Notes on Pathology of Embolism
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Embolism, from the Greek ἐμβολία, a plug, signifies a plugging, or obstruction of blood vessels by some object (embolus) which has been carried from a distance by the blood stream until it meets with a vessel whose calibre is too small to admit of its further passage. Such being the case it follows that the process goes on in the arterial system (although the origin of the embolus may be, usually is elsewhere) capillary system, or portal vein. The term embolus is applied to the obstructing particle itself, be it a blood clot, cardiac vegetation, fat globule, or whatever else. The result of such obstruction is known as embolism. Emboli arising in the venous system pass through the heart into the pulmonary circulation, those from the left heart, pulmonary veins, or arterial system, obstruct arteries or capillaries, those of the chylo-pulmonary system become
arrested in the various ramifications of the liver. Curious cases arise where a Thrombus is carried from the right to the left auricle through an open foramen ovale, to lead to embolism in the course of the greater circulation. Cohnheim and Litten reported cases of this kind and Gahn distinguished the process as Embolus paradoxicus. Whether considered from a medical or surgical point of view, the subject of Embolus and its results, embolism presents much that is of the deepest interest to the practitioner and in many cases of the gravest importance to the patient. In all probability, one is aware of the conditions necessary to give rise to an embolus, existing in an individual, and is not surprised to hear of their fulfilment, and then one fully realizes the suddenness of the attack, and in many cases the gravity and multiplicity of the symptoms.
The most frequent cause of embolus is thrombosis or clotting of the blood in the vessels, this usually taking place in one or other of the great veins as the Iliac, Scapular or Hypogastric.

"The process of coagulation of the blood is now understood to be due to the conversion into fibrin of a single body (Fibrinogen) under the influence of a ferment (Fibrinogen) which is derived from the Leucocytes." (Ballis' Physiology). Normally, the blood remains fluid whilst in the vessels, and coagulates after removal from them, and it may be supposed that whilst circulating in the vessels the fibrin ferment is removed as fast as it is formed by the Leucocytes, and so coagulation is prevented. If however the blood is brought in contact with a foreign body, it coagulates, so that in certain pathological conditions of the lining
of the blood vessels, the clotting is favoured, and a deposition of fibrin brought about.
Again there may be an excess of fibrin ferment in the blood and in such quantities as to cause coagulation.
Köhler has produced rapid coagulation of the blood in healthy blood vessels by the injection of a sufficient quantity of fibrin ferment. Showing that, an excess of such may cause clotting.
In other cases clotting may be due to injury to the walls of the vessels. Inflammatory changes in the surrounding tissues may be produce stasis in the blood vessels and subsequent coagulation.
Pathological changes in the vessels or the blood itself these changes being calcareous, syphilitic or such like. Diminution in the strength of the blood flow, pressure upon the vessels. Coagulation may occur in the
heart itself and very often there is reason to suppose that it occurs just prior to death when the circulation is at its lowest ebb, and the cavities are not completely emptied; this occurring more frequently in the right side of the heart.

This cause is frequent in Diphtheria from formation of Thrombi in the heart from weakness of the muscular tissue (Raverey Robinson) Buchanen Bristow think that in individual cases there is no evident source of embolus and that therefore conglutination of the Blood may occur in arteries as in the veins, this certainly does occur in aged people.

This anti-mortem clotting is known from that occurring post-mortem by the Clots being more or less decolorized former in Consistence and more fibrinous in structure, and are often to be found entangled amongst the
Carved Column.

Emboli may also originate from a Thrombosis in Diabetes, Typhoid Fever, or Puerperal Conditions.

Other causes of Embolism are portions of Depressions upon the Cardiac Valves which having been broken off are carried away in the blood stream. Calcaneous, or Atheromatous Matter from the lining membrane of the Blood Vessels, or the Cardiac Valves. New Growths may perforate the walls of Blood Vessels portions of these become detached to give rise to Emboli. Parasites, pigmentary granules, Droops of Fat from fractured bones or ruptured Fatty Liver, and Air Globules from wounded veins.

The Structure of Thrombi is of course the same as that of a Blood Clot originally, their ultimate composition and appearances depend chiefly upon the fact of their being rapidly or slowly formed.

If rapidly formed they are of a dark
red colour, and gelatinous in Consistency; formed of fibrin, in the meshes of which are entangled immemorable red and white corpuscles. This structure in time becomes somewhat drier and paler in colour, owing to the disintegration of the red corpuscles, and at the same time becomes more firmly attached to the walls of the vessel. It may or may not completely fill the lumen of the vessel.

On section its structure is seen to be tolerably uniform. Should the formation of the Thrombus be by a slower process—section shows it to be made up in a stratified manner, and to be harder, and more organized than the more quickly formed Thrombus. This stratification is evidently due to successive layers of fibrin and white corpuscles, the corpuscles adhering to the deposited fibrin.

Once a Thrombus has formed in a vessel
It increases along the lumen of the vessel till it comes in contact with a sufficiently powerful current of Blood to prevent further coagulation. The end of the Thrombus from this cause is usually tapering & becomes very liable to be broken off and carried away by the Blood current. If such a portion be broken off, this portion constitutes the Embolus. Again, the Thrombus or Clot may become broken up, and disintegrated giving rise to the same series of events from being arrested in some of the smaller vessels, and constituting secondary Emboli.

Once the Embolus is in the circulation it depends upon the size of it to decide where it stops, the usual place being at the bifurcation of an artery. Usually, from its irregular shape it does not at first completely occlude the vessel, but, gradually this occurs from a process of clotting, or thrombosis around
The Embolus. The size of the Embolus varies — it may be so minute as only to obstruct a capillary, or it may be large enough to obstruct one of the great vessels as the axillary, femoral or pulmonary. The fate of Emboli differs — they may become organized, and adhering to the walls of the vessel completely occlude it. Bowley says, the presence of an Embolus in a vessel excites changes in the vessel wall and new vessels are developed from the Vasa vasorum which penetrate the clot and surrounding thrombus — inflammatory exudation & organization follow causing occlusion of the vessel, or they become softened and disintegrate, or in rarer cases they may become absorbed. When the embolus becomes impacted in some abdominal organ as the spleen, liver, or kidney, we have formed what is known as a 'hemorrhagic infarct.' This being due to the Capillaries giving
way or more or less haemorrhage occurring in the tissue owing to an intense injection of Blood on the distal side of the Embolus. If the collateral circulation is sufficiently free, this injection gradually begins to subside, and the functions of the part return, if not, then the parts being deprived of their blood supply and the walls of the vessel being so much damaged exudation & emigration of corpuscles take place and a loss of vitality inevitably ensues.

Co operate explains the formation of Haemorrhagic Infarcts in the following manner.

The first effect of the blocking of a terminal artery, is the stoppage of all supply through it, the arterioles empty themselves by contracting and pressure in them is reduced to nil. Venous pressure though too is in excess of this and the blood regurgitates from the Veins to fill the Capillaries, and the arterioles beyond the clot's edge. The arterioles round about the area
dilate and their capillaries become full of blood, but, even now, the force of the stream in the latter is sufficient to overcome the resistance in only a few of the overlying capillaries of the obstructed area. Consequently, we should find such an area dark from containing stagnant venous blood, but surrounded by a ring of arteriolar redness, soon the darkness is increased by the escape of red corpuscles into the tissues without any rupture of vessels, just as happens in venous congestion. Finally, secondary thrombi of the veins and vessels in the area is bound to occur.

Colburn concluded, that when blood vessels with their vasa vasorum are deprived of circulating blood for a sufficient length of time they lose their power of retaining the blood, and allow first, the Leucocytes, Kargmunicis and Lympocyte, then the red corpuscles to escape from them, the escape taking place only through the capillaries & veins.
He also thought that, when emboli blocked terminal arteries the result was almost always hemorrhagic infarction, and that exceptions to this rule were due either to the veins of the part being valved or thrombosed, so that regurgitation was prevented, or to the part being so placed that gravity favoured strongly the return of blood by the veins in which cases the area remained pale and bloodless.

Some apparent exceptions were owing to the existence of fine arterial anastomoses with certain of a set of arteries, the great majority of which are really terminal. Thus anastomoses of the Bronchial artery with the Pulmonary artery might sometimes ward off an infarction of the Lung.

The v of the reasons why Infarcts are so much commoner on the
surface than in the substance of an organ, is, that, in the former situation, the whole base is almost absolutely cut off from collateral supply. Cattell however disputes the truth of Columbus's & placation of the whole process. He shows the infarction of the kidney which follows after amputation of the renal artery is not due to regeneration from the renal vein, but is even more intense when the vein is tied. The kidney swells greatly, becoming first congested, then, infarcted. The congestion begins in the subcortical zone of the cortex, and at the bases of the pyramids. Where the pelvis is attacked, and it is due to the continued supply of the organ by small arteries which spring from the lumbar, supra-renal, and phrenic arteries and pierce the capsule, and to others from
from the Spermatic, which run up along the sheath.
If the renal vein is left open the
kidney swells more slowly because
some of the blood entering from
these arteries escapes by the vein,
the stream is therefore away from
not towards the kidney.
But the most perfect proof
of the Enlargement being due
to supply through these —
arteries, and not to renal reflex
is afforded by this experiment.
The renal artery is rendered
terminal by sheltering the
kidney out of its bed of fat
and the artery is then lead.
Reperistation should now
occur from the open vein, but
as a rule it fails to do so.
A slight congestion of the
organ sometimes occurs, but
the gland remains much
lighter, and smaller than
its fellow of which the renal
artery only has been tied, and
never becomes the seat of —
haemorrhage per diapedesis.
It would seem therefore, that in
many cases the pressure in the
renal vein is not sufficient
to overcome the resistance of
the capillaries, and to produce
an infusion of them with
blood. When the main artery,
and its collaterals are tied,
niuch less would it do so
when the latter are pumping
blood in beneath the elastic
capsule and thus increasing
the intra capsular pressure.
If by constricting, poisoning the
pressure in the renal vein
is raised, Infarction is more
likely to occur, and it is pro-
duced in its severest form by
Clamping the Vena Cava Inf.
above the orifice of the Renal
vein.
Litten agrees with Colonheim.
in the fact that the red corpuscles escape by diapedesis, but simply on account of the stretching of the capillaries and small vessels by the mechanical congestion. It begins almost at once, after the ligature is applied, before anemia has had time to affect any marked change in the vessel walls, and no escape occurs in a kidney, shielded from its capsule, if a ligature on the renal vein be cut three or four hours after application. It would seem that the true reason why red corpuscles are found so frequently on the surfaces of the organs in which they occur, is not, that the base is almost entirely cut off from blood supply, but, that there be have entering small capsular arteries through which blood is still driven in the area.
Hamilton's Conclusions upon the subject of Infarction are as follow:

The infarctions of the spleen and kidney are due to the blood supply being cut off from the parts, and the most common cause of this is embolic plugging of the respective arteries.

They are not usually accompanied by hemorrhage unless in the zone of reaction which surrounds them although congestion and punctiform extravasations are possibilities in the early stages.

They are veritable necroses—the necrotic changes within them resembling those which follow the total removal of the blood supply in other protected parts of the body.

They are absorbed in the course of time and the formation of a depressed cicatricial follows.
Of the brain is accompanied by a similar necrosis, but as the arteries of the encephalon are terminal only in certain regions, the necrosis of the part supplied by an occluded artery is never so wide-spread as in the case of the spleen, and kidney. Hemorrhages may occur here owing to the attempt to nourish the part by collateral channels, and to the softness of the surroundings.

VII. The hemorrhagic infarction of the lung is simply an apoplexy due to various causes. By far the most frequent however is rupture of alveolar capillaries unduly distended by the regurgitant pressure resulting from valvular disease of the heart. Its wedge shape is caused by the shape of the bronches and the air vessels in which the effused blood is contained and not by the distribution of a terminal
branch of the pulmonary artery.
The lesion has usually nothing to do with pulmonary artery embolism but a hemorrhage from any cause if situated at the periphery of the lung will have the usual character of a hemorrhagic infarction.

The blood in these pulmonary infarctions is rapidly absorbed leaving no trace of its former existence.

Churchison and Bristow both hold that "embolic patches" arise spontaneously by conglutination in the artery and are not due to embolus.

Embolic changes in internal organs are nearly always wedge shaped from the distribution of the vessel and its capillaries and are usually found at the surface of the organ affected, the apex of the wedge being the seat of the infarction.
The results of embolism depend upon the circumstance whether the embolus completely shuts off the blood stream in the affected vessel or whether owing to its size or shape or exact position in the vessel the stream of blood is only somewhat diminished. If the vessel be not a terminal vessel the circulation is carried on by collateral vessels or partly by the vessel itself unless or until completely blocked. If the blockage is not at first complete it usually becomes so owing to the continued deposition of fibrin upon the original embolus giving rise to thrombosis. Cohunein found that the exclusion of blood from a vascular region for a sufficient length of time produced such impairment of the vitality of the vascular walls that when the blood was again allowed to circulate
the vessels dilated, the blood flow was retarded and the液体bougainie
and red or white Corpuscles escaped into the surrounding tissues.
An arrest of the circulation for a still longer period was followed by capillary haemorrhage and ultimately by necrosis of the vessel.
It sometimes happens that one or more of the arteries of the legs, arms, or even the Aorta itself become obstructed. The immediate result of this is serious impediment to the circulation through the complicated limb as shown by the impediment to or cessation of the flow of blood with increasing coldness and pallor of the part. In some cases the collateral vessels enlarge gradually and carry on the circulation so that in time the limb becomes normal. In other cases the circulation comes generally or in certain area — to a standstill
the affected parts gradually lose their clear, the surface is pale, but
covered with purple or colored spots, and the tissues become boggy and
doughy, while the aorta filled with a thin bloody fluid upon the dis-
colored portions, and sometimes soon supervenes.

Perhaps least in importance to thrombosis as a cause of embolus is Endocarditis. Here
the chief seat of mischief is the left side of the heart, where the valves
are exposed to friction. The formation of vegetations upon them
favoured. In acute Endocarditis, the deeper layers of the endocardium
become rapidly infiltrated with young cells and as these increase
in number, the intercellular substances become softened and destroyed, thus is produced
a soft tissue composed almost entirely of cells such as always
result from inflammatory.
processes in connective tissue. This new tissue as it increases fills up the superjacent endothelium and projects in the form of minute granulations and vegetations. Upon this roughened vascular surface fibrin is deposited giving rise to an excrescence, partly formed by the thickened into cardiacum mentioned above.

This new tissue may become partially organized or go on growing and ultimately degenerate fatty or calcareously. These excrescences on the degenerated portions of them being exposed to the blood current are easily detached and carried away to form emboli.

Wills or not or suggest that when embolic abscesses form in consequence of inflammation of the left side of the heart they may well be called arterial pyemia.
to distinguish the whole case by this title from the more common causes of Pyæmia due to surgical injuries or wounds; the difference being that in the latter case the disease generally affects the course of circulation of the Venous Blood, whilst in the arterial pyæmia the secondary suppurations are found in the course of the Arterial Blood. Another cause of Embolus is the entrance of Fat into the Circulation, and is commonly known as Fat Embolism — it was first discovered by Zenker in 1862. This frequently occurs in fractures when the cancellous tissue of the bones is broken up and lacerated, considerable quantities of fat being set free, and finding their way into the circulation. Obstruction and staining with Osmic acid of portions of the internal organs, members of Capillaries are found
To be plugged with this liquid fat. The fat globules may also gain an entrance from a rupture in a fatty liver, in any acute suppuration in fatty tissues, in acute nephritis, fatty degeneration of abscesses, from Typhoid or Blood in Diabetics in which case the fat globules are mostly found in the lungs. In Typhoid the fat globules are said to be smaller than those arising from a fractured bone or other means. In the kidney they are found principally in the Malpighian tufts. When in any quantity in the lungs the breathing is seriously interfered with and we then meet with edema and croupous pneumonia. Coats mentions a few cases on record in which extensive fat emboli has occurred in the smallest vessels of the brain, conjunctiva, skin, muscles, heart, intestines, liver and kidneys.
and has apparently been the cause of death. Handfield Jones has shown free fat in the smaller vessels arising from the disintegration of Atheroma.

The symptoms arising from fat emboli are in great measure due to the fact of the oil globules being too light to circulate freely and in their behaviour simulating air globules (Hamilton).

The fat in all probability finds its way into the circulation, either by the veins or lymphatics being at the time of injury subjected to greater or less pressure.

Scriba thinks that death arises from obstruction in the vessels of the Brain and never from pulmonary obstruction as mentioned above. Cohenheim denies that the symptoms and fatal issue are due to the fat at all, as large quantities can be injected into the circulation causing extensive embolism.
but not followed by death or even by serious symptoms.

Erichsen thinks that fat emboli may become impregnated with septic poison in cases of compound fractures or severe lacerations of adipose tissue and set up inflammation wherever they lodge.

The emboli from subcutaneous injuries are said occasionally to give rise to hemorrhagic infarcts around the point at which they are arrested and in some cases to causeadenoma of the lung by an expansive obstruction to the circulation but being perfectly indolent they never set up inflammation. Transtrous cells and small masses of blood from the often migrate or grow into blood vessels and are then carried off by the blood stream to distant organs being usually stopped in the Lung Capillaries.
or those of the liver thus giving rise to secondary growths which are as a rule other than the primary ones. These emboli are most frequent in the malignant Sarcomatous tumors (Lues and Syphillis).

Cohnheim in his Lectures on General Pathology, new London Society's Translation, Vol IV, p. 152 says:—

"The extreme frequency with which abdominal carcinoma is followed by metastasis in the liver and only in this organ, tells in the clearest way its distribution by means of the circulation. The metastases are true cancerous emboli i.e. they were originally cancerous thrombi which were carried off by the blood stream and how transported through the blood vessels as far as the narrowing of their calibre admits. In a precisely similar manner are formed most of the secondary"
Pulmonary nodules originating in primary malignant tumours of other regions, and it is very characteristic that Giant-celled sarcoma of Bone if it becomes generalised almost invariably gives rise to metastases in the Lungs. When the tumour cells are so small as to circulate freely throughout the system, their arrest is then little if at all controlled by the laws of embolism. The velocity of the flow being a very important factor in the process. The less the velocity, the more favourable the conditions for commencing proliferation.

According to Paget cases of rapid multiplication of Cancer may arise, from either, a cancerous diathesis, or a morbid condition of the Blood, but at the same time, he thinks, there has been some conveyance of cancerous
material by the Blood. 

Viehow's analogy to this is the 
similarity that exists between 
secondary deposits in cancer, 
and secondary abscesses in Pyaemia. 
Viehow on the other hand thinks 
that the fact of secondary deposits 
not necessarily occurring in 
the organ through which the 
Blood first passes rather 
neutralises against the Embolic 
theory. Again however has shown 
umerous cases in which new 
growths in the liver secondary 
to Cancer of the Large Intestine 
have had a similar structure 
to the Crypts of Lieberkuhn. 
Again it is a fact that the tissue 
from which the primary growth 
originates may arise in situations 
in which it is not normally present 
for example epithelium in the 
 glands and bones. 

Sir James Paget says "The Explanation 
of Cancers (so far as it may be distinctly
from them grow the) it is that which takes place through lymphatic vessels to their glands. The number of cases in which lymphatics have been traced from the primary growth to the nearest glands is sufficient to make it probable that the disease is often thus propagated by embolic extension from one to the other. But even when such tracts of cancer cannot be traced from the primary disease to that in the lymphatic glands, Mr. Simonds' suggestion is very probably true—that the disease is one of the lymphatics not of parenchyma or vessels of glands.

There can be no doubt that cancers grow into the veins, fragment of these being broken off by the blood current and carried away therein. Probably recurrent tumours
Other than cancerous are to be generated. Virchow mentions the fact of a cartilaginous growth in a testicle affecting the lungs. Volkmar and Richet also mention cases of tumors of bones affecting the lungs. While in this nature treatment of cancer also mentions pigmented masses from the liver being found in the small branches of the pulmonary artery. The parasitic origin of emboli is somewhat rare. The commonest forms being Trichina spiralis, the Scolex of Taenia Solium and Taenia Echinococcus. Hoppe mentions the case of a girl at 9 years where he found a large echinococcus in the middle and posterior lobes of the right hemisphere of her brain also a similar affection of her liver.
Hillson Paggs also mentions Cysticercus Cellulosae as infecting the Pia mater or ventricular space.

Rupprecht in his "Trichinen Krankheit" speaks of a form of Pneumonia in connection with Trichinosis which appears about the twenty sixth day of the disease, selecting the lower portion of the left lung and apt to prove fatal. The sputum consists of dark hemorrhagic blood. The Pneumonia is supposed to be due to Trichinosis embolism, the clots being derived from Thrombi which, forming in the Venous system are sent through the heart into the lungs. Strongyles armandus a parasitic worm is found in the mesenteric arteries of the horse and is a frequent cause of aneurism therein.
Goere and Gers both mention
Cæcum cerebralis as occurring
in the human subject.
Cruveiller Père André Guérin records
a case in which more than one
hundred cysticerci were found
in one cranium and about fifty
of these were located in the
Cerebellum.

The entrance of air into the large
vessels is also answerable for the
formation of Emboli; it is of
frequent occurrence in suicidal
wounds, occasionally it happens
during operations upon the neck
or jaws, and, also as mentioned by
Birch Hirschfield may be admitted
into the circulation after labour
when the uterine sinuses are
open before the formation of
blood clot, or by the removal
of one of these clots.

On the admission of the air into
the circulation it is supposed
that the air globules are too
to circulate freely with the blood. On post mortem examination the air is found churned up with the blood in the right heart, which is enormously distended, some however is in the Pulmonary artery and some in the systemic veins and the sinuses of the Dura mater. It seems that the right ventricle merely compresses the elastic air which again expands during diastole. The force of the heart is extended upon this and no blood passes to the lungs, and over-distension of the right auricle with air prevents the admission of blood from the Right Cava consequently both Pulmonary and entire Aortic Circulation cease. (Hamilton). Cotes has shewn that this confers upon the admission of air into the veins its very fatal character.
Panum. Rich. A. 1847. Thinks that air embolus is more dangerous than fat embolus. Cohnheim thinks air embolus is only fatal when the ventricle is entirely filled with air. Isolated bubbles being simply carried away by the circulation and arrested in the pulmonary vessels.

Of all the organs affected by embolism the brain is the most important and the symptoms presenting themselves depend greatly upon the seat of embolization. This may either be in the deep vessels, or the superficial ones if the former alone are affected the cortex and superficial white matter may not suffer. The commonest cause of cerebral embolism is acute or chronic endocarditis which gives rise to vegetations upon the cardiac valves one or more of which vegetations
may become detached. In cases of
of Mitral Stenosis the embolus
may originate from a clot in the
left auricle.
Hilton Fagg quotes two cases in
which the primary lesion was
in the heart itself, where a soften-
ing thrombus adhered to a
diseased portion of the arterial
wall. In many cases no clot
can be discovered on post mortem
examination although the
cardiac orifices or some lesion
of the vessels or thrombosed vein
may give one every reason to
depose an embolus has existed.
It may however have become ab-
sorbed or have broken down, hence
the condition of the brain substance
will point to the lesion being one
in which the Blood supply has
been cut off. In Gangrene of the
Lungs? There is Venous Thrombosis
and from Particles of Fibrin being
carried away to the Brain, we may
trace the origin of septic cerebral emboli, in the same way cancer of the lung may cause secondary deposits in the brain.
The commonest seat of infarction is in the arteries of the Sylvian fissure, the left middle Cerebral being perhaps the most common. This being—according to the opinion of most authorities on the subject—due to the fact of the vertebral artery arising from the Subclavian at nearly a right angle, thus reducing the possibility of an embolus passing into it. On the other hand the external and Carotid are nearly in a direct line. The middle cerebral is the direct continuation of the Internal Carotid and supplies the greater part of the Basal Ganglia, nearly all the Corpus Striatum and Lenticular Capsule, part of the Optic Thalamus and the greater part of the Motor Convolution.
The nutrient arteries come off from the first part of the middle Cerebral, and are so more exposed to obstruction from emboli, hence the softening of the central parts of the brain especially in the region of the Corpus Striatum.

It may be remarked that these vessels most frequently give way in the encephalitis of the brain. Brietower, Husz, and Gerhardt all mention cases in which there was found on post mortem examination to be an embolus on each side of the brain.

In addition to Endocarditis, as a cause of Embolism, may be mentioned rigid atheromatous or calcareous vessels in which the intima is changed, or syphilis, chronic renal disease, aneurism in which the blood clot has broken down and is being disintegrated and carried away by the Blood stream.
Malignant growths in various parts of the system may give rise to malignant Emboli in the Brain to causing secondary growths there, these are found to be of all kinds with the exception of epithelial types and are according to Hilton Fagge always multiple.

Hilton in his researches on the Blood in Sarc coma has found in Cultures obtained, a very small micrococcus - simple or double anaerobic and staining with difficulty - this being found frequently in malignant Emboli in the Brain.

Parasitic Emboli as hydatids of the Human Brain are not of very frequent occurrence. More mentions the case of a girl of 15 years in whom he found a large Echinococcus situated in the middle and posterior lobes of the right hemisphere also a similar affection of the Liver.
Cysticercus cellulose is mentioned by Hilton maggs as infecting the
the Ria Water and Ventricular
space. Parasitic growths are
most frequently found on the
surface of the brain, or in close
connection with the gray matter of
the convolutions, and are somewhat
frequent in the Ria Water.
Softening or Evanolissement arises
from the occlusion of the nutrient
arteries. Tallandau, Bonilland
and Durand-Fardel suppose as
they term it Ecephalomalacia to be
an inflammatory process. On the
other hand Pottin, Courvilhan,
Bright and Hashe say it is due to
some arterial disturbance.
In cases where an Embolus has
become impacted, and a fatal
issue has rapidly ensued, there may
be no noticeable change in the Brain;
or there may only be some congestion
from Venous pressure. As a result of
continued deprivation of Blood the
Brain substance seems to undergo a species of fatty degeneration or softening. The first stage of this softening is known as Red Softening and is only the early or acute stage of White Softening.

Gowers in Evans's Dictionary says Red Softening occurs very often in that part of a Brain affected by White Softening. Distillation of the Capillaries with Blood occurs most markedly in the Periphery, and Blood is actually effused chiefly by Perfusion of the Capillaries and in greatest degree perhaps by migration of Corpuscles.

In proportion to the amount of Blood mingled with the softened Brain tissue the Colour of the affected area is changed and thus Red Softening is produced when the amount of Blood is considerable. After a time the Blood effused in Red Softening degenerates and its tint becomes altered to yellow or orange and Yellow Softening is produced. Ultimately the colour
although at first moderate may be removed and white softening result but the pigment that gives rise to the yellow that may remain for many years. Red softening of the brain may be inflammatory in origin but in the great majority of cases it depends upon embolus or obstruction of venous sinuses. White softening is by many considered the ultimate condition of red softening but is without doubt not uncommonly primarily in connection with extensive thrombosis and atheroma of the vessels. Lesser structures break up very readily indeed. The kernel of the medullary sheath conglutinates and escapes from the primitive sheath and then breaks up into fine fat granules. The ganglion cells are some what more resistant but they also ultimately become granular and disappear. According to Dr. Green, the white softened tissue is then under the
microscope to consist of broken down nerve fibres, altered blood corpuscles, granules of fat, and, large compound granular corpuscles which result from fatty degeneration of the nerve cells, and, the cells of the neuroglia, and Anamboïd cells. The surrounding capillaries are dilated and filled with coagula and the granular corpuscles envelope their walls. Nervous structures break up very rapidly indeed. The hyperplasia of the medullary sheath coagulates and escapes from the primitive sheath and then breaks up into fine fat granules. The ganglion cells are somewhat more resistant but they also ultimately become granular and disappear. In more advanced stages this softened tissue becomes paler, and liquefies, or may gradually dry up and a process of repair take place by the growth of the surrounding neuroglia forming...
a fibrous network which contracts into a cicatrix containing amorphous crystals.

From anatomical conditions it sometimes happens that when a vascular territory is deprived of its normal supply by an embolus it cannot be supplied with blood from surrounding districts. A certain limited amount of collateral circulation is possible through the capillaries and the rare anastomoses, but it is only around the edges, so that the centre of the territory dependent upon the supply now cut off becomes destitute of circulating blood.

The extent of the mischief arising from embolism depends entirely upon the particular artery or arteries as the case may be, which become blocked. The size and position of the vessel is of very importance, as on this depends the fact whether large areas of the Brain are affected.
or merely a small area depending upon a terminal vessel for its blood supply.

An embolus in a cerebral artery does not however produce, as it does in other organs with a more abundant circulation and consequently poor collateral circulation, a large thrombo-haemorrhagic infarction. We may however find several small haemorrhages as it were overlapping each other and so producing a well marked "red softening."

In a few instances mentioned by Pepper, the Clot or embolus has become pervious by perforation from absorption of its centre or core so that the circulation has become restored, and the ill effects to a great extent modified. Although there is no reason to suppose that this takes place in a sufficiently short period of time to be of any
most advantage in restoring
the already seriously damaged
nutrition of the becrooked portions
of Brain.
Cases have been observed in which
the Whole of one hemisphere was
softened from obstruction of
the Carotid at its bifurcation.
Which Charcot suggests may
be due to an unusual distribution
of the arteries.
In a general way it may be
said that Embolus affects
the cortex of the Brain and
that hemorrhage affects the
Central ganglia.
In order of frequency of attack
the Brain occupies the third place
coming next to the Kidneys first
and then the Spleen second.
Hamilton thinks that the Kidneys
and the Spleen are is more frequent
ly attacked than the vessels in
the muscles of the Limbs but are
perhaps thought to be so, owing
to their showing the results of embolism more clearly.

Pyemic emboli in the vessels of the brain give rise as in other organs to secondary or meta-
static abscesses, although according to Loew and Stürk not very frequently. The same
authors say that these abscesses begin with the formation of a hard red solid infarctus
which begins to break down in the centre, and thus is formed a focus of suppuration
surrounded by highly vascular and hyperemic tissues. Rohtenkreutz says they are
usually situated in the deep parts.

Gallo has pointed out that in some cases hyperemic
infarctus or abscesses were found but in the brain, and
that in these cases the lungs are nearly always the seat of the
primary lesion. Other cases have been quoted by Sutton Biernev, Regent's Park, in which portions of some pulmonary thrombus were carried into the circulation. Abscess of the Brain has been known to follow various operations such as ligaturing piles, excision of an eyeball etc. and is commoner in the white matter than in the grey substance.

Gowers in Brain's Dictionary of Medicine mentions one of those rare curiosities of Pathology in the production of metastatic abscesses by the embolic obstruction of the vessels with fragments of Oldium Albicante.

In aneurysm of the cerebral arteries the most frequent cause is Emboillus. Pauchet has found that in a considerable proportion of cases of acute Endocarditis there is embolism with either -
fully formed or incipient aneurism.
Sir William Gull some time ago suggested that in young subjects aneurism should be suspected as a cause of cerebral hemorrhage.
D. Virkes originated the idea and he is ably supported by Hughlings Jackson, that the symptoms observable in an attack of Chorea are due to numerous small emboli in the vessels of the Corpus striatum and contiguous parts, with consequent scattered minute patches of congestion and softening.
Hughlings Jackson attempts to prove that the plugging of these vessels is in the nerve tissue forming the convolutions near the Corpus striatum, and that the tissue is not thereby destroyed but rendered unstable from under-nutrition.
resulting from a diminished supply of blood.

Cancéranx and Vidal both mention a case in which small numerous cancerous masses were swept away from the lungs by the Pulmonary veins and were found in the healthy heart and Aorta so that it may be easily understood that this may be a cause of cerebral embolism and softening of the brain.

Bristowe argues as follows against the embolic theory above.

"Obstruction of the arterioles has been observed only in a very small number of cases and it is doubtful if in these the obstructions were embolic or thrombotic. Besides which it is not only difficult to believe that showers of minute emboli should be distributed throughout the minute vessels supplied to one
Corpus striatum on one side of the Brain only, and that at some later period there should be a similar limitation of such embolic patches to the region supplied by the middle cerebral artery of the other side, but it is difficult to understand why large emboli should not be occasionally mingled with the smaller ones, and cause sudden hemiplegia by obstructing a large vessel, and why small emboli should not become blended by fibrinous coagulation around them into one or two concrete masses. The other hypothesis is that the same disease which affects the heart i.e. the valves of the heart or the joints in the heart also attacks the smaller cerebral vessels or the ultimate tissue of the central nervous organs—a view which might well explain
The intervention of Cardiac disease in Chorea as well as the dependence of Chorea on rheumatic fever.

Dr. Mackrell (New Ohio Review 1869) found, in the post mortem examination of a very severe case with maniacal symptoms, at the inner and outer aspect of the right hemisphere a large red patch of softening affecting to some extent the white as well as the gray matter, and a branch of artery which ran straight into it. Contained a small white tough fibrous connective tissue wedged into an angle of bifurcation, and connected on all sides with long black conglutina extending into the trunk of the vessel and its branches. On the outer aspect of the same hemisphere was a similar patch but smaller and limited to the superficial...
Circumstantial evidence of the constitution, in this nothing like an embolus was discovered, but the parts had been cut through in several directions before detaching the vessels themselves. In post-mortem examinations of fatal cases of Chorea there is never any mention of embolitic infarctions of the kidney and spleen so that it is somewhat difficult to suppose that all the emboli should have become impacted in the cerebral vessels and none in those of the organs mentioned.

Reynolds, in his System of Medicine, IV p. 291-292 mentions many cases of Temporary Insanity arising from Endocarditis and subsequent Cerebral Embolism.

In the Lungs, the most frequent origin of Embolus is the formation of a Thrombus in some vein. This Thrombosis arising in the course of some illness as Diabetes, Rheumatic, Gout, Typhoid Fever, or the Puerperal State, sometimes the Thrombus is formed in the Pulmonary Circulatory System itself, owing to some change going on in the Intima of the vessels, or the Scurfuration of Vins in Connection with some local inflammatory condition. The change in occurring in the Lung consequent upon Embolus depend upon the size of the Embolus if large the result may be sudden death, owing to an almost complete arrest of the Circulation, or a more gradual death, owing to the gradual arrest of the Circulation by Thrombosis around the original Embolus, or again the damage to the Lung may be strictly local.
Corti says that Pulmonary Embolism is fatal not altogether from the obstruction to the blood flow but also from the kidney's being deprived of its Blood from the Left Ventricle.

On examining such Lungs post mortem one finds them pale and over-inflated from the great efforts which have been made at inspiration during life, whilst the Right Ventricle of the Heart is dilated.

The appearance of the portion of Lung under the influence of Embolism was that known as an "Infarct" and was described as an area of tissue from which the Blood supply had been cut off, and being surrounded by a zone of intense hyperemia which resulted from the extra strain thrown upon the collateral vessels and being very characteristic. If there was any deficiency in the
Collateral Circulation, the vessels in the embolic area allowed re-circulation and hemorrhage to occur and the lesion became infiltrated with blood constituting an "hemorrhagic infarct."

One of the terminations of an infarct is Gangrene of the Lung of circumscribed extent and is due to the obstruction of the arterial branches supplying the particular part.

Jones and Skevington think that Gangrene of the Lung only supervenes when the infarction is of large size, and may at the same time be connected with some simultaneous obstruction of the Bronchial artery.

In the case of septic emboli originating say in the Cerebral lesions as in Otitis and becoming impacted in the Lung, we get gangrene and destruction of the Lung tissue and
Constituting Dynamic Abscesses. Nicksch found that Caoutchouc emboli introduced into the right side circulation through the jugular vein did not, in the dog, occasion any particular change when impacted in the lungs, Panum Cohrlein and Sethen showed artificial emboli to become encapsulated when introduced into the pulmonary circulation. According to Hamilton, infarction in the lungs is a complication from rupture of the alveolar capillaries from backward pressure arising from heart disease, the peculiar wedge shape being due to the shape of the air vessels and the distribution of the bronchi and not in any way connected with the distribution of blood vessels. Hemorrhagic infarctions of the lungs are usually peripheral and vary in size, then situated near the pleura they are wedge shaped.
but, when near the root of the lung they are somewhat rounded. They project upon the pleural surface, and are hard or tough, and of a purple leaden colour; the air vesicles are full of blood, and the interstices are compressed. Cohnheim stated that the 'infused blood in hemorrhagic effusions contained its fibrin, but Hamilton says—yes! and that a fibrinous network can often be detected within the air vesicles, the hard pulmonary feeling of the effusion indicating certain coagulation of the infused blood.

There is no evidence of the passage in the pieces of lung tissue implicated. Large half crystalline masses of hemoglobin separate soon after the hemorrhage has occurred. This infused blood is ultimately absorbed, leaving
no cicatraces, and there is moreover no tendency for these Infections to become Cheesy, nor to Slough, nor constitute pyemic abscesses unless of septic origin.

In Leuekemia cases of body-shaped Infections are quoted by Mr. Bryant who in 1866 excised a patient's Spleen who died after operation, post mortem the patches are said to have been found situated in the back portions of the Lungs they had distinctly fungous centres and red borders.

Dr. Power mentions Infections arising from plugging of the Pulmonary Capillaries by Leucocytes.

Hamilton mentions the fact of micro-organisms multiplying in surrounding spaces (septic Emboli) and resembling Croupous pneumonia, these collections being generally on the surface of the Lungs and where owing to a tone-
That limited arterial anastomosis they may cause local death, thus differing with their behaviour in the muscular system where the anastomosis is free.

Hemorrhagic suffocation of the lung has been explained by Kirchfleisch through the bursting or rupture of an artery. Upon the occurrence of the rupture he says the blood issues its way into a neighbouring bronchus with all the force of the abnormal pressure then existing in the lesser circulation. Thence by aspiration it is drawn into the corresponding lobule, filling it to the very last vessel. On the other hand the blood also rises in the bronchus and as the accumulation reaches the mouth of the bronchi it is sucked into them. Coagulation of the extravasated blood finally takes place, thus this formidable process is brought to an end.
To quote D'Arcy's Dictionary of Medicine: "When a branch of the Pulmonary Artery becomes obstructed by an embolus for instance by a fragment of conglutinum conveyed from the right auricle or one of the systemic veins, its territory becomes at once filled with Blood in the following manner. The Pulmonary Arterial Vessels do not communicate with one another—each branching separately to its capillary distribution—the Pulmonary Vessels on the contrary insensible freely, and moreover are not provided with valves. Thus when the outflow of Blood is arrested through the obstructed arterial branch various regurgitation through the capillaries from collateral pressure fills up the precluded vascular area with stagnating blood and the intra-alveolar tissues become speedily occupied with its effused corpuscles."
In some cases the vessels may not at first be accurately closed by the embolus, the onward current is then retarded instead of being quite arrested. The mechanism is however the same. In the liver according to Coats the Portal vein is in its distribution and end artery, but infarction does not occur from plugging. Embolism of the Hepatic artery is not of much consequence as it is a terminal vessel so that restoration of the circulation is rapid through anastomosis. If however the embolus happens to the Hepatic infarction has been observed in a few cases. Recklinghausen has in a few cases noticed Embolism of the Hepatic vein by backward passage by gravitation from the Inferior Vena Cava. Cohnheim and Letter died A. Y. 1878 p. 153 say that in the liver no changes
Take place, for the Capillaries of the Hepatic Acini are fed not merely by the Portal Vein but also by the Hepatic Artery. The Capillaries of the latter unite to form small veins which enter the Venules Intertubulariae. If now the entrance of Portal Blood into one or more Intertubular Veins be for any reason prevented there still remain the Intimal tributaries of the Portal Vein the supply through which is sufficient to avert necrosis of the organ and all severe disturbance of the circulation in the Hepatic Lobules.

As mentioned above, we may meet with Infarcts in the Liver giving rise to abscesses. These Emboli may be Pyemic or other wise. According to Rautheisen in the Embolic variety the portion of Hepatic tissue corresponding.
The distribution of the plugged vessel becomes intense congested and the circulation becoming entirely arrested it enlarges in mass.

In Pyemia Burch has observed that in liver abscesses retrograde movements of the blood from the right heart take place in the Vena Cava and in this way hepatic emboli may arise.

Gangrenous dysentery set up by embolism of the Portal vein Gangrene of the liver. In Pyemia also are found multiple abscesses of the liver of embolic origin so also in Pyemia following injuries to the Rectum do we find abscesses in the liver owing to the blood from the Rectum entering the Portal system of veins.

Malignant growths in the liver are usually secondary to some growth in the Stomach, Rectum.
or other distant organ but in somewhat rare instances they are met with as primary growths. Whether primary or secondary Carcinoma is most frequently met with of the malignant tumours. It is in the Liver that we most frequently find the Cystic form of some of the Intestinal parasites and notably that of the Taenia Echinocecum, and in all probability its frequency there may be explained by the fact that the Scolices in its wanderings gets into the Portal Circulation and being carried off by the Blood Stream is finally arrested in one of the Capillary Branches of the Portal Vein within the Liver. In the Kidney's Infarctions or Blocks are frequently met with. There is never the same stuffing or forcing of the Tissues with Blood such as he
meet with in the body, and a recent
Infarct may even be paler in color
than the surrounding parts. There
not being much liquefaction, the
Infarct shows as pale yellow de-
pressions — of a wedge shape with
the apex to the hilum of the organ.
The appearances of Infarct vary
in the Kidney, sometimes — and
especially if recent they may
be as distinct elevations upon
the surface of the Kidney and of
a dark red colour. They are
not however usually accompanied
by haemorrhage, unless in the
zone of reaction, which surrounds
them. The subsequent changes
which follow are degenerative;
forming a tough yellow fibrous
mass which ultimately becomes
fibrous tissue proper, this contracts,
becomes paler in colour, and
denser in structure, so causing
the characteristic punctate cicatrix
on the surface of the organ. (Hamilton)
Costs in his Pathology says that microscopically examined the kidney shows the outline of the tubes, the Malpighian bodies etc. In the strand of dead tissue are seen to be preserved distinctly enough even when it has a yellow colour. These are however extremely granular there has been a precipitation of Albumen within it a coagulative necrosis. Later on it becomes caseous and the outline of its elements are consequently blurred. The next zone is a zone of reaction the vessels within it are engorged and many of them will be found to have ruptured. Within it there is thrown out in the course of time a little cicatricial tissue. The dead piece of kidney substance becomes absorbed from the margin forwards and as its removal is taking place the cicatrix in the reaction zone contracts. A little
Calcareous matter is sometimes left after the absorption of the dead tissue has been completed and this finally becomes embedded in the cicatrices. Lister says the true septicemia or congestion is due to anastomosis of vessels pouring blood through the capsule and not as Cohnheim said to venous regurgitation. He has found necrosis of the renal epithelium two hours after he had ligatured the renal artery. He has also shown that the vessels in which the circulatation has been arrested retain their integrity much longer than was supposed by Cohnheim, and that the necrosis of the renal epithelium occurs long before the blood vessels themselves suffer. The nuclei of the cells disappear, their protoplasm coagulates or they become converted into softer gelatinous masses (coagulative necrosis) which have a remarkable tendency
to calcification. Hence the wedge-shaped white emboli of the type in the kidney are not produced by dislocation of hemorrhagic infarctions but are simply the result of the necrosis of the epithelium and the halo of injection which is seen surrounded by a due to inflammatory congestion caused by the presence of the necrotic patch. Plugging of the whole renal artery has occurred with very different results in different cases. In one case mentioned by Cohnheim the considered the effect to be a necrosis of the organ.

Herman Heber states the time occupied in the changes which are necessary for the production of a depressed cicatrix to be about thirty-eight days, while for the formation of a slightly developed eminence on the surface the time was ten days (Trans. Pathol. Soc. Vol. XIX, p. 166).
Pechlinghausen quotes a case which bears out the above statement of Beber. Where thrombosis of the renal artery resulting from injury gave rise to a yellow disfunction of the organ within eight days.

Dr. Charlewood Turner has found in many cases the papilla and portions of the Pyramids to be necrotic, and partially detached. This has been due to a thrombotic obstruction of the arterioles, or defect in the general nutrition. He regarded the necrotic portions of the Pyramids as analogous to the parts in the cortex which had undergone absorption, the difference in the process being due probably to the urinary secretion.

Witts and Hoax mention a case of obstructed renal artery where Bright's disease of both kidneys was already present; the arterial obstruction in this case was subclavian.
with anti-mortem clot from the
Left Ventricle. As Rich has also
shown granular disease of the
Kidney as the result of such an
Obstruction.
Jones and Siewert mention
Capillary EmboIosis as often
being regarded as Pneumonic
but not being necessarily so,
occuring generally in connection
with Acute Endocarditis.
Collesheim objects to the term
Capillary EmboIosis on the
ground that small arteries
are always blocked.
When the compaction occurs
owing to the surrounding hyper
emia the tubules may become
infiltrated with Blood thus
firing rise to the form of Hemal
Nephritis. Kiimeyer calls this process the
metastatic hepatitis of heart disease
and according to him a peculiar
form of renal hemorrhage

In the case of Pyemic Emboli of the Kidneys we meet with the metastatic abscesses usually in the Cortex, they are often multiple and surrounded with a thin zone of hyperemic tissue and are dependent upon arrest of circulation in a portion of the organ, this condition is very common in aortic disease and acute endocarditis, in some cases the cause is spontaneous thrombosis.

Gould in his Dictionary of Medicine mentions abscesses caused by lactic acid the deposition of which is found in the renal tubules of the newborn. Malignant Emboli may arise from any primary growth to giving rise to secondary growths in the Kidneys — notably from the testes. These secondary growths usually commence as renal nodules commonly in both Kidneys at once.
As the growth of the secondary deposits goes on all traces of renal structure are obliterated and the kidney is converted into a simple carcinomaous mass containing as time goes on—growing tissue, patches of Caucous fatty degeneration hemorrhagic effusious and tracts of liquefaction. According to Darwin the kidney comes next to the lungs in order of frequency among those organs liable to be affected by embolism. Embolism occurs is perhaps the most common form and most likely reaches this habitat through the blood stream. The spleen next to the lungs is most commonly affected by embolism this perhaps being due to the large size of the splenic artery. In the spleen we perhaps meet with the most characteristic appearances of "Infarction" owing to the anatomical arrangement of the vessels. This
Vessels forming what is known as a Pneumothorax and being very distinctly wedge-shaped, the apex of the wedge pointing to the hilus of the Spleen.

The cause of the Embolus may be disease of the Vascular apparatus of the heart or some other readily defined cause, but in many cases the stoppage is due to some spontaneous coagulation in the Vessels from diseased intima or some wounded condition of the Blood itself giving rise to spontaneous coagulation. This last condition may be readily supposed in the case of typhus fever where from sheer weakness there is a retardation of the Blood current. Hardell says that in inflammation of the Spleen the cause may be in the more important defects of the Blood itself or the relative quality or quantity of the
of the Corporacles, the tendency
to depositions of Fibrinose
corpora, and the liability
which there is to Congene
and tissue degeneration from
Embolism in either the Pulmonary
or Systemic circulation.

In obstruction: Heart disease the
Spleen is dark and hard, but
in Endocarditis with ulceration
it is generally large and soft.
Enlargement accompanies In-
farction and may be detected
by friction during life.

Once formed however, the Infarction
goes through the usual course
and ultimately forms — a cicatrix — we may find Cal-
fication of this cicatrix.

Embolic Infarctions appear on
the surface of the organ as pale
patches abruptly circumscribed
and generally situated on the
anterio thinner border of the
Spleen. If the Infarction does not
form a cicatrix, but go on to suppuration it may give rise to capsular peritonitis (waller's horn). Splenic cancer is never found alone which fact was first mentioned by Jenner. Malignant emboli of the spleen may be said to be very rare as shown by the fact that malignant secondary growths in the spleen are rare. When however they are met with they then arise from some abdominal viscera. Hydatid cysts are sometimes found in the spleen.

The blockage caused by altered blood pigment has been called microscopic embolism and many recent examples have been given. Virchow, Arscle, and Plauer all mention cases of this. In malaria there is splenic hyperemia and consequent destruction of red blood corpuscles, Freichs says that in this hyperemia of the spleen the
The stagnation is exceedingly great, and as a result there is the formation of large masses of pigment. Rindfleisch mentions pigmentation as occurring in the intravascular cords of the Spleen Pulp where the Blood flows most slowly and as the Intravascular Cords are not shut off from the cavernous veins by an impermeable membrane, filtration goes on between the arterioles and the venous radicles and in this way pigment flakes pass into the Blood. In those organs in which the Capillaries are the narrowest, pigment deposits are most marked.

In a pyemic embolus of the Spleen the deposit is well defined and always near the periphery with a well defined ring of reactive inflammation around it. The termination of the process may be simply the formation of a fibrous necrotic or there may be
formed a puriform creamy mass, which Rokitansky called Secondary Splenics, or a curious greenish brown or chocolate coloured pulp. True pyogenic abscess seem to differ from simple blocks in the rapidity of suppuration and in their concentric irregular shape.

Dr. Tyler quoted by Wilks & Knox has described micrococci in the vegetations from ulcerative endocarditis found in Spleenic infarctions to giving rise to secondary abscesses.

The above order of sequences is doubtless an etiological explanation of those inflammatory patches and purulent collections of smaller or larger extent which were by some of the older writers, Louis Abercrombie & others considered as examples of ischemic inflammation of the Spleen and of those consolidations which Rokitansky referred to the doctrine
of crisis.

Sachkovsky found that when he divided some of the nerves of the spleen, the pathological changes called hemorrhagic infarcts sometimes occurred in the parts of the spleen supplied by the divided nerves.

According to Siemens, the infarctions under the microscope reveal the persistence of the splenic tissue in them and the gradual disintegration of it by a fatty transformation. Whilst a quantity of leucocytic crystals always form in the infarctions, the presence of these blood crystals together with the superficial seat and wedgelike shape of the patches form the chief means of detecting making hemorrhagic infarctions from the local diseases of the spleen, such as tubercles or Hodgkin's deposits, etc.

Enlarging of the mesenteric artery,

The sloughing of the bowel & consequent
Tumors may cause death, but on the other hand a cicatrix may form or be left from the sloughing and recovery take place under what seem to have been very disadvantageous circumstances.

In Embolism of the Superior Mesenteric artery we find the ulcer known as the Embolic ulcer of Perniechi, there have however been only a few cases of this verified by post mortem examination. Embolism of an ultimate branch of the mesenteric artery with secondary thrombosis obstructing the vessels on either side will lead to an infarcted state of a small segment of bowel with tumor into its lumen, but embolism of the main trunk will cause gangrene of the whole intestine.

The portion of bowel which is affected is at first of an intense red colour gradually increasing to purple
and even to black. The extreme congestion of the vessels leads to the effusion of blood into the tissues which are uniformly coloured, decomposition rapidly takes place in the stagnating blood, and the products acted on by the sulphured Hydrogen of the Intestines become black, all traces of red colour soon becoming lost. Meanwhile the mucous membrane and muscular coats are swollen and swollen by the serum and blood with which they are infiltrated and a dark black to ash gray soft pulpy mass is finally thrown off from the healthy tissue.

Defend mentionous embolism of the mesenteric arterie in this experiment - Chin: Review 1871. Venice thinks embolus is a very common cause of ulcers of the stomuch and Intestine and this has been demonstrated by Ranuus experiments.
Further ligatured the Vena Portae and showed extravasations into the mucous and submucous tissues with subsequent ulceration. Both the shape and the most common positions of these ulcers point in many cases to their arterial origin, and the conical form with the base of the mucous surface corresponds to the ramifications of the capillary branches spreading outwards from a main trunk which has been obstructed in a deeper tissue, and thus resembling precisely the results of arterial embolism in other parts. When similar appearances present, Ehrlich Arch f. p. 287 has directly shown that the pressure under which the blood normally flows in the arteries is after closure of the superior mesenteric artery not even approximately capable of supplying the customary and necessary amount of blood to the small intestine through the
Duodenal, pancreatic duodenal and the colic branches of the superior mesenteric.

In the intestine we have great susceptibility to injury from an insufficient blood supply. If the circulation through the intestine cease or be greatly diminished even for a few hours its re-establishment may be no longer possible, but even if it should be restored the nutrition of the intestinal wall has generally suffered so much that Gangrene is no longer to be averted. It is therefore at once manifest that in Embolism of the mesenteric artery the intestine is not secure from necrosis by the existence of collateral circulation.

In the Stomach Poliostomek was the first to suggest the origin of Ulceration in hemorhagic erosion. Virchow considers that in Ulceration of the Stomach the ulcers originate
mainly in affectations of the vessels connected with the diseased area especially embolism or degenerative change in the arteries attended with arrest of circulation and necrosis or obstruction of branches of the Portal system of veins followed by interstitial hemorrhage.

Panum has attempted to corroborate Virchow's theory by this experiments of injecting dye into the abdominal aorta of dogs. He found that when the dye entered the arteries of the stomach the mucous membrane ulcerated.

It is open to question whether ulceration of the stomach is caused by arrest of the circulation at its commencement and begins as a hemorrhagic erosion - if so - then ulceration of the gastric mucous membrane should be very frequent as in those suffering from obstruction of the Portal circulation arising
from disease of the heart or liver, moreover, there should be more frequent traces of alteration from Embolism considering the frequency of such trances in other internal organs.

The Embolism of the Arteria Centralis retina the immediate effects are to render the Retina opaque and of a milky whiteness except over the Macula Lutea where the absence of Connective tissue prevents such change. Stedman in the International Encyclopedia of Medical Science quotes John who says that septic Embolus of the Arteria Centralis retina may cause Panophthalmitis. In Embolism of the arteria Centralis retina the functions of the Eye are destroyed and blindness results which is usually permanent from necrosis and softening of the Retina both sometimes accompanying tumor and atrophy of the optic nerve.

The usual situation of an Embolus
is just behind the Lumina Orbrosa.
In some cases the Clot is formed in the Artery itself and then it may be more properly considered under the heading of Thrombosis. Sudden occlusion of a coronary artery may give rise to softening and rupture of the heart wall or it may lead to the destruction of the contractile substance of that portion of the heart which is fed by the affected artery and afterwards to the formation there of so called pyo-carditic indurations.

Boeckart (Virchow's Archiv: Vol. 77) was perhaps the first to point out that changes analogous to those arising in other organs from Infarction occurred in the heart giving rise to what may be termed fibroid disease.

Hütter found dry greenish or yellowed patches in the heart substance which however still showed under the -
Microscope the structure of the muscular tissue which contrasted strongly with others which exhibited little or no structure and some fatty degeneration. It is possible that the above changes occur before the fibroid development.

Hülter traces these changes to obstruction of branches of the coronary arteries sometimes from Thrombosis and sometimes from Embolism.

Lochmann Legg (Brattle, Lecture 1883) doubts Hülter's views and opines that the changes in the coronary arteries were only such as might be found in them at the patient's age.

Richard Quain in his Lumbarian Lectures mentions embolic abscess of the heart as being usually found in boys with injuries to their bones or disease of their joints or bones.
Pyramie abscess of the heart may arise from left the embolus in the same way as in any other internal organ. Mitchell Bruce Laurin's Dict 1876 Vol:1 P:838 mentions pyramie abscess bursting either into the ventricles or causing further pyramie abscesses or else into pericardium.