diet, and taking the proper medicines. Others had a relapse, and were relieved by the same treatment. "I knew a man," says Galen, "who in the spring of each year was subject to palpitation. During a period of three years he was relieved by bleeding in the attack; the fourth year he prevented the attack by being bled beforehand. This man, notwithstanding, died before old age had fairly set in. The age, generally speaking, for such serious attacks is between forty and fifty, and they are often fatal by syncope."

As a type of actual organic disease of the heart and valves (possibly atheroma of the latter at the mitral orifice) I shall quote Galen's narration of the case of Antipater, a physician of some eminence practising at Rome:—"When between fifty and sixty years of age, Antipater was attacked by the ephemeral fever, on the cessation of which he examined his own pulse, with the view to further treatment. Detecting great irregularity in the movement of the arteries, he became at first greatly alarmed; but soon afterwards, feeling that the fever had entirely left him, he quickly took a bath; his physical power being exhausted by work and want of rest. He next adopted a guarded regimen, till the end of the third day, reckoning from the accession of the fever. Having experienced no relapse, he resumed his ordinary occupation; but he was greatly surprised, on feeling subsequently the same irregularity of the radial pulse. Meeting him one day, he held out his hand, and, laughing, asked me to feel his pulse. I, also smiling, replied, 'What enigma are you propounding?' He again, laughing once more, begged me to feel his pulse. I found great irregularity in the pulse, not only in that totality of the pulsations named systematic, but also in each single dilatation of the artery. Astonished to see him alive with such a pulse, I asked him if he felt the slightest difficulty in breathing. He answered, 'None whatever.' I watched each change that occurred, constantly feeling the artery of the wrist, during a period of six months. As he asked me at the commencement of what diathesis I supposed him affected, and how such condition could cause a similar pulse without fever, I replied, that in my *Treatise upon the Pulse* I had adverted to a similar irregularity. Indeed, I am of opinion that it results from an obstruction of the great arteries of the lung (mitral valve?). 'But,' said I to him, 'the obstruction could not become the cause of the inflammation of the viscous within you, for you would have fever. We must only suppose that the obstruction caused by thick and viscid humours on the formation of a crude tubercle, has produced in you a like diathesis.' 'But, then,' interrupted Antipater, 'I should have an asthmatic orthopnoea.' 'What you state,' I replied, 'is probable, but still not exact, for such an orthopnoea would very well be produced by the accumulation of this thick and viscid humour, not in the smooth arteries, but in the trachea.' I ordered him to adopt a regimen similar to that recommended in asthmatic cases, and to take medicines having the same properties as those used in such cases. After an interval of six months, he experienced slight dyspnoea and palpitation of the heart; at the onset, only once, then three, four, or even more attacks, the dyspnoea progressively augmenting to about the fifteenth day, then suddenly the breathing became extremely laborious, his powers failed, and he died quickly, in a similar way to other individuals suffering from disease of the heart."

Galen was, as I have before mentioned, the physician of the amphitheatre, and must in that capacity have had presented to him an almost boundless experience. He states, that gladiators suffering under well-marked traumatic inflammation of the heart die in a similar manner to those who have suffered from idiopathic disease. Should the wound penetrate one of the cavities of the heart, especially the left, death immediately follows, from the effusion of blood. But, on the other hand, if the wound does not enter the cavity, but is limited to its wall, the subject of the injury may not only survive the day on which the wound was received, but even the following night. Death eventually follows, as a consequence of the inflammation.

Lastly, with regard to the affections of the pericardium, Galen states, that there is nothing astonishing in the notion that effusion may take place into the sac sufficient in amount to embarrass the action of the heart, and prevent its perfect dilatation. This is inferrible from numerous dissections of animals, in whose pericardium an abundant collection of fluid was found. The following cases are given in illustration:—"A monkey was kept for dissection; but my engagements prevented my fulfilling, for a time, such purpose. It daily emaciated; after death, all the organs were healthy, with the exception of the pericardium. This tunic contained a cyst filled with fluid,

resembling hydatids. In a cock, instead of fluid we found within the pericardium a scirrhous tumour, resembling many thick membranes superposed (false membranes). It is, therefore, probable that in the human subject similar productions may occur." Galen further states, that uncomplicated inflammations of the pericardium, as well as other similar membranes covering other viscera, do not, as a rule, place life in jeopardy. I have elsewhere adverted to the fact, that the statistics by Bamberger of these affections fully justify the opinion of Galen.

Vacation Lectures

ON

FIBRINOUS DEPOSITION IN THE HEART:

ITS HISTORY AND PRACTICAL MEANING.

DELIVERED AT THE GROSVENOR PLACE SCHOOL OF MEDICINE.

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LECTURE II.

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

[Continued from page 103.]

THE symptoms indicating the presence of a fibrinous concretion in the heart are modified or obscured by many circumstances, as by the position of the concretion, the rapidity with which it forms, and by the nature of the case in which it occurs. Sometimes the symptoms of concretion stand forward alone; sometimes they are marked by other symptoms; in their course they may be presented in one of three phases:

- (a) They may be slowly developed throughout.
- (b) They may be developed slowly in the first instance, and rapidly at last.
- (c) They may be developed rapidly, appear in a marked degree, and run an almost independent course.

I would further note that the symptoms differ according to the side of the heart on which they are laid down; and they naturally divide themselves into two classes—the general and the physical.

I shall pursue, in the narration of symptoms, an order the reverse of that followed in narrating the conditions under which fibrine is deposited; for now we must have the broad symptoms brought out first—those symptoms which are least obscured, purest in their manifestation. We shall find symptoms of this kind well marked in all that class of cases where the fibrine is absolutely increased, the diseases of the acute inflammatory group; we shall find them best marked of all in those examples where the local inflammatory changes are developed in an early stage, or are not developed at all.

If my own observations are correct, the fibrinous concretions which form during acute inflammatory disorders are, as a general rule, developed on the right side of the heart. It is true that, in the *post mortem* examinations of such cases, small concretions will generally be found on the left side, the cavities of which are empty of all else; but the large concretion which has given rise to the symptoms will be found either in the right auricle, the right ventricle, or the pulmonary artery. The general symptoms arising from a concretion in these right cavities of the heart are such as might be anticipated by the physiologist who could devise means for checking the course of the blood through the inferior and superior cavæ. The supply of blood through the lungs toward the systemic side of the circulation is impeded; the chemical changes of respiration are reduced; and all the functions which depend for their manifestation on a due supply of oxygenated blood are disturbed, and in a greater or lesser degree suspended. The symptoms, however, are those of syncope, not asphyxia; *i. e.*, the leading processes of life are checked from mere absence of arterial blood, not from the presence of blood incapable of building up the tissues, and of endowing them and the brain with the essentials of the living forces. The symptoms, indeed, are analogous to

those which arise from the introduction into the veins of air, oil, or other material capable of so modifying the course of the blood as to prevent it from making the pulmonic circuit. The symptoms are, that the body gradually becomes cold, and of livid paleness, the coldness commencing first in the extreme parts; the beat of the arteries is irregular and subdued; the brain, being deprived of its blood, the mind wanders; the muscles, robbed of the same support, sink into restless prostration; sensation is in a great measure lost; the respiratory movements are irregular, though often persistent in action for a few moments after the heart has actually stopped; and the veins of the body, being charged with blood, are sometimes markedly prominent; while a tinge of blueness, arising from their congestion, is given to the more vascular parts, such as the lips or central part of the cheeks.

But there is one other symptom which beyond all these is striking, and I must refer to it particularly, because it is not only striking, but misleading. This symptom is a peculiar *dyspnœa*. The *dyspnœa* occurs not because the movements of the thorax are sensibly impeded; not because the entrance of air to the lungs is sensibly obstructed, for the murmur of respiration is audible enough, but for the reason that the supply of blood through the pulmonary artery is diminished. This form of *dyspnœa* is one common indeed to other modes of obstruction commencing in the heart; but in the cases I now name, it is most thoroughly apparent. It is the most distressing of all the forms of *dyspnœa* with which I am conversant. The asthmatic patient can find some relief in a certain position of the thorax; the phthisical patient can obtain relief on lying down in perfect repose; but the patient with this obstruction gains no moment of tolerance. He feels that in breathing, heaving, and sighing, there is no result; none of that steady continuous support which marks the natural life. You ask him where the oppression he feels is concentrated, and if he is conscious, he points to the heart. If he can speak, he tells you that he can fill his chest, that he has no pain in the strict sense of that term, but an indescribable restless, faint, sickening, deathly oppression. The very approach of your own body bending over the sufferer is insupportable. Whoever has suffered from loss of blood, whoever has carefully observed the symptoms incident to slow hæmorrhage, has felt or observed in a minor degree the symptoms I would describe.

The *dyspnœa* too, to the external observation, is special. The acts of respiration are rapid and yet deep. The breathing is mainly by the diaphragm, and the ale of the nose undergo that "working" which all practitioners recognise as an unfavourable sign in all acute diseases. The *dyspnœa* lasts to the end; after the mind ceases its function; after the body has become as marble; after the restlessness of body has ceased from sheer exhaustion; even some seconds after the heart has ceased to beat, the chest will heave, and the nostril play.

Such are the general symptoms, I say, of well marked concretions occurring on the right side, in the right auricle or ventricle, or both. But what are the special or physical signs? I would state at once, that in relation to the physics of the heart, there is no definite sign indicating the presence of concretion on the right side. The fibrinous mass may certainly interfere with the valvular machinery of the heart; the curtains of the tricuspid valves may be bound down to the cardiac wall, or looped up together by a transverse intertwining of fibrine threads with the tendinous cords of the valves; or the pulmonary semilunar valves may be overlaid with fibrinous deposit; but conditions here are not such as to produce murmur; they are only such as reduce or destroy the normal sounds. And, as the valvular mechanism on the left side is free, and is capable of producing the two sounds in their natural time and place, so the sounds are at most but modified in tone. I have in a few cases observed an irregular action of the heart; but this has been so rare, that I do not attach to it any special importance. Turning from the heart to the lungs, we find, however, in some instances, some physical facts on which to confirm a diagnosis of concretion based on general symptoms. In children, especially where during the existence of an acute inflammatory attack fibrine is laid down, there are presented the most striking indications of emphysema of the lung. In infants, this emphysema is sufficient to raise up the thoracic wall anteriorly into a rounded prominent projection. If you examine this chest with the stethoscope, you may find at no part sign of congestion nor of condensation. When this state obtains (and it often does obtain), the evidence is almost demonstrative as to the presence of concretion. The emphysema is a consequence

indeed, of the concretion. It is due, in this case, not to obstruction in some part of the respiratory tract, but to the deficiency of blood in the pulmonic circulation. The presence of blood in the pulmonary tissue in the capillaries covering the air-vesicles, is essential, indeed, to the integrity of the vesicles. We may take a narcotised dog, lay open his chest, keep up his breathing artificially by the double acting bellows, and see the heart beat and the blood make its circuit for an hour or more; there will be no emphysema of lung under moderate and measured force with the bellows, so long as the blood continues to make way. But if we put a check on the blood-current going to the lungs, although the same force be employed with the bellows, the air will infiltrate the pulmonary tissue, there will be emphysema. I make this observation from direct experiment; it is one of value to remember in disease in general, and of great value in this particular form of disease. At the same time, it is not invariably the case that emphysema accompanies fibrinous deposit. I am inclined, indeed, to the opinion that the emphysematous condition is most frequent in children. In adults the heart has a greater propelling power; and the lungs, charged with a small current of blood, give on physical examination indications of congestion rather than of emphysema; in persons of adult life, this is indeed the more common sequence.

In coming to a diagnosis, then, regarding concretion on the right side of the heart, we must carefully consider together the general and physical indications. The general indications are coldness of surface, pallor, prostration, restlessness, enfeebled pulse, and the peculiar *dyspnœa*, of which I have spoken at length. The physical symptoms, as regards the heart, are irregular or weak tumultuous action, with feeble sounds; as regards the lungs, emphysema or congestion. In the end, in the cases presenting these symptoms, the death is usually very easy; a syncopic anesthesia comes on, and the watching friends tell you that they were scarcely conscious when the sleep of life became the sleep of death.

The class of symptoms I have thus described marks peculiarly that condition of body in which the fibrine is in absolute excess—hyperinosis. In any one of the diseases which have been ranged under that head, we may have these signs manifested. They may occur before any decided local inflammation has been discovered, or at any time during the period of a local inflammation; in a word, there can never occur that systemic condition on which idiopathic inflammation is based, without some danger of deposition. It is true that in the majority of cases such risk is small, but it is always present; and what is more, the risk or tendency to deposition cannot be measured by the local signs of inflammatory mischief in any given case, nor yet by the general symptoms which accompany the local.

I have stated, in reference to these symptoms, that they may last for different lengths of time. This is the case. I have seen them developed almost in an instant, and run their irresistible course to the death in the period of a few minutes. I have seen them develop slowly, and extend over two or three days; once over as many weeks. These are the extremes on both sides.

The causes of these differences are rather accidental than otherwise. They depend on the manner in which the concretion is laid down, the extent of the obstruction which it throws in the way of the blood, and its position in the heart. When the symptoms have been ushered in suddenly, and have caused death in a few minutes, the event has usually transpired as follows:—The concretion has been laid down at the root or in the body of the pulmonary artery, either in the form of a hollow cylinder, shaped like the tube whose walls it surrounds, of which, indeed, it is a plastic cast; or as a plug or solid cylinder. The deposit may retain for a time a hold on the cardiac wall, so that the blood can float either through it, or around it, at first with but slight impediment. But, unfortunately, three evils are by this time set up: first, the blood-current is impeded; secondly, the surface of the concretion, the inner surface if the fibrine is laid down as a tube, the outer surface if as a solid cylinder, form favourable points for further deposit; and thirdly, as the size of the channel by which the blood is transmitted diminishes, the column of blood passing onwards is rendered proportionately slower. All these events lead to two grand results—narrowing of the blood-channel, and resistance to the blood-current. Occasionally, this process goes on until the passage for the blood is gradually reduced to small dimensions, and the blood circulating through it is cut off slowly, so that symptoms prolonged in character are presented. But, in other examples, the deposit, as the

pressure upon it from behind increases, breaks from its attachment at its base, is carried upwards by the force of the blood-current into the narrower part of the vessel, and, completely obstructing the life-stream, leads to inevitable and almost immediate death.

[To be continued.]

Original Communications.

THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CARDIAC DISEASES.

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VIII.—ENDOCARDITIS: PATHOLOGY. ACUTE AFFECTIONS OF THE HEART'S VALVES: THEIR DEGENERATIONS.

By the term endocarditis is understood inflammation of the whole or of a part of the membrane which covers the valves and the internal surface of the heart. Included also under this head are all those morbid changes which the valvular structures undergo in connexion with acute diseases, or which are indirectly the consequences of the endocardial inflammation.

Much obscurity still involves the pathological history of endocarditis; for now-a-days, happily, we have very rarely an opportunity of studying the disease in its early stage. The remarkable researches and experiments of Dr. Richardson appear, however, to throw some light upon this subject; and I shall, therefore, state shortly what he has observed in cases of endocarditis induced artificially by the injection of lactic acid into the peritoneum.

In considering this subject, it is necessary to keep in view the anatomical nature of the endocardial membrane, and the peculiar accidental circumstances which attend upon and influence its inflammation. This membrane is continuous with, and very similar in structure to, that which lines the arteries and veins. It consists of a stratum of fine fibres, bearing upon them a layer of very delicate epithelium. Blood-vessels ramify in the fibrous tissue, and it is from these that the pathological products of the inflammation are derived.*

The peculiar circumstances attending this inflammation are, the unceasing motion of the part inflamed; and the mechanical action of the blood, which is constantly flowing over and is in contact with the inflamed surface, and with any exudations which may escape from it.

The deep red colour, which the internal surface of the heart frequently presents after death, was once considered as an invariable sign of inflammation of the endocardium. It is, however, in almost all cases, the result simply of imbibition by the heart's tissues of the decomposed and dissolved colouring matter of the blood.

The redness of congestion—the true inflammatory redness—is described by Rokitsky as constantly presenting a pale rose-red colour, the tint of it being subdued by the superficial layers of the endocardial membrane; it assumes a stripe-like, ramifying appearance, corresponding to that of the vessels, and has not the appearance of a general saturation of the tissues. Small ecchymoses are seen here and there in the inflamed part.

In the first stage of the inflammation, Dr. Richardson (*On the Coagulation of the Blood*, p. 371, *et seq.*) found the endocardial surface “intensely vascular, approaching in colour to bright vermilion; the membrane also had a soft fleecy character, giving to it a velvety appearance”. The valves were at the same time swollen and vascular, and clear lymph oozed from them when pricked. This lymph appears occasionally to have transuded through the membrane, and may have thus laid the foundation for fibrinous deposits from the blood, by causing its coagulation—an important observation, if further corroborated. “On the surface of the valve (mitral) there was a small fibrinous deposit, which adhered to its position by

means of an underlayer of a white glutinous fluid, the like of which I found afterwards could be made to exude from the valve on puncturing it with a needle.” Small beads were at the same time developed in the margin of the valves, and also yielded a clear coagulable lymph when pricked, and then shrunk away.

In the second stage of the inflammation, the endocardial surface became less vascular, and had a pale pink colour. The valves also were less red, but were still thickened with the now semisolid exudation within them; and at points their tendinous chords were attached by loose adhesions to the endocardium. In the third stage, all redness disappeared; the exudation was firmer, and the valves and their beaded edges for a long time retained considerable firmness, and a pearly whiteness.

From these observations, it would appear, that the ordinary products of inflammation are exuded both upon and within the endocardium. There are, therefore, two kinds of endocardial deposits to be considered: The “beads,” the granulations, and thickenings, which are produced solely by the inflammatory exudation thrown out beneath the unbroken endocardial surface, and therefore within the valves; and the “vegetations,” etc., formed chiefly from the fibrine of the blood flowing through the heart, which is attracted to and deposited either upon the surface of the inflamed membrane, or upon the inflammatory matter that has exuded through it. These vegetations, therefore, are engrafted on the inflammatory deposit. It seems very probable, indeed, that a slight degree of roughness of the endocardial surface, resulting from the inflammatory action, may thus induce a deposition upon it of the fibrine of the blood; so that in this way the inflammation becomes, indirectly, a cause of the formation of fibrinous coagula. And this is still more probable in diseases, such as acute rheumatism, in which the blood appears to have a special tendency of separating from its fibrinous constituents.*

These abnormal products, thus formed upon or exuded within the inflamed endocardium, present themselves to us under a variety of forms, differing much in size, number, and consistence. At one time, they exist as minute granulations clustered together around the bases, or fringing the borders of the valves; sometimes they are attached, like polypoid bodies, to the valves, in an isolated form, varying in size from a pea to a hazel-nut. Then, again, they take the form of cauliflower excrescences, passing between and separating the valves, or fringing their torn and roughened edges. The fibrinous deposits are also frequently found fixed, not only on the valves, but also on the endocardium in their neighbourhood. They are mostly found fixed to that surface of the valves which is turned towards the current of the blood flowing through the heart. The different appearances which these granulations and vegetations, etc., present are readily explained, when we regard them as being formed, first, by the deposition of fibrine of the blood upon an inflamed surface; and, secondly, as the results of inflammatory exudation, having the endocardial membrane still unbroken upon them. Whenever this surface is broken or roughened by inflammation, a deposition of the fibrine of the blood (as it would appear) readily takes place upon the broken part.

The valves, and their chords, are not unfrequently ulcerated and ruptured, as a consequence of the endocardial inflammation. The tendons of the papillary muscles, and the valves themselves, are also sometimes fused together, or with the neighbouring parts, and so as to render the closure of the valves imperfect, or to diminish the aperture they surround. The aperture is also sometimes constricted by the deposit of inflammatory exudation in the tissues which form its circumference.

Endocarditis occurs more frequently, or at least its action is much more severe, in the left than it is in the right side of the heart.† The resolution of it is rarely perfect. Whenever we are able to diagnose the presence of endocarditis, we generally find that some appreciable consequences of the inflammation remain after the inflammatory symptoms have passed away.

* Virchow asserts, that these fibrinous formations are merely layers of fibrine separated from the current of blood which flows over the membrane. The only difference which exists, in his view, between endocarditis and endarteritis, is in the more acute course of the former, and in the tendency of it to the formation of papillary or warty excrescences. Both these facts he attributes to the greater vascularity and looseness of the cellular tissue which lies between the valves and beneath the inner layer of the endocardium.

† I am inclined to think that endocarditis attacks the right side of the heart more frequently than is generally supposed; and that we do not notice the fact, because its effects are much less severe, and the pathological signs of it less well marked. In most cases, when the mitral valve is much diseased, the tricuspid will, I believe, be found also affected.

* The idea, that the internal membrane of the heart or arteries is nourished by the blood which flows over it, is quite contrary to all physiological facts.