The Pathology of Endocarditis and Valvular disease of the Heart

by

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An excellent thesis, marked by accurate acquaintance with intricate questions.

Mark of inferiority.
The Pathology of Endocarditis and Valvular disease of the heart

Endocarditis

Endocarditis is the term employed to denote inflammation of the lining membrane of the heart. It occurs, though very rarely, as an idio-pathic affection, less often secondary to pericarditis from whatever cause this may originate, but by far its most common cause is Acute Rheumatism. Of 136 cases of the latter disease which came under Dr. Latham's notice he found that the heart was affected in 90. It has been a question whether endo- or pericarditis is the most common result of this affection. In the 90 cases before referred to in which the heart was affected, Latham states that 63 consisted of simple endocardiditis and only 1 of pericarditis, while there were 11 cases in which both existed. Of 474 cases collected from
Various sources by Fuller, endocarditis existed in 214 or in the ratio of 1 in every 2.25 cases of 204 of Rheumatic heart disease complicated of some kind. Endocarditis was present in 138 cases, pericarditis in 19, and endo-pericarditis in 38. As a result of these joint observations we find that in 294 cases of acute rheumatism with cardiac complication, endocarditis existed in 201 instances or in the ratio of 1 in 146 cases, pericarditis in 26 or 1 in 11.3 cases and endo-pericarditis in 49 or 1 in 6 cases. These results are not in accordance with the views of Dr. Holmes. He states that they occur in the following order of frequency:

1. Acute pericarditis with endocarditis
2. Acute pericarditis without endocarditis
3. Endocarditis without pericarditis

He makes this statement on the ground that pericarditis often remains a latent disease and cannot be detected by physical signs. This no doubt may happen in a small number of instances but we cannot suppose that it should do so sufficiently frequently to modify very great degree the results of Dr. Lathams and Fuller's statistics. What is the nature of the case -
region between Acute Rheumatism and Endocarditis? The former consists essentially of an inflammation of the fibrous tissue, particularly that connected with the joints. Now the lining membrane of the heart contains fibrous tissue, and it was formerly supposed that Endocarditis was a metastasis of the inflammation from the joints to the heart. But we cannot regard it in this light when the heart becomes affected the joints do not cease to be so, and sometimes the cardiac affection precedes that of the joints. We may more reasonably suppose that the two structures are affected by community of tissue.

The younger the patient is who labours under Ac. rheumatism, the greater is the tendency to the development of Endocarditis, in fact the exception to the rule seems to be rare, that almost all under the age of puberty persons affected with Ac. rheumatism, when the accompanying fever is great, will become the subjects of rheumatic carditis. It may be developed at any period of the rheumatic affection but the 15 Fuller's statistics would seem to indicate that the liability is greatest between the seventh and twentieth days of the disease.
and surfaces of the heart are particularly liable to be affected, this will probably depend on the greater quantity of fibrous tissue which exists in them.

Endo-carditis not unfrequently also supervenes on Bright's disease.

Symptoms & Physical signs.

These when well marked are those of an inflammatory affection, referrible to the Cardiac region; they are very much the same as those of peri-carditis. There is inflammatory fever, when the disease however occurs in connection with Rheumatism, this cannot be distinguished from the fever attending the latter disease. There is uneasiness about the Cardiac region, an anxious expression of countenance; the patient complains of a fulness or of a weight about his heart; its impulse is felt over a greater extent than it ought to be. Brill land thinks that this latter is in proportion to the inflammatory turgescence of the organ; but Hoke is of opinion that it is caused by the violent and abrupt action consequent on the inflammatory irritation of the organ. The importance attached to these symptoms
is greatest when they occur in the course of an
atherosclerotic. Very frequently however as far as
symptoms are concerned endocarditis remains a
latent disease; and often the only diagnostic indica-
tion is the physical sign to be spoken of here-
after. Should the circulation become impaired either
from a swollen state of the valves, or regurgitation,
or from congestion of blood in the cavities interfe-
ring with its free flow, the symptoms become
very much aggravated. The heart's action becomes
very irregular, the beats are unequal, quick and
intermitting, whilst the pulse is weak, small and
irregular, and sometimes beats are wanting in the
pulse which exists in the heart. There is deficient
circulation from the obstruction impeding the flow
of blood into the arteries. The free return of blood
from the lungs is also prevented by the same cause,
giving rise to a feeling of unhanding suffocation.
Delirium is very frequently present. Braubach
relates 2 cases in which these symptoms occurred.
Rupture of the chordae tendineae sometimes takes
place in the advanced stage of the disease. Stokes
thinks that this is most likely to occur when
endocarditis attacks a previously hypertrophied
heart. It has been said that the cardiac dul-
sef may be increased in endocarditis, thus increasing
action of the heart causing a greater portion of the
organ to come into contact with the walls of the
chest, and also from distension of its cavity by
blood. These must be only a very slight ef-
fect however in causing increase of the cardiac dul-
sef, and in those cases when it has been observed it
is more likely to have depended either on a previously
hypertrophied heart, or on a coexisting pericarditis
the presence of which had been overlooked. But
the great point in the diagnosis of endocarditis is
the existence of an endocardial murmur developed
generally at an early period of the disease. That
in diagnosis this should be of any value, of course we must
make sure that it does not depend upon old standing
disease, it must be a recent murmur. If it occurs
for the first time during the course of an attack of
acute rheumatism with any of the symptoms familiar
mentioned we may make ourselves almost certain
that it depends on endocarditis. Yet there is a
flaw of fallacy. The murmur may arise not
from any organic but from a functional cause.
Long continued exhaustion from disease may give
rise to a change in the blood itself causing the development of a murmur in all respects, nearly the same as that arising from organic disease of the heart. This however generally occurs late in the disease while the murmur arising from endocarditis is most commonly developed early. But ordinarily the greatest difficulty is to determine that a murmur is recent and to make certain of this it must be developed under careful observation. When called to a case of acute rheumatism we anxiously examine the heart. We may find no murmur, but on auscultating from day to day a murmur becomes developed. This is a case where we may be nearly certain though not absolutely so that the heart has become affected. But another case presents itself. We may not be called to the patient at the beginning of the disease and the murmur may have had time to be developed before we see him. How then are we to determine whether or not it is recent? Here we must exercise our judgement. We must find out if any of the other symptoms of endocarditis are present. We must ascertain if the patient has had acute rheumatism before.
Whether he was ever troubled with palpitation, or
darkness of breathing, or uneasiness about the
heart, previous to the present disease. Moreover
Certain characters have been laid down as exciting in
the murmur itself, by which we may guess
whether or not it is recent. It is more likely
to be recent then is softness of the blowing quality
lowness of pitch, or systolic rhythm or that which
would indicate Aortic obstruction or Mitral
Regurgitation; and as it will more probably be old
standing if then roughness of quality, high
pitch and diastolic rhythm.

Walsh, from his own observation, arranges
the murmurs of purely acute Endocarditis in
the following order of frequency 1 st Aortic Ob-
structive, 2 nd Mitral regurgitant, 3 rd Aortic Ne-
regurgitant, 4 th Aortic Obstructive and Mitral Ne-
regurgitant together and 5 th Aortic Obstructive
& Regurgitant together. He states that he has never
observed Acute obstructive Mitral murmur nor acute
tricuspid Regurgitant. The murmur is supposed to
be caused by the roughness of the Endocardium
covering the valves arising from deposits of Lymp
or fibrin.
But another question arises. Is the murmur invariable present in endocarditis? And this I suspect must be answered in the negative. Dr. Flinth of U.S. mentions a case in which it existed for one day only, careful auscultation failing to detect it the day previous and subsequently. This he attributes to the deposits of fibrin or lymph causing the murmur, having been washed away by the current of blood. Dr. Stokes relates a case in which the murmur occurred for a short period only during the progress of the disease, and he says that he has seen other cases which could not be explained upon any hypothesis except the absence of the murmur in endocarditis. Maldabe also says that he has seen distinct patchy redness with thin films of lymph on the anterior endocardium where there had been recent excitation of the heart without a newly developed murmur.

In extra auricular life endocarditis rarely occurs except on the left side of the heart. Several theories have been brought forward to account for this. Some thought that it was due to the greater amount of fibrinous tissue that exerts in the endocardium of
Je Morgan, his affectionately yours from the heart of Lincoln or James Lincoln.
the left side. Others that it was due to a greater stimulation of the left side by arterial blood, without specifying the nature of the stimulation. Dr. Richardson's explanation appears to be the most satisfactory. He injected into the peritoneal cavity of a dog a solution containing 10 per cent of lactic acid. The liquid became absorbed and he found that, in about 12 hours after he had made the experiment, there were signs of endocarditis. He performed various experiments of a similar nature and he always found that it was the right side of the heart which was affected, the peritoneum itself escaping injury. His explanation is that the lactic acid is taken up by the veins and goes into contact with the endocardium of the right side, irritating it and causing inflammation in the pulmonary circulation it undergoes some loss and is thus incapable of acting on the left side; in other words, in so far as the heart is concerned, the process is derived from the systemic current and is lost in the pulmonary. Now how does this explain the occurrence of Rheumatic Endocarditis on the left side? Dr. Richardson supposes that the chemical change whereby the
materies morbi of Acute Rheumatism is produced in the pulmonic circuit, that it consequently comes first into contact with the endocardium of the left side, causing inflammation there, and then it is decomposed or eliminated before reaching the systemic side so that it does not reach the right side of the heart.

Endocarditis rarely proves fatal in the acute stage. In Mathews' 63 cases formerly reported, none died in the acute stage; in 17 of them the endocardium regained its complete integrity, in the remaining 46 it was permanently injured. McDade says that he has never known the murmur of endocarditis entirely to cease after it has once been heard.
Valvular disease of the heart

Nature and Causes

Valvular lesions are commonly limited to the left side of the heart, the right being very rarely affected after birth. The disease may be of two kinds - obstructive, from constriction of the orifice, or some other cause, producing an impediment to the free onward flow of the blood, or regurgitant, from insufficiency of the valves allowing regurgitation of the blood backwards. Obstruction may exist without insufficiency and vice versa, but very frequently both are united. The valves generally affected are the mitral and aortic. We may have the two kinds of disease in all forms of combination, there may
visiting together, aortic obstruction & regurgitation, or Mitral obstruction & regurgitation, or as is sometimes the case, there may be aortic obstruction with Mitral regurgitation.

Adhesions may be formed between two values most commonly the aortic semilunar as the result of Endocarditis. If the partition between the valves thus united should be absorbed, and they thus converted into one, it would be very difficult to say at an examination after death, whether this was the result of disease or congenital malformation. If adhesions of the valves to each other or to the walls take place, I must prove an impairment to the free discharge of blood from the heart and must also allow regurgitation into the ventricle from imperfect closure during the diastole. According to Rehnitsky there are three varieties of valvular lesions, the result of Endocarditis, 1st. ossification of an eccentric deposit into the interior of the value. 2nd. ossification of endocardial deposit on the surface of the value, roughness of the endocardial membrane produces a tendency to the deposit of fibrin on its surface. This is proved by Simon's experiment of passing
a thread through an artery, the fibrin coagulates and adheres to the thread, presenting an appearance somewhat like the vegetations observed on the valves. It is supposed that their production is favoured by a superabundance of fibrin in the blood, as is the case during an attack of Acute Rheumatism. This deposit originally fibrinous may become in the course of time calcareous from the elimination of certain of its constituents and the deposition of others. Hence a gelatinous softening of their substance rendering them weak and liable to rupture. The valves of the heart may become atrophied and undergo atrophy, they become attenuated, their transparency is increased and their fiber perforations in their substance which may be so numerous as to give them a creniform appearance.

Vascular lesions may give rise either to obstruction or regurgitation or to both. Thus the presence of weakly constrictions in the valves will prevent their due apposition when closing and allow regurgitation, while at the same time some degree of obstruction will also be produced. Those which have undergone atrophy or gelatinous
Softening will also allow of regurgitation pro-
progressive to the extent of the lesion. The osse-
ification of the three semilunar valves together
is a common cause of aortic obstruction, this is
sometimes so great that the opening will scarcely
allow an ordinary quill to pass through. Re-

gurgitation may take place without any loss of
the valves at all, as when one of the orifices
becomes dilated and the valves remaining of their for-
mer size become insufficient to close the en-
larged orifice. This is a very common cause in cases
of mitral obstruction when the right cavities of the
heart have become dilated.

There can be no doubt that endocarditis is
the most common cause of valvular lesions.
Clinical observation shows that the great ma-
-jority of patients affected with them, have had
at some period or other of their lives, an attack
of acute rheumatism, a disease which we have
seen to be very frequently complicated with
endocarditis. But inflammation is not the sole
cause of the disease, ossification of the valves
may take place in advanced life independently
of endocarditis, as it does in the arterial sys-
Symptoms + Pathological Effects

On the Heart.

Lesions affecting the valves or orifice of the heart have been to be of a nature either to produce obstruction or admit of regurgitation. Let us see what effects would be produced on the heart itself by obstruction of the aortic orifice. In consequence of the obstruction, a greater force proportionate to the amount of obstruction would be required to be exercised by the left ventricle to propel the blood into the systemic vessels. This would cause hyper trophy of the left ventricle; then would follow dilatation of the left auricle, congestion of the lungs, succeeded by hypertrophy or dilatation of the right side of the heart. In mitral obstruction the left ventricle does not become dilated or hypertrophied but the right side of the heart is much more involved than in the corresponding lesion of the aortic orifice. Generally obstruction will produce hypertrophy while dilatation...
Habits
Regurgitation will cause dilatation. The
hyper trophy is commonly proportionate to
the amount of obstruction but this is not in
variably the case. Instances have been
observed where there has been enormous en-
largement while the contraction or insuf-
ficiency was small; while the size of the
heart has been observed to be only very slight-
ly increased where the amount of obstruc-
tion was very great. We know however
that the condition of the blood has a good
deal to do with the production of hyper-
trophy of the heart. When any of its el-
ements destined for execution are not eliminated
in consequence of disease of the eliminating
organs, as in the case with area or Bright's
disease of the Kidney, and the latter is not un-
concerned Concomitant of Cardiac disease,
the proper attraction does not exist between
the tissues and the blood. Greater force is
accordingly required to propel it through
the vessels and we find that in such cases
hyper trophy of the left ventricle takes place.
If Bronchitis or Emphysema should co-exist
with Cardiac disease, it also will be an
additional Cause of Hypertrophy affecting
the right side as there will be an obstruction
both in the lungs & heart to overcome.
Paris

Pain is not a common symptom in valvular
disease. There is however frequently a sense
of constriction uneasiness or undefinable
distress. The patient often cannot sleep
or rather he is afraid to do so, for some
patients say they have a sort of impression
that if they should fall asleep they will
never awake again. When they do get sleep
they sometimes suddenly start up in the
midst of it with a feeling of impending
dissolution. Patients affected with these
symptoms have an expression of anxiety
in their countenances. They are subject to
paroxysms of acute suffering, in which
pain however does not predominate, nor
is breathlessness always present either, but
great anxiety and the feeling of impending
dissolution appear to be the predominating
symptoms.
Palpitation

This is very common in valvular disease. It is caused by the hypertrophy of the aegid causing stronger contractions and making a greater portion of the heart to come into contact with the walls of the chest. Sometimes the action is simply more powerful than it ought to be, at other times it is irregular and intermittent. This symptom may be present in some patients without they themselves being aware of it. In others it is a cause of much annoyance and suffering. Generally it does not occur until there is hypertrophy of the aegid. Palpitation however is often a functional disturbance only, and when it is so, it generally creates in the minds of patients much greater uneasiness and alarm than when it arises from organic lesion. It is a matter of importance to be able to distinguish between palpitation caused by organic disease and that arising from functional disturbance only. The following characters may assist us in coming to a conclusion.
1st. Palpitation from functional disturbances is not felt when taking exercise or when the mind is occupied. Palpitation from organic disease is not usually increased beyond its habitual amount except when there is some obvious cause and more especially when taking exercise. The former usually takes place at night, the latter in the daytime.

2nd. Palpitation from functional causes always creates in the minds of patients great uneasiness and alarm. This is not generally the case when arising from organic disease.

3rd. Functional palpitation recurs in paroxysms in the intervals of which the heart is quiet. That arising from organic disease is less quiet but to a greater or less extent constant.

The Pulse.

In our estimation of the character of vascular lesions very considerable information may be obtained by a careful inquiry into the state of the pulse. With reference to this the most important points to be noticed are
its frequency, its regularity, its fullness and strength compared with the heart's action, the character of the impulse as felt by the finger.

In Aortic Regurgitation, the radial pulse is quite characteristic. In cases where the aortic values are in a state of integrity, they are able to support the column of blood, which has been forced into the aorta by the contraction of the left ventricle, during the diastole. But if they are insufficient, a quantity corresponding to the extent of the lesion passes backwards, the recoil of the walls of the arteries producing both a backward and an inward current. When the ventricle contracts and propels a fresh quantity into the aorta, it will meet a backward instead of an inward current. Sir William was the first to point out that under these circumstances the pulse becomes of a jerking character, called by him "the Pulse of unfilled arteries." It has also been called the "Collapsing Pulse," the impression given to the finger being the sudden disappearance of the arterial wave. Another
Britz Depressiveulse and Incipient Ante Babes.
very characteristic feature in aortic regurgitation
first noticed by A. M. Ogier is the visible pul-
sation of the arteries. The collision of the
backward and inward current formerly noticed
is such as to cause the vessels to be suddenly
thrown from their bed so as sometimes to
appear “like worms under the skin wriggling
into tortuous lines at each pulse.”
In aortic obstruction the pulse is not weakened in proportion to the amount of ob-
struction when this lesion is accompanied by
hyperplasphy. The diminished quantity which
the orifice can admit at a time is nearly
compensated for by the stronger contractions
of the ventricle. But in cases where the en-
largement of the heart has ensued with
diminished muscular power the pulse may
become irregular, intermitting and weak,
where there is extreme obstruction a marked
difference may be observed between the force
of the heart’s movements in the preceding and
the strength of the pulse.
Mitral obstruction is more strongly characterized
by a want of proportion between the elevated
The Pulse may become small, weak, and irregular, and it may be less frequent than that of the heart. If the obstruction is great, it will allow only a very small supply of blood to pass into the left ventricle, which probably may be often insufficient to fill it, the right ventricle being at the same time gorged. Under these circumstances the left ventricle contracting with the right may not transmit a stream into the arteries sufficient to communicate to them a sensible pulsation, while the action of the heart (chiefly the right side) is strong and vigorous, and thus beats may be present in the heart which are absent in the pulse.

In mitral insufficiency, the pulse is weak and small in proportion to the amount of regurgitation. Here also its strength and size are in striking contrast to the action of the heart. It is generally irregular, especially in advanced stages of the disease. Tonus transverse pulsation.

These are liable to take place in all valvular
lesions of the heart. They will occur more quickly on lesions affecting the right than the left side of the heart. The obstruction to the onward flow in the right side will produce a direct impediment to the free return of venous blood from the systemic circulation. The occurrence of pulsation in the veins generally corresponds to the ventricular systole. This is produced by the contraction of the right ventricle communicating an impulse not only forward but also in a retrograde direction into the veins of the neck. This of course involves insufficiency of the tricuspid valve. It may be mistaken for arterial pulsation but can be distinguished by pressing on the vein with the finger and observing whether the pulsation continues after the vein is emptied. In lesions of the left side of the heart venous turgescence is more common in internal than anterior vessels when we observe it, we must not necessarily conclude that there is valvular disease. It may be produced by other causes, anything which will produce obstruction to the return of venous circulation as for example emphysema or
Bronchitis will sustain the return of venous blood to the right side of the heart. It may also arise from tumours within the thorax or aneurisms pressing on the superior vena cava. Venous tenderness is a very important sign in valvular disease. It indicates that the lesion upon which it depends produced an amount of obstruction which has led to enlargement of the right side of the heart, and that there is a liability to the production of dropsy and haemorrhages.

Cardiac dropsy

Dropsy may arise in either to cardiac or renal disease and very frequently when due to both combined. In cardiac disease it generally appears first in the foot and ankles, afterwards in the face and then it passes up the lower extremities. It may ultimately infiltrate the muscular tissues of the whole body constituting general anaemia. The appearance of dropsy of the face caused by cardiac disease is quite different from that caused by affection of the kidneys. In the former there is congestion of the veins giving to the face a dusky appearance while in the
latter it has a pallid aspect generally without any appearance of lividity. Cardiac dropsy is not confined to the auricular tissue; the same cavities also may become affected although all are not equally liable to its occurrence. Waldeyer says that it occurs in them in the following order of frequency: 1st peritoneal, 2nd pleural, 3rd pericardial, 4th subarachnoid, 5th subarachnoid and 6th Juneau vaginalis. Ulceration of the lungs also frequently takes place in cardiac disease but we shall speak of it as another part of this paper.

We find very frequently that the dropsy takes place out of all proportion to the degree of obstructive or regurgitative disease. In some cases when the amount of obstruction is very great there is little or no dropsy while in others where the obstruction is scarcely discernible we have it to a great extent, even although there is no accompanying disease of the kidney. In the latter cases however there is always a derangement of condition of the blood in which either there is a derangement of the solids rendering it more watery, or imperfect clini
Bledsoe's cause of D. J. T. Falls
Transferred under revenue.
vation of certain of its matters destined for excretion or as is most frequently the case both of these an combined. Indeed it may fairly be questioned whether cardiac disease ever gives rise to dropsy when the blood is in its normal condition.

Mural obstruction or regurgitation are the lesions which most commonly cause death. It very frequently presents however in long standing disease of the aortic valves also Emboliens.

The occurrence of fibrinous deposits on the lining membrane of the heart and the presence of warty excrences on the valves have been already spoken of. The fibrinous coagula are very liable to undergo spontaneous disintegration giving rise to the production of little masses which are carried by the circulatory through the arterial system, until they come to arteries not sufficiently capacious to admit their passage. The warty excrences on the valves may also be detached during life and be carried by the current of blood in the same way. They thus filling up the arteries at the
place where they are impacted and prevent the further passage of blood through the vessel. A deficiency in the nutrition of the part which the artery supplies with blood consequently takes place causing softening or gangrene. It has also been supposed that the excrescences may disintegrate and break up in a molecular form, causing the granular matter which results to mingle with the blood giving rise to symptoms like pyaemia. The occurrence of these circumstances explains those cases by no means uncommon, in which hemiplegia with or without sensory supervenes on vascular disease. In cases of hemiplegia occurring in this connection, there may generally be found a nodule impacted in one or other of the arteries of the brain, a very common plane being the right middle cerebral artery, resembling in appearance and character a fibrinous Conglobina in the interior of the heart or Warty excrescences on the valves. That obstruction of the supply of blood to one side of the brain can produce hemiplegia is known by its occasional occurrence in certain cases.
of ligation of the common carotid artery of one side. If the embolus is detached from the left side of the heart, it may be carried to any part of the systemic circulation. Emboli have been found in the right common iliac artery. They are sometimes carried to the vessels of the spleen and kidney causing death of these organs. On the other hand should emboli be detached from the right side of the heart, they will be carried into the pulmonary circulation. If they should block off any of the primary branches of the pulmonary artery, death by asphyxia might be at once produced.

Effects on the Respiratory System

Congestion of the Lungs—This when arising from diseased orifices is caused by those affecting the left side of the heart only. It is an immediate result of the obstruction to the free entry of blood from the pulmonary veins to the left auricle. This impediment is most likely to arise in contraction of the mitral orifice, and the greater the degree of contraction the more marked will the congestion become. In this case there are two causes which
which are likely to favour its occurrence
1. the obstruction at the mitral orifice
2. the hypertrophy of the right ventricle, the
   consequence of the obstruction.

The second cause will act by pumping the blood
acitewed, with greater force into the lungs.
Mural regurgitation may also lead to pul-
monary congestion, although not so readily
as obstruction does. The acute orifice being
further removed from the lungs, disease affecting
it does not so readily give rise to congestion
as when the mitral orifice is affected, hy-
pertrophy or dilatation of the left ventricle re-
curring in the first instance. Chauvel & Brelton
have stated that pulmonary congestion most
commonly takes place as the result of disease
of the right cavities of the heart. It is difficult
to see however how this can occur. Obstruction
in the orifices of the right side would hinder
the return of blood from the veins and cause
within a diminution in the amount sent to
the lungs; and hypertrophy as the only other con-
dition which possibly could give rise to it
rarely occurs except in connection with dis.
case of the left side of the heart, or in connection with disease of the lungs themselves.

Congestion of the lungs gives rise to various phenomena of an important kind. There may be extravasation of blood, the pulmonary oedema, oedema of the lungs, haemoptysis, dyspnoea, cough or pulmonary embolism may occur in two forms—diffuse or lobular. In the former the pulmonary substance appears broken down or torn by the extravasated blood, and the extravasation is not circumscribed by the lobules. In this variety the impeded blood ooze through the walls of the capillaries into the air vessels and is partly expectorated combined with mucus. In the lobular form, Dr. Watson supposes that the extravasation takes place into the bronchi, and partly, by the convulsive efforts to respire, forced back into the lobules to which the bronchi lead. It is thus distinctly limited by the lobules.

Oedema of the lungs and an uncommon effect of pulmonary congestion. It is most likely to take place when the blood is in an anaemic con-
Dyspnea. This, like intravasation, one of the causes of dyspnea, and may even, if a great amount of the lungs is involved, cause death by asphyxia. Its presence is indicated by the presence of dulness on percussion and sub-costal rales.

Dyspnea almost always occurs in the advanced stages of valvular disease. This is caused by the impediment to the circulation thru the lungs and the consequent deficiency in the aeration of the blood. Being the result of congestion it is more frequent in diseases affecting the mitral than the aortic orifice. When it is caused by aortic lesions it takes place at a later stage of the disease and is preceded by dilatation of the left ventricle. But there are cases where a very great degree of mitral and aortic contraction must have existed for a length of time without any lead symptoms whatever. Dr. Stokes narrates a case where the aortic opening exhibited the most extreme degree of contraction from ossific deposit in a man of exceedingly active habits and who lived up to a few days before his death enjoyed...
uninterruptedly good health, dyspnoea in cardiac disease generally commences with a slight shortness of breath on exertion. In some cases it is only observed during exercise, while in others it occurs in paroxysms. The patient will often wake up suddenly out of his sleep with a feeling of suffocation. He cannot remain in the recumbent posture but has to sit up bending forward with his hands grasping his knees. There is an appearance of great anxiety in his countenance and his face, especially the lips, become livid. When the dyspnoea compels the patient to remain in the sitting or erect posture it is called orthopnoea. The suffering attendant on dyspnoea depends a good deal on the rapidity with which the pulmonary congestion causing it has taken place being much more intense when rapidly-developed.

As a rule the intensity of the symptoms referable to the respiratory system is in direct proportion to the amount of pulmonary congestion. These symptoms do not generally show themselves until a comparatively late period.
in the disease. When dependent on aortic lesions they are preceded by hypertrophy of the left ventricle and when caused by mitral disease they are followed by hypertrophy of the right ventricle. Effusion into the pleural sacs on both sides may take place as a part of general dropsey, causing compression of the lungs and producing an aggravation of the dyspneea proportionate to the amount of such compression. Sometimes pleuritic effusion occurs on one side only as a result of subpleuritic inflammation. In these cases it is often developed very insidiously without the presence of pain or any other symptom with the development of increased dyspneea. It is less likely to be removed by absorption than when it occurs otherwise than in connection with cardiac disease and will very likely tend to hasten the fatal termination of the case. The congestion of the mucous membrane of the lungs will act as a predisposing cause to Bronchitis. Should this occur it also will aggravate all the other symptoms.
Portal congestion

This type of congestion of the lungs is often the result of valvular lesion of the left side of the heart. When the result either of mitral or aortic disease it is preceded by pulmonary congestion and dilatation of the right cavities of the heart. The return of blood through the vena cava inferior being impeded, congestion of the hepatic veins first takes place, followed by the same condition of the portal system, and if the obstruction remains long enough compression of the excretory ducts will take place preventing exitation of bile or retarding its escape. When congestion takes place the organ becomes enlarged proportionate to the amount of engorgement, but its normal shape is generally preserved and it becomes more soft and laceable. It may assume its normal size under treatment sometimes however it remains permanently enlarged. The amount of enlargement can easily be ascertained by percussion.

Hepatic congestion is generally accompanied by a sense of weight in the right hypochondre
and frequently there is tenderness on pressure. Enlargement of the spleen generally precedes congestion of the liver from the obstruction in the portal vein.

Cerebral symptoms sometimes occur in connection with cardiac disease. They may arise either from emboli in the cerebral arteries which have been broken off in a former part of the paper, or from obstruction to the return of the venous blood from the brain. The venous congestion and the deficient amount of arterial blood transmitted to the brain causes a disturbance in the circulation giving rise to headache, vertigo, tinnitus aurium.

Disease of the kidneys is not uncommonly a result of valvular disease of the heart. Two theories have been brought forward to account for this:

1) That the renal disease is produced by the congestion occasioned on the impeded return of venous blood, this congestion terminating ultimately in structural change.

2) That it is produced by changes in the condition.
of the blood the consequence of deficient secretion as well as other changes which have been observed by Becquerel and others to take place in cardiac disease viz. diminution of the globules, fibrin and albumen and an increase in the coating portion.

It is probable that both of these causes combined will aid in producing structural degeneration. Albuminuria very frequently occurs as a result of congestion alone. Accordingly, it is frequently difficult to say whether the degeneration of structure has taken place. In the latter case however the presence of albumen is more persistent and the presence of one or other of the varieties of tube casts may generally be detected by the microscope. The tendency to general oedema will become augmented by this complication. Pleural and Cardiac oedema being combined.

Endocardial murmurs

It is not our intention to enter at any length into the subject of cardiac murmurs. The more important considerations to be taken into account in our investigation of their cause—
will only be taken into consideration. Obviously the first thing to be determined is, whether the murmurs that may be under consideration is dependent on cardiac disease or not, and this is frequently a very difficult point in diagnosis. In patients affected with anaemia having blood very much deprecating in quality, watery and with a deficiency of red corpuscles, it is very common on examination of the heart to discover a murmur, generally accompanying the first sound although not connected with organic disease of the heart. This is called an "anaemic murmur," the generally find it in pale anaemic patients, chlorotic young women, etc. It generally precedes the first sound of the heart, although not distinctly localized, yet is heard loudest at the base, and is communicated into the arteries of the neck. In forming our diagnosis we must bear these facts in mind. But a murmur dependant on organic lesion of the heart may occur also in an anaemic subject. It may likewise succeed the first sound and be heard loudest at the base. How are we to distinguish them?
The arrangement I have taken from a paper of Dr. 
Episcopus on "Neurovascular Murmurs in the Etiol.
Medical Journal for Novbr 1861
we observed previously that the aural mur- 
mur is no more so localized as the other, it is dif-
fused over a much wider space and it can 
be heard by placing the stethoscope over the 
behaved arteries or the vessels. If there is higher-
trubly of the heart and congestion of the lungs 
ever or it will very likely be dependent on 
organic disease of the heart. Having come to 
the conclusion that the murmur is imperative 
and caused by valvular disease, the next ques-
tion that arises is, what is its pathological 
character? To aid in determining this, it is 
necessary that two points be ascertained in 
connection with it: (1) the place of the murmur 
(2) its rhythm.

With regard to the locality in which it is best 
heard — there are four valvular orifices 
in the heart, disease of any of which may give 
rise to a murmur: (1) the tricuspid (2) the pul-
monic (3) the mitral (4) the aortic. The question 
that is as to which of these is the murmur pro-
duced. If ascertain this one source of fallacy 
must be guarded against viz: a deformity or 
displacement of the heart or great vessels. For
this purpose the percussion dulness of the heart must be accurately made out and its limits defined so that any abnormal condition may be observed. The presence or absence of subternal tumour should be ascertained, as well as the point of the aortic beat, and the character of the impulse of the right and left ventricle. These points having been studied we endeavor to find out the area of the murmur, or the locality in which it is heard, and it is chiefly from the observation of this that we determine which orifice is at fault in producing it. Now as there are four orifices so there will be four areas to which murmurs arising at these orifices will be communicated. The locality of these areas is a matter which has given rise to some difference of opinion, but Delandre arranges them as follows:

1) Area of the mitral murmur — this generally corresponds with the apex of the left ventricle, the apex being the only part of it in close cæsura to the thoracic wall. Having found this point by means of the aortic beat and observing that it is at the outer
Between the devil and the deep blue sea

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border of the cardiac dulness, if we find that a murmur can be localized to nearly a circle round this point, it will probably be of mitral origin. Mitral murmurs are generally very distinctly confined to this region and they are almost inaudible over the base of the heart.

(2) Area of the Aortic Murmur — This murmur is heard loudest over the base of the heart and the membranous of the aortic. It is heard also along the whole line of the aortic and is often intensified at the sternal ends of the second right costal cartilage. The cause of this wide diffusion is not very well known. It is generally louder over the right than the left side of the membranous aortic and has sometimes a special distinctness over the sternal end of the second right costal cartilage.

(3) Area of the Tricuspid Murmur — The occurrence of discord of the tricuspid value is very comparatively rare. When it does give rise to a murmur however it is heard over the right ventricle where the latter is uncovered by lung. This usually not heard
above the level of the third rib and may thus be distinguished from an aortic murmur. In some cases it extends to the xiphoid cartilage.

(2) Area of the pulmonic murmur. This murmur is of very pure recurrence, much more so even than the tricuspid. The diastolic pulmonic sound is distinguished from that produced by the closure of the aortic valves is heard generally at the junction of the third left costal cartilage with the sternum. Accordingly a murmur produced at this particular orifice might be supposed to be heard with greatest intensity at this point. The exact situation may vary slightly either from the lung covering the base of the heart more than usual as in Emphysema, or from being uncovered as in some other conditions.

11. St. rhythm

By knowing the locality in which an endocardial murmur is heard, we ascertained at what orifice it is produced; but a knowledge of the site alone cannot assist us in determining the nature of the lesion, e.g. whether it is obstructive or
Regurgitative or both; this we do by ascertaining its rhythm. We find out whether it accompanies the first or second sound of the heart, and whether it precedes or succeeds them or is heard in the intervals between. We shall not enter into any discussion as to what are the causes of the normal cardiac sounds, but before we can have a clear idea of the causes of endocardial murmurs we must ascertain what takes place during the first and second sounds of the heart respectively and during the interval between.

The events which correspond in point of time with the first sound are, the contraction of the ventricles, the first part of the dilatation of the auricles, the closure of the auriculo-ventricular valves, the openness of the semilunar valves and the propulsion of blood into the arteries. The second sound immediately follows the cessation of the ventricular systole and corresponds with the closure of the semilunar valves, the continued dilatation of the auricles, the commencing dilatation of the ventricles, and the opening of the auriculo-ventricular valves. The pause between the second and first sound
corresponds in its first half with the completed distention of the auricles and in the second with their contraction expelling the blood which they contain and completing the distension of the ventricles; the auriculo-ventricular valves being all the time open and the semilunar valves closed.

We shall now speak of the murmurs and of the relation they bear to these sounds.

1) The murmur precedes and runs up to the first sound. It occupies what normally is the last part of the pause corresponding to the auricular contraction and the passage of blood through the auriculo-ventricular orifice, the ventricle itself being passive. It is therefore an auriculo-systolic murmur. It will almost always be found that such a murmur depends upon contraction of the auriculo-ventricular orifice, and the consequent interruption to the flow of blood from the auricle to the ventricle.

2) The murmur succeeds and runs off from the first sound. This murmur corresponds to the ventricular systole and is caused by the passage of blood out of the ventricle. It is called a
ventricular systolic murmur. It may arise either from the blood being propelled in a retrograde direction from insufficiency of the auriculo-ventricular valves, or by an interruption to its progress through either of the arterial orifices from obstructive lesions existing in them. Consequently it may indicate either insufficiency of the auriculo-ventricular valves or obstruction at the arterial orifices.

3) The murmur succeeds and runs off from the second sound. This murmur will coincide with the diastole of the ventricles; it is called a ventricular diastolic murmur. Being diastolic it will depend upon the passage of blood into the ventricles, and this is generally in consequence of insufficiency of the semilunar valves allowing regurgitation.

The following conclusions may be drawn from our knowledge of the locality and rhythm of murmurs.

2) A murmur preceding and running up to the first sound heard within the mitral area will be due to mitral obstruction, over the tricuspid area to tricuspid obstruction.
(b) A murmur preceding and running off from the 1st sound: (1) if heard over the mitral area will depend on mitral regurgitation; (2) if heard over the tricuspid area will depend on tricuspid regurgitation; (3) if heard over the aortic area will be due to aortic obstruction.

(c) A murmur preceding and running off from the 2nd sound: (1) if heard loudest over the aortic area will be due to aortic regurgitation; (2) if heard loudest over the pulmonic area which however is very rare, thought to be due to pulmonic regurgitation.

Hines