A Paper

ON

TRAUMATIC TUBERCULOSIS, TRAUMATIC
PNEUMONIA, AND TRAUMATIC
PLEURISY.*

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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.† 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.

Traumatic Tuberculosis from Direct Inoculation.

Examples of this 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 by butchers, etc., from tuberculous cattle. Similarly tuberculosis may be inoculated by the accidental breaking of a glass or porcelain spitoon 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.

Alleged Traumatic Tuberculosis due to Injuries by which Tubercle Bacilli could not have been Introduced from Without.

Cases of this kind 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 military 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 blooming 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 hepatized. 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 the 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 twenty-five 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 hepatization 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 albumen.

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 pleurae there was evidence of older pleurisy, especially over one lung. The left epididymis (that is, where he had received the blow twenty-five 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 9 oz., 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 nine to sixteen 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 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 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, characterized by frightful dyspnoea and cyanosis, and, like the previously mentioned patient, died practically from suffocation about twenty-five 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—that is, cases in which acute miliary tuberculosis of the lungs is set up by injury to some part of the body which

* Cf. O. R. Wild, Ueber die Entstehung der Miliartuberculose, *Virchow's Archiv*, 1897, vol. cxlix, p. 65; and H. Ribbert, Ueber die Miliartuberculose, *Deut. med. Woch.*, 1906, vol. xxxii, p. 5.

† 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.

* Read at the Assurance Medical Officers' Association, May 4th, 1910.
† Cf. R. Stern, Ueber die traumatische Entstehung innerer Krankheiten, Jena, 1896 (with analysis of published cases); also A. Ascarelli, *Polietimico*, Rome, 1907, vol. xiv (Sezione pratica), p. 1025, and the papers of Hensell and Jordan, to which I shall refer later on.

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 9 years of age who died with general disseminated tuberculosis three or four weeks after an operation on a tuberculous joint. J. Orth¹ 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.²

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 anaesthesia 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 three and a half weeks after the inoculation. Had a bloodless manipulation been carried out, instead of the "open" resection of the joint, the boy might have fallen a victim to acute miliary tuberculosis as a result of tearing or crushing of 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. Weil³ describes 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 constituted 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), Hünermann (1893), Davidsohn (1899), and Westenhoeffer (1903). An analogous case is recorded by Hochhaus,⁴ in which the patient, a young woman aged 22 years, died forty-seven days after abortion. The necropsy showed the presence of a caseous focus in the uterus, and disseminated miliary tuberculosis of the lungs, peritoneum, etc.

Dr. C. T. Williams⁵ refers to the case of a gentleman who was thrown in the hunting field; several of his ribs were injured; 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 connexion 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.⁶ 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.⁷ In a man, aged 62 years, according to the account of Aufrecht,⁸ 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.⁹

Benda,[†] in three cases of miliary tuberculosis following

* F. Köhler, *Aerzt. Sachverst.-Zeitung*, Berlin, 1908, vol. xiv, 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. xxviii, 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. xii, p. 27; H. Toyosumi, "Intimatus tuberkel in den kleinen Lungenarterien," *Virchow's Archiv*, 1908, vol. cxcii, p. 403; and Thorel's communication on miliary tuberculosis with multiple tuberculous foci in the blood vessels (*Aerztlicher Verein in Nürnberg*, May 4th, 1905).

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 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. Ribbert⁹ thinks that in such cases the blood stream is not, as Weigert¹⁰ 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,¹¹ 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—for example, a blow with a blunt instrument or some 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 an infection happens to be forthcoming. That contusions of the thorax may actually injure the lungs and pleura has been well demonstrated by Külb in his experimental investigations in animals, to which I shall refer later on.

By a contusional injury of that kind a latent tuberculous focus (and experience from *post-mortem* examinations shows how frequently 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 recorded by O. Schrader¹² 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 thirteen 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 portion) or whether the lung disease was a tuberculous bronchopneumonia from the commencement. Great improvement in the patient's condition subsequently took place. C. Theodore Williams¹³ has recorded the similar case of a mate of a merchant vessel who fell from the mast to the deck and fractured

‡ The statistics on this subject, as is well known, vary considerably, doubtless to some extent according to thoroughness of examination and according to the criterion 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. Amongst recent work on the subject see the percentage statistics given by H. Beitzke of the frequency of tuberculosis in *post-mortem* examinations at the Berlin Pathological Institute (*Berliner klin. Wochenschrift*, 1909, vol. xlii, 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 minute 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, 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 every one by more or less suddenly falling victims to tuberculous affections, especially of the lungs.

three ribs. This injury was followed by the rapid development of well-marked symptoms and physical signs of phthisis (haemorrhage, 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. Nogués¹⁴ has described the case of a criminal who a few days after having been punished by 150 lashes, suffered from cough, dyspnoea, and haemoptysis. 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. Nicoletti¹⁵ tells of a labourer, aged 34 years, who after receiving a severe blow on the right side of the thorax, suffered from haemoptysis. 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,¹⁶ 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 local injury are commoner than cases in which the injury appears to have actually called a pulmonary tuberculosis into existence. R. Link¹⁷ gives the case of a strongly built brewer's drayman, aged 46 years, whose thorax was squeezed between two wagons. He remained three days in bed, but on the fifth day after the injury commenced work again, though suffering from dyspnoea and pains in his back. About six months later attacks of haemoptysis 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. Mosny¹⁸ 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, etc.

The question of a localized 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 inoculation)*—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, etc. 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.†

* 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 *Muench. med. Woch.*, 1904, vol. li, pp. 1689-91.

† For an excellent summing-up of this question see Jordan, *Muench. med. Woch.*, 1901, vol. xlviii, p. 1745. It must be remembered, however, that most cases of acute septic osteomyelitis after contusion (or

Similar experiments have been carried out with regard to the pathogeny of tuberculous diseases of bones and joints.

Schüller¹⁹ 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. Krause²⁰ 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 joints did not become tuberculous, excepting in a single instance (one of the rabbits).

Lannelongue and Achard²¹ found that it was not so easy to determine the site of a tuberculous process by experimental traumatisms 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 eight to 232 days after the injury, and none of the injured joints were found to be tuberculous. Friedrich²² 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, analogous 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. Honsell²³ who employed cultures of tubercle bacilli of high virulence, and also some of low virulence, came to the conclusion that in his experiments the localization of tuberculosis was not determined by traumatism.

E. Salvia²⁴ 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 localization 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 localization of tubercles resulted from slight traumata to the bones of the extremities. Definite localization of tuberculous changes was observed in the soft tissues of the articulations after slight chemical injuries.

Jordan²⁵ 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 military tuberculosis, whilst in cases of alleged traumatic tuberculosis of joints and bones in human beings there is generally no disseminated military tuberculosis present.

We conclude, like Honsell²⁶ 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.

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.

By Urban²⁷ 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 the 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. Pietrzikowsky²⁸ 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). Moser²⁹ 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 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,³⁰ in his experiments already alluded to, thought that he could localize 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, Orth³¹ 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. Luecke³² records a rather different kind of case, namely, one of tuberculous peritonitis in a boy, aged 12 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 (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 (that is, apart from the results of traumatism, etc.) ever have progressed sufficiently to become recognizable by ordinary examination or to give rise to any serious trouble. In any particular case, therefore, the questions we have to consider are:

1. Is there evidence that any clinically recognizable 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 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

* Cf. Port, Aerztlicher Verein in Nürnberg, July 18th, 1907; *Muench. med. Woch.*, 1907, vol. liv, p. 2013.

distance from the site of the alleged injury.* (I do not, of course, here refer to disseminated miliary tuberculosis following injuries.)

TRAUMATIC PNEUMONIA, TRAUMATIC PLEURISY, ETC.

I shall not stop to discuss such conditions as traumatic pneumothorax, haemothorax, pyopneumothorax, haemo-pneumothorax, 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 anaesthesia. There are numerous cases known of pneumonia-like symptoms following operation on the pelvic viscera, herniotomy, operations and injuries to the abdomen, operations on varicocele and varicose veins of the extremities, fractures of long bones, etc. Some of these may be examples of ordinary pneumonia due to the patient's diminished resistant power as a result of shock, etc. (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 anaesthesia, 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 haemothorax 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.⁵⁵

Külbs, in a series of dogs, investigated the effects of more or less severe contusions of the thorax, and found that considerable changes, mostly haemorrhages of greater or less extent, had been produced in the lungs, together with slight injuries to the 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 haemorrhagic 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, haemorrhage in the right upper lobe when the traumatism was applied over part of the left lung. Reineboth's⁵⁴ 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,⁵⁶ who in 1881 apparently first introduced the term "contusional pneumonia" (*Kontusionspneumonie*), it 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 haemorrhagic infiltra-

tion, such as Külbs produced by contusion of the thorax in animals, possibly associated with serous or haemorrhagic 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 8 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 skiagram taken later on. When I saw the boy on March 15th there was a good deal of dyspnoea, general uneasiness, and cough. Resonance to percussion was slightly impaired in the lower part of the right infrascapular 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, respirations 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 haemorrhagic infiltration (as in Külbs's 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 dullness 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,⁵⁶ 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 connexion it is interesting to note that Sir J. E. Erichsen⁵⁷ 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 hepatization of part of one lung.

The patient, a man 58 years of age, had several ribs on his right side fractured by being knocked 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 considerable dullness at the base of the right lung, and later on signs of consolidation of the right apex. He had decided fever (for some time about 102°-104° F., once higher), became delirious, and died on the fifteenth day after the injury. The necropsy showed grey hepatization 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. Bloch⁵⁸ 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.

* Vide, Port, loc. cit. Rare cases of acute tuberculous bronchopneumonia following abdominal operations performed under general anaesthesia may be theoretically explained in various ways. Cf. the case recorded by Mackey, Macdonald, and Sutherland in the BRITISH MEDICAL JOURNAL, 1910, vol. i, p. 745.

C. Rubino³⁹ 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 forty-eight hours later the first signs of pneumonia appeared on the right side.

Litten⁴⁰ attaches special importance to the lifting of heavy weights in the etiology 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 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 haemoptysis, 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.

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 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 was sent 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 fourteen 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 fourteen 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 connexion between pneumonia and trauma, though the interval between the two was as long as fourteen days; in

the case in question the injury was soon followed by signs of pleuritis, though the pneumonia did not apparently develop till fourteen 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 Külb's.⁴¹ 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 connexion between contusions of the chest and tuberculous pleurisy may be explained in the same way as that between contusions and pulmonary tuberculosis.⁴²

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* Souques ("Pneumonie contusive," *Presse médicale*, Paris, March 3rd, 1900, p. 109) refers to much of the older literature on the subject. Amongst older works on the traumatic origin of diseases of the thoracic viscera see especially R. Stern, *Ueber traumatische Entstehung innerer Krankheiten*, Jena, 1896.

† Cf. F. Pancrazio, *Acad. Med. di Padova*, March 31st, 1907, *Policlino*, Rome, 1907, vol. xiv (Sezione pratica), p. 781.

IN his annual report to the Aberystwyth Rural Council Dr. James concludes as follows: Were I to preach in this land of preachers and sermons my text would be "cleanliness is next to Godliness," and I would accordingly divide my discourse into three heads—first, cleanliness; secondly, cleanliness; thirdly and lastly, cleanliness. I would dilate upon the necessity of having clean air to breathe; clean, nourishing food to eat, and clean water to drink; a clean house to live in with clean surroundings. I would also emphasize the virtues of personal cleanliness and would condemn the mere ablution of hands and face as hypocrisy, humbug, and fraud. I would end my sermon by impressing on the minds of my hearers the truth that a damp dwelling house is a safe way to sanitary perdition.