Thesis on Diagnosis and Pathology of Rheumatic Endocarditis.
Thesis

on

Rheumatic-Endocarditis.

I have selected this subject for a thesis, partly on account of the deep interest which it excites, owing to the vital organs involved, in the mind of the physician, as well as from the important phenomena which usually attend the progress of such a malady.

John Robertson
Student of Medicine
University
Edinburgh
March 31st, 1863
Index

A description of the topography of the heart
and large vascular branches more immediately
connected with it. 1

The physiology of the valvular apparatus
of the heart. 8

Cardiac murmurs, the superficial areas over
which they are audible. 13

Rheumatic endocarditis—its general symp-
toms. 21

Rheumatic endocarditis—its pathology. 29

Rheumatic endocarditis—its physical
signs. 42

Cases illustrative of rheumatic endocarditis. 54
This diagram is intended to exhibit the position of the heart and great blood vessels in the chest and their relation to the thoracic parietes.
1. Topography of the heart and blood vessels:

An accurate knowledge of the position of the heart and of its several parts, is absolutely essential for the discrimination of numerous diseases pertaining to the circulatory system. The heart is placed obliquely in the chest, lying beneath the lower half or two-thirds of the sternum, and occupying more of the left than the right half of the thorax. Its form is that of a blunt cone, flattened on its under surface, with its base directed upwards, backwards, and towards the right side, opposite the spinal column, and corresponding to an interval between the fifth and eighth dorsal vertebrae; its apex inclines downwards, forwards, and to the left, striking the thoracic parietes during life, when normal, below the fifth rib, and near the junction of that rib with its cartilage. Dr. Williams of London, describes the site of the apex beat, as two inches below the left nipple, and one inch on sternal side.

The heart projects half an inch on the right side of the sternum, and within the same
distance of the left nipple by transverse measurement. A portion of the right auricle with a small section of the right ventricle is placed on the right side of the sternum, between the third and fifth ribs; behind the sternum are situated the larger portion of the right ventricle, a less proportion of the left, the remaining part of right auricle and the root of the great vessels.

On the left side of the sternum, the greater portion of the left ventricle, the left auricle, and a portion of the right ventricle near the heart's apex, all lie. The base of the heart is situated on a level with the upper border of the third costal cartilage, while its lower border is defined by a line drawn across the sternum from the articulation of the sixth and seventh cartilages on the right side to the spot of the apex beat. It is retained in position partly by the bag of the pericardium, which invests it, as well as the large vessels attached to its base.

The relation of the lungs to the heart:
In consequence of the oblique position of the heart in the chest, only a small portion of its superficialis comes in contact, anteriorly, with
the parietes of the thorax. The base as well as the
greater portion of the left ventricle is directed a-
way from the sternum and costal cartilages, and
placed deeply in the cavity of the chest; only a
small triangular portion of the right ventricle come
into apposition with the chest walls, the rest of the
organ being overlapped by lung substance. From
the level of the second costal cartilage, down to the
fourth, the inner margins of each lung run parallel
and almost close behind the middle of the ster-
num; consequently they overlap the vessels at the
root of the heart. Below this point, the left lung di-
verges from the right, curving pretty nearly in the
course of the fourth left costal cartilage, but project-
ing more or less over the apex, while the right descends
almost perpendicularly behind the sternum as low
as the attachment of the ensiform cartilage, and then
follows the direction of the sixth right costal car-
tilage. From this observing the relative position
of the lungs to the heart, the latter will be found
embedded between the two respiratory organs, with
the exception of a very small portion of the left ven-
tricle, and the triangular space above mentioned
which will be subsequently described. I may
here state that the area of this part of the central organ which remains uncovered by pulmonary tissue varies slightly during every act of respiration being only two square inches both inspiration when the lungs are inflated.

The Aorta arises from the base of the left ventricle, and inclines upwards, forwards, and towards the right side of the sternum, lying behind this bone till it reaches the second right costal cartilage (whence termed the aortic cartilage) when it suddenly sweeps across the manubrium of the sternum on a level with the lower border of the first cartilage, downwards and backwards to the left side of the third dorsal vertebra. The superior vena cava is situated on its right side and the pulmonary artery on its left. The carotid and subclavian arteries along with the external jugular veins may be easily recognised, especially the former in the living subject, and the latter become prominent when there exists a turgescence of the venous system, so that a description in their case would here be superfluous.

The pulmonary artery springs from the base of the right ventricle behind the sternum, and close to the articulation of the third left costal
cartilage with the sternum; it inclines upwards to the left side until it bifurcates opposite the second costal cartilage (hence designated the pulmonary cartilage). 

2. For the purpose of having a clear perception of the various phenomena which constitute the true cardiac sounds, it is advisable to notice briefly the structure and mechanism of the valvular apparatus.

The internal lining membrane of the heart is a thin transparent fibrous membrane, being composed of thin but distinct layers, the free surface of which consists of an epithelial stratum of polygonal cells resting on a fibrous elastic tissue (Bain). On the left side of the heart this membrane is continuous with the lining membrane of the pulmonary veins and aorta; on the right side of the heart prolonged into the venous and arterial orifices; but it seems more opaque on the left than on the right of the organ.

The heart is a hollow muscular organ, consisting of four chambers or cavities, two of which are situated on opposite sides of a longitudinal septum which divides this organ into a right and left half, viz. an auricle and a ventricle. Each auricle communicates with its own ventricle by means of an auriculo-ventricular
orifice, which is provided with a valvular apparatus: the tricuspid valve guards the right, while the mitral valve guards the left auriculo-ventricular opening. The tricuspid valve consists of a fold of the endocardium, between the layers of which numerous fibrous tissue is found. The valve consists of three segments which are united at their bases to form a tendinous ring round the margin of the aperture between the two cavities. One of these triangular flaps looks towards the ventricular septum; another is placed towards the anterior wall of the cavity; while the larger of the three is so situated that when not in action it partially covers the pulmonic orifice. In the interior of the right ventricle are found bands of muscular fibre called "columnae carneae," of which there are three distinct sets, the most important being those that are directed from the apex towards the base of the ventricle (musculi papillares); these consist of small muscular bundles attached by one extremity to the walls of the ventricle, while their opposite extremities end in small tendinous chords (chorda tendinae), by means of which they are connected with the ventricular surface of the valve. The tendinous chords arising from a given papillary muscle become attached to the ventricular surface of the two adjacent valves, between which
they are placed, as well as the smaller intermediate lobes, at three distinct places: some at the base, others toward the middle, and the last set are inserted a little within the thinner marginal edge of each segment.

The mitral valve is similarly constructed, except that it consists of only two lobes; hence its name bicuspid; the large of these flaps lies between the aortic and auricular orifices, the number of chordae tendinae and muscular papillae correspond to the number of segments of either valve, those of the left auriculoventricular valve being, however, much larger and stronger than those of the right.

The semilunar valves, both aortic and pulmonic, being situated at their respective orifices, and similarly constructed, will be considered under one general description. The aorta and pulmonary arteries, which form the main trunks of their respective systems, are connected to the heart, chiefly, by means of their middle coat or fibroelastic tunic; the cardiac orifices from whence these vessels originate, being enclosed with white fibrous tissue, become firmly united by means of their septums to the middle tunic of these arteries. Along the lines of these septums, the lining membrane of the heart, becoming continuous with that
of the arteries, forms three semilunar segments, which are strengthened by processes of fibrous tissue continuous with the septums. These segments or valves are attached to the fibrous rings at the roots of the arteries, by their convex borders, while their margins are traversed by thin fibrous cords at three distinct points, the central one constituting the "corpus arteriosus:"
The physiological action of the valvular apparatus.

The mere hydraulic pressure of the blood, is stated by physiologists, as accounting for the closure of the valves. During the heart's systole, the right and left auriculo-ventricular valves, which had been partially closed by the amount of blood in the two ventricles, shortly previous to their contraction, are now suddenly and forcibly closed, so that not merely do the free margins of each curtain come in contact for that narrow portion of each lobe external to the insertion of the chordae tendineae; all round their free edge is folded inwards towards the auricular opening, in close apposition with a similar fold on the adjoining segment. But the mere contact of these lobes with each other is not sufficient to prevent regurgitation; they must be retained during the whole period of ven-
tricular contraction in a certain position. Shoda remarks that where the chordae tendineae are attached to the margins of the bicuspid and tricuspid valves, little semilunar pouches are formed corresponding to the extent of insertion of each cord, which swells out by the blood putting the valves on stretch and so sufficiently close the orifice. The musculopapillae and chordae tendineae prevent the valves from being pushed back into the auricles, by the contraction and relaxation of the former during the systole and diastole of the heart's action; whereas the tendinous cords during dilatation of the ventricle maintain the valves in their proper position for their simultaneous action.

The arterial valves are closed, owing to the reaction of the elastic arterial tunics upon the column of blood sent into the arteries, so that, by the sudden recoil of the wall of the artery, not only is the blood propelled forwards along these vessels, but is likewise forcibly driven backwards towards the ventricles; when slipping between the valves and the walls of the artery, these are instantaneously closed and with such perfect adjustment, by means of the "sinulae" and cartilaginous nodules placed on their free margins, that they approximate each other more completely in proportion to the pressure they sustain.
The mode of production of the normal cardiac sounds.

The sounds elicited by the heart are two in number, systolic and diastolic, or first and second sounds, the latter terms being those usually employed by the physician. The first sound is a dull, deep, prolonged sound, and most audible at the heart's apex, while the second is clearer, more superficial, and a shorter and sharper sound than the first, being best heard over the sigmoid valves, on the left side of the sternum, opposite the inferior margin of the third rib. In stating the causation of the cardiac sounds, the writer found it somewhat difficult to determine how far the various causes, as stated by medical authorities, contributed to the production of these phenomena, more especially those in connection with the first sound. The heart, in consequence of its being a double organ and both sides acting in unison with each other, is provided with a special nervous arrangement which serves to control the heart's rhythmic movements, and by means of which its separate parts work harmoniously together in producing that rhythm or synchronicity of action which characterizes the movements of that organ. From this it appears that both sides of the heart have a share...
in the production of the two sounds. The sudden expansion of the aorta and pulmonary arteries, along with the vibrations of their tendinous cords, seem to be the principal cause of the first sound. The rushing of blood through the aorta and pulmonary arteries, and the throwing back of the semilunar valves—the impulse of the heart diastole against the thoracic parietes, and the muscular contractions of the ventricles, are all merely secondary in the production of this sound, and only produce audible phenomena when some morbid condition of the heart is present. The second sound is synchronous with the ventricular diastole, and appears to be mainly produced by the sudden closure and tension of the semilunar valves.

Before considering the subject of cardiac murmur, it is necessary to notice shortly the succession of actions or physiological facts that constitute a cardiac pulsation.

The following table of Dr. Hinch shows very well the order of succession in which the actions of the heart take place during a period of eight parts each. The numbers representing eights of a second.

<table>
<thead>
<tr>
<th>1st Sound and Impulse</th>
<th>2nd Sound</th>
<th>Pause</th>
</tr>
</thead>
</table>
The following diagram of Dr. W. J. Airdhers is intended to exhibit the whole audible and tangible phenomena of the heart's action, and their relation to the physiological movements which cause them.

From the preceding table and diagram, it will be observed that the auricles contract immediately before the ventricles, the latter contracting afterwards, producing the first cardiac sound, then follows the ventricular diastole coinciding with the heart's second sound, which is followed by a period of rest, the whole constituting one cardiac pulsation. In the normal condition of the heart's action, the cardiac sounds indicate or correspond to the active movements of the organ, bearing a definite relative to each other, as well as to the interval of rest, giving rise to the heart's rhythm. This rhythm is liable to vary according to the rapidity or slowness of the heart pulsation: should the cardiac beats be rapid, then the sounds succeed each other very closely, while the period of rest is proportionately diminished, but the reverse is the case when the
Pulsations of the organ become clear. In proportion to the rapidity of the heart's action, arises the difficulty of recognizing the first and second cardiac sounds, and should there exist an irregularity and feebleness of these audible phenomena, it is almost impossible to distinguish between the two sounds.

Cardiac murmurs—Their relation to the superficial areas over which they are best audible. From the peculiar configuration of the heart, its position in the thorax, and the relative situation of its four orifices, as regards each other, it will be observed that from the mere position of the valves, very little information can be gained as regards the exact seat of the murmur. By merely examining that particular region. Therefore in order to arrive at an accurate diagnosis on this point, we must first note the seat of the murmur and the direction in which it is propagated. 1. Mitral murmurs: A systolic mitral murmur is considered the most common of all organic murmurs. It is the result of regurgitation of blood from the left ventricle through the mitral opening back into the auricle. It is best heard a little above the apex of the left ventricle, by being transmitted along the tendinous cords of the myocardium.
trial valve, and gradually diminishes in intensity as the stethoscope is carried upwards until the middle of the fourth left costal cartilage is reached, where they usually become inaudible. In many instances the murmur is recognized with great intensity in the left axilla and near the inferior margin of the left scapula. Also a greater intensification of the pulmonic second is often noticed to occur in connection with this murmur, and when well marked becomes an important diagnostic phenomenon. Thoda was the first who directed his attention to this point, and accounts for its production thus: owing to the retardation of the flow of blood in the lungs and pulmonary veins, the pulmonic arterial tunics become thickened and consequently contract more powerfully for the propulsion of the blood along its proper channel; so originate an intensified second sound with its maximum intensity at the pulmonic cartilage. A diastolic mitral murmur is supposed to be a rare murmur, in consequence of the muscular walls of the auricle being much thinner than those of the ventricle and so less forcibly contracting on its contents, and giving rise to a fuller current; do not produce any acoustic phenomena, even where there exists considerable mi
ternal constriction. But both systolic and diastolic initial murmurs, more frequently occur together, because there are few-marked conditions of the auricular orifice, which give rise to distinctness, but will at the same time permit of free regurgitation. It is heard in great intensity over the left apex of the heart and gradually ceases to be audible as the ear approaches the base of the organ.

A systolic aortic murmur is usually heard over a considerable extent of the surface of the body but more especially at mid-sternum on a level with the third intercostal space, also with great distinctness at the aortic cartilage, and not infrequently audible in the great arterial trunks in the neck and extremities. It may be quite distinct along the whole length of the aortum, but the fact of its being so readily transmitted along the systemic circulation distinguishes it from all the other vascular murmurs. A diastolic aortic murmur is produced by the incompetency of the aortic valves and regurgitation of blood into the ventricular cavity. This murmur is likewise transmitted along with the arterial current but to a much less extent than the systolic brief, it is heard with the greatest distinctness over the base of the heart, and when very intense, becomes audible at the apex.

A systolic pulmonic murmur like the aortic is the
result of obstruction to the onward flow of blood from the right ventricle into the pulmonary artery. In examining this murmur, it must be borne in mind that the left lung is liable to overlap the left base of the heart during inspiration, and more especially when its anterior margin is in an emphysematous condition; therefore apply the stethoscope during expiration over the pulmonic valves, and in the interval between the second and left costal cartilages, close to the sternum, where the murmur is heard with the greatest intensity. It is further recognized by its superficial character and being quite imperceptible along the sternum back or systemic circulation. A diastolic pulmonic murmur is, along with the preceding, of very rare occurrence and duluble over the same surfaces.

A systolic tricuspid murmur is heard over that portion of the right ventricle which remains uncovered by lung substance towards the lower end of the sternum and ensiform cartilage; such a space is described as included within a triangle whose limits are as follows: The right side extends from the right margin of the sternum to a little above the third left costal cartilage; the lower side reaching from the fifth and sixth left costal cartilages to the lower margin of the fifth rib where it joins the corresponding left costal cartilage, while
the left side is formed by a slightly curved line, convex towards the right side, joining the free extremitities of the other two sides. A murmur of this kind is rarely heard above the level of the third rib. It is almost imperceptible at the left apex. It is usually the result of an hypertrophied and dilated right ventricle, rendering the bicuspid valves incompetent, and so allowing regurgitation of blood into the auricle, which ultimately becomes dilated, and producing distension and visible pulsation in the jugular vein during the ventricular contractions. A diastolic tricuspid murmur is in all probability the rarest of all the cardiac murmurs, when audible will be heard in greatest at the xiphoid cartilage, and diminishing in force towards the heart's apex and quite inaudible at its base.

Having discovered the existence of an endocardial murmur and the seat of its maximum intensity, the next point to be ascertained is to determine the rhythm of the murmur, or in other words, during what period of the heart's action does the murmur occur. In attempting to find out the relation of a murmur to the various acts that take place during one cardiac pulsation, the phenomena which accompany the distinct periods of cardiac activity and qui
Essence must be kept in view, e.g. whether the murmur is synchronous with the systole or diastole of the heart; whether it precedes, is simultaneous with, or immediately follows either the first or second sound; and if the interval of rest between the second and first sounds is entirely free from, or is partly or completely occupied by one or more murmurs. All the endocardial murmurs that are not of complex origin are thus readily determined and localised, but where a combination of murmurs happen during a cardiac pulsation, in the same organ, and where they substitute the normal sounds of the heart, a difficulty in the diagnosis of such will be experienced by one who is not a great adept in the use of the stethoscope. But by carefully observing the difference in character of what at first seems one murmur, and its relation to the heart’s impulse or radial pulse, as well as the seat of its maximum intensity, it will be found to consist of several murmurs that have coalesced into one.

A further aid in the discrimination of such murmurs, that are sometimes surrounded by causes of uncertainty which are apt to confuse the mind, will be derived by attending specially to the condition of the pulse, for the purpose of discovering any visibility in the radial or ulnar arteries, and so detect...
atheromatous or calcareous degeneration of the arterial tunic's of the systemic circulatory system, but more especially to notice the charactel of its impulse as communicated to the fingers. Also, by means of inspection, palpitation, and percussion of the precordial region, along with the previous history of the case and the nature of the accompanying symptoms, an accurate diagnosis will, in the majority of instances, be attained.

By the term 'rheumatic-endocarditis' is meant, an inflammatory disease affecting the lining membrane of the cavities of the heart, more especially its valvular apparatus, and supervening as one of the most dangerous complications during an attack of acute rheumatism. That endocarditis is a more frequent complication of acute rheumatism than peri-carditis, and that the former malady is more frequently produced by it than by any other exciting cause, will appear from the following statements. Dr. Latham remarks that from cases sufficiently numerous, endocarditis occurs nine times as acutely rheumatic for peri-carditis once—that simple endocarditis constitutes more than two-thirds of all rheumatic cardiac affections, and simple pericarditis only one thirtieth, and that peri-
carditis is more frequently found in combination with endocarditis than alone. Dr. Hope enumerates a list of the exciting causes that originate endocardial inflammation, but states that by far the most frequent is that of acute rheumatism: and Dr. Watson mentions these startling facts. I have known only three persons pass through acute rheumatism with an untouched heart prior to the age of puberty; and in two of these I am by no means certain that the articular disease was genuine rheumatism. These observations inculcate this most important principle on the medical practitioners, that whenever a patient is the subject of one or several arthritic attacks, the heart should invariably be made the object of a most careful examination for the detection of organic disease, and more especially those premonitory symptoms recognised and described by Dr. Latham as the harbingers of endocarditis, viz. an undefined modification of the cardiac sounds, which does not amount to a murmur, along with a certain peculiarity in the heart's action, which can only be ascertained by an experienced and skilful physician. By the discovery of such local signs and the constitutional state of the individual, we might be able to anticipate the disease in question, and by the timely ad
ministration of judicial remedies, save a fellow-sufferer from the appalling consequences of permanent organic lesion of the endocardium. It has been said by some medical authors that endocardial inflammation is the result of metastasis, or a transmission of the cardiac affection from the more distant articulations of the extremities to the lining membrane of the heart; that such may occasionally happen I do not deny, but that the majority of instances do not originate from such a cause is proved by the fact that rheumatic endocarditis is sometimes established a day or two previous to the articulareffect; that both diseases may simultaneously by coexist, and that the cardiac symptoms only begin to manifest themselves just as the articular pains have disappeared.

Next proceed to notice the general symptoms of the special subject under consideration, as they materially aid the diagnosis of organic lesions of the heart, apart from the physical signs as revealed by the stethoscope. In discussing the more characteristic symptoms which accompany the early and progressive stages of rheumatic endocarditis I shall state and treat of those in the first instance, which are "primary" or the direct sequelae of the seat
of disease, and subsequently, of those which are of secondary origin and manifest themselves in organs which have a close relationship with the heart. One of the primary symptoms which are usually witnessed during the early stage of the cardiac affection is pain felt across the substernal region and attended by a high inflammatory fever. In some cases the patient only complains of a feeling of uneasiness over the situation of the heart or a sensation of tightness or constriction across the lower part of the chest, which is characterised by distinct remissions and exacerbations, while the countenance assumes an expression of the most intense anguish and deepest agony when such pain partakes of the character of angina pectoris, by extending upwards along the left side of the thorax and stretching down the left arm. Palpitation, or a violent action of the heart becomes an important diagnostic mark only when viewed in combination with other existing morbid or pathognomonic phenomena and should it become greatly intensified or of a tumultuous nature after the performance of some slight exercise and afterwards gradually resume its former impulse and frequency, then it receives an additional confirmation as indicative of disease. An irregularity of the cardiac pulsations, as evinced
by the feebleness and unequal pulsations in the radial arteries, is an important symptom in certain organic vascular lesions on the left side of the heart; as, for instance, in cases of mitral obstruction and regurgitation, when the free transmission of the flow of blood through the cavities of the heart becomes impeded, and consequently the left ventricle, owing to the diminished supply of arterial blood, contracts imperfectly on its filled contents, hence the intermittence, irregularity, and inequality of the pulse. These external manifestations when symptomatic of organic mischief within the heart, during an early period of the disease, not only continue to persist as the malady progresses, but usually become intensified in their character, while a new train of symptoms originate in various organs in the more advanced stages of endocarditis, concerning which a few explanatory remarks will now be made. In consequence of the close connexion which exists between the circulatory and respiratory organs, as well as from their mutual dependence on each other, any serious deviations from the healthy or normal standard in the vital functions of the one will materially interfere with the equally important functions of the other. Hence it is that
we are called upon, during the last stages of cardiac disease, not so much to palliate its more immediate morbid phenomena, as those unequivocal yet still more distressing complications of paroxysmal attacks of suffocative coughs and dyspnoea, which ultimately prostrate the patient's strength and exhaust the powers of life. Cough although a frequent is not always a constant symptom, and when present is the result I conceive of displacement of the lungs by an abnormally large and hypertrophied heart, as well as from the unbalanced condition of the circulation or the exsicotic disproportion between the amount of venous blood in the respiratory organs and the supply of atmospheric air for its ablation. Its character is that of a short, dry cough, although in some instances it is accompanied with a copious watery expectoration. Dyspnoea is almost never absent during the advanced stages of endocardial disease, more especially in those cases where there exists an impediment to the flow of blood in the left chambers of the heart from a permanently contracted state of the mitral orifice, or from a retrograde current through that opening, as the result of valvular incompetency. Consequently, the lungs become congested and engorged.
with venous blood, the pulmonary artery becomes thickened and dilated, and the right side of the heart dilated and hypertrophied; subsequently the abdominal viscera become involved in the diseased action, particularly the liver and spleen, which, unless previously detected by the physician, will soon have his attention directed to these organs by the patient manifesting symptoms of uneasiness and tenderness over the hepatic and splenic regions. As the obstruction to the systemic venous circulation advances, the constitutional disturbance becomes more marked by a sturgence of the jugular and superficial thoracic veins, while the integumentary system assumes a dingy dusky hue as observed in the cyanotic appearance of the countenance, resulting from the circulation of imperfectly aerated blood throughout the system. General anasarca, with drophial accumulations in several or all the serous cavities, passive hemorrhages from the mucous membranes, especially adenectasis, constitute several of the more characteristic signs of an advanced case of rheumatic endocarditis. When the patient expectorates pure or mixed blood, during the progress of cardiac disease, and there coexists a mitral obstructive or
regurgitant murmurs the probability is that sanguineous extravasation has resulted into the pulmonary tissue (the so-called pulmonary apoplexy) and lacerated the parenchyma of the lung from the excessive congestion and mechanical obstruction of the pulmonary vessels, or from the capillaries of the mucous membrane of the minute bronchial tubes lacerating and pouring their contents directly into the respiratory passages. Another class of symptomatic phenomena referable to endocardial disease originate from a disordered cerebral circulation and result in a derangement of the cerebral functions. The brain may become intensely congested from an obstruction to the return of the systemic venous circulation to the right chambers of the heart, producing stupor, delirium, coma, cerebral apoplexy with their dreadful effects. Or equally serious destructive miliary changes spring from obstruction to the arterial circulation within the cranium by the plugging of some of the smaller cerebral arteries by fibrinous concretions which, on becoming detached from the valvular apparatus on the left side of the heart, are transmitted along the systemic circulation till they reach the seat of obstruction.
I may here mention that embolism may, instead of resulting in paralysis, terminate with all the marked characters of diffuse phlebitis or pyaemia, by the fibrous masses soften and disintegrating, while the disorganized matter mingling with the blood contaminates that fluid. Tauber's cellulitis supervening in the more advanced cases of endocarditis, indicates that the liver is so intensely congested with venous blood, that the bile has become already in the hepatic ducts owing to the pressure exerted by the engorged and distended veins, and that recovery under such circumstances is of rare occurrence. The feelings of lassitude and exhaustion, headache and disturbed sleep, are morbid sensations of minor importance, but still diagnostic in particular stages of the disease. Droopy, when the result of endocarditis first manifests itself by an oedematous condition of the lower extremities which almost entirely disappears by the patient assuming the recumbent posture for some period of duration, hence the limbs appear less swollen towards morning than during the evening, and as this oedema advances to the stage of general anasarca, the urine, owing to a congested state of the kidneys, becomes scanty and albuminous.
where they are firmly impacted. This embolism, as pointed out by Dr. Kirk, may induce delirium, coma, or a sudden insensibility, from a deficient supply of blood to certain portions of the encephalon, and unfrequently, hemiplegia from a softening and disintegration of brain substance. Similar detached fibrinous clots are often arrested in some of the minute ramifications of the hepatic, splenic and renal arteries, producing a turgescence of their respective organs, but the vessel most liable to suffer from these floating vegetations is the middle cerebral artery. The general aspect of the patient, the emaciated and suppressed respiration, and the peculiar almost pathognomonic condition of the pulse in certain instances, form prominent features which frequently indicate endocardial disease.

Pathology of Rheumatic Endocarditis:

It is only since a comparatively recent period that Bouchlau directed the attention of the medical profession to this species of endocardial inflammation, and its most important pathological effects. In a previous section of this thesis I mentioned the structure of the endocardium and that of the cardiac valves, and now I proceed to observe the trans-
formations and disorganization of texture which these undergo, as the morbid productions of rheumatic endocarditis. Inflammation of the lining membrane of the heart, and more especially that portion of it which invests the valvular apparatus, is a very frequent concomitant of acute rheumatism, so that the following remarks will bear particularly, on the most important seat of the diseased action, while at the same time, applicable to the inflammatory changes in the remaining portions of the endocardium. This membrane, being devoid of blood vessels, receives (it is supposed) its supply of nutritive materials by imbibition from the successive waves of blood which constantly glide over its free surface, rather than from the minute capillaries that permeate the subjacent cellular tissue, hence the 'matrices' marks of acute rheumatism, according to the ingenious theory professed by Dr. Richardson accounts for its liability to repeated inflammatory attacks, while the more decided fibrous organization of the left valves, by exciting more strongly the injurious influence of the circulatory poison, explains why the left side should be more frequently the seat of inflammation than the right side of the heart. From the disease, during its earlier stages, seldom terminating fatally, the successful morbid alterations which mark its progress are very rarely observ
red in the human subject. But from experiments made on the lower animals by Dr. Hope and more recently by Dr. Ward Richardson, the subsequent post-mortem appearances were observed and detailed. First, an increased vascularity, or an inflammatory redness, with a swollen condition of that portion of the lining membrane which enters into the structure of the different vascular surfaces and becomes continuous with that of the blood vessels, along with a peculiar velvety appearance and occasionally the exudation of lymph with minute beads of semi-transparent film, are the earliest structural changes that can be perceived on its free surface. Dr. Kekhtenashev observed that the actual seat of the inflammation is the cellular substance lying under the endocardium, and that the products deposited on the inner surface of the heart must have penetrated through the permeable texture of that membrane, and that the redness observed does not reside, but is merely transmitted through it. But the greater probability is that both of the internal coats are simultaneously involved in the diseased action, although the seat of irritation must have originated in the fibrous, and afterwards communicated to the cellular tissue. Second, the vascularity or intense redness assumes a more subdued
tint until it ultimately disappears, while the valves become leprous and swollen, but considerably thickened and beaded, from the former consistence of the sub-stratum of infiltration, as well as the effusion of lymph on its free surface or deposited within its substance, acquiring greater consolidation. Third, this exudation begins to diminish by the process of absorption though in most cases very imperfectly, leaving the margins of the valves permanently thickened and corrugated, with the more perfect formation of the fibrinous vegetations that so closely adhere to their edges, when coincident with these alterations, the smooth, glistening, translucent appearance of the internal cardiac tissue is substituted by an opacity, whiteness, and a rough, granular, or velvety aspect which constitutes a suitable nidus for the production and deposition of these curdy concretions which form the most important as well as the most frequent valvular lesion in connexion with rheumatic endocarditis. I therefore purport to enter somewhat minutely into their morbied anatomy, causation, seat, and mode of production.

Fibrinous vegetations. When these excrescences are recently formed and immediately examined they are found to consist of a soft, friable, imperfectly organized
or fibrous material, of a pinkish-red colour, and varying in size from a small plum head (when first observed) to that of a midlet-seed or walnut. Gradually they change their colour and appear of a greyish-yellowish hue, as their texture acquires a more compact and firmer consistence, until they become perfectly colourless and in certain instances possess the firmness of fibro-cartilage. When these fleshy granulations are of recent formation, they are easily detached from the slight adherences that bind them to the surface of the membrane, but as they slowly undergo structural transformations of a more permanent organisation, connections of a more tangible kind are contracted, till finally the two different textures become amalgamated to each other. Besides consisting of several layers of imperfectly developed fibres, minute granules and oil globules may be detected, diffused throughout their substance and should they be present for a considerable length of time, not only may they degenerate from stellomatous and atheromatous deposits, but become partially or completely ossified. The most favourable termination in these transparent gelatinous accretions which lead to complete resolution of the valves, are, entire absorption of the morbid deposits during the earlier stages of the disease, or a gradual diminution in their size.
By becoming incorporated with the valve texture, while the living membrane attains its former smoothness and polish. But unfortunately, complete separation of such lesions is an incidental event of rare occurrence, and those who confide too implicitly in these happy results, will, in the majority of instances, find their most sanguine expectations utterly disappointed. More frequently the destructive transformations in these abnormal products have a tendency to further disorganization, viz., ulceration and laceration of texture, occasioned by this separation, rupture of chordae tendineae, or even suppuration of the muscular substance of the heart; but in the milder forms of the disease less severe metamorphoses may ensue, such as permanent indurations, contractions, or adhesions of one or all of the valvular segments, or subsequently infiltrated with otheromatous or osseous deposits. Fabulous vegetations are found deposited on the ventricular surface of the valves in the direction of the current of blood, and usually along the crescentic folds of the semilunar and mitral appendages on the left side of the heart. They vary in their distribution, some occurring singly, others in small clusters, or agglomerated into callosflour excrescences, while a few form pedunculated
masses that dangle freely in the ventricular cavity. The reason why the vegetations should be so regularly deposited along the double semicircular flumes of the sigmoid valves may be accounted for by the difference in structure between their free margins and the base of each curtain; whereas the doubling up of the former on the latter towards the ventricular diastole appears to concentrate the foreign products, simply by their mechanical pressure. In attempting to deduce the mode of production of these feathery structures concerning which various medical authorities have entertained different views, I shall only observe the conditions most likely on which their original formation depend. The blood of patients suffering from acute rheumatism is always found in a state of hyperplasia, from an excess of fibrin, varying in proportion from six to eight parts per one thousand, although in a few instances increasing as high as ten parts in one thousand, but never diminishing in ratio below four such parts. Consequently, Balth and Bouillaud were the first to inquire into the probable cause which led to the depopulation of the fibrin from the blood, as the latter was the earliest observer who had noticed the disease. He thought that lymph diffused on the free
surface of the valves was the source of such fibrinous
accumulations, which, undoubtedly in not a few instan-
ces do thus originate, notwithstanding the arguments
adduced in support of an opposite theory Dr. M'Kee
of Glasgow, by obstructing and artificially irritating the
lining membrane of a vein in a living animal, com-
pletely failed in producing the exudation of lymph
on its free surface, but whenever blood was readmitted
along this irritated portion of the vessel, the deposi-
tion of fibrin on its surface was readily recognised. Dr.
Brockhoff Vienna took fresh blood from a turtle &
injected one-half of the fluid into the heart of a
recently killed turtle, while the remainder was sent
into the heart of a similar bird that had been dead
for some time previously, and it was observed, that
in the former case, the blood remained in a fluid
condition for a considerable time when in the latter
it speedily coagulated. Again, it is a well known
fact, that, when a patient dies and sectio cadaveris
is immediately performed, the blood contained in
the cardiac chambers & neighbouring blood vessels
will be found to coagulate much sooner than in
the more distant or peripheral set of vessels. Hence
the induction from these statements is that the
blood has a natural tendency to coagulate, but that this disposition is prevented by the counteracting influence of the healthy tissues, that during life there exists an intimate, vital, reciprocal relationship or intercommunication between the blood and living tissues, and anything that will interfere with or alter these vital phenomena is liable to promote or induce a coagulation of the blood from a deposition of its fibrinous element. This alteration is produced in either of the following ways: first, by an abnormal condition of the blood; or second, by an abnormal condition of the tissues, so that these pearly excrecences owe their origin to both of these causes. That the fibrinous constituent of the blood in acute rheumatism is present in abnormal proportions we have already stated, but to what cause is this excessive increase of the fibrin ascribed seems at present an undecided question; although Andral's observations are entertained by eminent pathologists as the most probable if not the true explanation of the case. C. F. Shackleton in his essay on the blood mentions the direct conversion of albumen as a supposed source of the increase of fibrin, but one great objection to this is that the former is not necessarily decreased
notwithstanding the rapid increase of the latter, and
presuming the albumen had decreased in amount, the
fact of its being diminished could be otherwise read-
ily accounted for. G. Kummermann advanced
a different theory, by endeavouring to prove that fi-
brous is an effete product in the blood during a state
of health, and that its increased proportion depends
upon the greater amount of waste which the tissues
of the body undergo during the progress of the in-
flammatory fever, but we find this statement entirely
false when compared to the facts observed in a case
of typhoid fever, for instead of finding an increase
or excess of fibrin, the blood actually presents a dimin-
ution in the proportion of that element. Professor
Vicrow regarded the increase of fibrin as due to the
production of a fibrinogenous substance exuded in the
inflamed part, which however is not true fibrin until
it becomes absorbed into the circulation & oxygenated
in the lungs. But when the tissues in the vicin-
ity of an inflamed joint are swollen by the infil-
tration of a thin serous exudation whenever the
inflammatory process subsides, the swelling rapio-
ously diminishes & ultimately disappears without leav-
ing a trace of its existence, which transition is
incompatible with the exudation of a fibrinogenous substance. The only true explanation which appears satisfactory to account for the excess of fibrin in patients of the arthritic diathesis as well as inflammatory attacks in general, is the conversion of the coloured blood discs into that material by their solution in inflammatory blood, possessing apparently a solvent property for disintegrating these corpuscles to a much greater extent than the same fluid in its normal condition. That the greater solubility of the red corpuscles is the chief source of the increased fibrin is confirmed by observing the striking disproportion between them during a healthy and diseased (inflammatory) state of the blood; for when the fibrinous constituent is present in an abnormal degree the blood discs become proportionately diminished, & vice versa, thus proving the transformation of the one into the other. Again an abnormal condition of the tissues may induce a morbid tendency of the fibrin of the blood to coagulate and separate as a distinct deposit, which morbid products we have already seen occur most frequently on the valvular apparatus as the result of rheumatic endocarditis. The lining membrane of the heart from which these vegetations usually originate had in almost
every preparation that came under my observation all
the characteristic structural appearances of inflammation;
and the modus operandi of this diseased action-in
the production of fibrous concretions. I formerly noticed
and the inference-deducible from such remissions seems
(to my mind-at least) perfectly legitimate and satis-
factory, viz. that in consequence of the valves being the
seats of an inflammatory process, their vitality is very
much diminished, or even impaired when in a state
of mere irritation so as to cause more especially when
associated with a hyperplastic condition of the blood,
the deposition and formation of organised fleshy ex-
crescences. As under these theories and hypotheses ad
infinitum have been from time to time advanced and
propounded by as many authors, on the causation of
the coagulation of the fibrous portion of the blood
during life, any attempt to enumerate much less to
enter into any lengthened disquisition upon the merits
or demerits of such supervacuous suppositions would
not only be a digression from the immediate subject of
this paper, but labour that would have a premature
abortive termination. Therefore I shall only briefly
allude to two of the more recent arguments brought
forward by Prof. Virchow & Dr. W. Richardson.
Virchow contends that the fibrinous exudation (which substance does not appear to be present at all) produced by rheumatic inflammation is deficient in oxygen and does not exhibit in consequence any tendency to coagulate until it becomes exposed to the influence of atmospheric air, when it manifests coagulating properties or the power of spontaneous coagulation. That this doctrine is fallacious in the extreme is sufficiently evident from the fact that we have no conclusive or demonstrative proof of this deficiency of oxygen in such fibrinous products but on the contrary it would appear that they are freely supplied with that aerial element being the "sundae" of a protein substance, and the presence of oxygen is not absolutely essential for the accomplishment of fibrinous coagulation, as it can be gradually produced when brought in contact with any of the simple gases, or more speedily even in vacuo; still I am inclined to entertain the opinion that the blood in this abnormal condition undergoes such a chemical transformation during the process of aeration that the elementary constituents of certain compounds become exchanged or decomposed so as to form the component parts of new combinations, which so modify
the existing relations between the fluid and solid portions of the blood as to lead to the deposition of fibrin.

Dr. Richardson from experimental inquiry into the chemical agencies influencing the coagulation of the blood, attributes the fluidity of blood in the living subject to the presence of an alkaline volatile principle ammonia, which chemical agent seems to possess in an eminent degree the property of dissolving or effecting the solution of fibrin, and that by the elimination of this volatile solvent from the blood, the fibrin which had been previously held in solution is transformed from the fluid to the solid condition. And so this distinguished physiologist promulges and advocates this remarkable and important pathological induction, that the fibrin of the blood is held in solution by a volatile solvent being mechanically kept in contact with the circulating fluid through the instrumentality of the living tissues and not depending upon a vital force emanating from the or from that internal surface being the seat of inflammation and subsequent fibrous deposits, which some modern pathologists consider as destitute of experimental proof and probability. But if such be the case, what are we to infer from the experimental observations I have formerly quoted? Why do such fe
rinos granulations so frequently located on the valves of
the heart, and, especially, if such valves be lacerated as the
result of disordered action, why do similar organized masses
form around their margins? or how did analogous produ-
tions occur in Dr. Hope's experiment on the ass? But
although it has been shown that some alkaline
volatile principle resides in living blood, which partly
accounts for its fluidity, it still remains a subject for
future investigation to determine how far this fluid
condition of the fibrin depends upon such a chemical
agency, and if such a volatile solvent be ammonia? The
practical deduction from the preceding lines of argu-
ment both vital and chemical points to the probabili-
ity of both exercising a healthy action on that fluid;
probably a more correct explanation of the problem
might blend them harmoniously into one. It has
been repeatedly observed that in rheumatic endocar-
ditis, the left side of the heart is almost always the chief
seat of the disease, and this fact has originated the idea
that some morbid material exists in the circulation
which is hyperaerogenated in the lungs, and so converta
into an blood poison whose irritating properties excite
inflammation of the endocardium. By neutralizing
the alkalinity of the blood, destroy the mutual attrac
ive influences which suspend the fibrin in solution, consequently leading to its precipitation. Whether this acid be lactic acid or some other acid of an analogous character that constitutes the rheumatism-producing poison requires further experimental proof, but, that such an acid-quantity of the blood may be propagated by any imperfect excretions of the amylopectous or saccharine materials of the food would corroborate the production of lactic acid in the pulmonic system, if such could be eventually proved.

Physical Signs

In describing the various phenomena observed in the morbid anatomy of the heart when the seat of acute rheumatic endocarditis, as well as the more recent hypothetical doctrines advanced in attempting to determine the causation of these pathological appearances, I omitted to mention Prof. List's explanation of the production of such morbid products on the valvular appendages. He supposes that in consequence of the diseased conditions of the valves, the amnionia present in that portion of the blood which comes in contact with their surface is rapidly eliminated through the impaired textures and by permitting the fibrin to become deposited in exceedingly minute gran
rules which constitute the most important structural changes in the lining membrane of the heart. I now pass on to the consideration of the physical signs of rheumatic endocarditis as revealed by a stethoscope examination of the chest, and will notice I avail myself of the important assistance afforded by the other means employed in physical diagnosis during the course of the following remarks. Dr. Stokes records, in these words, what appears to his estimation to be the object of a careful stethoscopic examination: 'It too often happens when the existence of a valvular disease is determined, that great labour is expended in ascertaining the exact site and nature of the affection. Long and careful examinations are made to determine whether the disease exists at the right or left side of the heart, whether it be a lesion of the mitral, tricuspid, or the semilunar valves; a contraction or dilatation; an ossification, a permanent lumen, or a warty excrescence. Now, though in some cases these questions may be resolved with considerable accuracy, it is also true that in a large number their determination is of comparatively trifling importance! I therefore propose to enquire into this mode of physical diagnosis in accordance with the spirit which pervades the commencing portion of
this paragraph, but view in an entirely different aspect the vital importance attached to the position of a murmur, as I shall subsequently endeavour to explain, from the concluding remarks of the above observation. The most frequent structural transformations resulting from endocarditis have been already considered, and may be conveniently arranged under the four following heads: 1. A thickened condition of the valves. 2. A deposition of fibrin on the inflamed valves. 3. Ulceration of the margins of the valves. 4. Ulceration of the chordae tendineae. From these marked productions spring incompetency of the valves, a roughening of their surface, a narrowing of the valvular orifice and consequently an obstruction to the circulation or a dilatation of these openings, and more or less regurgitation. It is quite evident, therefore, that the current of blood in its onward passage through these abnormal apertures and cavities of the heart will be more or less interfered with, the particles of which in their normal relation glide noiselessly along, are violently impinged against each other and the valvular textures, so as to elicit sensuous vibrations which sound as synonymous with cardiac murmurs. Such acoustic phenomena vary
wieldy in character as regards their quality and intensity, according to the particular lesion from which they originate, and the relative proportion between the obstructing and the propelling powers, as well as the density of the medium through which they are transmitted. Consequently, although we find the endocardial friction usually assume a soft blowing character, they are not unfrequently rough and rasping, shrill and musical in their tone; yet, if the muscular walls of the heart be in an hypertrophied condition, and exerting violent and forcible contractions in order to overcome some organic obstruction, the sound, which otherwise would go to the ear with its harshness and intensity, is now only faintly audible, one of a subdued muffled quality. Again, if the cavities of the cardiac chambers be unusually atrophied, dilated cavities and large orifices, the normal sounds of the heart in such a case would be abnormally clear and a murmur would be heard with great distinctness and intensity, but supposing that some organ to contract with great force, while the mechanical impediment to the circulation continued to exist, such a murmur would cease to be audible. In the one case, the flow of blood is forcibly in-
pelled along a contracted orifice, whereas the same column of blood driven less forcibly through the same aperture in the other produces no manifest or tangible phenomenon. Hence it is that the well-informed medical man does not attach much importance to the mere loudness or volume of sound as an indication of the amount of endocardial mischief present, but by attending to the seat of maximum intensity of the murmur to the direction in which it is propagated, the period of the heart's action with which it is synchronous, the character of the pulse, along with the duration of the disease and the collateral circumstances of the case, will infallibly determine the nature, situation, and in most cases the probable termination of the malady. I have previously mentioned that the left side of the heart was almost invariably the site of rheumatic endocarditis, and that when the right side became involved in the diseased action, it was merely in a secondary form and not the primary or original source of morbid production; and it is by viewing the subject in this light that we intuitionally enter short into a disposition of the stethoscopic physical signs which enable us to discriminate the various forms of endocardial murmurs along with the
cause or lesion upon which they depend. During the
systole of the heart, the ventricle contracts upon the
blood contained within its cavity, and forces it along
the aorta, but a portion of it may regurgitate through
the auricular orifice at the same time, hence a brief
simultaneous with the (left) ventricular systole may
either depend upon aortic obstruction or upon an au-
ricular regurgitant murmur. An aortic murmur
of obstruction is heard contemporaneous with the first
cardiac sound and is usually a prolonged hollow mur-
mur, most distinctly audible over the base of the heart
and between the second and third right costal carti-
lage as well as along the systemic circulation. A pe-
culiar thrill is sometimes felt over the cardiac region
perceptible in some of the large vessels in cases of
old standing, and the pulse is seldom modified an-
left the obstruction at the aortic opening is great
when there results a weak and feeble through reg-
ular pulse. The obstruction depends upon a thick-
ening and glueing together of the semilunar valves
or upon dense fibrous vegetations around their
margins, which impair their natural elasticity so
as to impede the onward arterial current. This form
of valvular lesion, in its early stage, does not produc
much distress until it leads to an hypertrophy of the left ventricle when the disease becomes much more dangerous. A mitral regurgitant murmur is a bruit produced by a reflex current through the auriculo-ventricular opening and corresponding to the heart's systole or immediately following it. This murmur is generally of a soft, blowing character and attains its maximum intensity over the left apex of the heart. The retrograde current into the left auricle leads to its dilatation with a slight thickening of its walls in a very few instances, but never amounting to a true hypertrophy. The pulmonic circulation is retarded in its progress towards the heart, causing a permanently congested and engorged condition of the lungs and ultimately leading to a dilated and hypertrophied right ventricle. When such a state of matters exists, it is quite evident how this form of valvular affection should become complicated with haemoptysis, the result of pulmonary sanguineous extravasation, dyspnoea, orthopnoea, cough, dyspnoea &c. By adjusting the hand over the praecordial region a distinct fremitus or purring tremor (Laennec's frissonnement cutaire) is felt in most cases of mitral insufficiency and is a pathognomonic symptom of the disease, from its not being trans-
mitted along the arterial circulation. In consequence of the variable amount of blood which recedes at each ventricular systole along the patent mitral opening, the pulse feels small, feeble and very irregular, and often intermitt by two or three cardiac impulses succeeding rapidly each other and corresponding to only one radial pulsation. The valves may become incompetent from a permanent thickening of their edges and from the presence of fibrous deposits, which here found associated with mitral obstruction as well as regurgitation, ulceration of the margins of the valves or of the chordae tendineae without an auricular systolic murmur and lastly from a simple dilatation or a more mechanical displacement of the valvular curtains of the auricular orifice. This form of cardiac disease proves more rapidly fatal than the preceding valvular affection, but is certainly not so likely to terminate abruptly by a sudden death as the analogous complication of the aorta.

Again, when a cardiac bruit is simultaneous with the second sound or the hearts diastole, it is at once apparent that it originates from the entrance of blood into the ventricle, and consequent by the result of aortic incompetency a mitral obstrucion. An auricular systolic murmur is a
bruit of mitral obstruction, and always occurs immediately before the first sound of the heart. It is most distinctly heard about the fourth left interspace and towards the heart's apex, and in most cases of mitral obstruction, there exists a patency of the bicuspid valves which permits of a backward current into the auricle or a mitral murmur of regurgitation, but in many instances where a regurgitant murmur is heard, one or no presystolic sound can be detected by physical diagnosis, still a constriction of the auriculo-ventricular aperture may be present, although the auricular contraction were so feeble and the auricular current so few that sonorous vibrations were not produced.

The chief point to be kept in view regarding the existence of mitral murmurs whether distinctive or regurgitant, is this viz. their relation to the first normal cardiac sound, if the bruit immediately precedes this sound then a contracted mitral orifice or obstruction will be ascertained to exist, but a murmur produced by a leakage of the bicuspid valves is either synchronous with the first sound or immediately follows it. The symptoms of mitral obstruction are similar to those I have mentioned in connection with a well marked instance of mitral regurgi...
gitation. According to the amount of obstruction to the arterial circulation will be the severity of the attendant symptoms and the important structural changes that result as secondary complications, the left auricle dilates and its lining membrane may become slightly thickened but the muscular tectures do not become hypertrophied; the pulmonic circuit gets engorged; and the right ventricle may become so dilated and hypertrophied that the whole anterior surface of the heart is occupied by the enlarged muscular cavity which can be observed pulsating freely in the epigastric region. As the obstructive effects such morbid enlargement in the right ventricle a tricuspid murmur of regurgitation may be discovered over the lower margins of percussion dulness near the ensiform cartilage which hitherto is the result of a dilated tricuspid. But while such structural transformations are operating in the right chambers of the heart, the left ventricle is undergoing an entirely opposite metamorphoses, since the usual & normal quantity of blood is prevented from reaching that ventricular cavity it becomes less active and energetic in its muscular contractions and the consequence is that its muscular parietes get atrophied and enfeebled
and the whole chamber is diminished in its dimensions so that the entire organ appears, as it were, to have rotated or twisted on its own axis from the left apex receding behind the heart, whereas the apparent dislocation is simply owing to the hypertrophy and dilatation of the right with a corresponding atrophy and diminution of the left ventricle. The pulse from the impaired ventricular action and the limited supply of blood sent along the systemic circulation, notwithstanding the violent impulsive movements of the right ventricle and the frequent though powerless contractions of the left, will be found weak and thready though not necessarily irregular. The pathological alterations of structure which constitute these organic obstructions are inflammatory adhesions of the mitral valves and adjoining chordae tendineae with deposits of fibrin and in cases of old standing an ossified aperture of a button-hole appearance is not unfrequently met with. This form of cardiac disease is in most instances a deadly affection, and as rapid in its termination as it is mortal. An aortic murmur of regurgitation corresponds to the second cardiac sound and frequently replaces that normal sound as well as occupying more or less of the period of rest between
the two cardiac sounds. Generally in such cases there exists a ventricular-audible obstructive murmur, and upon placing the stethoscope over the base of the heart a double blowing murmur is audible, corresponding with the ventricular systole and diastole. This fruit de soufflet is distinctly marked along the great blood vessels of the neck and extremities, along the whole extent of the sternum, and the regurgitant murmur with special distinctness towards the aortic cartilage and with less intensity over the left apex. The characteristic murmur is that depending on aortic regurgitation, and originates from a permanent insufficiency of the semilunar valves, thus allowing a reflux current into the ventricle, which produces at no distant date a dilatation of its cavity with an hypertrophy of its walls. This enlargement of the organ further tends to increase the inadequate closure of the arterial opening, and hence arises from the augmented action of the left ventricle and the dilated though incompetent aortic orifice, a most remarkable visible pulsation in the large arterial trunks, with a well fruit de soufflet, and a rushing thrill communicated to the fingers analogous to an electric shock resulting from the retrograde current into the ventricular long
A characteristic case of a double aortic murmur came under my observation lately, which well illustrates the physical signs and general symptoms of this form of vascular disease.

William Brown, aged 22, a printer, case man. From his history, symptoms of cardiac disease referable to acute rheumatism have had two attacks of rheumatic fever, the first one most severe about two years ago. For the last six months, complains of palpitation, and pain over region of the heart, left shoulder, and across mamillary sternum. The patient appears of a pale anaemic cast, and slender built frame, exceedingly nervous, and agitated by the slightest exertion. His countenance betrays marked angina-like suffering. The chest presents the appearance of one bearing on fulminating tumors. The cardiac motions of the diffused apex have been observed between the sixth and seventh left ribs and extending for fully an inch beyond the line of the nipple. The heart is enormously enlarged. Measurement of percussion dulness transversely at nipple five inches dulness upwards along the sternal width confined to the margins of that bone until mamillary sternum is reached when it extends for a slight distance on either side of it. By palpation a most distinct premitus perceived over cardiac dulness, and the shock or stroke emanating from the
brachial artery is easily transmitted through a column of six hands; a marked pulsation is felt in the jugular fossa and distinct visibility of superficial arterial trunks from the temporal arteries of the head to dorsal arteries of the foot. A double blowing murmur audible over the base of the heart and particularly over the manubrium of sternum at its junction with second right costal cartilage, also very distinct at ensiform cartilage, and second sound pretty well marked over the heart's apex. The first sound or murmur is faintly heard over the apex, but very loud in all the other localities mentioned and the 'but de soufflet' is heard wherever there is a visible artery. The ascending portion of the arch of the aorta is extremely dilated and might easily be mistaken for an aneurismal sac. The patient once spat blood although no mitral physical signs could be detected. Fibrinous vegetations are probably the organic mischief which has rendered the papillary valves incompetent for the efficient closure of the arterial orifice.

The morbid conditions which may produce the above phenomena are inflammatory products on the surface of the valves, with a subsequent thickening and contraction of their tissue-nucleation and rupture of their substance. The prognosis here is
most unfavourable, and is certainly the most likely of all the cardiac vascular lesions to terminate suddenly in death, and probably on this account, that the hypertrophied muscular walls undergo fatty degeneration which so enfeebles their vital contractility that upon some slight excitement of the circulation, the diseased organ receives a stimulus which it is incapable of counteracting, becomes suddenly paralyzed, and results in most fatal syncope. Such cases of auricular disease are often attended by agonizing cerebral symptoms such as sleeplessness, and a sensation of approaching dissolution.

Another case of auricular insufficiency that I had an opportunity of observing.

John Gillies, age 46. Had an attack of acute rheumatism about ten years ago while living in England, and was successfully treated for his complaint in the Durham Infirmary. About four months ago was suddenly attacked by a sense of giddiness in the head, followed subsequently by a fit of insensibility, after regaining consciousness, discovered that he had been deprived of the power of speech and his one side of the body completely paralyzed. He now suffers from paroxysmal attacks of palpitation, heartburns, and pain over the ensiform cartilage and sternum with a feel
Acute rheumatism is liable to attack individuals between the ages of fifteen and thirty-five or forty years of age, but children are sometimes subject to it, and the mitral valve in their case is the more frequent seat of disease than the semilunar, whereas in adults both valves are equally liable to be affected. Dr. Watson states that children suffer from acute rheumatism so early as the third or fourth year, and that in such young patients cardiac affections are most apt to become developed. A case illustrative of this statement occurred recently in the person of a young boy, John Trask, aged 8 years, a pale anemic-looking boy, who suffered from severe rheumatic pains about twelve months ago. When the hand is placed over the heart's apex a distinct presystolic thrill is felt, occurring immediately before the impulse of that organ. Cardiac dulness considerably increased, extending from the upper margin of the left costal cartilage to one-fifth cartilage, transversely, from middle of sternum to a little within left nipple. On applying the stethoscope over the apex of the heart between the fifth and sixth left costal cartilages a distinct presystolic murmur is heard, and over the base of the aorta, a systolic murmur with the first sound, along with an accentuated second sound. The pulse is somewhat feeble though regular and breathing quite tran-
ing of fluttering at his heart. The patient is rather anemic in appearance; pulse, full and jerking though regular in rhythm, on percussion cardiac dullness slightly increased, especially in the transverse direction. By auscultation a single murmur following the first sound is heard in greatest intensity over the apex of the heart, while over its base—a little to the right of junction of third left costal cartilage with sternum—a double murmur is detected, the first is a soft systolic burst of a blowing character; but the second sound is accompanied by a very distinct musical murmur. These murmurs are the result of an elongated vegetation which, by its attachment to the anterior valve, hangs freely in the circulating current, and produces somnolent vibrations which assume a musical character. And the fit of unconsciousness with the existing paralysis is accounted for by the presence of an embolism followed by strophic softening of the substance of the brain.

By thus taking into consideration the diagnostic physical signs and concomitant symptoms of each individual valvular lesion, the physician can generally ascertain the true nature of the disease, and should he fail sometimes in accomplishing a cure, still he can in most instances alleviate human suffering and prolong life.
gall when confined to the recumbent posture, has
lead no dysmictosis, or any other complication.
Here there exists a contraction of the mitral orifice
most probably the result of abnormal fibrous deposits
and the semilunar valves are similarly affected while
the accentuated second sound depends upon an
hypertrophy and dilatation of the right ventri-
acle.