connection with any other renal condition, or with any other morbid state with which we are acquainted. This is evinced by the hardness of the pulse, as evident to the touch, the absence of diastolic collapse giving to the vessel a cord-like persistence; by the force with which the sphygmograph must be brought to bear upon it before a graphic tracing can be obtained; by the abnormal pressure required to obstruct the vessel, as measured by the sphygmometer of Dr. Handfield Jones; and by the alteration in the sounds of the heart, especially by the accentuation and frequent reduplication of the second sound: evidences, as they have been interpreted, the first of the abnormal force of the diastolic recoil, owing to the repletion of the aortic system; the second, of the slowness with which, from the same cause, the left ventricle empties itself, so that the sound which succeeds upon the completion of the left systole is produced not synchronously with that which follows upon the right, but appreciably later.

It is not my purpose, however, to dwell upon this portion of the subject, since it has been brought before the College recently, and with an ability and completeness to which I could not hope to attain.

THE GOULSTONIAN LECTURES

ON

THE NATURAL HISTORY OF PULMONARY CONSUMPTION.

Delivered at the Royal College of Physicians, London.

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LECTURE I.—*Concluded.*

LET us turn for an instant to the comparative pathology of these modern days. I have here a series of diagrams, copied, by his permission, from the second part of Dr. Klein's work. On the table is a drawing of the naked-eye appearances of the lung in a case of acute tuberculosis in a child, together with microscopical preparations of a case of the same kind, made for me by Mr. Lithgow, our assistant-curator at St. Mary's; the latter show perivascular growth, with some lobular collapse, and but very slight catarrhal product. [A description of the diagrams, etc., followed here.]

In these two diagrams (Figs. XXIV and XXVI), it is possible to see in the lung of the guinea-pig, as well as in that of the child, certain histological products which it has been attempted, not for the first time in the history of "tubercle", to set up as the specific elements of that growth. Happily these giant-cells will soon be relegated—if they be not even now relegated—to the list of ordinary epithelial cells, the limbo in which rest already the pathognomonic tubercle-corpuscle and cancer-cell. As my friend Dr. Klein informs me, and as I am quite prepared to believe, the giant-cell is but an epithelial cell growing under certain conditions. But

"Each month is various to present
The world with some development",

and the specific element refuses still to be unrecognised; this time it appears under the form of the particular cells drawn and described by Rindfleisch in Ziemssen's *Handbuch des Respirations-Apparates* (vol. ii, p. 161. Leipzig, 1874). I am perfectly prepared to see these cells, also, sink to the same position as the Riesenzellen. I confess that I do not perceive why, in the pursuit of a supposed scientific truth, the same common-sense which men use, or ought to use, in the ordinary affairs of life, should not exert itself; and when through the long list of specific structures, started by one author only to be hunted down by another, we find always the same untimely death, I believe that the very best thing we can do is not to join in the shadowy chase, but to abjure, as something that oppresses us, with no fault of our own, this "specific" incubus.

With the results shown in those diagrams and described in his work by Professor Klein, the researches of Dr. Burdon Sanderson and Dr. Wilson Fox in the main agree. But the latter writer goes much further, and states that he finds "in the lungs of patients dying of phthisis almost identically the same changes as those found in the lungs of children dying of acute tuberculosis"—a sentence to which I cannot altogether give my adhesion. There are cases, no doubt—cases of which I hope to speak more at length hereafter—in which the

physical signs and symptoms of lung-disorganisation are followed by death, with all the symptoms of this "acute tuberculosis". But there are also cases, of not infrequent occurrence in the adult, of this same "acute tuberculosis" with almost the same series of clinical symptoms, and the same pathological changes, as far as I am able to make them out, as those found in artificial tuberculosis, and in the general tuberculosis of the child. The only case of the kind which I remember to have come almost entirely under my own observation was that of a young man, about twenty-one years old. In him there were no abnormal signs on percussion; the respiratory sounds were everywhere coarse. There was increase in temperature and pulse-rate; his chest and abdomen were covered with sudamina; he was admitted into hospital as a case of probable acute tuberculosis, or doubtful typhoid; within a short time, he died with symptoms of tubercular meningitis, and his body presented the expected changes.

And it was, I think, from these comparatively rare cases in the adult that observers and writers argued that "ista tubercula (seu tumores)"—I use the words of Morton—underwent a further, and that a cheesy change, resulting—not in these cases, which, if the argument be true, must be said to die before such changes can take place—but in cases the clinical symptoms of which are entirely different, in a larger or smaller destruction of pulmonary tissue. That I am not, Quixote-like, opposing some image of my own brain, is, I think, shown by the fact that writers of the present day still class these cases of "acute tuberculosis" with "pulmonary consumption"; though it is but fair to state that Dr. Douglas Powell has, in his small work, drawn the distinction which others, though by no means all, have failed to make. I need not travel far for an illustration. In a very valuable, because very true and practical, though it may be a very simple, paper on the different forms of pulmonary consumption by my colleague Dr. Peacock—a paper published originally in *St. Thomas's Hospital Reports* for 1870—are given two cases, "selected from numerous instances of the same kind"; together with a third, which differs from them in several respects; all three classed by the writer under the name "acute constitutional phthisis". Dr. Peacock's first case is that of a young lady, aged 22, who died on the twentieth day of her illness of apnoea. Four days before death, "the respiratory sounds were found everywhere harsh..... but there was no rhonchus and no marked deficiency of the resonance on percussion in any part of the chest." These signs were slightly modified the day before death. On *post mortem* examination, the body "was thin, but not emaciated". There were old pleuritic adhesions over a large portion of the left lung; the right lung was collapsed. "Both lungs were copiously infiltrated throughout their whole substance with miliary tubercles, having an average diameter of from half a line to a line, and uniformly of a yellowish-white colour..... In no part had any of them undergone softening." Nothing is said about the head or abdominal organs. The second case is also that of a young woman, aged 24, who died after an illness lasting over three weeks of capillary bronchitis; here was the same absence of phenomena on percussion and auscultation; but little emaciation of the body, a chest sufficiently broad and expanded; both lungs contained a copious deposit of small, round, yellowish-coloured and semitransparent-looking bodies, which in no part had softened; the bronchial glands were softened to a brown-coloured pulp; the abdominal viscera were free from tubercle. Dr. Metzquer, in a *Clinical Study of Galloping Consumption*, published in Paris in 1874—and I may mention, in passing, that, to the majority of French writers, galloping phthisis is synonymous with acute tuberculosis—Dr. Metzquer gives two similar cases. Case 1 is that of a female aged 28-30, free from history of hereditary phthisis, and always well till she was taken one day with pains in the head and back, intense fever, rigors, and ringing in the ears. There were no signs on percussion or auscultation, though a few days later there were rhonchus and *râles* in both lungs, and streaks of blood in the sputa. Death occurred a month after the commencement of the illness, and there were "tubercles in all organs". The second case, that of a man aged 43, is precisely similar: the same absence of hereditary phthisis and physical signs on the part of the lungs; the same general tuberculosis without softening. In both these cases, there was some diarrhoea and slight jaundice before death. It would be easy to multiply from modern literature cases of this kind, but I prefer to turn back again to the commencement of the century. I quite agree with M. Thaan that Bayle's cases numbered 4, 14, and 15, named by him cases of granular phthisis, do not answer (see Andral, *op. cit.*, iv, 10), either clinically or pathologically, to this "acute tuberculosis"; but, in spite of the distinction Bayle makes between the *miliary tubercles* found in the lungs of his first case, a woman aged 20, who seems to have exhibited as her chief symptoms those of "tubercular peritonitis", and the *miliary granulations* in the lungs of his second case, a postillion aged 24, who died, as the four cases I have just quoted from Drs. Peacock and Metzquer did, from

apnoea, both of them agree in these three points, that neither had any of the physical signs of phthisis pulmonalis during life; neither was emaciated, but rather both were remarkably plump; and both presented pathological products elsewhere as well as in the lungs. His cases 7 and 8, of men aged respectively 45 and 24, are very similar. They are the more interesting that, in addition to the lung-changes found, the cerebral symptoms which were chiefly exhibited during life left some after-death results; while the author himself adds in the title of the last, "Death from a disorder of the brain" (Barrow's translation of Bayle: Liverpool, 1815, p. 173). In Case 7, "there was near an ounce (of serum) at the bottom of the skull, which was as turbid as that contained in the lateral ventricles". (*Ib.*, p. 171.) In Case 8, there was what some pathologists would call now-a-days a "scrofulous" tumour in the medulla oblongata, and a collection on the surface of the brain of miliary round bodies of a dull opaque white, which may have been only Pacchionian bodies. But both cases again agree in these points: the most marked symptoms during life were not on the part of the lungs; the pathological products were presumably those found in general tuberculosis; the writer distinctly tells us there was no emaciation in either.

It would be but time wasted to quote similar cases. My own views as to the pathological products found in all these cases of "acute tuberculosis" are briefly these: There are found frequently together, sometimes alone, numerous vesicular pneumonias, collections of lymphoid cells round the vessels or round the smaller bronchi, or—and the distinction from the two last is by no means easy—proliferation of the nuclei in the wall of the capillaries themselves. Each of these products to the naked eye is exactly like the typical tubercle of authors, and, without injection of the tissue and microscopical examination, cannot be distinguished from either of the others, except that, in cases where the vesicular pneumonias are the sole or almost sole production, the lung on section does not present to the hand that feeling of points standing out from the surface; though later, in a shorter or longer period after removal from the body, the lung-tissue collapses and sinks away, leaving the solid nodules more prominent. Such was the case here,* in which even the skill of Mr. Hurst did not catch them in their original superficial flatness.

I have purposely abstained from quoting cases of general tuberculosis in children under my own observation, because I find that they are only examples of what has been stated and illustrated by authors, who have expressed in a far better manner than I can do ideas which carry with them, at least to my mind, the impress of truth. But if, in addition to the evidence not only adduced for the purpose by writers like Buhl, but unconsciously present in the observations of older workers, there were needful, for my own decision, facts within my own knowledge and my own reasoning, they also are forthcoming to uphold the statement of others beside Buhl that acute miliary general tuberculosis is a disease of absorption and infection. And this conclusion, in the very case I am about to state, is the exact contradictory of the older theory—that of Laennec—inasmuch as he considered the larger areas of softening and destruction to be due to the smaller disseminated product, which we hold to be the consequence of some older caseous centre. The one case I wish to mention is this. At the commencement of January 1871, a boy aged 10 was brought to the Children's Infirmary with marked dulness at the left apex; no increased vocal resonance or tubular breathing; tympanitic resonance over the right chest, which measured a quarter of an inch more round than the left. The pulse was 90; the temperature 98.2. Towards the end of the month, there supervened diarrhoea and night-sweats. Admitted into the hospital, the boy improved in general health, without much change in the physical signs up to the commencement of April. In the second week of July in the same year, he was brought back. The left side was flattened from above downwards. There was absolute dulness over the whole left front; less absolute behind. On coughing, there was tubular breathing, and slight crepitation at the end of expiration. The voice-sound was rather ægophonus and diminished in loudness. The right side was normal. There was no cough; no pyrexia to hand. The bowels were now constipated. Four days later, there was sickness. On the 25th of the month, he was readmitted in the drowsy state of tubercular meningitis. Early on the morning of August 3rd, the fifteenth day after the certain diagnosis of the tubercular meningitis, he died, his temperature the night before having been 102.3; and on the afternoon of that day we made a *post mortem* examination. The result was as was to be expected: the left lung consolidated; the vesicles filled with cheesy deposit; in the apex, a cavity which communicated with a half-chalky, half-cheesy mass in the position of the

thymus, making its way apparently into the pericardium; perivascular nodules in the pia mater; small miliary tubercles elsewhere; cheesy bronchial glands. The only comment I can make on this case is this: As a matter of reasoning, it must either be allowed that the antecedent presence of miliary tubercles which, according at least to the language of some, had caused the changes in the lung, gave no fatal indication till they appeared in the adventitia of the pia mater; or—and I confess the conclusion is easier to my mind—the presence of these perivascular nodules was in all the organs of the body simultaneous, or almost so; later than, and in every probability secondary to, the lung-changes which had already proceeded. There is no need to look upon the cheesy mass in front of the trachea as the original focus; though, as a pathological curiosity, its connection with the lung, and the thinning it was causing in a portion of the pericardium, is extremely interesting. There is one more point connected with this case, to which I may allude later on, which shows the probably hereditary character of what I assume to be the original disease. A younger sister and brother, of whom the latter died, both exhibited symptoms of pulmonary change, commencing, as it did in this boy, at the left apex. I cannot lay my hand on any notes of, nor do I remember making, a *post mortem* examination of the younger boy. In all probability, it was not allowed.

In everything I have said about this so-called "tubercle" to-day, I have confined myself altogether to that description of it by various writers as applied by them entirely to its supposed presence in the lungs. Unhappily, as I have already hinted, conclusions made as to its occurrence or supposed occurrence there, whether histologically as homologous growths, or pathologically as heterologous products, have been referred to the occurrence of "tubercles", neither histologically nor pathologically similar, in other organs. Backwards and forwards, from its implied type in the lung, its somewhat less defined repetition elsewhere, has this word "tubercle" been tossed, till, if it could speak for itself, it would make of men the same complaint which men, according to Plautus, make of the gods: "Dii nos quasi pilas homines habent." (*Capt. Prolog.*, 22.) If any support for such a proposition be needed, the latest standard works on pathology supply, in their woodcuts, the most various microscopical illustrations, stamped all of them with the very same name—"Tubercle".

THE PREVENTION OF PYÆMIA IN HOSPITAL PRACTICE.*

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IN what I am about to say, it is not at all my intention to enter or invite discussion on the nature or treatment of pyæmia, further than to describe what is meant by the term. The subject is too abstruse and complicated for any paper or any oral discussion, and, in proof of this, I would refer to the unsatisfactory and unproductive result of that discussion which took place two years ago at the Clinical Society. The subject of that argument was professedly, Does pyæmia occur in private practice, or is it entirely or chiefly confined to hospitals? No general agreement was arrived at even on a practical point of this kind, and this was chiefly due to a want of agreement as to what should or should not be properly termed pyæmia. Many of the speakers on that occasion confounded together cases of septicæmia; that is, cases where, as in dissection-wounds, or after parturition, a poison is at once absorbed before suppuration is established; cases of abscesses following eruptive fevers, such as scarlet and typhoid fevers; others, again, claimed as pyæmia cases of peritonitis and pelvic cellulitis following lithotomy or herniotomy; and some even spoke of local pyæmia.

I would, however, confine the term to the cases having these general symptoms: it attacks mostly, but not entirely, those who have wounds, or have undergone operations; it comes on not immediately, but generally after one or two weeks, with violent rigor, followed by profuse perspiration. To constitute true pyæmia, the rigors and fever-fits must be repeated frequently, once or twice a day, or every few days; the wound in all likelihood alters but little, or looks dry and secretes pus scantily. Rapid exhaustion follows; a sallow skin always, frequently more or less jaundice; delirium at times; signs of pneumonia or pleurisy. These are the main characteristics of acute typical pyæmia, illustrations of which may be, I grieve to say, abundantly found in the records of this hospital, and it is of this acute typical, or if I may so call it, hospital form of the disease, to which I now refer; and I would ask, in the first place, is this a common disease in the

* Drawing of lung of child: acute tuberculosis: catarrhal pneumonia most marked.

* Read before the Norwich Medico-Chirurgical Society.