The purulent matter was allowed to escape by incisions made into the larger punctures and into the fingers.

He is now (September 23rd) slowly recovering from a state of great prostration, under the use of nutritious diet and quinine.

Remarks. The chief features of interest in the case thus briefly outlined are—

1. The total absence of any evacuations per annum until the tenth day after the operation.

2. The persistence of the vomiting for fifteen days after the operation; the vomited matters being distinctly faecal during a portion of the time.

3. The occurrence of pyramidal symptoms with deposits of pus, in patches of various sizes, under the cuticle, over a great portion of the body, and the symmetrical crippling of the little fingers of both hands from pernicious inflammation.

The result of the case seems to show the advisability of withholding any secondary operative procedure in the way of opening out the wound again for the purpose of further exploration, the operator feeling satisfied that the construction has been sufficiently divided, and the protruded parts have been completely returned into the abdomen.

MIDDLESEX HOSPITAL.

HEMOCCEL.

Under the care of A. Shaw, Esq.

S. E., aged 46, a bricklayer, was admitted into the Middlesex Hospital, under Mr. Shaw's care, on August 9th, 1859, for a tumour in the right scrotum.

Five months ago, he began to observe what appeared to him an enlargement of the testicle. The swelling gradually, without pain, increased to the size of his closed fist. A surgeon, to whom he applied, after examining the tumour in the dark with a candle, proposed to lance it for him; but as he suffered little inconvenience from it, he declined to have that done. On a Sunday, eight days before his admission, while sitting, and in the act of crossing his right leg over the left knee, he felt a sudden pain in the part. During that night, the swelling gradually enlarged to twice its former size; the scrotum also became blue; but he had no pain worth noticing.

He continued at his work as usual; all that he did was to cover the swelling during the night with wet cloths; and in about three days it had returned to its former size. The builder's strike having commenced at that time, he thought it a good opportunity for having his complaint cured.

The tumour, on his admission, was of the size of a closed fist; it was irregularly globular, being at its upper part somewhat square shaped, tending to lobes slightly projecting from it. The situation of the testicle could not be exactly ascertained by its figure; but from the peculiar pain produced from pressure, it was supposed to be at the back part and lower down. In front of the penis, where examination was performed, but in a most indistinct manner, and that chiefly where the lobes projected. The cord was sound. He had scarcely any pain. On examination in the dark with a lighted candle, no transparency could be detected, whether by looking through a tubular stethoscope, or in the common way, without that assistance.

The treatment consisted for the first week in elevating the parts on a pillow, and keeping them constantly moist with a lotion of hydrochloride of ammonia. For the following six days, mercurial ointment was applied; and that was succeeded, for a similar period, by the ointment of iodide of lead. No perceptible diminution of the tumour was observed.

August 20th. A puncture was made with an exploring needle in a prominent part of the tumour, in front, while fluctuation was the most distinctly felt; and as blood ran along the groove, the trocar for hydrocele was next introduced, when an ounce and a half of dark coloured blood was drawn off.

September 4th. Till to-day, broken down clots of blood have been discharged somewhat copiously into the poxilium; and from the diminution of the tumour, the scrotum has become flaccid. No inflammation has been set up.

September 15th. Since then blood has ceased to be discharged the matter which comes from the wound is soro-purulent. The quantity has been gradually diminishing, and now amounts to only a few drips in the day. A poultice of cotton wool was ordered to be used.

September 20th. The testicle and scrotum have returned to nearly their natural condition. The former is a little enlarged, but otherwise appears healthy. The discharge has ceased, and the wound is closed.

September 27th. He left the Hospital cured.

Original Communications.

THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CARDIAC DISEASES.

By W. O. Markham, M.D., F.R.C.P, Physician to St. Mary's Hospital, London.

IV.—PHYSICAL SIGNS OF PERICARDITIS.

We have seen, in speaking of the causes of pericarditis, that the disease presents itself to us under two distinct forms, viz.: rheumatic and non-rheumatic. This is a fact which should be kept in mind in considering the diagnosis of the disease; for we shall find that the signs and symptoms of the two forms of pericarditis differ much the one from the other. In non-rheumatic pericarditis, the inflammation is generally of a latent kind, being insidious both in its origin and its progress, and from an absence of all local symptoms of its presence, not unfrequently entirely escapes observation during life.

In rheumatic pericarditis, on the other hand, we observe the inflammation in its typical form, and note the characteristic signs and symptoms which attend it. Local signs of the disease—viz., more or less pain and a feeling of oppression about the precordia—are usually present, if not always in the earliest at least in the later stages of its course. Even in rheumatic pericarditis, however, the local and general symptoms do not, as a rule, give us the earliest information of its existence.

We carefully watch for its physical signs in all cases of acute rheumatism, and it is the stethoscope which first apprizes us of its presence. Acute local pain, moreover, and I have ever endeavoured to show, is not a usual or necessary accompaniment of simple pericarditis, but depends rather upon the pleuritis, which so often accompanies pericarditis. When, indeed, as often happens, local pain is absent, we may assume that the pericarditis is not associated with pleurisy. It is well, however, to be aware that in some cases, even when pain exists, it may be overlooked, in consequence of the severity of the associated arthritic element of the rheumatic fever.

The Heart's Sounds, Impulse, etc. At the very onset of the inflammation, the heart's sounds are generally louder, and its impulse stronger and more extensively felt over the precordia, than natural. The force and nature of this action are, we may assume, increased by the local irritation, as well as by the general febrile condition of the patient. But as the inflammation advances, and when exudation gathers in the pericardium, the movements of the organ become feebler; its impulse irregular and trembling; and its sounds at last weak and altered in character, or superseded by morbid sounds. When considerable quantity of effusion is taken place into the pericardium, and the patient lies recumbent, the heart naturally gravitates to the back of the pericardium; and, in consequence of this, a considerable quantity of fluid is interposed between it and the thoracic walls. Under such circumstances, the natural sounds and the impulse of the heart may become very weak, so as sometimes to be almost imperceptible. And the reason of this is, that the heart's movements are impeded by the thickened layer of the effusion upon it. The contractility of the muscular structure, moreover, is affected by the near contact of the inflammatory process which
is going on in the serous membrane; its nutrition and its vitality being thereby impaired: the result of which is, that the organ is at last partially paralyzed and the heart, though still beating, is no longer able to do its duty. We we infer that small amount of exudation will have as enfeebling an effect upon it, as a much larger amount would have under ordinary conditions—that is, so long as the heart is not already palsied, though we must not forget that a heart already hypertrophied, may become the seat of pericardial inflammation; and in such case, we may expect the sounds and impulse to remain loud and distinct, even when a considerable amount of effusion exists in the sac.

The physical signs characteristic of pericarditis are: double or single friction-sound heard over the pericardial region, and increased dullness of percussion there. Occasionaliy, though very rarely, a tactile fremitus also is perceptible to the hand at the same part.

Friction-Sound. A double or single friction-sound heard over the heart, synchronous with its movements, and confined to the pericardial region, is the surest diagnostic sign we possess of the presence of pericarditis. It is, indeed, the first positive sign which enables us to determine with certainty the local character of the inflammation. Fever, pericardial pain or tenderness, and all the other general symptoms of pericarditis, may exist; yet, until this friction-sound be heard, we cannot with certainty affirm that the pericardium is attacked. The friction-sound appears at a very early stage of the inflammation; it may remain audible through the whole period of the existence of the inflammation; or it may be heard at the commencement of the process, and by the time the absorption of the fluid part of the exudation has taken place. The friction-sound usually accompanies both the systole and the diastole of the heart; occasionally it is heard during the systole only; in some very rare cases, it is confined to the diastole. The smallest amount of lymph exuded on the surfaces of the pericardial membrane, will, I believe, give occasion to the friction-sound during the heart’s movements, provided no fluid come between them; it is not impossible, indeed, that mere dryness of the pericardial surfaces may be sufficient for its production.

The loudness and character of the friction-sound are modified by several accidental circumstances. Thus, the intensity of the sound depends not only upon the roughness of the surface of the fibrous deposit, but also upon the area of the roughened surfaces which rub upon each other; upon the force of the heart’s action; and upon the amount of fluid diffused into the sac. These accidents necessarily modify the loudness and clearness of the sound; but still, with rare exceptions, its essential character is ever that of a rubbing sound. Whatever particular well-known sound fancy may please to liken it to, it is still a friction-sound, bearing but one import to the physician’s ear. Its varieties are only degrees of loudness or weakness of the rubbing, of the rub being regular or irregular, and reaching up to a rough cracking and even scratching sound.

Conditions necessary for the Production of the Friction-Sound. These are the above from what are the conditions requisite for the production of the friction-sound; viz., the deposition of solid or semi-solid matters on the surface of the membrane; the contact of its opposing surfaces; and a certain degree of force in the action of the heart. Serous, hemorrhagic, or purulent effusions, will not of themselves occasion a friction-sound; nor will the sound arise when the heart’s action is very feeble, nor whenever such an amount of effusion is present as suffices to prevent the surfaces coming in contact, and so rubbing on each other—a condition, indeed, which very rarely obtains in a complete manner; that is, at all parts of the pericardium.

The amount of exudation suffices, in any given case, to prevent the production of a friction-sound, cannot be determined. The size of the heart itself; the amount of solid deposit on the serous surfaces; the weak or strong action of the heart; the situation of the deposit—these, and other circumstances, which cannot be decided with any degree of certainty in such cases, necessarily modify the result. This much, however, I believe, is certain—that the sounds described by Dr. Watson, "no conceivable amount of fluid will of necessity totally annul friction-sound." When the quantity of serous fluid is large, the friction-sound, if it still exist, will be heard chiefly about the base of the heart; the friction-sound may be entirely lost, and then the friction-sound is, when it is no longer audible over other parts of the pericardial region. The reason of this no doubt is, that the opposing surfaces of the membrane are less liable to be separated by the serous fluid around the roots of the great vessels and at the base of the heart, than at other parts of the organ.

Sometimes, although very rarely, friction-sound does not avail us as a sign; for it may be absent altogether, or it may have been present and passed away before the patient comes under our observation. Its duration is uncertain; it may disappear after a few days, and it may remain constantly present for very many days together. Friction-sound is, however, in the case of the patient, or in adhesion of the pericardial surfaces, or in the resolution of the inflammation; and, probably, sometimes in the formation of "white spots.

Difference between Endocardial Murmurs and Pericardial Sounds. It is in certain rare cases difficult, if not impossible, to distinguish the pericardial friction-sound from an endocardial murmur. A great authority in stethoscopy asserts, indeed, that there is no kind of endocardial murmur (with the exception of the whistling murmur) which may not resemble a pericardial friction-sound. We certainly not unfrequently meet with cases in which the most practised ear will hardly venture to draw the difference between the friction-sound of the murmur from the more characteristic of its sounds. In such cases, we must endeavour to enlighten our diagnosis by other signs: we must judge of the nature of the sound by the circumstances of the case; by the presence or absence of coexisting signs of endocardial disease. The friction-sound is generally double; it does not coincide exactly with the heart’s sounds; it commences rather before the systole, and it is heard; and when a murmur is present, the friction-sound is in the direction of the current of the blood flowing along the vessels out of the heart. The friction-sound, moreover, do not replace the heart’s natural sounds, except when they are so loud as to overpower the sounds of the murmur. Dr. Sibson has shown, increases the intensity of the pericardial friction-sound in certain cases, but does not, he says, affect an endocardial murmur.

This latter remark, however, must not be received as invariably true, and all these differential signs, indeed, must be accepted with a certain degree of caution, and for the following reasons: An endocardial murmur may be, and very frequently is, present in pericarditis, and may thus, consequently, complicate the pericardial friction-sounds. The heart’s action may be so rapid, that the relation in time of the pericardial murmur to the heart’s sounds cannot be determined. A pericardial friction-sound is, indeed, an accompaniment of the pericardial region; thus, I have heard a friction-sound precisely resembling, as far as the ear could judge, a rough aortic systolic bruit, conveyed up to the top of the sternum, in the case of a phthisical patient, who had been for some time under medical treatment for an inflammation of the pericardium. The others signs, also, proposed for the recognition of the pericardial friction-sound, are modified by accidental circumstances, by the force of the heart, the quantity of fluid effusion, etc. Even pressure by the stethoscope does not appear to be an infallible sign. Dr. Walde asserts that he has heard a mitral murmur increased in force by it; and it seems very probable, that pressure over the site of the pulmonary artery will in some cases excite or increase a murmur there. It must also be remembered that the effects of the pressure can hardly be made to bear upon the pericardial surfaces in persons advanced in life, and in whom the walls of the thorax are firm and unyielding. Neither can the pressure be exerted upon the affected parts, except when it can be made to bear directly against the roughened surfaces of the pericardium, and therefore not in cases where the friction-sounds proceed from the sides and back parts of the heart.

* Dr. Watson says, according to his experience, that pericarditis is invariably complicated with endocarditis. It is; but, however, he remembered that a systolic bruit at the apex of the heart and over its aortic valves is not unfrequently present in many cases of acute phthisis. The presence of such a murmur, therefore, in the course of the rheumatic pericarditis, is no sure sign of an aortic bruit, or, on the other hand, and, indeed, it is not invariably a sign of endocarditis under such circumstances; and, unlike the aortic bruit just spoken of, is rarely found to disappear with the conclusion of the rheumatic pericarditis.
A pericardial friction-sound may also be confounded with an exo-pericardial friction-sound, arising from the rubbing of the outer surface of the pericardium against some opposing surface of the organ, or its pericardial cavity, when this is covered with an exudation.

Hence, there appears to be no absolute sign by which these murmurs may be distinguished from the one of the pericardial friction.

Sounds—certain bronchial rales, for example—which sometimes resemble friction-murmurs, often arise within the lungs; these, however, and likewise pleuritic friction-sounds, are readily distinguished from pericardial friction-sounds by the circumstance of their cessation during the arrestment of the respiratory movements.

A blowing murmur—endothoracic—is occasionally observed along the left costal border under the left ventricle, in cases of pericarditis, in which, after death, no valvular lesion is found to exist.

The cause of such a murmur may be attributed either to pressure upon the aorta, produced by the exudation of lymph upon it; or to the loss of its elasticity, or some other alteration of its coats, caused by the inflammatory process; or to irregular action in the heart's muscular movements, involving those of its columnae carneae, whereby the function of the auriculo-ventricular valves is rendered, temporarily, incomplete; or again, it may possibly, I have thought, be ascribed to the pericardial adhesions, these being of such a character as to prevent the walls of the ventricles from contracting freely, so that the mitral orifice is left partially unclosed during the heart's systole.

**Pericarditis: Percussion Signs.** Effusion into the pericardium, is, by the ordinary degree of præcordial percussion dulness, but not always in proportion to the amount of fluid present. When the effusion is very considerable the percussion may be extensively dull, reaching over the greater portion of the anterior surface of the left side of the thorax, and considerably beyond the right border of the sternum. Increased resistance, also, is, in such case, felt by the finger on percussion. The dulness arising from pericardial effusion is more marked than that arising from hyper trophy and dilatation of the heart. Practised from day to day, percussion often enables us to follow the increase or diminution of the fluid in the pericardium; it does not, however, give us any accurate information as to the actual amount of effusion which has taken place. There are, indeed, several sources of error, which may mislead us in judging of the amount of effusion by percussion; and these it is well to note. A small amount of pericardial effusion, for instance, may be associated with hyper trophy of the heart, with malignant disease, with enlargement of the left lobe of the liver, with aneurism of the aorta, with condensation of the lungs, and with pleuritic effusion; and consequent ly the extent of the pericardial dull percussion-sound may, in such cases, be increased out of proportion to the amount of pericardial effusion. A careful analysis of the signs and symptoms present, however, often enables us to fer to a tolerably correct diagnosis as to the nature of the coexisting diseases, and thereby prevent our falling into the errors referred to.

On the other hand, there is emphysema of the lungs, or distension of the posterior part of the lung, the amount of fluid, the parts being inordinately extended, and consequently overlapping the heart to an unusual extent; or when the lungs are adherent to the thoracic walls in front of the pericardium; or where there is atrophy of the heart—the amount of effusion present may appear, on percussion, to be less than it really is.

An inflamed stomach, also, often modifies considerably the percussion sound over the precordial region.

As a general rule, we find in health that about two and a half to three inches of the heart's surface is uncovered by the lungs. This uncovered surface may be rudely likened to a triangle. A line is drawn regularly down, from the apex to the left of the centre of the sternum, from about the fourth to the sixth or seventh rib, defines the position of the anterior edge of the right lung, and constitutes the right side of the triangle; its left consolidation, or clear sound to percussion; and the posterior surface of the lungs, the opposite portion of the triangle, being clearly excited by the contiguity of the pericardium. Such consolidation will, of course, of itself occasion increase of the præcordial dull percussion-sound.

I have observed cases of this nature after death, where the anterior border of the diaphragm, by consequence of the respiration being carried on by the anterior portions of the lungs, lay far over the distended pericardium, which was obliterated or covered by the pleural effusion, or the pleura, and the lung, and even when the distended pericardium reached beyond the right border of the sternum.

*Pneumonie consolidation of the parts of the lungs which overlap and surround the posterior surface of the heart, being occupied and excited by the contiguity of the pericardium.*

Sterno-costal articulation, down through the cartilages of the fifth and sixth ribs; these two lines meet above, directly beneath the level of the fourth cartilage; the base joins the sides above the cartilages of the sixth rib.

The dull percussion is often ill-defined at the base of the triangle, because the left lobe of the liver in many persons lies in close contact (the diaphragm intervening) with the lower border of the heart.

Between this surface of the heart and the thoracic walls, only fat and intercellular tissue naturally intervene; hence, when dulness on percussion exists beyond the limits here indicated, we may suspect an abnormal condition of the parts beneath. The dulness in itself, of course simply indicates that solid or fluid matters, containing no air, lie beneath the thoracic walls in such parts where a certain amount of air ought naturally to be present; the diagnostic value of the dulness must be decided by other signs.

The extent of dull percussion-sound varies during respiration and expiration: on a full and deep inspiration, when the lungs almost wholly cover the pericardium, the dulness is hardly perceptible; on a deep expiration it becomes proportionally increased.

The fluid effusion collects, in the first instance, about the base of the heart and the roots of the great vessels; and if in its situation that the abnormal dulness is first observed; but as the quantity of effusion increases, the dulness reaches across to the right, or beyond the right border of the sternum, upwards towards the clavicle, and towards the left lateral region—and that is, in the direction of the heart's breadth from the right, behind, the heart, sinks downward and backwards in the pericardium when the patient lies recumbent; and its apex is pressed somewhat to the left of its natural position.

In the first instance, gather from the above a consideration that pericardial dulness is, in itself, no certain sign of pericardial effusion; but, judged by the light of other signs, it becomes so. Pericardial effusion commences at the anterior, pleuritic effusion at the back sides of the thorax. In hypertrophy of the circulation, in pericardial dulness is not so well marked, nor is the resistance so complete as in pericardial effusion. And besides this, hypertrophy has its special signs to distinguish it; in effusion the dulness commences suddenly, and is most markedly in an upward direction; in hypertrophy, downward and towards the left side, or in the direction of that particular part of the heart which is enlarged. In fact, in these cases, there are very few instances in which the careful observer can be deceived in his diagnosis, especially if he has watched the progress of the pericardial inflammation from its commencement.

Enlargement of the heart, particularly of its left lobe, occasionally pushes the heart upwards and to the left; encroaching on the precordial region, and increasing much the precordial dulness. Such an enlargement of the heart would hardly fail of being recognised.

It is necessary to refer particularly to the other abnormal conditions which may produce increased precordial percussion dulness; their coexisting symptoms will generally enable us to distinguish between them and pericardial effusion.

Together with the friction-sound, and other signs of pericardial effusion, a *fremirntus* is sometimes, but not often, felt over the precordial region, when the heart is laid thereon. It is doubtless occasioned by the circulation of the blood rising to the friction-sound. It has been observed in cases in which effusion of lymph has taken place external to the pericardium, whereby the pericardium has been united to the thoracic walls.

In young subjects, and in those in whom the elasticity of the ribs and their cartilages permit of it, a bulging of the precordial region is sometimes observed where the pericardial effusion is considerable.

Distention of the external jugular veins is not unfrequently observed in the advanced stages of pericarditis, and when present it indicates obstruction to the circulation through the heart; the degree of the distension is, in fact, a good test of the amount of the impediment. When the veins do not collapse, but remain constantly distended during inspiration and expiration, we may be sure that the circulation is seriously obstructed.

*[To be continued.]*

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*Dr. Graves (Lecture 11, 1861) relates a case of this kind, where the base of the heart was pushed up as high as the first intercostal space by the left lobe of the liver, and fluid suddenly effused in the abdomen.*