ON A FEW POINTS CONNECTED WITH THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF PERICARDITIS.

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Pericarditis is a very frequent and a very dangerous disease: it attacks those persons most exposed to changes in temperature and climate, and least likely, from position in life, to be able to take precautions to prevent, or use remedies to cure such a disease. It is consequently of the utmost importance to society that such an affection should, if possible, *admit of easy detection*; and that its *nature and treatment* be plain and straightforward to medical practitioners in general.

In Dr. Bartolomé's Essay on this subject, published in this Journal of the 5th of May, he remarks, "The *diagnosis* of pericarditis is not always easy;" it has been mistaken by Corvisart, Andral, and Laennec, for other diseases;—that the "fibrous portion of the pericardium is that first affected;" and he advances a mode of treatment so contrary to that which I have been in the habit of considering as the one most approved of by all the late pathological writers on this disease, that I trust he will allow me to notice his statement, with the laudable motive of searching for truth.

I shall confine my remarks to pericarditis only, although I am perfectly aware that endocarditis, with all the resulting effects of valvular disease, atrophy, dilatation, and hypertrophy, are generally its consequences; but space will not allow these to be named.

The following order may be as well observed:—

1st. The pathological condition of the membrane involved, and its terminations.

2nd. The best mode of determining, by examination during life, the actual condition of the part.

3rd. The most approved method of treatment as described by the best authors of the present day.

I.—The Pathological Condition of the Part.

Although the pericardium is composed of both fibrous and serous textures, yet the same arteries supply both, and it will depend upon the condition of the blood supplying the inflamed capillaries what the exudation corpuscles produce.

Dr. Bartolomé says, "very probably pericarditis generally, if not always, first attacks the fibrous pericardium," then, afterwards, "the most common causes besides rheumatism, are blows, pressure, inflammation propagated along the lungs or pleura, &c." He gives no proof of the fibrous being the membrane first affected, and is "at a loss how to describe the first stage of the disease." But, if pericarditis arises from inflammation propagated along the serous membrane of the pleura and lungs, it is not very likely to affect the fibrous membrane first.

I am not aware of any other author having laid any stress upon the fibrous membrane being immediately implicated. I have had a great many opportunities of

examining the parts after death, having during the disease anxiously and narrowly watched its progress; and until I see by dissection that I am wrong, I shall feel inclined to agree in the opinions which I quote below.

The same degrees of congestion, nervous and vascular irritation, determination of blood, obstruction of capillaries from *atonic* enlargement, and adhesion of white corpuscles in the vessels; distension of blood in the arteries *before*, and emptiness of the veins *beyond*, impeded or arrested circulation *at*, and increased circulation *around*, the obstruction; with change in the whole quantity of blood by increase of fibrin and diminution of the secretions, exhaustion and depression either from excessive excitement or the presence of pus or other foreign matters in the blood, must attend this as well as all other inflammations.

If the blood be excessive in red particles, as in very robust and healthy persons, there will be coagulating lymph, serum, and blood exuded; if deficient in red particles, as in anæmic, lymphatic and leucophlegmatic subjects, tubercular deposits will be formed; if fibrin predominate, the serous membrane will be covered with a thicker and more adhesive layer of lymph, and of the most plastic character. But the blood may be so altered in quality, and the quantity of its salts and red particles so much increased, as may produce even osseous deposits. The fibrin may be so low in character as to allow only *aplastic* lymph to be secreted, in which case very slight, if any adhesion, would take place; and pus, curdy matter, yellow tubercle, &c., would be deposited. It might be *euplastic* and become highly organized; or *cacoplastic*, and susceptible of only a very low degree of organization, particularly in the chronic stage, thus giving origin to fibro-cartilage, grey tubercle, &c. It therefore depends on the character of the blood as well as upon the character of the membrane inflamed, what the product of inflammation will be; and this easily accounts for the various statements of many of the old authors,—of bony, bloody, purulent, curdy, fibrinous, and various other effusions having been found in cases of pericarditis.

It is very evident that some of these products of inflammation are much more adhesive in their nature than others, and I have taken the opportunity of naming all, for the purpose of giving the greatest latitude possible to the difficulty this will give us in diagnosis. But it is well known that pericarditis, like rheumatism, is a disease of a highly inflammatory nature, and oftener attacks those whose blood is loaded with red particles and fibrin, than the weak and anæmic; and those who have had the most extensive experience in examining such cases after death, agree in saying that *adhesion of the pericardium* is by far the most frequent result.

The first and immediate effect of pericarditis is.

effusion of a small quantity of coagulating lymph and serum on both sides of the pericardium; this increases according to the activity of the attack, or the success of remedies. It may go on to such an extent as to bulge out the bag of the pericardium enormously, or it may stop short, and only produce so much mischief, as to impede the smooth motion of the heart in its natural capsule. There may be the other deposits mentioned before, but there *must* be both effusion of lymph and serum.

The general opinion amongst authors, without one exception, I believe, is, that after every case of pericarditis, the lymph unites the pericardium to the substance of the heart, and the cavity is thus either partially or wholly obliterated; and this is considered the most efficient cure which the parts are capable of admitting. For some years I have had the opportunity of watching numerous cases of this disease, in the practice of our late lamented and talented associate, Dr. Favet, as well as in my own; and there are at least two specimens of his in existence, and one of my own, which he added to his collection, where, after the most positive proofs of pericarditis having existed from acute rheumatism, verified by several medical gentlemen and students, the patients quite recovered under the specific influence of mercury, and died afterwards of some other disease. On dissection, the pericardium in each case was found *unadherent throughout*, and the parts which had been inflamed are quite distinct and easily discerned from the rest, being of a much lighter colour, thicker, and raised from the surface of the uninflamed part of the membrane. This is a circumstance deserving notice, for it gives great encouragement to the practitioner to treat the disease very actively, with so good a prospect of perfect success.

II. The best Mode of detecting Pericarditis during Life.

Dr. Bartolomé remarks, "All authors seem to agree, that the most unequivocal sign is the presence of pain over the region of the heart, particularly if aggravated by pressure." He lays great stress upon the quality of the pulse, as a diagnostic symptom, stating in one place that it is "the loose throbbing pulse of acute rheumatism, when the fibrous membrane alone is inflamed, but will vary in proportion as the serous membrane becomes affected;" and in another, that "it is the jerking immediately felt along the arteries, that distinguishes the pericarditic pulse from that produced by merely accelerated circulation." But he afterwards says, "we cannot form our diagnosis from the pulse alone." He mentions the "*bruit de soufflet*," which is *pathognomonic* of inflammation of the *endocardium*, but passes over very slightly the "*craquement de cuir*," and "*bruit de cuir*," and does not notice the "*bruit de frottement*."

The symptoms, as described by Dr. Watson, to whom, simultaneously with Dr. Stokes, we are

indebted for the first information of the diagnostic physical signs, are,—a singularity of manner and peculiar expression of countenance, strange aspect of distress in the deportment, palpitation, oppression at the epigastrium, catch in the breathing, dry cough, inability of lying on the left side; pain in the region of the heart, increased by inspiration, pressure between the ribs, or upwards under the diaphragm; stiffness, and pain in the left shoulder, extending into the arm, elbow or wrist; and *delirium*, often *wild* and *furious*, not dependant upon any disease of the encephalon.

All these symptoms put together would not be enough to make us sure that this was pericarditis; for truth, in all its kinds, is most difficult to win, and truth in medicine the most difficult of all. Clinical observation, though never blind, was, until lately, always deaf; but now, with the aid of the ear, when, with even some of these symptoms, we find the *attrition murmur*, Dr. Latham says the disease cannot be mistaken for anything but pericarditis.

Before the true *attrition* or *friction-sound* is heard, there is almost always, and generally for some days, a kind of *clanging, harsh, churning, creaking, or crumpling* sound heard over some part of the præcordial region, *not* conveyed to the aorta, pulmonary artery, or carotids. This is not constantly present.

In pericarditis there are the fluid products of inflammation as well as the solid: there is serum as well as lymph; and the signs of fluid effused within the pleura and pericardium are the same. The fact of its existence, and the measure of its accumulation within the pericardium, can only be known by the degree and extent to which the præcordial region, and perhaps, some space beyond it, may be *dull to percussion*. Thus, *dulness* sometimes occupies a part, sometimes the whole, of that region; sometimes it reaches beyond, as high as the *second*, and even the *first left rib*; sometimes it extends *beneath the whole length of the sternum*, except about an inch at the top, and even *beneath the cartilage of the ribs on the right side*.

This *dulness to percussion* is a most important sign, and hardly inferior to, or less diagnostic of, the pathological condition to which it points, than the *attrition, friction, or to-and-fro murmur* itself, which is caused by the rubbing of the two layers of membrane on each other, after they are covered with lymph.

But there are two other physical signs of this disease which often but not always attend,—1st, an *undulatory motion*, visible to the eye, *between the cartilages of the second and third, or the third and fourth ribs, on the left side*, or in both situations at the same time; 2nd, a vibratory motion, like the purring of a cat, *felt just over the same places as the undulatory motion is seen*. These two last signs sometimes attend disease of the semilunar valves of the aorta and pulmonary artery, and are therefore *not* pathognomonic.

During the life-time of Corvisart and Laennec the physical signs of pericarditis were not discovered, and it is no wonder they speak with so much doubt about it. But since Dr. Watson, of London, and Dr. Stokes, of Dublin, distinctly ascertained that *increased dullness on percussion*, and the *peculiar friction to-and-fro sound*, were pathognomonic of this lesion, no one who has once listened attentively to this sound can ever again mistake it, or misinterpret its meaning.

In all acute cases of pericarditis I believe this *friction-sound* can be heard distinctly if listened for. The harsh churning sounds spoken of before are generally the precursors of this more distinct sign, but they are not always to be heard. The *to-and-fro friction-sound* is always evident, and if watched quite distinct.

In cases where a very large quantity of serum is exuded, the *friction-sound* may only be heard during the first few hours or days of the disease, the fluid having so far separated the pericardium from the heart as to prevent its touching, *at which time the dullness of percussion would be more marked*. But under the influence of proper remedies, this fluid becomes absorbed in a few more days to such a degree, as again to allow the membranes to rub against each other, *and then the friction-sound re-appears*. I believe this very rarely happens, but I have certainly noticed it myself in cases which I have narrowly watched.

The difference of density in the fluid in the pericardium does not alter the *nature of this friction-sound*, for it is *conveyed immediately from the outer layer of the pericardium*, and appears to proceed from a surface immediately under the ear. This sound is perfectly distinct from the *systolic bellows-murmur of endocarditis*; but when endocarditis exists at the same time, as is often the case, this bellows-murmur is heard as well, and frequently confuses, for a time, the friction-sound.

I would recommend any one who is sceptical on this point to read Dr. Latham's late work on "Diseases of the Heart," in two volumes, 1846-47. I name this work above all others because Dr. Latham has taken his data from ninety cases of pericarditis, attended by himself in St. Bartholomew's Hospital, all of which were open to the observation of the medical staff of the hospital and students; and he is a physician of such decided talent and probity as to carry conviction to every unprejudiced reader.

But there still may come a difficulty, which I will briefly allude to. In a case of pericarditis which has been apparently cured by treatment, and where most of the pericardium has become adherent; what will be the pathognomonic physical sign, should another attack of inflammation make its appearance? Here auscultation is not to be entirely depended upon. The *impulse will be great, tumultuous*. There may be some slight friction-sound if a very large part of the pericardium still remain *un-adherent*, but generally there

is little or none. If the former attack was cured *without adhesion*, (a circumstance by no means impossible,) then I presume the very same physical signs which have been already described will become again pathognomonic. In most second attacks the endocardium, valves, and cavities become diseased, and during acute inflammation these sounds are often difficult to diagnose, unless the ear be well accustomed to such abnormal sounds.

It may be well to mention a combination of sounds originating in disease of the *semilunar valves* from *endocarditis*, which sometimes simulates this friction-sound of pericarditis, and might mislead an ear unpractised in auscultation. I allude to a *direct* as well as a *regurgitant* murmur existing in the *semilunar valves* of the *aorta* or *pulmonary artery*, and not unfrequently met with in the aorta. The character of this sound is different to the friction-sound, although the peculiarity is difficult to describe. The *length of both sounds* in the *pericarditic friction* sound is the same, but the *systolic is longer* than the *diastolic* sound in that originating in the semilunar valves. A very little education of the ear will be found sufficient to distinguish the difference. The *valvular sound* is also heard *louder* at the *top of the sternum* than at the *apex* of the heart, and is conveyed by the vessels, whereas the *intensity of the friction-sound* is generally in the *opposite direction*, and is *not conveyed* by the vessels. We now come to—

III.—"The most approved Method of Treatment."

Dr. Latham, in 1847, says, "In foreign practice *no mercury* is used from first to last, but all the power of common antiphlogistic remedies is brought to bear upon the disease, and thus its symptoms are mitigated or subdued, yet they return again and again, and are again and again mitigated or subdued, and so the patients are kept alive for a week or ten days, and *then they die*, in the great majority of cases."

M. Bouillaud's treatment of pericarditis is of this antiphlogistic description; he never uses mercury; and in his treatise on it, he says almost every case is found on dissection to have the pericardium adherent. In inflammation of the pericardium the products or exudation of the inflammatory action are deposited in a shut sac. There is not only congestion, great nervous and vascular irritation, and determination of blood, with their usual consequences, but a large quantity of lymph and fibrin are exuded, which, so far as I know of the remedies for inflammation, can only be checked, stopped, or absorbed, *during the time the system is under the specific influence of mercury*.

Bleeding, both general and local, is undoubtedly invaluable, and ought to be carried to such an extent as to cut off the supply of a certain quantity of blood to the part, and decrease the quantity of fibrin in the blood. Purging to a certain extent is necessary, but that will not absorb either the serum or lymph. *Opium* is of the *greatest benefit*, by soothing the excessive irritability:

of the nervous and vascular excitement, and relieving pain; and counter-irritation, particularly blistering, is invaluable, by exciting the action of the absorbents near the seat of the lesion, and in that way relieving the distension of the vessels. But all these remedies combined will not prevent a case of pericarditis making progress to that stage which ends in adhesion and premature death.

I have carefully examined the works of Stokes, C. J. B. Williams, Watson, Elliotson, Hope, Copland, Joy, and Latham, and I find my opinions borne out by all of them. I am convinced that *mercury* has the power of doing *something more* in inflammation of the pericardium than venesection and other antiphlogistic remedies can do; and that, upon this something being done, the life of the patient often depends.

Dr. Taylor, one of the physicians to University College Hospital, who took the highest honors both in the College and University of London, has published in the *Lancet* forty cases of pericarditis, which he has treated in the Hospital under the constant inspection of the pupils. I have read them all, and he has never once omitted to do all in his power to bring the patients under the specific influence of mercury; *where he has failed to do so his patients have died*, but where he has succeeded his patients have generally recovered. These cases are worth reading, being written by one whose knowledge of the disease is more extensive than that of most authors.

In *iritis* the influence of mercury is quite visible in removing effused lymph, and it thus obviously promotes absorption as well as prevents effusion. In syphilitic ulcers mercury soon removes the callous indolent margin. I have heard an opinion canvassed that mercury destroys the red particles of the blood, and produces a disposition to erythematic inflammation which is incompatible with healthy or plastic. But, if this be true, how can healthy lymph be thrown out, and granulations formed, in such numerous instances under its influence, as we are constantly in the habit of observing? It is more probable that the specific action of mercury *changes the condition of the blood*, and *diminishes the quantity of fibrin and white corpuscles*, as we find mercury most useful when the blood is buffed, and in serous and fibrinous inflammations, where effusions take place to a great extent.

Dr. Latham says, "my experience tells me that whenever the exocardial murmur has ceased early, *salivation* has first taken place." And in the relation of the events of the ninety cases, there were *two* in which he *could not* produce the specific action of mercury. *These two died*, and only one more. In another place, he says, "not in a single instance did the *exocardial murmur* cease to be audible, until *salivation* appeared."

Again he says. "In English practice, mercury is

given from first to last, but it is for a time as if it were not given at all, for it produces no sensible effect. Common antiphlogistic remedies, however, are able again and again to mitigate and subdue symptoms; and so, at the end of a week or ten days, the patients are *still alive*, yet they are *ready to die*; but in a great majority of cases they do not die. *Salivation arrives late, and seems to save them.*" !!

Colchicum, tartarized antimony, and aconite, ought to be mentioned as remedies, which occasionally, very much allay the excessive action of the nervous and vascular system. They require caution and judgment in administration, perhaps more so than those before mentioned.

In my own practice I have been in the habit of treating these cases by general bleeding to a certain extent, followed by cupping, leeches, and blisters; but I have placed my chief dependance upon well regulated doses of calomel and opium, and frequent frictions with strong mercurial ointment, until ptyalism is produced.

It would have been easy for me to give numerous other quotations to prove the necessity of putting pericarditic patients under the specific influence of mercury as *quickly as possible*, but space will not allow. I trust, however, that I have not uselessly intruded on this Journal, and refer, in conclusion, to the words of the illustrious Sydenham:—"Ars medendi (si quid ego judico,) in eo potissimum cardine vertitur, ut certa aliqua et consummata undique ac fixa Methodus Medendi in publica commoda tradatur, eam intelligo quae, satis magno experimentorum numero corroborata suffultaque, huic vel illi morbo devincendo suppar invenitur." Sydenhami Opera, Praef. Ed. Tert., 16.

Rotherham, 8th May, 1847.

CASES AND NOTES FROM HOSPITAL AND PRIVATE PRACTICE.

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CASE XIX.

HÆMATURIA; ASCITES, AND GENERAL ANASARCA.

An inkeeper, aged 45, of leucoplegmatic habit, requested me to visit him on the 4th of October, 1845. His medical attendant, whom I met in consultation, stated that his habits had been very irregular from excess in drinking; that he had for some years exhibited more or less of the hæmorrhagic diathesis, as evidenced by bleeding from the nose and gums; and that the commencement of his present attack was hæmaturia, followed by general dropsy. He had been out of health for a lengthened period, but the present severe symptoms had existed only for about a week. I found him in bed, his head and shoulders elevated by pillows, and breathing with difficulty; countenance