existed. He believes that at the operation or injury a few bacilli pass into the blood-stream and lodge on the *tunica intima* of a blood-vessel. In the intima a tubercle then

* Woodcock, *Brit. Med. Journ.*, 1903, Vol. 1, p. 490.

† A Primavera, *Giorn. Internaz. delle Scienze Mediche*, 1907, Vol. 29, p. 296.

‡ Aufrecht, *Deut. Arch. f. Klin. Med.*, Leipzig, 1908, Vol. 94, p. 230.

§ F. Köhler, *Aerztl. Sachverst.-Zeitung*, Berlin, 1908, Vol. 14, p. 154. Köhler (*ibid.* p. 32) has likewise recorded a curious case of pulmonary tuberculosis developing after an injury to the skull.

|| Benda, *Verhandlungen d. deut. Gesellschaft für Chirurgie*, 1899, Vol. 28, part I., p. 48. Cf. also W. T. Longcope's description of a case of tuberculosis of the aorta in a child with general miliary tuberculosis, *Johns Hopkins Hospital Bulletin*, 1901, Vol. 12, p. 27; H. Toyosumi, "Intimatuberkel in den kleinen Lungenarterien," *Virchow's Archiv*, 1908, vol. 191, p. 403; and Thorel's communication on miliary tuberculosis with multiple tuberculous foci in the blood-vessels (*Aerztlicher Verein in Nürnberg*, May 4th, 1905).

develops, becomes caseous, and finally ruptures, liberating into the circulating blood great numbers of tubercle bacilli, which are thus distributed throughout the body and give rise to general miliary tuberculosis. H. Ribbert* thinks that in such cases the blood-stream is not, as Weigert† believed, all at once, so to speak, "flooded" with tubercle bacilli. From the results of his microscopical investigations he is inclined to suppose that relatively small numbers of bacilli reach the circulating blood at a time, but that these bacilli are able to multiply quickly, apparently because the patient is in some way "predisposed," and his tissues prove a favourable soil for the bacillary growth. "Spontaneous" disseminated miliary tuberculosis may, of course, result from rupture of tubercles into the lumen of arteries, veins, or the thoracic duct, but Huguenin,‡ when he insists that in such cases the veins in the lungs are the usual sites for the irruption of tubercle bacilli into the blood-stream, has great probability on his side.

(B) *Pulmonary Tuberculosis.*

Cases are certainly occasionally observed in which an injury to the thorax is followed after an interval by active signs of pulmonary tuberculosis. One can readily conceive that a "contusional injury" to the thorax (*e.g.*, a blow with a blunt instrument or sudden violent compression) might bruise or otherwise damage the lung and so lower its resistance as to give rise to a pneumonia or other infection if the specific cause of such infection happens to be forthcoming. That contusions of the thorax may actually injure the lungs and pleura, has been well demonstrated by Külbs in his experimental investigations in animals, to which I shall refer in Part II. of the present paper. By a contusional injury of that kind a latent tuberculous focus (and experience from *post mortem* examinations shows how frequently

* H. Ribbert, *Deut. Med. Wochenschrift*, 1906, Vol. 32, p. 5; *cf.* also O. R. Wild, *loc. cit.*

† C. Weigert, *Virchow's Archiv*, 1879, Vol. 77, p. 269, and 1882, Vol. 88, p. 307 ("Ueber Venentuberkel und ihre Beziehungen zur tuberculösen Blutinfektion").

‡ Huguenin, *Société Médicale de Genève*, July 4th, 1906.

latent tuberculous lesions are present in the lungs and thoracic lymph-glands of apparently healthy individuals*) may be squeezed or torn in such a way that tubercle bacilli are mechanically disseminated over the neighbouring lung tissue, the vitality of which is, moreover, temporarily lowered by the injury so as to render it a favourable nidus for the growth of the bacilli. In a case reported by O. Schrader † the patient, an apparently healthy man aged 29 years, was admitted to a hospital two days after an injury to his thorax, and in due course the diagnosis arrived at was pneumonia of the lower and middle lobes of the right lung. But 13 days after the accident tubercle bacilli were found in the sputum, and Schrader discusses the question whether the tuberculosis was engrafted on a "contusional pneumonia" (see later on, Part II.), or whether the lung disease was a tuberculous broncho-pneumonia from

* The statistics on this subject, as is well known, vary considerably, doubtless to some extent according to thoroughness of examination and according to the criterium of what is to be regarded as constituting a tuberculous lesion. Birch-Hirschfeld in 826 *post mortem* examinations on persons who had been killed by accidents, or who had died from acute diseases, found evidence of recent, chronic, or healed pulmonary tuberculosis present in 20·7 per cent. Brouardel, at Paris (in medico-legal necropsies) found the percentage higher. Various pathologists have found evidence of old or fresh tuberculosis in 48 to 95 per cent. of all corpses examined. Among recent work on the subject, see the percentage statistics given by H. Beitzke, of the frequency of tuberculosis in *post mortem* examination at the Berlin Pathological Institute, *Berliner klin. Wochenschrift*, 1909, Vol. 46, p. 388. References are given by Beitzke to other recent work on the subject. It must, of course, be remembered that very many cases, though tuberculous from the strict pathological point of view, never become tuberculous in the ordinary clinical sense of the term. The presence of latent tuberculous foci is of great importance, though in the ordinary course of events they may remain quiescent and become obsolete. Many of us are doubtless tuberculous from the strict pathological point of view, even though we enjoy good health, and a contusional injury, if it involves our "weak spots," that is to say, our "*tuberculous spots*," may suddenly render us tuberculous from the ordinary clinical point of view as well as from the pathological point of view. These considerations serve likewise to explain why muscular men, such as athletes, runners, football-players, boxers, and persons who live healthy open-air lives, and who, owing to their splendid physique, might be supposed to be specially resistant towards tuberculosis (*but are, it must be remembered, peculiarly liable to contusional injuries and strains*), sometimes surprise everyone by more or less suddenly falling victims to tuberculous affections, especially of the lungs.

† Schrader, *Berliner klin. Wochenschrift*, 1897, Vol. 34, p. 1001.

the commencement. Great improvement in the patient's condition subsequently took place. C. Theodore Williams* has recorded the similar case of a merchant vessel's mate who fell from the mast to the deck and fractured three ribs. This injury was followed by the rapid development of well-marked symptoms and physical signs of phthisis (hæmorrhage, expectoration and cavity formation), upon the injured side of the chest. He made, however, a quick recovery and returned to his occupation. F. de A. Nogués† has described the case of a criminal who, a few days after having been punished by 150 lashes, suffered from cough, dyspnœa and hæmoptysis. Pulmonary tuberculosis developed, and it is stated that the anterior parts of the lungs were very little affected in comparison with the posterior parts, in comparison, that is to say, with the parts which may be supposed to have been chiefly affected by the traumatism. Nicoletti‡ tells of a labourer, aged 34 years, who after receiving a severe blow on the right side of the thorax, suffered from hæmoptysis. In about two weeks' time he was able to return to his work, but soon developed signs of tuberculosis at the apex of the right lung, and tubercle bacilli were found in his sputum. A hammerman, aged 44 years, with tuberculous family history, whose case is described by J. Weir, § developed signs of tuberculosis of the right lung a few weeks after receiving a blow on the right side of the chest, but some months later he seemed to be on a fair way towards recovery.

Though many more illustrative cases of this class might be referred to, I am inclined to think that examples of pulmonary tuberculosis which seem surely to be connected with an injury to the thorax, must on the whole be very rare. Doubtless, cases in which an already obvious pulmonary tuberculosis is clearly aggravated by such a (local) injury are commoner than cases in which the injury appears to have actually called a pulmonary tuberculosis

* C. T. Williams, *Polyclinic*, London, April, 1900, p. 235.

† Nogués, Annotation in the *Lancet*, London, May 26th, 1888, p. 1044, from the *Boletín de Medicina y Farmacia* (Barcelona).

‡ See V. Nicoletti, at the Societa Lancisiana at Rome, July 23rd, 1904, quoted in the *Muenchener Med. Wochenschrift*, 1904, Vol. 51, p. 1939. Cf. A. Ranelletti's case, described at the same meeting, quoted *ibid.*, p. 1939.

§ Weir, *Brit. Med. Journ.*, 1903, Vol. 1, p. 1196.

into existence. R. Link* gives the case of a strongly-built brewer's drayman, aged 46 years, whose thorax was squeezed between two waggons. He remained three days in bed, but on the fifth day after the injury commenced work again, though suffering from dyspnœa and pains in his back. About six months later attacks of hæmoptysis commenced and he developed signs of active pulmonary tuberculosis at the upper part of the left lung; at the base of that lung there were signs of old thickened pleura. In this case there were grounds for supposing that the injury may have exerted an evil influence on a previously quiescent tuberculosis. E. Mosny† mentions the case of a workman who whilst at work fell and fractured some ribs. This was followed by pleurisy, acute manifestations of pulmonary tuberculosis, and death. But the man was known to have had pulmonary tuberculosis before the accident and the court naturally took this fact into consideration in deciding on the amount of compensation to be paid to the victim's widow and children. On the other hand, had it not been for the accident, the man would have probably lived and earned money for several years.

(C) *Tuberculosis of Bones, Joints, &c.*

The question of a localised tuberculosis developing in the region of, and as the result of, an injury (by which no direct inoculation could have been effected) has been chiefly discussed with reference to tuberculosis of the bones and joints. In these cases great discretion should obviously be observed in attaching importance to histories of injury, for with regard to such exposed parts as the knees, ankles and long bones of the extremities, a history of some slight traumatism or other is nearly always forthcoming.

It may be remembered that many experiments on animals have been carried out to ascertain whether the localization of the manifestations of artificially produced general septic conditions could be determined by local traumata (without local inocula-

* Link, "Verein Freiburger Aerzte," June 30th, 1905. *Muenchener Med. Wochenschrift*, 1905, Vol. 52, p. 2202.

† Mosny, *Presse Médicale*, Paris, September 6th, 1902, p. 858.

tion*) that is to say, whether the site of the local septic lesions (when the circulating blood contained septic microbes) could be determined by contusions, injuries to bones, &c. The results of such experiments were to a great extent affirmative, and have thrown much light on the pathogeny of diseases like acute infective or septic osteomyelitis.† Similar experiments have been carried out with regard to the pathogeny of tuberculous diseases of bones and joints.

Schüller ‡ injected tuberculous material (tuberculous sputum, material derived from tuberculous lungs, lymphatic glands, etc.) through a tracheotomy wound into the lungs of dogs and rabbits, and on the same day injured one of the knee-joints of the animal experimented on. In this way he apparently succeeded in producing not only general tuberculosis, but also tuberculous synovitis of the injured joint, accompanied sometimes by tuberculous osteomyelitis in the neighbourhood. Later on F. Krause§ injected pure cultures of tubercle bacilli subcutaneously into guinea-pigs and intravenously into rabbits. Directly before or immediately after the inoculation, or after a variable lapse of time, a joint was injured or a bone broken. The fractures in every case healed by the formation of bony callus without the development of local tuberculosis, but many of the injured joints became tuberculous (15 out of 44 in guinea-pigs, 14 out of 28 in rabbits); the uninjured

* In regard, however, to the possibility of septic organisms being sometimes introduced into the body at the time of the traumatism through unnoticed abrasions and scratches in the apparently unbroken skin see Durlacher's paper on acute osteomyelitis in the *Muenchener Med. Wochenschrift*, 1904, Vol. 51, pp. 1689-1691.

† For an excellent summing up of this question see Jordan, *Muenchener Med. Wochenschrift*, 1901, Vol. 48, p. 1745. It must be remembered, however, that most cases of acute septic osteomyelitis after contusion (or other injury without open wound) might be equally well explained by the awakening to activity of septic microbes previously resting in a quiescent and latent condition in the bone-marrow.

‡ Max Schüller, "Experimentelle und Histologische Untersuchungen über Entstehung und Ursache der skrophulösen und tuberkulösen Gelenkleiden," Stuttgart, 1880.

§ F. Krause, "Die Tuberculose der Knochen und Gelenke," Leipzig, 1891, pp. 80—102.

joints did not become tuberculous excepting in a single instance (one of the rabbits).

Lannelongue and Achard, in a work on "Traumatism and Tuberculosis,"* found that it was not so easy to determine the site of a tuberculous process by experimental traumatism as one would surmise it to be from the account of Schüller's experiments. By various methods they inoculated guinea-pigs with tubercle bacilli, and directly afterwards, or later on, produced local injuries of various kinds in joints or bones. The animals died 8 to 232 days after the injury, and none of the injured joints were found to be tuberculous. Friedrich† introduced cultures of tubercle bacilli of low virulence through the carotid artery into the left ventricle, and thus succeeded in producing a tuberculous joint affection in rabbits, analagous to that in human beings, which took four to nine months to become clinically manifest. But it was remarkable that the tuberculosis in Friedrich's rabbits picked out the joints not subjected to traumatism rather than those in which he had produced an experimental injury. Similarly, B. Honsell,‡ who employed cultures of tubercle bacilli of high virulence, and also some of low virulence, came to the conclusion that in his experiments the localisation of tuberculosis was not determined by traumatism.

E. Salvia,§ however, another of those who made experiments of this nature, injected a virulent culture of tubercle bacilli intravenously into rabbits (by a vein of the ear) and subjected them to different forms of *slight* traumatism. He found that in the thin, flat spongy ribs the trauma always decided a localisation of tubercle, which involved the whole thickness of the bone. In regard to the subjacent lung there were more tubercles (and more rapid growth of tubercles) in the parts nearest to the injured ribs. No special localisation of tubercles resulted from slight traumata to the bones of the extremities. Definite localisation of tuberculous changes was

* Lannelongue and Achard, "Sur le Traumatisme et la Tuberculose," *Comptes rend. de l'Acad. des Sciences*, Paris, 1899, Vol. 128, p. 1,075.

† Friedrich, *Deut. Zeitschrift für Chirurgie*, Leipzig, 1899, Vol. 53, p. 512.

‡ Honsell, *Beiträge zur klin. Chirurgie*, Tübingen, 1900, Vol. 28, p. 659.

§ E. Salvia, *Il Policlinico*, Rome, 1904, Vol. XI. (Sezione chirurgica), p. 367.

observed in the soft tissues of the articulations after slight chemical injuries.

Jordan,* in an excellent summary of the results of experimental investigation of the kind (a summary that I have largely made use of) points out that in animals the positive results of Schüller and Krause were obtained on the basis of a disseminated miliary tuberculosis, whilst in cases of alleged traumatic tuberculosis of joints and bones in human beings there is generally no disseminated miliary tuberculosis present.

We conclude, like Honsell † and Jordan conclude, that so-called traumatic cases in man must be explained as the rendering manifest and active of a pre-existing tuberculosis, however limited in extent, quiescent and latent such pre-existing tuberculosis may have been. Mr. S. G. Shattock, in a similar way, I believe, explains the onset of tuberculous bone disease after local injuries in children. He thinks that there is a gradual leakage of tubercle bacilli from tuberculous lymphatic glands, and that some of the escaping bacilli are carried by the blood-stream and lodged in the bone-marrow, where he supposes they may remain latent until some injury to the bone lowers the tissue resistance, thus favouring the growth of the pathogenic microbes and setting up local tuberculous lesions.

By Urban ‡ also an explanation of the same kind seems to be accepted. He relates the case of a labourer, aged 47 years, who was supposed to have previously enjoyed good health. On August 26th, 1895, he slipped and sprained his right ankle. He continued at his work, however, till the end of the month, but had then to give up owing to increasing pain. Excepting this local spontaneous pain and tenderness to pressure no definite signs of disease were discovered till September, 1896, when an operation was performed on the right foot and the presence of undoubted tuberculous changes discovered. It became afterwards necessary to amputate in the

* Jordan, "Ueber die Entstehung von Tumoren, Tuberkulose und anderen Organerkrankungen nach Einwirkung stumpfer Gewalt," *Muenchener med. Wochenschrift*, 1901, Vol. 48, pp. 1741—1746.

† Honsell, *loc. cit.*

‡ Urban, *Muenchener Med. Wochenschrift*, 1899, Vol. 46, p. 346.

right leg. Urban accounts for the slow development of the signs of disease in this case by supposing that a small tubercle in the middle of one of the bones of the foot, hitherto latent, was disturbed and awakened to activity at the time of the injury, but that no symptoms, excepting pain and tenderness, developed until the tuberculous process reached the outer surface of the bone, about a year later.

E. Pietrzikowsky * thinks that about 20 per cent. of all (clinically obvious) tuberculous affections of the joints and bones are causally connected with some traumatism, which is very rarely a fracture or a dislocation, but usually an injury of lesser degree, such as a sprain or a bruise. (Compare the results of bone-fractures in Krause's experiments and those of slight traumata in Salvia's series already alluded to). In what he regards as genuine examples of such traumatic tuberculosis, the interval between the accident and the development of obvious signs of tuberculosis is neither very short (few weeks) nor very long (not more than a year at the outside). Moser † concludes that a traumatic origin of articular tuberculosis can never be accepted as proved, but only as being more or less probable. He thinks the alleged injury should have been a considerable one and should have been immediately followed by symptoms of some kind, which symptoms, however, need not necessarily persist until the appearance of definite signs of tuberculosis. The interval between the injury and the manifestation of tuberculosis must be at least four to six weeks, and the shorter the interval, the less likely is the joint tuberculosis to be traumatic. According to Moser, the pathological explanation may sometimes be that the injury, by lowering the power of resistance, prepares the way for the development of tuberculosis, but is more usually that an old quiescent, and latent, tuberculous focus is rendered active and obvious as the result of the injury.

Needless to say, an already manifest, but chronic, tuberculous process can be rendered acute as the result of traumatism. Such a

* E. Pietrzikowsky, *Zeitschrift für Heilkunde*, Vienna, 1903, Vol. 24 (Surgical portion), pp. 187-272.

† Moser, *Aerztl. Sachverständigenzeitung*, Berlin, 1906, Vol. 12, pp. 69, 91.

transformation (exacerbation) of a chronic local tuberculosis into an acute one may occur within a few days or weeks after an injury, and a very rapid development of signs of acute local tuberculosis should always give rise to a suspicion that a clinically obvious (though chronic and possibly quiescent) tuberculous affection was present before the injury was received.

Tuberculous lesions connected with local traumatism (contusions, etc.) may doubtless occasionally occur also in other parts of the body. Salvia,* in his experiments already alluded to, thought that he could localise tuberculous changes in the livers of rabbits (after intravenous injection of a virulent culture of tubercle bacilli) by repeated slight percussion over the abdomen below the costal margin. Similarly, Orth† squeezed the kidney of a rabbit, into whose ear he then intravenously injected a culture of tubercle bacilli. After some time a post-mortem examination was made on the rabbit and it was found that the tuberculous changes were more pronounced in the contused kidney than in the other. R. Luecke‡ records a rather different kind of case, namely, one of tuberculous peritonitis in a boy, aged twelve years, which was apparently due to local traumatism. The boy fell down whilst skating, and another boy trod on his abdomen. Acute abdominal symptoms followed, and fifteen days later, when a laparotomy was performed, a condition of diffuse tuberculous peritonitis was discovered. A subsequent *post-mortem* examination demonstrated the presence also of caseous mesenteric lymph-glands and some old peritoneal adhesions. In a case like that it is of course quite easy to understand how a traumatism of the alleged kind can set up diffuse tuberculous peritonitis.

What part does traumatism play in particular cases of alleged traumatic tuberculosis?

The answer to this question must often be difficult and must generally be a doubtful one. We have to admit that in all cases

* Salvia, *loc cit.*

† Orth, "Gesellschaft der Charité-Aerzte," Berlin, June 27th, 1907, *Berliner klin. Wochenschrift*, 1907, Vol. 44, 1394.

‡ Luecke, *Berliner klin. Wochenschrift*, 1903, Vol. 40, p. 409.

(excepting those in which an inoculation with tubercle bacilli from without occurs—a class which we are not now dealing with) tuberculosis of some kind, whether latent or not, must have been present (that is to say, tubercle bacilli must have been present) before the trauma was received. In short, cases of contusional traumatic tuberculosis may, as I have already pointed out, be roughly divided into two classes: those in which obvious tuberculosis existed, and those in which no tuberculosis, recognizable by ordinary clinical methods, existed, previously to the traumatism. In the former class of cases the traumatism merely accelerated the progress of (produced an exacerbation of) an already existing, that is to say, clinically manifest, disease, and it is quite likely that the disease would have ultimately progressed even in the absence of traumatism. But in the second class the patient's condition previously to the traumatism is accepted as having been just like that of any other apparently healthy person, and (knowing as one does that very many apparently healthy individuals carry with them, hidden away in their bodies, minute tuberculous foci which never give rise to serious trouble), one has no right to suppose that the latent tuberculosis in question would, under ordinary circumstances (*i.e.*, apart from the results of traumatism, &c.), ever have progressed sufficiently to become recognizable by ordinary examination, or to give rise to any serious trouble. In any particular case, therefore, we have to consider the following questions:—

- (1) Is there evidence that any clinically recognisable tuberculosis was previously present or not? If there was already obvious tuberculosis, what was its probable distribution, extent and activity at the time when the trauma was received?
- (2) Is it likely from our pathological knowledge and from our study of the individual case in question that the alleged traumatism played any part at all in the outbreak of tuberculosis?
- (3) Is the whole of the illness to be regarded as practically the direct or indirect result of the traumatism? If, on the other hand, the injury merely caused an exacerbation of

a previously existing (clinically obvious) tuberculosis a medical estimate of the extent of the actual damage done by the injury should be as far as possible arrived at; that is to say, the difference between the probable condition without any traumatism and the condition actually present as the result of the injury should be estimated.

In order to answer the last two questions a decision in regard to the first question must be arrived at. Then the nature, position, extent and violence of the traumatism have to be investigated; the lapse of time between the injury and the onset of symptoms of tuberculosis has to be ascertained; and one has to consider whether the existing tuberculous lesions and symptoms are of a nature pathologically likely to have followed the traumatism in question.

I am quite aware that the pathological answer in many cases will be by no means identical with the medico-legal (or rather legal) answer. In every case, however, the exact pathological consideration of the data should be required. Of special importance is the knowledge that an outburst of traumatic tuberculosis (apart from that due to actual inoculation) is, pathologically speaking, according to the experimental and clinical evidence already referred to, the result of the disturbance of a pre-existing, though generally unrecognized and often quiescent, tuberculous focus. It is therefore quite conceivable that a man may have an encapsuled caseous tuberculous nodule not at the site of the main injury, but at some distance from it (for instance, on the opposite side of the thorax *), and that the injury may yet be sufficient to rupture it (in the case of the lungs violent respiratory movements connected with the excitement and accident might play a part in this), so that the tuberculous disease may first show itself at some distance from the site of the alleged injury.† (I do not, of course, here refer to disseminated miliary tuberculosis following injuries.)

* Cf. Port, Aertzlicher Verein in Nürnberg, July 18th, 1907, *Muenchener Med. Wochenschrift*, 1907, Vol. 54, p. 2013.

† *Vide* Port, *loc. cit.* Rare cases of acute tuberculous broncho-pneumonia following abdominal operations performed under general anæsthesia may be theoretically explained in various ways. Cf. the case recorded by Mackey, Macdonald, and Sutherland in the *British Medical Journal*, 1910, Vol. 1., p. 745.

PART II.

TRAUMATIC PNEUMONIA, TRAUMATIC PLEURISY, &c.

I shall not stop to discuss such conditions as traumatic pneumothorax, hæmothorax, pyopneumothorax, hæmopneumothorax, pleurisy, empyema, septic pneumonia and pulmonary gangrene, when obviously due to penetrating wounds and their complicating infections, nor shall I deal with pneumonia or pulmonary gangrene following operations or injuries to the upper air passages, such as cases due to the introduction into the bronchi of foreign bodies or septic particles during dental operations under general anæsthesia. There are numerous cases known of pneumonia-like symptoms following operations on the pelvic viscera, herniotomy, operations and injuries to the abdomen, operations on varicocele and varicose veins of the extremities, fractures of long bones, &c. Some of these may be examples of ordinary pneumonia due to the patient's diminished resistant power as a result of shock, &c. (in the same way psychical shocks may probably favour the development of pneumonia in susceptible individuals), others may be due to the sucking in of septic particles from the mouth during general anæsthesia, and other cases may be in reality of embolic origin. But I cannot enter upon such cases here. Nor need I consider traumatic pneumothorax and traumatic hæmothorax when obviously merely mechanical results of fracture of ribs or severe contusional injuries. In regard to cases of "traumatic pneumonia" in the most limited sense, that is to say, "contusional pneumonia" (which may or may not be associated with fracture of ribs), I shall first of all refer to the results of some recent experiments on animals by Külbs.*

Külbs, in a series of dogs, investigated the effects of more or less severe contusions of the thorax and found that considerable changes, mostly hæmorrhages of greater or less extent, had been produced in the lungs, together with slight injuries to the

* Külbs, "Lunge und Trauma," *Arch. f. exper. Path. u. Pharm.* Leipzig, 1910, Vol. 62, p. 39.

pulmonary pleura. In a few cases, after very violent blows on the thorax, the lung tissue and pleura were found considerably torn. Sometimes the whole of a lobe was infiltrated with extravasated blood, sometimes the hæmorrhagic infiltration was nodular in distribution, mostly in the deeper portion of the lobe. In the animals killed some days or weeks after the injury the process of repair, the development of granulation tissue, and then of ordinary scar tissue, was observed. The pulmonary changes were not always found limited to the region of the thorax where the blow had been received. This was more noticeable when the front or side of the thoracic wall than when the stronger and less yielding dorsal portion had been struck. Only in the former case could a "contrecoup" effect be obtained, for instance, hæmorrhage in the right upper lobe when the traumatism was applied over part of the left lung. Reineboth's* results in rabbits differed somewhat from those obtained by Külbs in dogs, and Külbs has satisfied himself by actual experiment that in rabbits the ribs are less elastic and more fragile than in dogs.

Contusional Traumatic Pneumonia.

Though actual pneumonia has not been produced in animals comparable to "traumatic pneumonia" in human beings, nevertheless, as Külbs points out, changes such as he produced in the pulmonary tissue by traumata, must favour the local growth of pathogenic microbes, should such find their way to the injured parts. Traumatic pneumonia in human beings may, indeed, be well-explained as due to the growth of pneumococci or other microbes previously present in the respiratory organs in a quiescent and latent condition, but awakened to activity as a result of the trauma and the lowered resistance of the patient's tissues. As pointed out by Litten,† who, in 1881, apparently first introduced the term "contusional pneumonia" (*Contusionspneumonie*), it

* Reineboth, "Experimentelle Studien über Brustcontusionen," *Deut. Arch. f. klin. Med.*, Leipzig, 1901, Vol. 69, p. 144.

† M. Litten, "Kontusionspneumonie," *Deut. Med. Wochenschrift*, 1907, Vol. 33, p. 499. See also his first paper, *Zeitschrift für Klin. Med.*, Berlin, 1882, Vol. 5, p. 26 and p. 48.

does not seem clear that all cases clinically classed as traumatic pneumonia are really examples of typical microbial pneumonia. The signs in some cases may be due to hæmorrhagic infiltration, such as Külbs produced by contusion of the thorax in animals, possibly associated with serous or hæmorrhagic effusion into the pleura. In cases that I have seen myself (and can refer to through the kindness of my surgical colleagues, Dr. E. Michels and Dr. J. P. zum Busch) I have had considerable doubt as to the precise nature of the change.

A boy, Albert G., aged eight years, was run over by a cart on the morning of March 13th, 1909, and was brought at once to the German Hospital. On admission his temperature was 97° F., but rose to 101·2° F. in the evening. On the evening of the next day (March 14th) the temperature was 103·4° F., and then began gradually to fall. There was some bruising of the back of the thorax, but no evidence of fractured ribs was obtained either by ordinary examination or by a skiagram taken later on. When I saw the boy on March 15th there was a good deal of dyspnœa, general uneasiness, and cough. Resonance to percussion was slightly impaired in the lower part of the right intrascapular region and there were bronchitic sounds at the bases of both lungs. He complained of pain in the lower left part of the front of the chest. By March 18th the temperature had fallen to normal and he had no pain, but he still coughed. Pulse 116, respiration 34, to the minute. There was then decided impairment of resonance and diminution of breath-sounds at the base of the right lung, and occasional dry bronchitic sounds could still be heard at both bases. After that the boy's general condition gradually improved, and he had no further fever excepting on one occasion (100° F. on the evening of March 22nd). For some time there was relative deficiency of the inspiratory breath-sounds over the right lung. The signs in this case might perhaps all (including the fever) be accounted for on the supposition of traumatic hæmorrhagic infiltration (as in Külbs' experiments) in both lungs, but especially in the right one.

A little girl, Edith S., aged 4½ years, had her chest run over on

September 16th, 1898, and was brought on the same day to the German Hospital, where she was admitted under Mr. R. W. Parker. No ribs were broken and there was not much external bruising. On admission, the temperature was 102° F, and examination of the chest revealed bronchitic signs only. On September 19th, however, there was dulness to percussion, with very clear bronchophony and a few scattered crepitations over the whole of the lower back portion of the left lung. There was less fever than on admission, but the patient was in a semi-conscious condition, and was often "grinding her teeth." By September 22nd, the abnormal signs in the chest had practically cleared up, there was no fever in the morning, and the patient was quite cheerful.

In a little girl, aged 8 years, whose case has been recorded by W. P. Cockle,* great pain in the right side was complained of when on one occasion her father suddenly lifted her up by the thorax. She said something had "given way." The temperature two hours later was 100° F., and the breathing was very frequent. Next day the temperature was 103° F., and the pain continued. Later on pleuritic friction developed at the site of the pain. After a week the temperature gradually fell to normal, the friction and pain disappeared and the child recovered completely. Here again the injury may have produced not a pneumonia in the strict sense of the term, but a pulmonary change similar to those experimentally produced by thoracic contusions in dogs (Külbs).

In this connection it is interesting to note that Sir J. E. Erichsen† claimed that cases of so-called "traumatic pneumonia" were either mere bruising of the lung and not dangerous or else were due to an inflammation of spreading septic character and dangerous to life.

I can certainly instance one fatal case (at St. Bartholomew's Hospital, 1892) in which the post-mortem examination showed grey hepatisation of part of one lung. The patient, a man 58 years of age, had several ribs on his right side fractured by being knocked

* Cockle, *Brit. Med. Journ.*, 1902, Vol. 1, p. 1333.

† Erichsen, *Science and Art of Surgery*, London, Ninth Edition, 1888, Vol. 1., p. 860.

down and run over by a cab. On admission immediately afterwards his temperature was found to be 100° F. He was a gouty and bronchitic subject and his urine contained albumen. Afterwards there was impaired resonance at the base of the right lung and later on signs of consolidation of the right apex. He had considerable fever (for some time about 102°--104°F., once higher), became delirious, and died on the 15th day after the injury. The necropsy showed grey hepatisation of the upper part of the right lung. This seems to have been a case of actual pneumonia supervening on severe contusion of the lung.

E. Bloch* mentions the case of a man aged 41 years, who when suffering from slight bronchitis fell down in such a way as to severely strike the lower posterior part of the right half of his thorax. The next day his temperature suddenly rose to 103.1° F. and the physical signs of pneumonia developed at the base of his right lung. A kind of crisis occurred on the fourth day of the illness, and later on some pleuritic signs cleared up spontaneously.

C. Rubino† speaks of a man, aged 53 years, who, as he was commencing to carry a very heavy stone on his right shoulder, suddenly felt pain in the region of his right scapula. This pain was accompanied by cough and dyspnoea, and 48 hours later the first signs of pneumonia appeared on the right side. Litten‡ attaches special importance to the lifting of heavy weights in the ætiology of "contusional pneumonia," he knows of several cases in which fatal pneumonia supervened soon after such exertions. Thus, when a heavy carriage had just been lifted, one of the men employed in lifting it fell down and had to be carried home. Two days afterwards he was found to have pneumonia, which soon proved fatal. Another man, when helping to lift a heavy stone flag with a lever, complained of pain in the chest and spat up some blood. Signs of pneumonia developed two days later and fatal gangrene of the lung followed.

An interesting case of doubtful nature that occurred not long

* E. Bloch, *Muenchener Med. Wochenschrift*, 1898, Vol. 45, p. 967.

† C. Rubino, *Gazzetta degli Ospedali*, Milan, 1904, Vol. 25, p. 837.

‡ Litten, *loc. cit.*

ago in England was that of a man who was violently thrown when hunting and fell heavily on his left shoulder and side. He then rode home wet through. Signs of pneumonia developed very soon afterwards, and he died seven days after the injury.

Many other cases, resembling some of the preceding ones, have been recorded, and a good many papers on the whole subject of traumatic pneumonia have appeared from time to time.*

In regard to the symptoms of traumatic pneumonia it is probable that in the more favourable cases there is less expectoration than in ordinary non-traumatic pneumonia. Early hæmoptysis, due to the mechanical injury of the lung, seems to be a characteristic of some, but by no means of all, cases. External bruising is of course often present, but is by no means necessarily a marked feature, and may be absent altogether. A crisis may occur, as in typical examples of ordinary pneumonia. Recovery is the rule, but the proportion of fatal cases, according to Litten, is considerable. Chronic bronchitis, alcoholism and degenerative conditions of the blood-vessels doubtless act as predisposing causes † of contusional pneumonia, and favour the severity of the attack. The presence of latent pulmonary tuberculosis perhaps also favours the occurrence of contusional pneumonia. M. Wassermann ‡ records the case of a tailor, aged 24 years, with quiescent pulmonary tuberculosis. He received a fracture of the sternum, and this was apparently followed both by lobar pneumonia and by miliary tuberculosis.

With reference to the diagnosis of contusional pneumonia, Litten claims that a traumatism of suitable kind should be ascertained to have preceded the lung symptoms, that there should naturally be no evidence that the patient had pneumonia before the traumatism, and that the time intervening between the injury and the onset of

* Souques ("Pneumonie contusive," *Presse Médicale*, Paris, March 3rd, 1900, p. 109), refers to much of the older literature on the subject. For numerous references to the literature see especially R. Stern, *Ueber traumatische Entstehung innerer Krankheiten*, Jena, 1896; and the recent epitome of the subject by L. Mueller in *Centralblatt f. d. Grenzgebiete d. Med. u. chir.* Jena, 1910, Vol. 13, Nos. 1-5.

† Cf. F. Pancrazio, Acad. Med. di Padova, March 31st, 1907, *Policlinico*, Rome, 1907, Vol. 14 (Sezione pratica), p. 781.

‡ Wassermann, *Charité-Annalen*, Berlin, 1899, Vol. 24, p. 184.

the pneumonia should not be too long. The condition of the patient during the intervening period varies in different cases. Patients brought to the hospital after the injury may go home, and may even commence work again before the development of signs of pneumonia. I know of a case in which the patient was first of all brought to a hospital in a drunken condition with a scalp wound. This was seen to and he went away, but a policeman brought him back again, saying that he complained of a pain in his right side. He was then treated for fractured ribs and allowed to go home; but he could not sleep, and the pain in his side continued. On the third day he was admitted and looked like an ordinary case of pneumonia, with signs of consolidation of the lower part of the right lung, orthopnoea and rusty expectoration. Recovery took place with a sort of crisis on the seventh day.

The shortest interval between the traumatism and the development of pneumonia, according to Litten, is ten hours; this was the interval in a case he observed himself, and likewise in a case of Aufrecht and in another one of Birch-Hirschfeld. Stern fixed the interval at between a few hours and four days. The average is, perhaps, about two days. Litten now thinks that an interval of six days is possible, but longer periods have been recorded. For instance, in one case von Leyden regarded an interval of 14 days as not impossible. The case was that of a workman who fell from a ladder so as to receive a blow on his thorax. He continued at work for 14 days, and then developed pneumonia, from which he died a few days later. In the discussion on Litten's paper, Fürbringer mentioned that in one case he had also recognized a connection between pneumonia and trauma though the interval between the two was as long as 14 days; in the case in question the injury was soon followed by signs of pleuritis, though the pneumonia did not apparently develop till 14 days had passed.

Traumatic (Contusional) Pleurisy.

One or two cases in which contusional pleurisy was associated with contusional pneumonia have already been referred to. It must likewise be remembered that the physical signs of pleurisy may

be due to slight tearing of the pleura from severe contusions of the thorax, such as occurred in some of the dogs experimented on by Külbs.* A very important point is that signs of pleurisy after injuries to the chest may be due to tuberculosis. Pleurisy, with or without fluid effusion, has certainly sometimes been associated with signs of pulmonary tuberculosis following thoracic traumatism. Such cases belong really to the first part of my paper, and from the pathological point of view, the connection between contusions of the chest and tuberculous pleurisy may be explained in the same way as that between contusions and pulmonary tuberculosis.‡

DISCUSSION.

THE PRESIDENT: I am sure we are all very much indebted to Dr. Parkes Weber for this very elaborate, interesting and important paper, which we shall all be glad to study more thoroughly at our leisure. In the discussion I hope our members will bring forward facts from personal experience bearing on the subject. My own experience has not been large in this connection. Of course one has heard about many cases of traumatic tuberculosis; but they seldom come before one at a chest or general hospital. On the other hand, cases of traumatic pneumonia are not uncommon.

The whole subject of the relation of injury to pre-existing disease is of great interest. It is difficult to tell how far an injury is the cause of some acute attack which occurs just after it. The severity of the illness may be out of all proportion to the injury. In that connection I should like to mention a case which impressed itself on my mind some time ago. A young man was brought to me as he was somewhat out of health, but a most careful examination showed nothing definite in the way of physical signs. It so

* Külbs, *loc. cit.*

‡ See Part I. of this paper.

happened, however, that before he left my house he was taken ill with shivering and vomiting, and he went home and straightway developed acute pneumonic tuberculosis, from which he died a few weeks later. How far that attack was connected with the examination no one can tell. Injury may be mental, such as fright or shock, as well as physical. When the conditions of the body are ready for it, a small stimulus only is needed to bring about an explosion. Even the mental perturbation produced by a medical examination by two doctors might suffice. It is the spark which fires the train. One has seen cases of acute miliary tuberculosis and cases of acute meningitis which have followed fright as well as injury. With regard to trauma in its various forms, I think there is a connection between its action and the tuberculin reaction. An injury or a mental shock probably induces an auto-inoculation, and the effects are very much the same as those of an overdose of tuberculin, which we know is capable of lighting up a latent focus of tubercle into activity.

DR. THEODORE WILLIAMS: I am sorry to say that it is some time since I had the pleasure of attending this Society, but I have always taken great interest in it, and I am very pleased to be present this evening to hear one of the best papers I have heard for some time. In one way I am proud of it, because it is written by my former house physician. The only fault I have to find is that it is so good a paper that it might have been divided into two, one dealing with traumatic tuberculosis and the other dealing with contusional pneumonia and pleurisy. That would have made two excellent papers and have probably evoked two very good discussions. However, we must not complain because the present paper is a very rich one. I thank the author very much for his notice of some of the cases I have mentioned at the Polyclinic from time to time, and I should like to add to them two more that really came from my late father's note-books. It may be interesting to the members to know that the first time my attention was drawn to the subject was when I was writing a thesis for my M.D. degree at Oxford in 1869. Recently I came across a copy of that old thesis in which I

dwelt particularly on injuries to the chest and other parts of the body as causes of tuberculosis, and there are two cases in the thesis, one of which would come into Dr. Parkes Weber's first class. A gentleman, aged twenty-three, whose mother died of consumption, was seen on April 13th, 1861. Five years previously his left eye was destroyed by an accident, and two months later cough and expectoration came on and he had hæmoptysis amounting to two ounces. At that time he presented well-marked signs of tubercle in the right lung. He went on very well, but it seems to me that this was a case that started from the decided injury to the eye. The other case was a steward on board one of the Royal yachts. He was seen on July 9th, 1863. Eight years previously he had fallen on the left side of his chest, and had experienced occasional pains there ever since. He had had no cough or expectoration, but had always had a pain in his chest, and then suddenly he developed a cough and had loss of flesh and well-marked physical signs on the right side and also hæmoptysis. He went on like that for some time. I do not think a cavity formed, but there was very well-marked tubercular consolidation. That case would come into Dr. Weber's second class.

I was very much interested in the part of the paper where Dr. Parkes Weber dwells on the probability of signs of tuberculosis being *developed* in the lungs after an injury, and I think he comes to a very right conclusion there. Most of them were due to an old nidus in the lung. Over and over again I see cases where there has been some old history and then there has been an injury of some sort, a fall from a horse, or a blow, and symptoms gradually develop. It seems to me that both surgeons and physicians ought to be careful about these cases. It is a very common thing now in surgery, if there is a tuberculous joint, to excise it, and surgeons think very little of dealing with such cases, although very often they may do a great deal of harm by letting loose the bacilli. You may get an acute tuberculosis supervening. We physicians must also bear it in mind. In questions of examination I think we take pretty good care.

Another thing is that where there is some history of tuberculosis

or some old lesion, or a history of hæmoptysis, and such like, we must give some sort of caution to the patients. If I found a patient who had had well-marked disease of the right lung, although I could not find very many signs remaining, I should decidedly caution him against the kick of a gun in shooting, or anything of that sort, or boxing. There are a number of these occurrences which might evoke fresh activity in some old nidus.

Dr. Parkes Weber has alluded to infection—I thought we had had enough of that subject at the Medical Section of the Royal Society of Medicine the other day. I should be sorry to tie myself to the opinion that most of us had had tuberculosis, for instance. I think even the German statement, “Jedermann hat am Ende ein bischen Tuberculose,” is rather doubtful, but there is no doubt that there are a great many cases of quiescent tuberculosis. The worst of it is that the evidence in many of the cases is *post mortem* evidence, and that, as a rule, means hospital *post-mortem* evidence. The number of *post-mortems* done in private practice is very few. In certain hospitals in Paris a large portion of the bodies, when examined, show signs of old mischief at the apices, and I believe my father and Dr. Hope were the first to publish the fact in England, taking it from the famous Andral's *post-mortem* examinations. At that time an enormous number of *post-mortems* were done in one hospital every day, and my father and Dr. Hope attended, I think, for a year, and gathered a large amount of valuable information which they turned to account. I think that something like 50 per cent. of the cases shewed signs of old tubercle, but it should be remembered that the patients belonged to the worst classes in Paris. The people who die in hospitals are the people who are likely to contract diseases, and I do not think you can conclude that all the private patients who die have tuberculosis; I think that an exaggeration. I do not know whether I have tubercle in either of my apices; it would not make me very unhappy if I had, at my time of life. I do not think we have evidence enough to make such a general statement as my friend, Prof. Osler, hazarded the other day, I believe, half in joke.

There is one thing that has puzzled me: I do not quite see how this paper bears on life assurance.

DR. PARKES WEBER: It was meant to have a bearing more upon accident insurance.

DR. THEODORE WILLIAMS: I did not quite understand that, and your statement very properly answers my question. I think the paper is one that must be thoroughly studied. Not only are there some very good conclusions in it, but our friend has got together some capital authorities, so that we have not only good advice but a very good paper of reference. I can congratulate Dr. Parkes Weber heartily on the paper, and I congratulate his excellent father, Sir Hermann Weber, who has been President of our Society, and whom we look upon as our Nestor in many things, and I shall tell his father that the paper is worthy in every way of Sir Herman himself. Long may both father and son live to be ornaments to the medical profession.

It was agreed to prolong the meeting for half an hour.

DR. T. D. LISTER: I should like to join my congratulations to those of Dr. Theodore Williams on Dr. Parkes Weber's most excellent and exhaustive paper, which gives a splendid summary of the pathology of the subject. It will be looked upon by insurance experts as a mine of information in cases in which traumatic tuberculosis, traumatic pneumonia, and traumatic pleurisy are alleged to have occurred, and will be extremely valuable to them in supporting any theory they wish to enunciate in a court of law. From our point of view as an Insurance Association, the paper is summed up in the three questions which Dr. Weber propounds, and in the classes of disease which he puts forward at the beginning. In Class B he speaks of injury to the lungs, but I gather he means where the injury to the lung can only be inferred—where there is no obvious injury to the lung. An injury to the thorax or in the neighbourhood of the lung does not necessarily cause an obvious injury to the lung, and yet such cases are followed by tuberculosis, which strongly suggests a direct etiological relationship. I remember a case of a young carman attending in my out-patient depart-

ment. He was struck in the right lower thorax by the pole of a cart, and about a year afterwards he developed a local tuberculosis. He was believed to be in good health before, and there was no evidence of any pre-existing disease. The relationship of the injury to the disease is a matter in which it is extremely difficult to form any certain opinion because of the latent tuberculosis with which we are all familiar. But in this case he was believed to be well before the accident, and the situations of the injury and the disease were in correspondence.

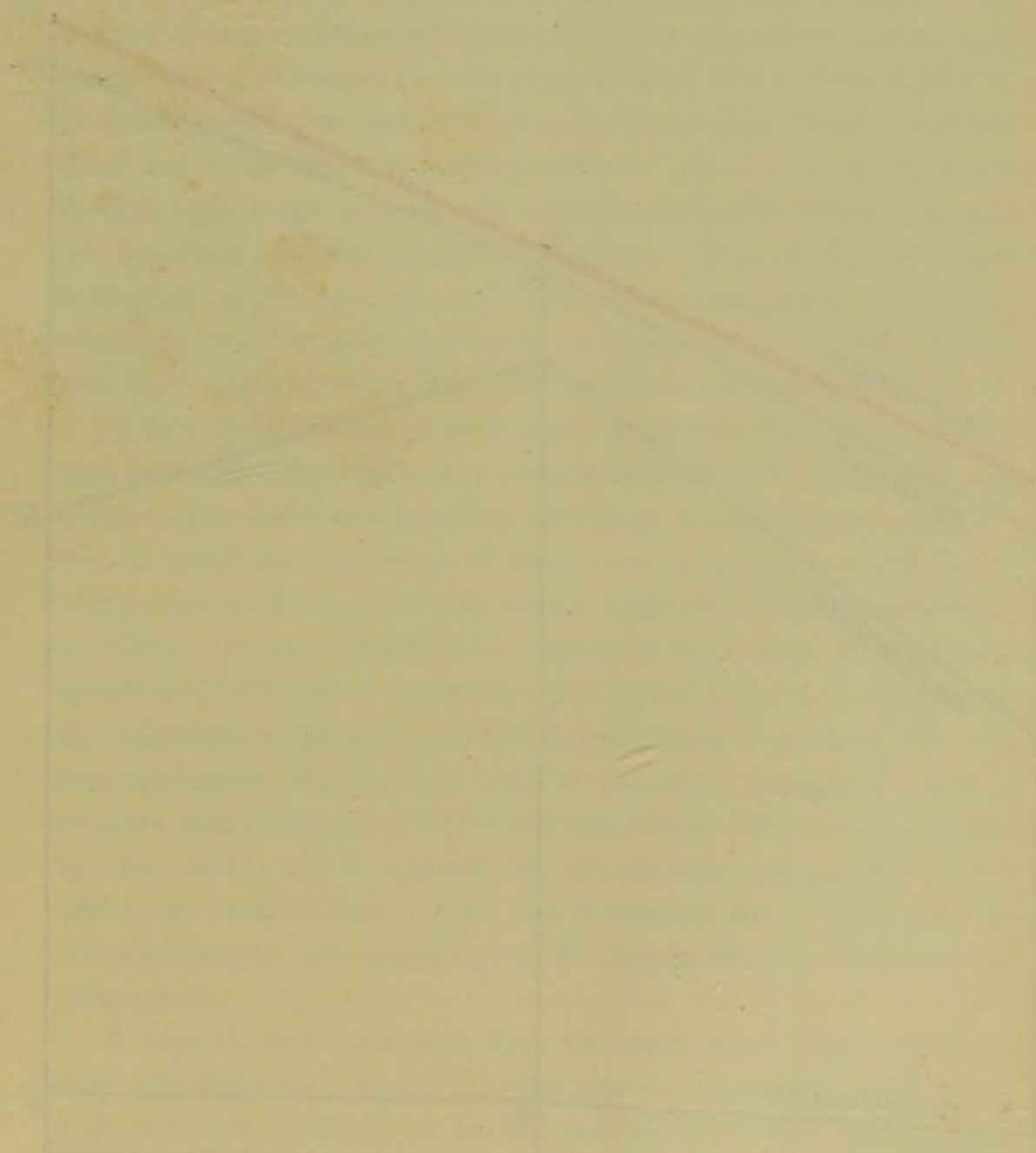
With regard to traumatic tuberculosis of the peritoneum, quite recently I saw a case with a doctor, of a child aged $3\frac{1}{2}$, supposedly healthy, who, in running along a passage, fell on her face. Within a few hours she had vomiting and acute pain in the abdomen and she was constipated for 48 hours. The medical attendant gave her aperients without any result. She passed a little mucus by the bowel, and he suspected obstruction and asked me to see her. She had that text-book symptom, "a sausage-shaped tumour" in the abdomen. The case was provisionally diagnosed as intussusception, with a reservation as to tubercle, and I called in a surgeon to see her. He entirely agreed, and on opening the abdomen, a large caseous mass of glands was found, with some recent peritonitis, of too early a date to show the new tubercles scattered about the peritoneum, but obviously the commencement of traumatic tuberculosis. The child did well afterwards, rapidly became convalescent, and went away to the seaside, and is now apparently in good health, some five months afterwards. That is an example of the latent cases where one may suspect other conditions and find them to be due to a traumatic tuberculosis. But such cases are rather beside the question from our point of view as an association for the duty of life and other forms of assurance. The problem for us seems to me to be more to try to put before the offices, as the result of the papers and discussions here, such information as may help the medical officers of the companies or the managers of the companies to assess claims and premiums and risks at their proper value, or to arrive at the truth as to the possible results of injury, and so secure greater uniformity of opinion.

This subject of traumatic tuberculosis, I would suggest, is capable of being approached from a different standpoint. Recently I had occasion to investigate some statistics to try and find whether there was any evidence of the relationship between the liability to traumatism and the liability to tuberculosis. The whole trend of Dr. Parkes Weber's paper shows, I think, that we must regard the possibility of a latent tuberculosis as being almost a normal condition in the human civilized body, and that the pneumococcus, which we know to be a normal inhabitant of our respiratory mucous tract, is possibly possessed of a not unusual colleague in the tubercle bacillus. I do not say that we all have tuberculosis; I think Dr. Theodore Williams is wise in asking us to restrict the significance of the statement that "we are all the subjects of tubercle"; but I can imagine the possibility that we are all more or less the carriers of latent tubercle bacilli, and therefore, in regard to traumatism, we have to consider the possibility of trauma either setting free latent tubercle bacilli, or causing them to become relatively virulent by diminishing resistance. If it is true that the tubercle bacillus is a normal inhabitant of our respiratory organs or tissues, then if trauma can act as suggested, we ought to be able to find that amongst those occupations which are more liable to traumatism there would be also a greater liability to tuberculosis.

In endeavouring to find some statistics to support such a view, I found it impossible to obtain statistics of the comparative liability to non-fatal accidents, and difficult to obtain mortality figures of occupations in which the conditions were fairly comparable. For instance, if we take various groups of sailors we should find probably that some of them would show a very high mortality from accident and a very low liability to tuberculosis. That high liability to accident might be explained by the excessive mortality from catastrophes from many sailors of one class being drowned in one accident; so that no diagram based on such figures would be pertinent to the subject. If we take the general labourers, agricultural labourers, wharf labourers, and labourers in industrial centres—I am speaking of such figures as are given for comparing the labourers of the country in the supplementary report to the Regis-

OUTDOOR RAILWAY WORKERS

Comparative Study
of the
Health of
Outdoor Railway Workers
in
Albany



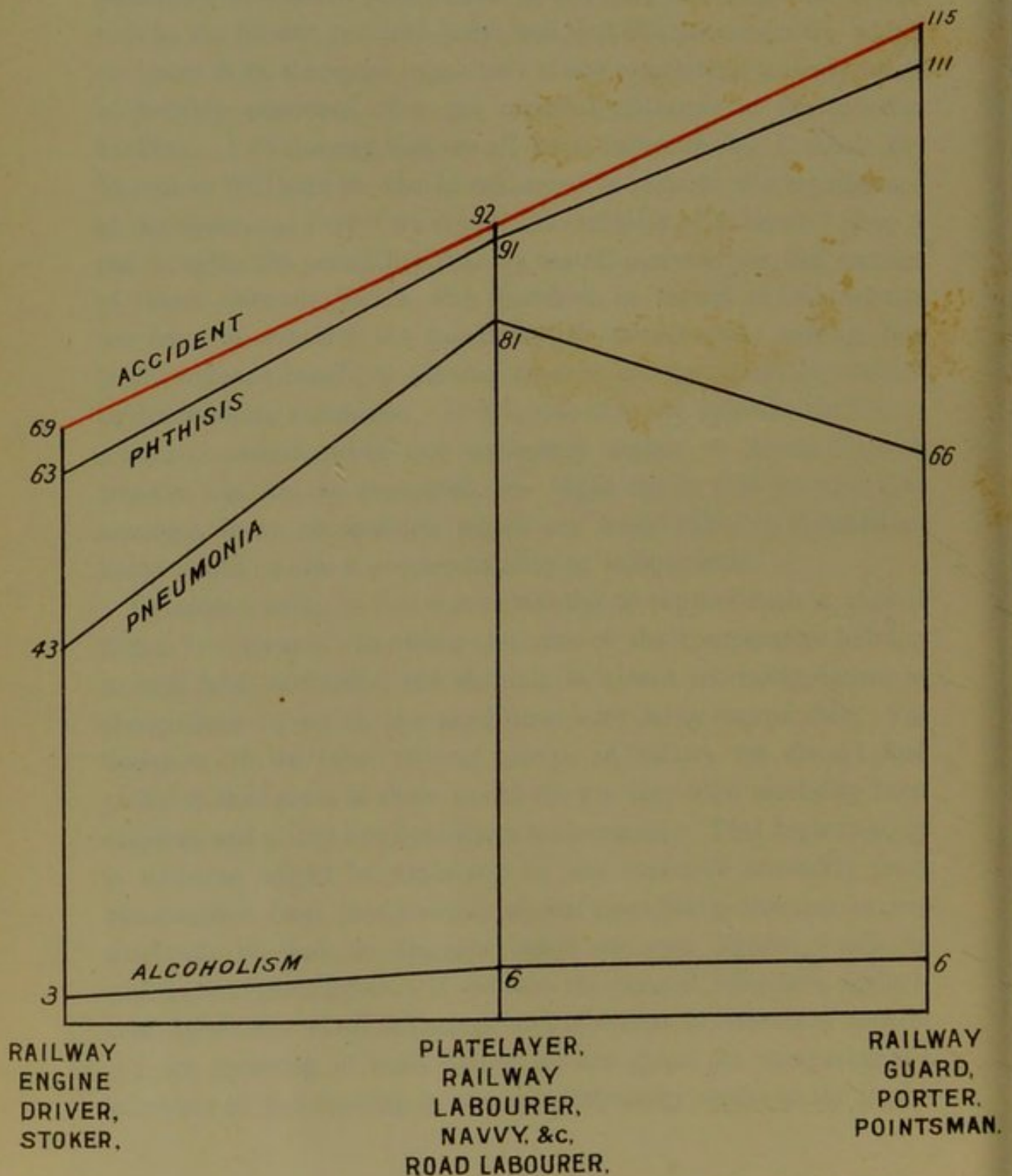
Y-axis label: HEALTH
X-axis label: RAILWAY WORKERS
Legend: SOLID LINE, DASHED LINE

Author: J. H. ...
Date: ...

OUTDOOR RAILWAY WORKERS.

Comparative Mortality
FROM
ACCIDENT, PHTHISIS, PNEUMONIA,
AND ALCOHOLISM.

ACCIDENT IN RED.



THO^s D. LISTER, M.D.
INVEN. ET. DELIN.
1910.

trar-General's returns—we should agree that there are a great many different forms of labour with scarcely comparable environments. Industrial conditions in cities as compared with those which exist in the agricultural districts are so widely diverse that it would be impossible to arrive at any safe deductions from such figures. On the other hand, there seems to be, in looking through the list of occupations analysed in the returns, one set of workers whose work is more or less strictly comparable, and where there is no catastrophe death-rate, in the sense that no great number of men were liable to be annihilated in one accident so as to disturb the mortality figures. That set is the outdoor railway workers. In the returns they are divided into three groups—the engine-drivers, the pointsmen and the labourers, and the porters and guards. Almost all their work is entirely in the open air, and under fairly comparable conditions. Accidents occur in one's and two's, and preserve a very fair average over the large population that is measured in the returns.

It is very interesting to find, if one compares those three groups, that as the phthisis death-rate rises in the three groups the accident death-rate is absolutely parallel. In other words, taking a population in which the conditions of labour are comparable, a liability to death from accident is directly related with a liability to death from phthisis. In such a population I think we have a set of figures in which the incidence of mortality may be taken as the measure of the incidence of the risk. In the diagram that I produce, the red line represents the accident risk in the three groups of railway workers, and the phthisis and other respiratory risks are shown also by lines, and it will be noticed that the phthisis and accident risks are almost parallel lines. That is a suggestion for a new mode of investigation of this question of the relationship of accidents to tuberculosis.

I, myself, feel convinced that we must inquire more carefully than is usually our custom, into the previous history of our patients suffering from phthisis, as to the possibility of there having been pulmonary strains or contusions. Subsequently to getting out those figures for the engine drivers, an engine driver was admitted to my bed at the Mount Vernon Hospital, a man between 50 and 60, who

had passed many medical examinations, a presumably average healthy engine driver. He was admitted with early phthisis of his right apex. I asked him whether he had had any injury to his chest, and he told me that two years previously he had dislocated his right shoulder. There seemed to be a very close association between the occurrence of the dislocation and his admission within two years to the wards of a consumption hospital suffering from early phthisis in the near neighbourhood of the injury. He is a resistant man and is apparently making a good recovery. I have made other figures dealing with the general labourers and miners of the country, but in miners the statistics are disturbed by the fact that there are various catastrophes in various districts which do not happen in the same year, and the averages are always upset. In regard to the various classes of labour, other disturbing factors are present, as I mentioned just now. I think the outdoor railway men's chart is of some use as an illustration, and that the figures may be taken as representing in that population the measure of the incidence of the two risks of accident and phthisis. You will see that the phthisis and accidents follow each other.

DR. COLLIE: What are the numbers on which these diagrams are based?

DR. LISTER: I think something like 60,000 drivers, 140,000 guards and porters, 170,000 labourers, speaking from memory; it is a very large population. The number of deaths is respectively 1,262, 5,054, and 6,508.

DR. SELFE BENNETT: I desire to join my thanks to those already expressed to Dr. Parkes Weber for bringing this subject before us and for the excellent series of cases he has adduced. Happy is he who knows the causes of things, and in the matter in question it appears to me we are justified in taking the popular line of argument, *post hoc propter hoc*. The number of cases known to exist of the connection between injury and phthisis seem to justify us in concluding that there is a very intimate connection between

the two. These cases were observed over a long course of years, before the rise of bacteriology. We are told that some people would go so far as to say that everybody is tubercular, and it appears to me that this is the conclusion that must be arrived at in consequence of the modern teaching of the non-heredity of phthisis, a theory which excludes the soil and only concerns itself with the seed.

One moral seems to be, to let sleeping dogs lie, and not to disturb ankylosed joints, as Dr. Theodore Williams has suggested. As an instance of this, it was very unfortunate for the owner of the diseased epididymis mentioned in the paper that that testicle should have been the one to have been injured. We are requested to bring forward cases from our personal experience, and here is one which is known to me. A man of thirty, who was an experienced Alpine climber, had a fall in the Alps. He rolled for some distance down hill and lay for some time exposed until assistance could be procured, and then he was carried to the nearest inn and laid up with a sprained ankle and bruised ribs for a period of six weeks. He then returned to this country, and after four months of ill health died of acute phthisis within six months of the injury. There had been no hæmoptysis and little cough, but there was intractable diarrhœa and fever, with rapid wasting, and those were the prominent symptoms for the last two months of life. The parents both attained eighty years of age and their five other children showed no signs of tubercular tendency. Is it not almost a certainty that in this case there was contusional pleurisy and probably slight pneumonia during the period of resting whilst the injuries to the ribs and the ankle were the most notable features immediately recognised and attended to? That was a case which came under my personal observation, and I take it it is *à propos* to the matter we are discussing to-night.

MR. SHILLITOE: Dr. Parkes Weber's first case alluded to an injury to the epididymis. I do not know whether it is still the practice of Sir Frederick Treves in cases of tubercular epididymitis, but some years ago he always scraped them in preference to removing the testicle. It always seemed to me that the scraping

must have been a much less radical operation than the removal of the organ. I do not know whether Dr. Parkes Weber has had any experience of scraping the tubercular foci from a diseased epididymis, but I should have thought it might give rise to general military tuberculosis.

DR. R. J. COLLIE : There is one aspect of the case I should like to draw attention to. A very large part of my work consists in acting for employers in cases of accident, and I look forward with some dismay to the effect of this paper being printed and circulated and finding its way into the hands of gentlemen of somewhat doubtful morals, who act systematically for plaintiffs in actions at law. I think when one hears it alleged that violent inflation of the lung may itself be the cause of traumatic pneumonia, one might have very serious difficulties in resisting bogus claims. I do not mean that one should not have these matters thoroughly discussed, but I cannot help feeling that one will be met with a large number of cases where the subject is not treated in the scientific way in which we treat it here, but the mere dogmatic statement in Court will be made that anyone who happens to have the early stages of phthisis and meets with an accident and subsequently dies of his phthisis will have set up for him an unjust claim. I heard only recently from one of the medical officers of the Home Office that they have very great difficulty with this subject already; in cases like plumbism where men have also phthisis, the phthisis is put down as the consequence of the plumbism. I am very much indebted to Dr. Parkes Weber, for his paper which has opened up wider ideas of these things in my mind, but I have my misgivings about publicity.

THE PRESIDENT : I should like very much if Dr. Parkes Weber, in reply, will bring out what is the practical outcome of his paper, and in what way it may be of service to the Insurance offices, to medical officers, and to managers.

DR. PARKES WEBER, in reply, said : I have to thank all those

who have taken part in the discussion, not only for the facts they have furnished and the suggestions they have made, but also for their kind remarks, especially those of Dr. Theodore Williams, with reference to my paper. I do not intend to discuss everything that has been said, and I think I have been only asked one direct question, and that was by Dr. Shillitoe in regard to the danger of scraping out a tuberculous focus instead of cutting it out. As far as I know, facts do not point to there being particular danger in scraping easily accessible tuberculous foci, but, *a priori*, one would certainly think there was more danger from scraping a tuberculous focus than from excising one. A contusion is, after all, very different from a surgical scraping: the latter is an "open" surgical operation, though perhaps less "open" than the cutting out of a focus with a sharp knife, whereas the former is comparable to a bloodless surgical manipulation—that is to say, to taking hold of a bone and breaking down adhesions in a joint without making any external wound.

I was exceedingly interested in Dr. Lister's statistics, and I have no doubt that working in that direction will throw light not only upon the question of traumatic tuberculosis, but on other traumatic diseases of microbic origin. I want to lay stress upon a particular point, namely, that one sometimes hears of vigorous men becoming victims to pulmonary or other forms of tuberculosis, people one would have thought the least likely of all men to suffer from the disease; I refer to occasional cases in athletes, boxers, &c.

DR. LISTER: I had two cases of 'varsity oars that suffered from phthisis, men who had been always healthy.

DR. PARKES WEBER: To people who write on congenital or acquired predisposition to tuberculosis this has been very puzzling, and one sees it sometimes put down that a splendid physique, open-air life, and all the rest of it do not necessarily save people from tuberculosis. The consideration of "traumatic" tuberculosis throws an immense amount of light upon this subject. Those athletes and boxers and men of splendid physique are just the men

(from the severe efforts they make occasionally, strains, &c., and from their being specially liable to contusional traumatism) who are likely to stir up a minute latent tuberculous focus and to get traumatic tuberculosis of some kind.

DR. LISTER: Dr. Theodore Williams assumed that there is a tuberculosis and not a tubercle bacillus wandering about which reaches the focus of traumatism.

DR. PARKES WEBER: I think it is extremely unlikely that tubercle bacilli could be for a long time wandering about at large in the human body. I cannot imagine bacilli wandering about without inducing some inflammatory reaction on the part of the tissues, and I take it that the presence of minute tubercles or foci containing tubercle bacilli is all that is required from the theoretical point of view.

The consideration of traumatic tuberculosis, in the way I have endeavoured to represent it in this paper, does, I believe, really throw light upon those anomalous cases of persons of splendid personal physique and athletic habits unaccountably falling victims to tuberculosis. I hope that the facts and cases which I have brought together in the paper may form a kind of theoretical basis for understanding some of the possible results of contusional injuries, especially those of the thorax.

The meeting then adjourned.

B.

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