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Vacation Lectures

ON FIBRINOUS DEPOSITION IN THE HEART:

ITS HISTORY AND PRACTICAL MEANING.

DELIVERED AT THE GROSVENOR PLACE SCHOOL OF
MEDICINE.

By BENJAMIN W. RICHARDSON, M.A., M.D., M.R.C.P.,
Lecturer on Physiology at the School, and Senior
Physician to the Royal Infirmary for
Diseases of the Chest.

LECTURE II.

*Conditions of Disease in which Deposition of Fibrine occurs in
the Heart: Symptoms and Diagnosis of Concretion.*

GENTLEMEN,—From points connected with the structure of fibrinous deposits, and the proofs that these deposits are in many instances laid down prior to death, we pass naturally to consider those conditions of the system which are favourable to deposit—in other words, the diseases in the course of which deposits are formed. To understand clearly the question here involved, we must consider the modifications to which the fibrine of the blood is subjected under various diseased states, and especially those modifications leading to deposit.

1. In various conditions the fibrine is, or seems to be, diminished in the blood. These conditions are indicated in diseases in which the hæmorrhagic tendency prevails. True typhus, and yellow fever, are diseases of this nature. The effects of many poisons, as the cobra poison, the vegeto-alkaloids, the alkalies, and several organic acids, are manifested in this apparent deficiency of fibrine. In these cases, the blood, as Plato would express it, “is no longer under the natural guardianship of its fibres”; so it exudes into the soft structures, giving rise to petechial spot, black vomit, melæna, hæmaturia, and, in fact, to any form of hæmorrhagic discharge, according to the degree of pathological change.

I have said that, in these cases, the blood seems to be deficient in fibrine. I say *seems* to be deficient, because I wish to guard myself from saying absolutely that the blood is so deficient; and I would impress this qualification earnestly, because, from some late inquiries on the effects of alkalies and alkaloids on blood containing, when drawn, the normal amount of fibrine, the same apparent decrease of fibrine has been produced by the mere addition of these agents in excess to the blood. We need not, therefore, bind ourselves to the idea that, in diseases attended with great fluidity of blood, less fibrine is really produced in the system; we may rather infer that, as all evidence tends to prove, there are conditions in which the blood is surcharged with a body having the power of holding fibrine in solution—of reducing the fibrine, in fact, into a form of albumen. In such condition of blood, whether my explanation of its cause be correct or not, this that I am about to say shall be correct: the blood, while in the condition named, never, either before or after death, yields a fibrinous separation. Ordinarily, it does not coagulate even as red clot, but at most it becomes a thick treacly mass, like newly drawn blood treated with an alkali and afterwards mixed with albumen.

2. There is a second variety of blood, not widely removed from the above named kind, in which there is nevertheless a peculiar tendency to separation of fibrine; and this without any necessary increase of the fibrine itself, or the presence of those systemic states in which the combustion of the body is increased. The older writers were accustomed to notice this separation of fibrine in scurvy. They spoke of the stream of

blood in that disease as having two colours, a purple and a white; and of a loose buffy coat forming on such blood. I am convinced to proof that the condition here expressed exists—exists sometimes, as it would seem, by hereditary predisposition; at other times, by acquired disease, especially by disease derived from peculiarities of food. I do not pretend to be master of all the facts connected with this subject, but I will narrate such few as I know. I once attended a child covered from head to foot with purpuric spots—a miserable puny child, cold always, and emaciated. This child died suddenly; and I traced the cause of the sudden death clearly enough to a large concretion of fibrine which had been laid down in the right auricle. I also knew an instance of extreme anæmia terminate in a similar way; the only difference being, that the concretion had formed in the pulmonary artery, and not in the auricle. In a case of purpura at this very time under my observation, there is every disposition to effusion of blood under the skin, and large purpuric blotches are produced from the slightest cause; but, some time after this effusion has occurred, there is a hardening of the matter at the effused point; and it is observable that the blood of this patient, although it is so easily effused, is not incapable of coagulation when it escapes from the body.

3. There is another condition, in which the fibrine of the blood undergoes relative increase. It may be difficult at first to understand what is meant by relative increase. I mean by it, then, simply a state in which the fibrine is not abnormally increased, nor the system under any condition for the production of an increase of fibrine, but in which, the fibrine remaining normal, the watery constituent of the blood is below the usual standard. This condition is not uncommon; for, water being the grand menstruum in which the fibrine is held in solution, any reduction of it in amount will favour materially the tendency to deposit. It may seem, at first sight, that this statement runs counter to that which was made above relative to anæmia; but there is no contradiction. Let me explain. Water is, as I have said, the menstruum in which the fibrine is held in solution. But fibrine will not remain in solution in water simply; the water must be the menstruum also for an agent having the power of dissolving the fibrinous constituent. This agent, as we now have proof beyond every kind of dispute except sheer cavil, is an alkali of the volatile series. Now, in order that fibrine should remain in solution, there must be a fixed relationship existing between the menstruum, the solvent, and the substance dissolved. If the substance dissolved is but dissolved, and no more, as fibrine is in blood, precipitation of fibrine can be induced by addition of more water, because, in this addition, we distribute the solvent over a larger surface; we weaken, that is to say, the solvent power of the solution; and the result necessarily is, that the dissolved substance is proportionately precipitated. On the other hand, if from a solution of fibrine, as in blood, we draw off by osmotic action the watery part, together with the solvent, the fibrine is necessarily increased in relation to the portion of water and solvent left behind. In both cases alike, there is, therefore, a tendency to the deposition of fibrine. Out of the body, I can produce in experiment all these phases; and in the bodies of the dead we constantly meet with the results of these changes, if we are observant observers.

The best marked cases in which the fibrine is found separated after death, as the result of loss of fluid from the system, are those where, during life, there has been an exhausting and rapid flux from the alimentary canal. In cases of cholera, the fibrinous deposit so commonly found after death is formed in this way; and the same may occur in other disorders affecting the alimentary system. I have seen them, as so produced, in death from mesenteric disease; and once in a debilitated person, after a fatal purging, induced primarily by self-dosing and overdosing with black draught. Deposition, brought about by loss of fluid, will further happen from rapid elimination through other channels than the bowels, as from the skin in the colligative sweating of phthisis.

Once more; the fibrine may be relatively increased, not

in proportion to the water, and not in proportion to the mass of blood, but in proportion to its own solvent. The old writers were accustomed to state that concretions were the result of what was called languor of the circulation. We cannot adhere to that phraseology at this time of science, because the phraseology is imperfect; but we may, nevertheless, accept that these writers did truly express a partial explanation of a very interesting fact. In many cases of great prostration of the body and of the powers of the circulation, where, notwithstanding, life is slowly destroyed, the blood making languid way for many hours—in such cases (and I may add, in the majority of such cases), the fibrine is left deposited in the heart. The cases themselves include: (a) deaths from shock, where the life is not extinguished at once, but in which the act of death has been very considerably prolonged, say for a period of from three to four days; here the fibrine may be separated in a firm and decided mould; (b) deaths from the prolonged effect of some poisons, which depress the heart without adding any solvent substance to the blood, such as opium; and (c) those instances of disease where death is preceded by great degenerative change, and by slow and certain exhaustion. I have met with *ante mortem* concretions in examples of this nature, after death from mere senile decay, fatty degeneration of the heart, and slow alcoholic poisoning. The concretion, however, in these cases, is often but indifferently developed. It is gelatinous in character, very heavy at first from the presence of water, but containing in reality a very small amount of fibrine. Dr. Gould, whom I have noticed once before, laid great stress on debility as a cause of deposition. He opined—and the opinion has been reiterated by Dr. Meigs of Philadelphia—that, owing to the stasis produced in the blood during a fit of syncope, the commencement of a fibrinous deposition may be traceable, in some instances, to an attack of syncope. Nor is it to be denied that many arguments lend a colour to this view of stasis as a cause of concretion. The observation made by Lancisi respecting the mode in which fibrine is deposited in aneurism, the first point of deposit in the sac of an aneurism being that at which the stasis is most marked, was alone of sufficient importance to establish the stasis hypothesis for a long period; and the more so, because this hypothesis is not opposed to the truth, but is only faulty in that it does not express the whole truth. The position of the question is as follows:—When blood yet fluid is placed entirely at rest in a closed cavity, and under a pressure equal to that of the circulation, there is no separation of fibrine. On the contrary, blood so enclosed is in the best condition for its fibrine to remain in solution. Here is an argument, then, in direct opposition to the pure stasis view; flatly disproving it, indeed, as a bare hypothesis. Blood, therefore, from which *ante mortem* concretion is formed in the asthenic, is not blood in a state of absolute rest. No; it is blood undergoing slow and languid motion, in process of which the volatile solvent of the fibrine, a product of an active nutrition, is in the first place imperfectly supplied, and, secondly, is eliminated in the expirations of the skin and lungs, to the loss of the blood. The relationship existing between the fibrine and its solvent being thus destroyed, the fibrine is precipitated, and is found in the separated condition after almost all forms of death from asthenia, excepting those where a solvent poison is present as part of the disorder.

4. The last condition favourable—most favourable—to fibrinous separation, is that in which the blood undergoes an absolute increase of fibrine—that condition which the old men were content to designate as disease “attended with sizzly blood”, and which we moderns, following Franz Simon, call “hyperinosis” or superfibrination. We include under this head a large group of diseases marked by inflammatory symptoms, and always running an acute course; pneumonia; croup; diphtheria; acute rheumatism; erysipelas; inflammatory diseases of all the serous and mucous tracts; the inflammatory reactive fever succeeding on surgical operations; inflammatory lesion in the uterus and peritoneum after parturition; and cerebral congestive excitement.

In thus naming certain local symptoms indicating what we are pleased too often to consider as pure diseases, I write to make the subject intelligible. In my opinion, these local changes are all secondary to one grand systemic derangement, a primary and essential feature of which is increase of fibrine—hyperinosis.

Still farther, I maintain that there are cases in which the hyperinosis is not only preexistent to any local manifestation of disease, but in which it is so marked, and the deposition of fibrine in the heart as a consequence of the increase is so rapid, that death takes place from the obstruction, before any local

change is developed. Instances of this nature, while they are not by any means numerous, are, as a general rule, overlooked. There are, nevertheless, to be found in our literature certain extraordinary examples of this nature, worthy certainly of notice. One of the very best observers of disease in the last century, Dr. Huxham, in a letter written by him to Dr. Mortimer, describes what was sometimes called an epidemic of cardio polypi. In this letter, it is narrated that several seamen, brought during cold weather into England from the West Indies, were seized with short coughs without expectoration, continual and violent palpitations of the heart, intermitting trembling pulses, anxiety, and pain and sinking at the heart, so that they could not lie down; they had heavy dead countenances, some pains in the side, but very little fever. Twenty men thus soon died. Two of these men were opened, and in their hearts were found monstrous polypi, tough and adherent to the walls of the ventricle. One of these weighed an ounce. They were on both sides the heart; the largest on the right side.

A still more remarkable illustration of a similar epidemic is described in the *Edinburgh Annals of Medicine* for the year 1800, by another equally good observer, Dr. Chisholm. I have often noticed this valuable paper, but now I will give it almost entire. It is entitled “A Short Account of the Epidemic Polypus in Granada in 1790.” The sufferers were negroes, who were subject to peculiar local influences at the time of the seizure. These influences Dr. Chisholm first describes. The foreground was the open sea, with an extensive and bracing beach of sand. On the left was a hill of considerable size and ascent, the base sides of which, with the reflecting surface of the sea, produced in dry seasons an immense degree of heat. The background was a marsh, extending from the sea to the mountains. From this marsh deleterious vapours were exhaled. Immediately behind the marsh, a ravine began, through which rushed a current of wind of great coolness. The negro houses of the plantation were built on the hill on the left, chiefly on its slope and towards the edge of the marsh. The negroes were, therefore, at once exposed to excessive heat, a cold chilling current of air, and the miasma of the marsh. Their diet was chiefly vegetable food. They had been employed, immediately before the appearance of the disease, in clearing the surface of the marsh, and in holing land for the reception of cane-plants. They were much given to the destructive habit of eating a species of pipeclay very abundant in Granada.

The disease commenced in the end of September, was most prevalent in the latter part of October, and went off in November. The whole number of the sick was about forty, of whom seven died.

The symptoms were, pain at the pit of the stomach and head, and difficult respiration, attended with dry skin, small quick pulse, and slight dry frequent cough. No febrile heat accompanied these symptoms. The surface, on the contrary, was remarkably cool; but there was a heaviness and dulness of the eye, anxious features, and depressed spirits. This state continued for three days; then the pulse became quick, from 120 to 140, and intermitted, attended with a penetrating pungent heat, which produced a pricking sensation on the hand of the person feeling the pulse.

The disease now became somewhat intermittent, the intermission lasting eight or nine hours. During the paroxysm, the struggle for breath, the aggravation of all the other symptoms, and the *very quick*, interrupted, and evidently visible, as well as audible palpitation of the heart, produced a scene of uncommon horror. The paroxysm was succeeded by a cold clammy sweat, and a state of approaching syncope. The second paroxysm generally put a period to the existence of the patient. The disease was also distinguished, during the latter stage, by an almost constant, disagreeable clammy sweat, overspreading the face, the upper extremities, and the body as low down as the scrobiculus cordis, all below remaining parched in a most remarkable degree. The disease seemed sometimes inclined to terminate by a metastasis. One instance of this was remarkable, wherein a patient, after labouring under all the symptoms peculiar to the disease before the intermittent period, found himself all at once, and without an evident cause, relieved of them; but he perceived at the same instant an excruciating pain a little above the elbow, and nearly about the middle of the thigh. He continued ever afterwards quite free of all the symptoms of the polypus; but they were succeeded by a large abscess in the parts in which he felt the pain. That in the arm disappeared gradually; but the other became so large as to occupy the whole of the under part of the thigh. The cure was effected by passing a seton through the whole length of the tumour; by the use of two dozen of

Madeira wine, bark, and a calomel pill with opium, three times a day.

The audibleness of the palpitation of the heart in these cases, Dr. Chisholm remarks, may be considered an exaggeration; but in one instance, particularly, a gentleman (Mr. McSween), to whom the negroes belonged, heard distinctly the palpitation from an adjoining room.

Dr. Chisholm states that, at the onset of the epidemic, he knew not how to act until dissection showed him; and adds, that having no suspicion of the heart being the seat of the malady, he did not examine that organ in the first two bodies opened; but finding the brain and all other organs in a state of health, he opened the heart in the third body, and discovered what he considered might be the cause and seat of the disease. In the right ventricle of the heart he found an enormous polypus, which extended considerably into the pulmonary artery. On extracting it, the body which was contained in the ventricle, measured two inches in breadth. In the fourth body there was a large polypus in the right and left ventricle, besides one in the right auricle. The hearts of the fifth, six, and seventh were just similar; and in these five, except one, where the lungs were diseased, no other morbid appearance could be found.

It is interesting to remark, as an aid to our therapeutics, that Dr. Chisholm, after he had discovered what he considered to be the cause of death, viz., the concretions in the heart, changed his plan of treatment. On the very first appearance of symptoms, he took blood, and then freely salivated, with mercury. The results, he adds, were most happy; not one that was salivated died.

I can offer, on my own part, no such a narrative as that given by Dr. Chisholm; but I can very definitely refer to not fewer than six well marked cases in which death, taking place rapidly, with no other foreboding symptoms than a rigor and succeeding slight febrile heat, was traceable by the *post mortem* inquiry to deposit of fibrine in the right side of the heart; the deposit filling the auricle, to the exclusion of blood, and extending, in the course of the circulation, from the auricle into the ventricle and pulmonary artery.

In other instances, as we have seen, the deposit of fibrine is subsequent to the development of one or other of those forms of local mischief to which we assign a specific name. The obvious features of such cases are distinguished by the presence of the local change, but the essential characters are the same in all examples, that is to say, the fibrine is abnormally increased. The increase may extend from the normal two and a half or three parts per thousand, to six, nine, or it has been said to twelve parts per thousand. This latter figure is possibly derived from an error in analysis, but the fact of a great increase is without a shadow of doubt; the result of the increase is also often equally striking. The excess of fibrine, beyond a limited extent, cannot remain in suspension in the blood, and the heart, under such conditions—if so plain but expressive a term be allowed me—*churns* out the fibrine, and becomes the receptacle of the separated mass.

I can give, at this moment, no explanation of the first cause of increase of fibrine in the cases I have named, except by entering upon the whole question of hypercausis and its origin; which task I would rather avoid, or at least retain. It will be more instructive, at this time, to pass to the subject of the symptoms of concretion, concerning which all is demonstrative.

[To be continued.]

CHILDREN'S HOSPITAL IN EDINBURGH. A house (No. 7, Lauriston Lane, Edinburgh), has been fitted as a Children's Hospital, and was to be opened on February 1. The objects of the institution are:—1. To provide for the reception, maintenance, and medical treatment of the children of the poor during sickness, and to furnish with advice and medicine those who cannot be admitted into the hospital. 2. To promote the advancement of medical science with reference to the diseases of childhood, and especially to provide for the more efficient instruction of students in this department of knowledge. 3. To diffuse among all classes of the community, and chiefly among the poor, a better acquaintance with the management of infants and children during health or sickness, and to assist in the education and training of women in the special duties of children's nurses. *Consult. Phys.*—Prof. Christison, Dr. C. Wilson. *Consult. Surg.*—Prof. Miller. *Ordinary Phys.*—Dr. G. Weir, Dr. P. Newbigging, Dr. J. M. Duncan. *Resident Med. Officer*—Mr. W. B. Robertson. *Med. Com.*—Drs. A. Peddie, Smith, Struthers, and H. D. Littlejohn.

Illustrations

OF

HOSPITAL PRACTICE:

METROPOLITAN AND PROVINCIAL.

DORSET COUNTY HOSPITAL.

CASES OF NEURALGIA, ETC., SUCCESSFULLY TREATED BY NARCOTIC INJECTION.

Under the care of CHARLES COWDELL, M.D., Physician to the Hospital.

[Cases reported by D. W. PHILLIPS, Esq., House-Surgeon.]

At the anniversary meeting of the British Medical Association, held at Edinburgh in July 1858, a lecture was delivered by Dr. Alexander Wood on the method of treating neuralgia by narcotic injection, the narcotic chiefly recommended by him being a solution of morphine. This method had yielded many successful results.

It has since been tried in some of our British hospitals (see report of cases in St. George's Hospital, *BRITISH MEDICAL JOURNAL*, January 8, 1859), and more thoroughly, perhaps, in the Parisian hospitals. In these last, the sulphate of atropine appears to have afforded the greatest relief.

In the following cases, a solution of morphine was tried in one with promise of a good result; but the patient would not permit a continuance of the treatment. In the other three, a solution of atropine (two grains to the ounce) was the narcotic employed, being slowly injected by means of the small *navus-syringe*, now in common use, which has a screw piston, whereby the quantity can be both measured and injected *guttatim*. A similar instrument was employed by M. Hérard, of the Lariboisière Hospital, in Paris (*Journal de Médecine et de Chirurgie*, etc., 1859; art. 5680, p. 337, and art. 5694, p. 392). It is delineated in the journal referred to (p. 394).

CASE I. Susan Ellis, aged 20, domestic servant, was admitted into hospital September 29, 1859, with the following statement:—"She had an attack of acute rheumatism five months ago, with delirium at intervals during three weeks; and on recovery she was seized (sixteen weeks ago) with an acute pain in the left hip. The hip has been repeatedly leeches and blistered without benefit." She cannot stand or sit. She is extremely anæmic, and in the region of the sciatic nerve, experiences violent pain, which is much aggravated by pressure. There is no hip-disease; the contour is normal; the muscles of the thigh and leg are very flabby; the ham-strings are contracted and tense; the knee is bent, and the foot drawn up. She has, during the whole time, been unable to walk without crutches. She was ordered meat diet, cod-liver oil, and a mixture of iodide of potassium.

Sep. 30th. She passed last night, as usual, without sleep, and tortured with pain. Half a drachm of the solution of atropine (two grains to the ounce) was ordered to be injected into the track of the nerve to-night.

Oct. 1st. She slept well all night. She says she has not had so good a night for sixteen weeks. She felt drowsy immediately after the injection.

Oct. 5th. The injection was employed last night. She slept well, and this morning she is quite free from pain.

Oct. 6th. There is still no pain. She was ordered ammonio-citrate of iron for the anæmia. The iodide of potassium was omitted.

She was treated for anæmia from this date to November 10th, when the report runs thus:—

She has had no pain in the hip since the last injection on October 4th. Two injections only were employed, at an interval of three days, the first of which gave immediate relief; the second removed pain, soreness, and contraction altogether. She can walk with ease, experiences no pain on pressure over the nerve, and has gained flesh rapidly. She was discharged well.

CASE II. Henry Parsons, aged 25, plasterer, was admitted with chronic rheumatism December 1st, 1859. He had for ten days before admission had so much pain in his loins, that he was quite unable to turn in bed. He had been unable to work for six or seven weeks. He was treated with guaiacum and Dover's powder, and an iodide of potassium mixture, until January 12th, 1860, when, as he complained of additional pain in the lumbar region, the atropine injection was ordered.