ON
TUBERCULAR PERICARDITIS,
WITH PATHOLOGICAL AND PRACTICAL REMARKS.

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Received January 18th—Read February 23rd, 1847.

It may appear to many members of this Society almost a work of supererogation to describe another variety of pericarditis, after the extended inquiry into the causes of that disease by Dr. Taylor, which was published in the 28th volume of our Transactions. Considering how much attention has been paid to diseases of the heart during the past twenty years, their history might indeed by most persons be regarded as nearly complete, and such an opinion has been expressed upon several occasions, and was so by M. Andral, when he published, in 1829, the 1st volume of the *Clinique Médicale*. That able pathologist commences his clinical history of cardiac diseases with the following remarks :—" Since the publication of the immortal researches of Corvisart, and of numerous other works subsequent to his which have still further enlarged the dominion of science, the history of diseases of the heart and its membranes ought to be regarded as almost complete." Notwithstanding this prediction, how much has our knowledge on this subject been extended since the publication of Andral's valuable work, by the labours of Hope, Bouillaud, Bertin, Latham, Watson, Taylor, and others.

Dr. Taylor, in his essay on the causes of pericarditis (*loc. cit.*), has enumerated, and I think correctly, the three following as the most influential causes of that insidious disorder; —(1) Rheumatism; (2) Granular degeneration or Bright's disease of the kidney; and (3) the extension of inflammation from contiguous tissues to the pericardium. Although Dr. Taylor states that he believes other causes operate in the production of the disease, he does not intimate that he is acquainted with the variety which I am about to describe as Tubercular Pericarditis.

In a review of the causes of pericarditis as they are detailed by different writers, Dr. Taylor has not quoted any one who describes tubercular granulation of the pericardium as the exciting cause of inflammation of that membrane. Dr. Latham, in his elegant and instructive "Lectures on Diseases of the Heart," only incidentally alludes to this variety of the disease of the pericardium; although in the course of that physician's large hospital experience, it is most probable that some cases of this insidious and latent disorder must have occurred, but passed by unnoticed. Dr. Latham states that his experience of pericarditis is, like that of other physicians, mainly derived from what it is as an accompaniment of acute rheumatism. He remarks that he has seen the disease under other circumstances, but it has been very seldom; so seldom indeed, that he has little acquaintance with other conditions, external or internal, conducing to it. He can neither tell whence to look for it nor when to expect it, except when it occurs as a part of acute rheumatism (vol. i. p. 136). This author also remarks, that the pericardium is not exempt from those diseases which result in formations different from the natural structures of the body, such as tubercle, carcinoma, &c. But these diseases have not any special pathology respective to their seat within the heart, nor any distinctive history, diagnosis or treatment (Lect. 24, vol. ii. p. 120). It is hoped that the present communication may extend our knowledge on this subject, and point out other conditions of the system when pericarditis may be expected to occur.

The existence of tubercular deposits on the pericardium, both on its serous and fibrous surfaces, has been known to the morbid anatomist long anterior to the possession of any method of detecting pericarditis in the living person.

Dr. Baillie has recorded an instance of tubercles in the pericardium, and remarks that this disease is very rare there, and "could not have been guessed at in the living body." (Morbid Anatomy, p. 9.)

Laennec, in his celebrated treatise (vol. ii. p. 658), states that he had met with two cases of the development of tubercle in the false membrane of pericarditis; and which he thought had caused the acute to pass on to chronic pericarditis.

M. Louis reports that in 112 fatal cases of phthisis he had found the pericardium covered with false membranes three times, thus exhibiting evidences of pericarditis occurring in the course of phthisis, but he there makes no allusion to the presence of tubercles in the false membranes. In this author's Essay on Pericarditis, he relates a case of grey tubercular granulations of the pericardium, which he considered had acted as the exciting cause of the pericarditis. Andral has recorded two cases of chronic pericarditis (Clin. Med., vol. i. Obs. ix. & x.), which, from the details, may be regarded as examples of tubercular pericarditis; although it does not appear that that physician considered the inflammation of the pericardium as the consequence of the deposit of tubercles which were discovered in the fibrinous exudation. Dr. Taylor has concisely related (*loc. cit.*, Case 6, p. 457) the particulars of a case of pericarditis, the cause of which he could not assign, and which, if contrasted with those about to be related, will, I think, appear to have been an instance of tubercular pericarditis.

Rokitansky (Handbuch der speciellen pathologischen Anatomie, Band i., p. 378-9) has also described very accurately the various conditions of the pericardium when tubercle is deposited on that membrane; but he is of opinion that the tubercles result from the transformation of the fibrinous matter exuded during prior attacks of pericarditis, and not

that this inflammation is caused by the previous deposit of tubercle within the sac of the serous membrane. I shall again advert to this opinion of Rokitansky, which I believe is an erroneous explanation of the pathological phenomena.

This brief retrospect of our present knowledge of tubercular disease of the pericardium will render the following cases more intelligible and instructive.

Case 1.—Dominico Casa, an Italian musician, ætat. 24, was admitted, under my care, into St. Bartholomew's Hospital on Jan. 4th, 1844. His aspect was healthy; he complained of some pain in his right side and shoulder, of general weakness and slight cough. His respiration was frequent though not laborious. His pulse was excessively feeble and irregular, occasionally intermitting. The sputa were at times slightly tinged with blood. Upon auscultation, there was found great feebleness in the respiratory murmur throughout the left lung; but otherwise nothing morbid was detected in either the heart or lungs. He continued to improve in health up to the 22nd Jan., when, in consequence of more blood being detected in the sputa by Dr. W. S. Kirkes, who was then acting as one of my clinical clerks, auscultation was again employed, and the following note taken:—

Jan. 22nd.—He has slept well; tongue clean; bowels confined; pulse 92, rather jerking, but soft; complains of pain in the left shoulder and præcordium; cough remains; the sputa are white, rather flocculent, and contain a few globules tinged with blood.

Auscultation.—A harsh double friction sound is heard all over the cardiac region, also to the top of the sternum, and to some distance to the right of the sternum. It is most intense, and distinctly “to and fro,” at the base of the heart. There is increased dulness in the cardiac region, and to a slight extent beyond it. An exaggerated respiratory murmur is audible in the upper part of the right lung, with a very feeble respiratory murmur throughout the left lung.

He was ordered—Ol. Ricini ʒfs. statim. Hirud. x. re-

gioni cordis. Hydrarg. Chlor. gr. ij. cum Pulv. Ipecac. Comp. gr. v. 6tâ quâque horâ. Rice milk diet.

Leeches were applied to the chest on each of the two following days; the mercurial pills were continued: no change took place in the general symptoms or in the auscultatory signs of pericarditis.

Jan. 25th.—There is mercurial fœtor of the breath, and the gums are slightly tender. He has passed a good night; the cough is troublesome; the sputa more abundant; pulse 108, soft and feeble. The friction sound remains, having become more intense and more decidedly a “to and fro” sound towards the apex of the heart. The dulness on percussion continues the same. No change in the auscultatory signs of respiration. He was ordered a blister to the chest; to take the calomel and opium every night, and an expectorant draught three times daily.

On the 27th, the friction sound was less harsh, and heard less extensively towards the base of the heart and upper part of the sternum; it remained unaltered about the apex.

On the 29th, the pericardial friction sound was less harsh and of more limited extent; the friction sound with the diastole becoming less audible, and having a softer, moister character.

On the 31st, the intensity of the friction sound had still further subsided, and it could only be heard in the proper cardiac region; there was great dulness on percussion in the same space, but fair resonance immediately around it.

The mercurial pill at night and the expectorant mixture were still continued.

Feb. 3rd.—A very faint soft friction sound may still be heard in the cardiac region, but the sounds of the heart are audible through it. Dulness on percussion over the præcordium is diminished. There has been no prominence of the cartilages of the left ribs from the commencement; indeed, the cartilages appear depressed: this peculiarity has perhaps arisen from the constant pressure of a French organ, which he has been in the habit of carrying about with him.

Feb. 6th.—The friction sound is no longer audible, having been detected during fifteen days. Both sounds of the heart are free from murmurs. The dulness in the præcordium is more limited. He is quite free from pain; his cough is troublesome; the pulse 100, small and soft.

The mercury was discontinued.

Feb. 19th.—He complains of weakness and nocturnal perspirations; pulse 120, feeble and irregular; cough continues troublesome; his appetite is bad.

R. Quinæ Disulph. gr. j. cum Acidi Sulph. dil. ℥v. ter die. Port wine ℥iv. daily.

March 4th.—He sleeps well and takes his food with appetite, nevertheless he loses flesh and has a permanent flush on the cheeks; the pulse 120, feeble; the cough continues; the sputa consist of a clear mucus, having disseminated through it specks of pearly-looking matter, with occasional streaks of blood.

Auscultation.—Exaggerated coarse respiratory murmur on the right side. Inspiratory murmur feeble, and expiratory murmur prolonged and loud on the left side. Impaired resonance on percussion over the upper third of the left lung.

He continued the Quinine mixture, with an opiate at bedtime and the occasional employment of counter-irritation on the chest, without any material change in the symptoms of advancing phthisis until the 22nd of April. It was then remarked that his countenance was more anxious, his circulation more accelerated (pulse 116), and his nights had become restless. Upon auscultation, a friction sound was audible beneath the left mamma, extending round to the base of the same lung; there was also a diminished respiratory murmur with dulness on percussion below each scapula. On the following day he complained of pain about the right mamma, and, upon auscultation, a rubbing sound was audible between the clavicle and mamma. It was now evident that he was suffering from double pleurisy. These friction sounds accompanying the respiration varied in intensity and extent during the twelve succeeding days. At the same time the

dulness on percussion, posteriorly, increased from below upwards. This attack of pleurisy was treated by one abstraction of blood by cupping from the back, by the application of two blisters, and by small doses of calomel and diuretics.

On May 6th the pleural friction sounds had quite disappeared, having existed fourteen days, but the physical signs of extensive effusion on both sides of the chest were more decided. Blisters were again applied to the chest, and more stimulating diuretics employed.

On May 9th, the respiratory murmur was coarse and exaggerated in the upper lobes of each lung, and altogether absent in the lower lobes; there was considerable dulness on percussion over the lower half of both lungs.

On May 10th this patient died, without any material change in the symptoms.

The body was examined twenty-eight hours after death; it was much emaciated, and the limbs were rigid.

Lungs.—The right pleura was about two-thirds filled with bloody serum; the left pleura about one-third filled with serum, in which were floating a few shreds of coagulated lymph. The right lung was much compressed and shrunken; the free surfaces of the right pleura were covered with a thick layer of false membrane: this latter contained numerous opaque yellowish tubercles, about the size of millet seeds, deposited singly and in groups. The surface of the false membrane was slightly reticulated; there were numerous adhesions, some firm and others soft, between the opposed surfaces of the pleura; at the base of the lung the pleura was firmly adherent to the diaphragm. The substance of the lung was tough, inelastic, and contained very little air; there were a few small groups of recent tubercles in the upper lobe, less numerous in the middle and lower lobes.

The left pleura was in the same state as that on the right side; the upper lobe of the left lung contained plenty of air; the lower lobe was rather compressed: a few groups of tubercles, of the same description as those on the right side, were observed throughout the lung, but most numerous towards

the apex. The absorbent glands about the trachea and larger bronchi were much enlarged, and filled with tuberculous matter.

Heart.—The surfaces of the loose and reflected pericardium were rendered entirely and firmly adherent by a layer of lymph, studded with minute tubercles. The external surface of the pericardium was also adherent to the pleura by a false membrane, in which were deposited tubercles similar to those in the false membrane lining the pleura. The cavities and valves of the heart were natural.

Abdomen.—The peritonæum was very much thickened by false membranes, in which were deposited numerous crude tubercles of larger size than those of the pleura. The convolutions of the intestines were agglutinated into a mass by adhesions. The liver was covered with a thick, firmly-adherent false membrane; its substance was healthy. The spleen contained a few tubercles, but was otherwise healthy. The left kidney congested; the right healthy. The brain was not examined.

Andral has related a case of chronic pericarditis* complicated with double pleurisy, which is very analogous to the foregoing. The symptoms of cardiac disease were very obscure; and although pericarditis was complicated with double pleurisy, and both disorders went through their various stages up to the fatal termination, there was very little complaint of pain by the patient. Nothing but the physical signs revealed the extent of thoracic disease during the life of the individual.

In Andral's case, there was found a layer of false membrane, an inch in thickness, between the free surfaces of the pericardium; and in this false membrane were discovered numerous small tubercles, some crude, and others softened. The left pleura was distended with sero-purulent effusion; while the right was agglutinated by false membranes, in which were detected numerous tubercles similar to those deposited within the sac of the pericardium. There was also

* Clin. Med., vol. i., *Maladies du Cœur*, obs. x.

abundant serous effusion in the peritonæum. The state of the lungs is not recorded, although they are reported as having lesions in them.

The particulars of the following case were communicated to me by my friend, Dr. Baly. The patient was only occasionally seen by myself during his last illness :—

Case 2.—John C——, ætat. 19, of a leucophlegmatic complexion and habit, but of rather a strong frame of body, had enjoyed good health previous to his confinement in Millbank Prison. He had been already more than two years in that prison, when, on the 12th of Feb. 1842, he was attacked with severe dysentery. From the acute stage of the dysenteric attack he recovered under a mercurial treatment (calomel combined with opium) ; but on returning to a diet of animal food, and before he had quitted the infirmary, diarrhœa, unattended with pain or any feeling of illness, came on. The alvine evacuations were almost totally devoid of bile, and for this reason moderate doses of mercury, combined with opiates, carminatives and astringents, were given to check the diarrhœa. This relapse took place in the first week of March 1842. No permanent benefit was derived from the remedies employed, although the diarrhœa was from time to time, for a day or two, arrested by the astringent medicines ; but it recurred as often as the use of them was remitted, and at the same time the patient became more and more pallid, thinner, and feeble. About the 20th March he was ordered to take the Sulphate of Iron with Infusion of Calumba and Tincture of Opium every eight hours, and some brandy was allowed him. In spite of these measures he became weaker.

On the 30th March, it was remarked that, in addition to his increasing debility, the respiration was hurried and the pulse 108. The chest was examined by auscultation, and nothing but a rather exaggerated vesicular murmur was detected. Attention was not particularly directed to the state of the heart, for he had no pain in the præcordial region, did not complain of palpitation, and the frequency of the pulse

was regarded as a consequence of the hurry of respiration, and the state of general debility. During the week following, the symptoms remained the same in kind, but became exaggerated in degree. The debility and dyspnœa rapidly increased, and the pulse, concurrently with the respiration, was more accelerated. No increase of diarrhœa occurred, to account for the rapid failing of the vital powers. He died on the afternoon of the 5th of April 1842.

Upon examination of the body after death, the colon was found extensively ulcerated; the largest ulcers were not more than a quarter of an inch in diameter, and their edges were devoid of vascularity. Both lungs were thickly studded with tubercles, for the most part in the yellow crude state, varying in size from a millet seed to a small pea, not in masses or clusters, but pretty equally disseminated throughout all parts of those organs.

The two opposed surfaces of the pericardium were firmly attached to each other, over a great extent, by a thick deposition of firm false membrane. Where the surfaces of the pericardium were not attached to each other, they were covered with thick layers of false membrane, which had a somewhat reticulated texture. The whole substance of the effused lymph was stained of a deep red colour; coagulated blood and bloody serum were intermingled with it at various points. In one part near the base of the heart, where the false membrane was thickest and firmest, upon separating the layers of the pericardium, three or four small yellow tubercles were distinctly recognised imbedded in the lymph. Their size was about that of a grain of pearl barley.*

A drawing of the heart was made on the day after the death of the patient, and is preserved in the Museum at St. Bartholomew's Hospital.

* I much regret that, from an accident, the false membranes in other parts of the pericardium were not examined, so as to determine the amount of tubercular deposit there; but it appears probable that a similar condition existed in all parts.

Case 3.—John P. K., ætat. 18, a delicate man, of spare habit of body, was admitted under my care into St. Bartholomew's Hospital on May 30th, 1842, and gave the following account of himself. He was a native of Brighton, and had no regular occupation. He had been sentenced to the hulks in November 1840, and had remained there until July 30th, 1841, when he was transferred to the Millbank Penitentiary. His health had been good, according to his own statement, up to September 1841, when he began to suffer occasional attacks of diarrhœa, and on May 9th, 1842, he was placed in the Infirmary of the Prison, on account "of an increase of diarrhœa and general feelings of illness." During the following five days he was treated by Dr. Baly for the intestinal affection, when that physician observed an unusual dulness on percussion over the præcordial region, and, on auscultation, a friction sound in the same situation was distinctly audible. Calomel and opium were administered to a considerable extent, but the gums were not affected by the mercury. The extreme weakness which this patient now began to manifest, and the continuance of diarrhœa, induced Dr. Baly to suspend this treatment, and to order Ferri Sulph. gr. j. cum Pulv. Opii gr. fs. ter die; also Brandy ʒij. daily.

His strength appeared to be rapidly failing, when his pardon was granted on May 30th, and he was admitted under my care into St. Bartholomew's Hospital the same day. He was much exhausted by the removal, and presented the following symptoms on his admission :—The countenance pallid, and rather anxious; the voice weak; pulse 128, small and weak; tongue clean and moist; bowels much relaxed; respirations 28 in a minute, hurried, but not difficult. He complained of his extreme weakness, and of a short dry cough, but of no pain, except when pressure was made in the intercostal spaces in the cardiac region or beneath the margins of the ribs in the left hypochondrium. Upon auscultation, a friction sound was heard over the præcordial region and quite

across the sternum to the right side. The respiratory sounds were healthy.

He was ordered, in reference to the diarrhœa, *Ferri Sulph. gr. j. c. Opii gr. fs. 6tâ quâque horâ*, and a diet of bread, arrow-root, two eggs, milk, and $\frac{3}{4}$ j. of port wine daily.

He slept well the night of his admission, and on the following day his condition was unaltered, excepting that his bowels had acted only once in twenty-four hours. The pericardial friction sound remained the same. He was ordered *Hydrarg. Chlor. gr. v. c. Pulv. Opii gr. j. horâ somni*.

June 1st.—Pain was still felt upon pressure in the cardiac region: respiration natural, pulse 112: bowels not open.

The friction sound was less intense, and the proper sounds of the heart could be heard through it. Dulness on percussion in the cardiac region scarcely more than natural. He was ordered *Hydrarg. Chlor. gr. iij. c. P. Opii gr. fs. horâ somni. Olei Ricini 3fs. cras mane*.

June 2nd.—The friction sound could no longer be detected, having been heard during twenty days; but the dulness on percussion was rather greater than normal in the cardiac region.

From this date he continued to progress satisfactorily, and on June 9th, when he quitted the hospital, contrary to my advice, there was no pain, dulness on percussion, nor morbid sound detectible by auscultation. His weakness was still considerable when he left the hospital.

Although the event, in this last case, did not allow us the opportunity of verifying our diagnosis of pericarditis, nor of confirming our opinion that it was an instance of tubercular pericarditis, still the history of the individual, the circumstances under which the illness arose, so perfectly analogous to those which accompanied the preceding case; the absence of any rheumatic symptoms, and the favourable progress of the patient, when he was removed from the influence of various causes which had long been depressing his spirits and his constitutional powers,—all these circumstances appear to

justify the opinion already expressed as to the nature of the disease with which this young man was affected.

The cases of tubercular pericarditis here recorded are but three; but it must be remembered that all our knowledge of disease, which is worth the title of knowledge, is derived from the observation and comparison of cases. In rare diseases, the experience of the individual physician must be small; and one of the great advantages of this Society is, that, from the assemblage of numbers and from their accumulated experience, a more complete history of a disease may be compiled than can be drawn up from the limited number of facts in the possession of any individual member.

Before concluding this communication, I will venture upon a few pathological remarks on tubercular pericarditis. First, with respect to the circumstances which appear to favour the development of tubercular pericarditis. I have no statistical records of the cases of phthisis pulmonalis, which have passed under my notice at St. Bartholomew's Hospital, and which would enable me to estimate the frequency of tubercular affections of the pericardium in phthisis. Louis reports that, in 112 fatal cases of that disorder, he found the pericardium covered with false membranes three times, but he does not state whether tubercles were looked for in the false membranes. It is probable that many more phthisical patients must have passed under his notice who were similarly affected, although the disease was never suspected nor looked for, because simple pericarditis often occurs without being detected during life, and does not commonly end fatally. So far as any conclusion may be drawn from a limited number of cases, it would seem that tubercular pericarditis is not so much to be looked for in ordinary cases of phthisis, where tubercular disease, in the form of granulation and infiltration, invades the different lobes of the lungs, and rapidly runs through its disorganizing processes; but this disease is rather to be anticipated when individuals, previously healthy, are exposed for a length of time to the influence of the most

powerful exciting causes of the tubercular diathesis. Dr. Baly, in his elaborate and valuable essay, "On the Mortality of Prisons," (Transactions of this Society, vol. xxviii.), arrives at the conclusion, "that not merely pulmonary phthisis, but tubercular disease in all its forms, is the frequent result of the long-continued influence of imprisonment on bodily health." The fact that two cases out of the three just recorded, occurred among persons who had long been confined in Millbank Prison, tends to corroborate the opinion advanced by Dr. Baly. The circumstances under which the first related case occurred, are not very dissimilar to those of the other two. The patient was an Italian musician, who, having been tempted to leave his sunny clime, found himself doomed to spend a northern winter, and such a severe one as that of 1843-4, wandering through the dreary streets of this metropolis, daily wearied with his burthen, dispirited, badly fed and clothed, and passing the long winter nights in a close, ill-ventilated lodging. Such is the class of persons among whom this disease should be sought after or suspected, and particularly if the constitutional symptoms of tubercular disease are well marked, although the physical signs of pulmonary disease are but slight and equivocal.

Other questions of pathological interest here present themselves, and are worthy of investigation. What relation do the tubercles and the inflammatory fibrinous exudation in the pericardium bear to each other? Which lesion is cause and which effect? Or do both lesions result from contemporaneous morbid actions?

Laennec and Rokitansky (*loc. cit.*), who have both described this lesion of the pericardium, have, I think, erroneously supposed that the tubercles are deposited in the false membranes of pericarditis, subsequent to, and as a consequence of, the inflammatory process. Whereas, it appears to me more consistent with our present knowledge of the exciting causes of inflammation and tubercle respectively, to regard the inflammation of the serous surface of the pericardium as resulting from the previous accidental de-

posit of tubercle on the membrane, and to ascribe the production of the tubercles to the operation of causes quite independent of inflammation, and acting prior to the inflammation. Rokitsansky, indeed, broadly affirms that tubercles occur in the pericardium most commonly as a product of inflammation, and very seldom under any other form. His explanation of the formation of tubercles on the pericardium is nearly as follows:—"An attack of pericarditis deposits an exudation, the peripheral coagulated portions of which undergo either complete or only partial conversion into tubercle. When a second attack of pericarditis ensues, it frequently happens that the deepest or oldest layers of the exudation become converted into tubercle; or, it may be that the most recently-deposited layers of fibrin, having become tubercular, are covered by a villous shreddy substance deposited from the fluid effusion. The tubercles, which are often very large and aggregated into masses, are sometimes deposited quite close upon the muscular substance of the heart. These tubercles seldom go on to softening, for most commonly death soon occurs, either from the pericarditis itself, or from deposit of tubercles elsewhere, or from the general cachexia."

Laennec has also advanced a similar opinion, and gone so far as to state that these tubercles, deposited in the false membranes after an attack of acute pericarditis, cause the inflammation to become chronic, thus regarding them as an effect of acute and a cause of chronic inflammation.

It appears to me a more simple and philosophical explanation of the phenomena, and one more in accordance with our present knowledge, to state that the tubercles are deposited in the first instance as a result of tubercular cachexia, and that these foreign bodies then excite an inflammation of the serous membrane on which they are deposited, and that fresh crops of tubercles springing from the same state of constitution keep up the inflammatory action, and it is thus that the acute is converted into chronic pericarditis. The situation of the tubercles, as described by Rokitsansky, in close contact with, and almost imbedded in, the muscular substance of the

heart, points out that they are deposited there prior to the fibrinous exudation with which they are covered. A similar explanation of the pathology of that variety of chronic peritonitis which is connected with the presence of miliary or tubercular granulations on the peritonæum is adopted by Dr. Watson, who states (Lect. 66, 2nd edition) that he is in the habit of regarding the tubercular granulations as the cause and not the consequence of the peritoneal inflammation with which they are found associated. Most pathologists admit that tubercles, like other foreign bodies, may act as irritants to the tissues with which they are long in contact, and thus excite inflammation. Andral,* Lombard of Geneva, Carswell, and some others, explain the softening of tuberculous matter through the intervention of a purulent secretion poured forth from surrounding inflammation excited by the tubercular deposit. It is not surprising, then, that inflammation should be excited on the surface of a serous membrane by the deposit of tubercle there, and that such inflammation should lead to an abundant fibrinous exudation.

If such be the correct explanation of the origin of this variety of pericarditis, I think the ordinary treatment of that affection, viz., by repeated abstraction of blood, and by bringing the system under the full influence of mercury, is quite inappropriate. Such remedies, by impairing the powers of the constitution still further, would certainly favour the continued development of tubercle in the pericardium and elsewhere, inasmuch as it is from cachexia that a similar condition of other serous membranes is observed in the same individual. Tubercular pleuritis or peritonitis is frequently found co-existing with the tubercular pericarditis, and it is quite uncertain which serous membrane may have been the primary seat of deposit of the tubercles. If depletion be practised in these cases, a very moderate abstraction of blood will be sufficient; mercury should be used sparingly, and every precaution taken to prevent ptyalism. Counter-irritation by

* *Precis de Pathologie*, vol. i. p. 415.

blisters over the cardiac region, saline diuretics with compounds of iodine, and the speedy removal of the patient from the influence of those causes which are known to favour the development of the tubercular cachexia, will probably be found the most efficacious remedies in obviating the fatal consequences of the chronic pericarditis produced by the irritation of tubercular deposit.