Congestion & Anaemia of the Brain
Remarks meant as introduction to the Thesis

The student of medicine when studying for his professional examinations thinks it useless to commit to memory minute histological and anatomical descriptions which he finds in his text books. The text books of anatomy and histology do not tell him the exact signification of the detailed accounts which they contain in a particular point of a subject. But in after years when the student has become a busy practitioner when he is face to face with the great battle of professional life—when he finds that nothing in medicine nor surgery can be advanced with the satisfaction of a true scientist without special regard to those minute points which he once in his day dreamed thought useless for the University Curriculum.

It is the same thing with an author of Exerciser—he also must grasp anatomical details. Therefore we shall begin by giving a brief description of anatomical and physiological points, which is necessary for a distinct understanding of the subject under discussion.

The Circulation of the Brain

The Circle of Willis which is formed by the anastomoses of the two internal Carotides and the anastomosis of the branches of the basilar, supplies the brain by its numerous branches. But the brain circulation must not be regarded as merely an ordinary Circulation. It is peculiar in several respects, which will shortly be noticed. But taking the teaching of Charcot as a generally accepted view, we will briefly summarize our opinions as follows:
Diagram of the arterial circulation at the base of the brain (After Charcot).
Circle of Willis

The Circle of Willis is bounded behind by two posterior cerebral arteries, on either side is the posterior communicating branch, which establishes communication between middle cerebral and posterior cerebral arteries. In front the internal carotids terminate, the terminations are broken up into two branches, the internal branch of which passes through the Sylvian fissure to be distributed to the side of vertex of the brain, the internal branch lies close to the corpus callosum, it is known as the anterior cerebral artery. From the main vessels of this Circle six groups of vessels are given off, which lie within or behind the hemispheres.

Charles has divided them into:

1. Anterior median group of ganglionic branches
2. Posterior median group
3. Right anterior lateral group
4. Left anterior lateral group
5. Right posterior lateral group
6. Left posterior lateral group.

The anterior cerebral, the middle cerebral and the posterior cerebral besides giving the above mentioned branches terminates in fine ramifications in the brain matter. Hence it is said that the Circle of Willis gives two systems of vessels.

1. Central Ganglionic System
2. Cortical System

The Central Ganglionic system gives off the six groups mentioned above. The anterior median group comes from anterior cerebral and anterior communicating. The posterior median group is from the posterior cerebral and anterior communicating. Anterior lateral groups of right and left sides are derived from the middle cerebral.
Distribution of arterial arteries (arterial network)

Parietal arteries

Gastric arteries

Colonic arteries

Pancreatic arteries

Biliary arteries

Reserve arteries

Venous network
Distribution of Middle Arterial artery (after Charcot).
The perforating cerebral groups of the right shift come from the fossa
lateralis cerebri. The vessels belong to this system are called
"terminal vessels" which neither supply nor receive arteriolar
branches.

**The Cortical System.** It is formed by terminal branches of Ar-
terial, middle & perforating cerebral arteries. These vessels enter the Ri-
malis and give off nutrient arteries which perforate the Cortex of the brain
in a perpendicular manner. The nutrient vessels may be divided into 2 groups.

**First, the Medullary arteries.**

Passing through the grey matter they penetrate into the centrum
ovale. They intercommunicate by way of fine Capillaries & thus act as if
were from independent & small systems by themselves

**Second, the Cortical arteries.**

Strictly speaking the vessels are not terminal vessels, with the
medullary vessels they form a network in the middle zone of
grey substance. Thus the middle zone of grey substance is richly
supplied with blood as compared with its outer grey zone.

It must be noted that vessels of one area cannot be injected
through the vessels of another area as a rule. Therefore obstruction
of main branch will cut off circulation in the area supplied
by the vessel & thus producing local brain degeneration in the area
of cerebral Cortex.

A few words about middle cerebral artery

The middle cerebral artery, the artery of cerebral haemorrhage,
supplies the motor area. Entering the Sylvian fissure it gives
branches to the insula of Reil through the anterior perforated
space to the corpus striatum & to those parts of the frontal &
parietal lobes adjacent to the fissure of Rolando. Thus when the
main artery of the left side is plugged, this is right hemisphere &
aphasia, wherein the right vessel is plugged. There is left hemiplegia.
when a branch only is blocked, the motor paralysis is partial.
openings temporary, as the anastomotic branches of the left
middle artery due to time repair the lesion. The left middle cerebral
is said to be more often plugged than the right, because, it is ar-
gued, a vegetation is more likely to pass with the blood stream
into it than into that of the right side. This statement is not how-
er, borne out by statistics: probably more left cases are reported
because of the interesting clinical features — aphasia being
present. Acute rhematism, gout, arteriosclerosis, poliomyelitis,
granular disease of the kidney, syphilis or injury, are the
chief causes of disturbance of arterial circulation in the
medial motor area. And he who knows himself to be the sub-
ject of one or more of these conditions should try to avoid ex-
travagant exertion, such as running to catch a train, straining at stool, 
so on.

The common site of cerebral haemorrhage is in the neighborhood
of the corpus striatum, a vessel from the middle cerebral
which runs straight up from the beginning of the middle cerebral
artery into the lentiform nucleus; probably it is
the direction of the course of these branches from the main
trunk which causes them to burst, under the shock of the
ventricular contraction.

The posterior communicating artery passes from the base of
the internal carotid to join the posterior cerebral. Just then
also the carotid gives off the anterior choroidal artery, which
enters the descending cornu of the lateral ventricle, and
supply the hippocampus, the choroid plexus.

* Tillmann - Traite d'anatomie topographique avec
applications à la chirurgie

Principles of the Circulation of the Brain.

The brain is a very active and very delicate organ. It is important therefore that it should receive an abundant supply of blood (which it does), that it should not be subjected to shocks from the surrounding vessels or to pressure from overfull veins. These last ends are attained by:

1. The External Carotids of the Anterior and Middle Carotids, which carry blood to the brain.

2. The carotids joining dividing into the three branches before entering the brain substance.

3. The Central spinal fluid or the Continuity of the Sub-Anterior space of the Cranial Cavity or the Spinal Canal. This Continuity is of great importance, because by this means the risk of pressure on the brain substance is obviated as is explained further on.

Physiological Points in Connection

With Nerve Tissue generally

Nervous Substance depends principally for its nutrition on the proper supply of blood. Active nerve substance absorbs oxygen and eliminates Carbonic acid gas. The chemical substances which may be produced by the decomposition of nervous matter during a state of activity are quite unknown.

One of the conditions of nervous excitability is that there be a period of repose or activity, a high state of activity though
lasting only for a short time, prolonged activity soon produces a feeling of fatigue, if too long continued may abolish excitability altogether. On the other hand, a lengthened period of absolute rest improves excitability; if this state be continued too long, there may be wasting and degeneration of the nerve.

Heat up to a certain temperature, augments whilst cold diminishes the excitability. A deficient supply of blood diminishes the excitability. Slight drying increases excitability, as may be frequently seen in the nerve muscle preparation used in physiological experiments. Potash, potash salts, the acids or abolish, whilst a weak solution of common salt conserves excitability. According to Eckhardt, it is independent of oxygen. But this theory is not accepted by physiologists. But whether Eckhardt's view is right or wrong it is certain, in any case, that the functional processes of nerve change a nerve fibre depend upon blood which contains oxygen. Carbon dioxide and nitrogen in their normal proportions.

* Muller's Physiology by Budy. Vol I

Nouveaux Elements de Physiologie Humaine. Beaux...
Consideration from anatomical point of view

In thinking over the anatomical facts in connexion with the construction of the cranium, it will be observed that the cranial wall is indifferent to pressure from without or within. This is due to complete ossification of bones of the cranial vault. But there is an...other facts, the importance of which can scarcely be exaggerated; it is the existence of a fluid made around the cerebro-spinal axis.

The fluid which is enclosed within the cranial vault—a fluid, indeed, to the well known law of physics relating to liquids—is nearly incompressible to any pressure which may be exerted upon it.

* Traité pratique d'anatomie chirurgicale: Richet 1888

1. Intracranialisation of Munro-Kellie Theory

In 1783 Munro-Kellie recognised these facts. They formulated a certain theory. It was this after maintained that their views were, that the accumulation of an abnormal quantity of blood within the cranium is an impossibility in view of the anatomical conditions of the brain. But the statement is absurd; moreover, it is not in accordance with the views which these two observers meant to imply. They did not say, they could not by any possibility an abnormal quantity of blood enter the brain, but what they maintained was not the quantity of blood which the cranial vault contains is not susceptible to any variations.

Observations on the histological facts associated with Encephalothini

There exists in the Encephalothini a circulus by which the pressure arising from any increase of the circulus fluid is eliminated. The sub-arachnoid space of the spinal cord is in direct communication with the sub-arachnoid space of the cerebral hemispheres. Therefore if from any cause there enters an abnormal quantity of fluid into the vessels of the two hemispheres, a corresponding quantity of Cerebrospinal fluid passes down into the sub-arachnoid space of the spinal canal, thus avoiding pressure to which otherwise the brain mass necessarily would be subjected. Between the tunicia adventitia and tunicia proper of blood vessels is the lymphatic sheath of Batini; between the outer tunicia and the cerebral hemisphere there is a space although apparent only in a state of strain which is known as the space of the dip.

But more than this. Researches of Kuyers and Paez have shown that all around the nervous elements of brain substance there is a sheath of lymphatic tissue. Thus it happens that the hemisphere is as it were filled up by a fluid network which yields to the increased variations of liquid in the cranial cavity. Donders asserted that under the influence of inspiratory pressure the vessels of the brain must show variations in their calibres. This statement has been verified by Giacomini and Morel and Frankel. By these observers the vessels have been seen to dilate, from 0.1 mm. to 0.14 and 0.07 to 0.16. It is clear then that in spite of the special physical conditions which rule over the Central Circulation, the capillary meshes of Encephalothini are susceptible to be increased with an excess of fluid. When this happens it will of course
Constituted Cerebral Congestion.

Further proof of the relation of Blood to Encephalon.

Cerebral spinal fluids.

We can go further to say that there are other evidences to show that there exists an intimate relation between Cerebro-spinal fluid and blood of the hemispheres: observe the diminution of the amount of blood in Hydrocephalus, which is generally coincident with Cerebral Anemia: observe that it is in atrophy of the brain that the blood & Cerebro-spinal fluid show a corresponding augmentation: observe that in the hypertrophy of Cerebral hemispheres when some kind of pathological condition has diminished the capacity of the Cranial Cavity, that the blood & Cerebro-spinal fluid undergo a simultaneous diminution.

Causes of Cerebral Congestion

It may be classified into two classes

1. Participating or Remote Causes

2. Exciting or Immediate Causes

1. Participating Causes

Heredity has been considered as one of the participating Causes.

The disease is more common in men & more frequent in the
2. **Exciting or immediate Causes**

1. Increased pressure in the arterial system more especially an increased blood pressure in Carotid & Vertebral arteries.
2. Compression of thoracic portion of aorta or of abdominal aorta or of any other portion which prevents circulation to other parts of the body leads to an increased & excessive flow into the Central vessels.
The sudden cessation of habitual flows, such as menstrual flows, the bleeding from the haemorrhoids or the piles, are all prone to be determined causes.

(3) Contraction of peripheral vessels caused by the cold stage of intermittent fever or by the severity of a winter's cold. Watton relates of cases of death occurring during severity of winter in London. But the congestion caused by cold weather offers to us phenomena which are too complex to be entered into here. It is sufficient to mention that besides the cutaneous contraction of the peripheral capillaries, the warm motor centre loses its equilibrium by the irritation which reaches the by impressions conveyed along the sensory nerves of the skin.


(4) The most ordinary causes of cerebral congestion which are due to irritation, are, protracted watching, mental exhaustion, exposure to the sun, and all those conditions which produce gouty diathesis or fevers of specific origin. It may be noted however that congestion produced by pyrexia presents rather the character of passive hyperemia rather than that of the active variety.

* Cerebral hyperemia the result of mental strain or emotional disturbances. New York 1877.

(5) With regard to fevers, Malarial fever & acute Mania, have been specially mentioned as active agencies in producing the disease.

(6) Drugs such as opium & alcohol have been said to produce cerebral congestion by dilating the blood vessels or causing irritation of vascular walls. In the case of alcohol the fact may have certain resemblance of truth. But with
regard to opium, it is still a matter of uncertainty. Recent
observations & experience have shown us that large doses of
opium cause cerebral anaemia & not hyperaemia. The
Cerebral hyperaemia which has been observed in certain
cases of post-mortem examination is due mainly to the
slowing of respiratory & circulatory movements, the agency
being of long duration in poisonous doses of these drugs.

(viii) Faults of Vaso. Motor Mechanism

Congestion due to disturbances of
Vaso. Motor Mechanism

Congestion of Cerebrum Caused principally by Vaso. Motor
Mechanism is rare. Because the dilatation & contraction
of Capillary blood vessels are necessary to produce
this condition. But the nervous influences by which this
must be done can not primarily be brought to bear upon
these minute vessels. According to Kolliker the Smallest
Capillaries are destitute of Vaso. Motor nerves. He believes
the Capillaries which are larger than ulterior Capillaries
or arterioles are supplied with Vaso. Motor nerves. The
initial seat of dilatation is on these vessels. These Causes
which may bring about this condition may be enumerated
as the digestive processes, moral emotions, Synaptes,
turns & cutaneous irritation.

* Consideration sur l'ischemie et l'hyperaemie Cerebrale
These de Paris 1879

Seeing - Cerebral Congestion. Medical Times 1880.
The Physiological Explanation of Congestion

Due to Nerve Action

Excitation carried by nerve terminations such as by gastric filaments of vagus, by cutaneous filaments of sensory nerves, or even by processes set up in the cells of cerebral convolutions is brought to the vasomotor centre situated in the bulbous portion of cerebro spinal axis. Influences thus brought, react upon the centre. From this centre impression irradicating by nerve plexus going to the vascular area of the cerebral hemisphere dilates the minute vessels producing active congestion. It often happens that the dilating impression is sent out only to cerebrum, but at the same time stimulus passes out to the area of distribution of facial and trigeminal nerves causing dilatation of the face and conjunctiva.

* Sur la distribution des artères nourricières du bulbe rachidien (Arch. de Physiol. Normale et pathol. 1873).
  Disorders of Cerebral Circulation. London 1876.
  Veillet Pratique d'anatomie chirurgicale Paris 1835.

Passive Congestion

The causes which will produce passive congestion can be easily suggested by remembering the physiological and anatomical details.

They are as follows:

1. Compression of jugular veins, or of Superior Vena Cava
2. Various tumours of cervical & thoracic region, preventing
The return of blood from cerebral hemisphere towards the heart

iii. Violent respiratory efforts such as is produced by severe and prolonged coughing, playing upon wind instruments, singing or general convulsions

iv. Contraction caused by the umbilical cord round the neck of the newborn

v. The tricuspid insufficiency, which disturbs the equilibrium of caval circulation

vi. The stenosis of mitral valve is believed by some authorities to be one of the factors

vii. Emphysema & Pulmonary sclerosis. Conditions such as those which bring about the dilatation of the right ventricle & tricuspid insufficiency; and here is the explanation why in the chronic diseases of the lung a passive congestion of the encephalon may be expected

viii. The atelecasis of the newborn

ix. The Compression of the inferior Vena Cava:

When the inferior Vena Cava is compressed, the increased flow of blood through the Azygos Veins disturbs the return of blood by the Vena Cava Superior. But there have been found cases in which partial obliteration of Vena Cava inferior has not produced this effect.

x. The arteriosclerotic & calcareous degeneration, which deprives the arteries of their elasticity, ultimately leads to the weakening of cardiac impulses, which may end in cerebral congestion. The slight paralysis of the heart & physical & chemical modifications which the blood undergoes may also be considered as some of the causes. In cholera for example the conditions are more or less
Coincident, hence passive congestion of the brain is a
frequent occurrence in this disease.

* On disorders of the Cerebral Circulation

Burrows London 1886.

Remarkable Case of Meningeal Congestion without inflammation
British Medical Journal 1873.

On Cerebral Congestion: Roland London med. gazette 1846

Pathology & Morbid Anatomy

Phenomena which may cause error in diagnosis

It is better to keep in mind the following facts:

1. The existing congestion after death may become less
or even disappear.

2. On the other hand the Cerebral vessels may become
stiffer after death. In such cases it is better to examine
Carefully the state of the Ventricles. In Congestion the
Ventricles also will be found congested.

3. If the body has been lying in dorsal decubitus,
the occipital region will be found surcharged
with blood. Bear in mind this is only a post
mortem appearance.

4. Prolonged death agony. Various kinds of asphyxia
may produce cerebral congestion.

It may here be noted that the congestion which
is pathological & congestion which is cadaveric
may easily be distinguished. In the former case
the arteries within the Cerebral tissue are in a
state of turgidity, whereas in the latter case the
lesion is found only on the membrane & surface.
Comparative Vascularity of Blood

Vessels of Cerebrum

Let us observe for a moment the distribution of blood vessels in these regions which will lead to an exact understanding of the pathological condition.

1. The grey matter contains more blood vessels than the white substance.

2. The Corpus Striatum contains a large number of blood vessels in its cortical zone. According to Morgagni its transverse section will show one or two blood vessels even of considerable diameters.

3. The optic Thalamus also is supplied with a considerable number of blood vessels although in a lesser degree as compared with Corpus Striatum.

4. The Corpus Callosum may be mentioned next in order.

5. The Cerebellum is more vascular at the periphery of the area of Corpus Denticulatum.

6. The Proliferation is also vascular.

7. Notice must also be taken that the infant brain is much more vascular than that of an adult or of the old.

The Localisation of Cerebral Hyperaemia.

The exact position of cerebral hyperaemia is different in different cases. Ordinarily it is found in the substance of the two hemispheres. But one cerebral
hemisphere may be more affected than the other. The same irregularity is found as to the localities when the vessels of the peritoneum and meninges are involved. But whatever may be the condition it can be easily stripped off the nervous tissue. Another important fact is that in Simple Conestion the membrane is never adherent. With the Cerebral hyperseemia there may be found inflammation in the deeper membrane of the eye-ball.

Pathological Changes which are seen
in post mortem examination

The vessels of diastis are congested. The meninges contain more or less dark blood which runs out in abundance. The vessels of the brain are turgid, the brain surface is seen to be injected. The injected vessels may be traced within the substance of the cerebral hemispheres. The hemispheres assume firmer consistence. If they now be cut, the cut surfaces will present punctiform spots which are the gaping mouths of cut vessels.Ether believes that these vessels dilate double of their normal capacity. In a very pronounced hyperseemia there is a transudation in the Pia Materia very often in the cerebral tissue.

The increase of fluid within the Cavities of the ventricles and also in the sub-arachnoid space is not found in acute Congestion. It is a result of Chronic Congestion.

The vessels are distended, tortuous, crowded, compressed. The cut surface looks like the mouths of little open tubes.
This is the acute appearance of Durand-Jardin.
Archant believes that it is the dilatation of the pia-vasa-
cular canal of this, or not as Parachape says, a pia-
organic condition of the vessels with secondary thickening
of neighbouring elements.

This form of congestion is partial; but it ends, in
permanent lesion which is charmed into. It produces
hydrocephalus by atrophy of the affected part and by
compensatory dilatation of the ventricles.

The condition of Fascianian bodies

If the fascianian bodies are examined, they will be
found to have undergone a kind of granular degener-
ation; but although the above statement is true in many
cases, it is not true in every case. Hyper trophy of fasci-
arian bodies has been seen in instances where there has
not been Congestion of any kind.

Pigmentary Changes

The pigmentary changes are found with surrounding
tissues. Where Central haemorrhage has been repeated,
the colouring of the tissues is well marked. But what
could be the cause of this pigmentary change? Some
authorities say it is due to changes going on in the
old haemorrhage. Conheim believes that it is due to
the disintegration of the blood red globules of the blood.

Re Peri-Vascular changes as seen

Under the microscope

In the Chronic Congestion, the microscope reveals some.
times a multiplication of the peri-vascular nuclei, an
evidence of nutritive disturbance around the tissues
of the vessels which are Complicat'd by Congestion.
Schiefer & Defize have examined children who
have died from Convulsions, and have found that
with an intense hyperaemia of the brain there is
associated a considerable proliferation of the
peri-vascular nuclei, indicating the existence of the
Meningeal Phlegmosia of slight degree. It is a secondary
lesion, a lesion which properly speaking should not
be found in these Cases. But these lesions depend
not so much upon the intensity of the congestion as
upon its superficial appearance. Defize examined one
Case where a Convulsive attack resulted in quick
death. There was no proliferation of the peri-vascular
nuclei in spite of hyperaemia of a very severe kind

* Traité de Remolliissement du Cerveau. Durand- Jankel Paris 1843
Dissertation de Celbris et Medullas Apiniadis Systematil.
Vascularum Capilairum in Estado Sano et Morbo. Eikko, Trajecti
ad Rhenum 1858.
Traité des Maladies inflammatoires du Cerveau. Calmeil
Paris. 1859.
Nouveaux éléments de Pathologie intense. Bouchard
1886. 90.
Manuel de Pathologie intense. Diderot, 1897.

**Symptoms**
Various authors have described various forms of tre.
bral Congestion. Each of these has its Merit. But without
Measuring to speak against these divisions, we may say that Cerebral Concretion can be divided advantageously into:

1. The Simple Variety
2. Serious Variety
3. Apoplecticform Variety

Slight form of Cerebral Concretion:

a. It is principally characterized by excitation in the areas of sensibility.
b. There is alpahalgesia, painful and lancinating. Light, heat, movement, noise, anger, much this pain & makes intellectual processes almost an impossibility.
c. The retinal Concretion brings on illusions of vision, dizziness also is present at the same time.
d. Sleeplessness follows, v if sleep can be produced it is disturbed by dreams. v the sleep does not relieve the pain of headache.

Symptoms when this form is more severe:

a. Vertigo appears & vomiting comes on by excitation of the medulla, but this vomiting is not accompanied by gastric disorder.
b. The same cause brings about slowing of the heart. The pulse is slow, full and hard.
c. Accompanying the headache there is a painful sensation of heat sponsorship in the head; the heating of the Carotid artery is exaggerated & cutaneous hyperesthesia produces contraction of the painful, and the face & conjunctiva are suffused with blood.
observation sur efficacité de la Compression de Carotid. - Bland.

1. In Cerebral Constrictions there may be absence of the last symptom.

Although the flushing of the face is almost constant in slight forms there is no binding relation between the state of the vascularity of the brain and that of the face.

It has been said that the engorgement of the Cerebral vessels is compensated for by Anæmia of certain parts. Because at times it has been found that when the slight form of Cerebral Constriction becomes intensified, there is an absence of redness of the face.

Bibliothèque Medical LXXII.

2. The most intense and most serious forms of hyperæmia are those which are due to the dilatations of the Cerebral blood vessels caused by irritating agents.

The onward course of simple Variety of Cerebral Hyperæmia.

In the first form of hyperæmia there is no intellectual trouble, the inactivity of the patient is voluntary; his faculties are not altered; but he avoids to put his faculties into action on account of the suffering which it involves.

There is no disorder of movement, the Constipation is slight—sometimes severe. This kind of Constriction is produced with great ease.

It is often found in the phlegmatic, in patients suffering from organic diseases.

It is brought on by the slightest Cause, after a copious meal, after prolonged watching or intellectual fatigue.
The duration of this phenomenon is variable; it disappears after a few hours of rest, but it often persists for many days and only disappears under appropriate treatment.

* Recherche sur l'apathieia. Pachoux

The Severe Variety

It follows the preceding variety or comes on suddenly. It is characterised by mental disturbance and by delirium. Disorders of senses are among the first phenomena in a great number of cases. The patient suffers from violent headache. He is often subject to illusions and hallucinations. In other cases, the morbid excitation is manifested not on sensory apparatus, but when the organs of ideation and therefore the perversion of ideas is the initial symptom. It is not caused by impression from the external world, but the delirium originates in the brain as the result of changes going on in the substance of it. Whatever may be the origin of delirium it manifests itself by originations of impulses and determinations which react upon the motoric apparatus producing muscular movements and disorders of actions. The patient wants to leave his bed. He wishes to follow or run after imaginary phantoms which attract or frighten him. He speaks, he cries; and inimical to appeals addressed to him he wishes to escape, to tear away materials which may have been put in bed to keep him in his place. This state of super-excitation continues for some
time. Then comes a change. The pulse accelerates, perspiration bathes the skin, & the congestion offers an image which may be mistaken for meningcal inflammation with intense fever. But here the term's milk will play its part. Notise the temperature. the temperature is insignificant or may not show any variation from the normal. These alarming phenomena, if they go on for some time without abatement, are gradually replaced by intellectual torpor, the muscular agitation ceases and the limbs become inert. There may be involuntary evacuation, inspiration is stertorous. To thehyper excitation succeeds a depression & the patient falls gradually into a Comatose Condition.

Symptoms in other Cases

In other cases the delirium is only an isolated condition. It is only a shade of the picture. There is general weakness of all the limbs, there is sometimes circumscribed paralysis with certain individuals especially with the old. These serious forms of congestion make their appearance with a certain action of delirium during the night; the patient wakes, he gets out of his bed, he does certain disordered actions of which he has no knowledge. From a bed in the hospital, he wakes up and goes to sleep in another bed which may be lying empty in the ward. The day appears; he becomes calm & composed. But he is dull morose & weak. This condition may go on during


Many nights. This form is insidious. It may follow an ordinary attack of delirious Congestion which has been described above. There is an abundant discharge of serous mucus material from the Conjunctiva & the buccal cavity. Durand-Farrel has described this variety as occurring generally in the old.


The Symptoms in the Adult & Children.

The severe form of disease is rarely accompanied by general Convulsion. The cases which have been described as such are true or Symptomatic Epilepsy. In Children Convulsion is the most striking symptom. But with them it is not so very frequent as has been maintained by many authors. If you exclude so-called Convulsion which Convulsion produces, if you exclude Meningitis—especially tubercular Meningitis—-it will not be difficult to understand that Cerebral Congestion as a primary disease is a very rare affection in Children. But I admit that as a primary disease it is still to be found in Children.

Meningitis & Cerebral Congestion in Infants.

It is important for a physician to remember that there are groups of symptoms which are common to Meningitis & Cerebral Congestion of infancy. But what are those symptoms? Partial or general.
Convulsions with symptoms of abortion, nausea, headache, contraction of pupils, vomiting & constipation. The symptoms which differentiate it from meningitis are absence of high temperature & the enjoyment of perfect health before the commencement of the disease. This variety of congestion has been described by Bland. Convulsions are united with phenomena of paralysis, a state in which the patient is from the commencement profound insensibility, from which the patient can only be awakened by sudden or strong excitation. The course of the disease is very short; it rarely lasts more than a few hours; at least it does not continue over a day. But it terminates in death. Post mortem examination reveals the existence of congestion more or less severe.


**The Apoplectic form of Congestion**

The symptoms of this variety are quite different from the others. The brain, all at once loses its power of being excited by stimulus. The sudden total disappearance of consciousness follows. Suddenly seized, the patient falls & is unable to raise himself; the limbs become paralyzed. Urine & feces escape; but his reflex excitability & sensibility remain intact. After a few hours the patient comes round she gradually recovers; and his recovery may be complete. Slowly or slowly, he regains strength & power. In two or three days the painful symptoms with which the disease made its appearance gradually disappear. Sometimes consciousness & volition are re-established, but slight paralysis comes on after that, which may last from twenty-four to thirty, six or forty-eight hours.
Localisation of the lesion

Now where is the lesion which causes the paralysis. Take notice that Volition is clear. Therefore the paralysis is due to some depression of Conductivity of the motor nerves. There are evidences to prove that the paralysis may sometimes take the form of hemiplegia. According to Decambré the post mortem examination shows a Cerebral Concretion of pronounced Charactel in the side opposite to that of paralysis. But Laennec has found Concretion in both sides, in this case the phenomena of unilateral paralysis cannot be easily explained. The Condition can however be met by one of the two following hypotheses-

1. The Concretion which is found on both sides of the brain after death was not there at the time of death.

2. The hypoxemia of one half of the brain has produced considerable cerebral atrophy in the other side.

The above facts are merely hypothetical. The exact nature of the Concretion has yet to be explained: it is certain however that there may be a possibility of unilateral symptoms such as paralysis and convulsions with a generalised Cerebral Concretion without haemorrhage.

The delirious form of Concretion may very often show symptoms which will correspond with those of a spasmodiform.

The Passive Concretion

In the passive Concretion notably those which are produced by the diseases of the heart or by the Compression of the Arteries. Thoracic Ganglia. Cases are on record where passive concretion has been produced by the Compression of the Sanguineous Veins. The character and course of the disease are peculiar.
There is a period of sleepiness, this is followed by a persistent headache, & the symptoms of nervous depression now become dominant: the pain in the head diminishes & disappears, perhaps by anaesthesia caused by an excess of carbonic acid in the blood; the ideation is slow & sluggish; the memory is feeble; some dreams & hallucinations with a well marked torpidity which announces the advancing coma into which the patient falls. This is its special characteristic. But it is asserted that this stupor or coma is not a result of congestion, but it is the outcome of exuded exudation which is produced by the increase of pressure in the nervous system. The nervous system being in a state of stasis.

**Criticism of the Theory**

But a critical examination of the phenomena is not difficult, the symptoms are not produced by compression, because the resistance of cerebro-spinal fluid is the continuity of the fluid of the cerebrum with that of the spinal cord, as has been stated above, will effectively prevent any such accident.

But granting there is no such mechanism as that which has just been mentioned, the pressure will produce symptoms not of that kind which is produced by dilatation of brain, but it will give rise to symptoms which are produced by depression. It is necessary to take notice of pressure of another element. The influence of gases - oxygen & carbonic acid gas which the blood contains - upon the exsiccating of the nervous tissue. In stasis it also in congestion for reason quite being longer in the hyperemic part the blood loses its oxygen & becomes charged with carbonic acid gas. An aberration such as Brown-Segard
holds that the blood being thus deprived of the property which stimulates the tissue elements is no longer capable of per. forming its function; the consequent depression follows. But take into account also the nerve paralysis which the deep Credo the corresponding stimulation. Take into account also the sedation of the brain—an sedation which is produced by intra-vascular pressure.

**Differences of clinical pictures in different Cases.**

The first phenomena can be interpreted as taking place by reason of the slowness preventing the renewal of the blood which makes it to be suffused with carbonic dioxide gas. The second phenomena can be explained as a well-established fact that a super excitement excitation of any kind is followed by exhaustion. The duration of this exhaustion of powers will depend upon the nature of the part of kind of stimulus, etc.

The third explains the phenomena of depressed observed sometimes from the beginning of active congestion, such as the asphyxialiform congestion which has been mentioned above. The combination of these pathologic conditions makes the clinical phenomena to different in different cases. But we should not leave another point unnoticed. With the increased flow of blood as well as in stasis, there must necessarily go on certain molecular changes in the intracellular nutrition of the brain. These molecular changes must react on the nervous system. Thus it may produce a class of functional disorders which must not be overlooked. This is not merely a hypothetical assumption. Dr. Buhl has demonstrated that even a simple variation of the proportion of gases in the cerebral tissue will
produce serious phenomena & an ordeal of an acute nature.
* A Case of Cerebral Constriction with peculiar reaction of the
Constriction Cubo, aievi de contre de la vue (Bellis Arch. Med.
Beloq 1880).

Diagnosis

In diagnosing Cerebral Constriction it should be always be
remembered that there is:

1. Absence of abnormal sleep;
2. The diffusion of symptoms;
3. The rapid disappearance of the lesion.

The Brain fever & apoplectic form haemorrhage.

In all other febrile diseases of the brain, the rise of temperature
is an important factor, the symptoms are diffuse, therefore the
lesion cannot be a Cerebral lesion. The rapid disappearance
of lesion allows us to distinguish it from Constriction or apoplectic
haemorrhage. If after an apoplectic attack the symptoms
persist beyond three or four days - then it must be admitted
that blood vessels have been injured. Then it is not a Simple Con-
genation. And in the case of rapid death it must be admitted
that a differential diagnosis is not possible.

Alcoholic intoxication & delirious Constriction.

In these cases the diagnosis must be made not so much from
the symptoms which are before us, but from collateral evidences.
Ask & ascertain the previous history of your patient, about his
habit of life, & his occupation, his food & drink. Then Consider
if this is a probability of delirium tremens, and the following phenomena which is present will go in favour of establishing the diagnosis that your patient is suffering from alcohol poisoning. These are:

1. The hesitation & uncertainty of the words which he speaks.
2. The tremulousness of the lips and hands.

But is it saturnine intoxication? Examine the cause of death at the gums. In the case of lead poisoning, there will be a blue line at the gums. In saturnine intoxication, a blue line appears at the gums, near to the teeth, after lead in small quantities has found its way into the system for any considerable time. It is caused by the metallic deposits in the tissue of the gums, being converted into a sulphide by the action of sulphuretted hydrogen degenerated from decomposition of fragments of food remaining between the teeth. It is best marked over the region of the incisors, it is absent or indistinct where the teeth are away.

The difference between Syncope & Epilepsy

In Syncope the arterial pulsation is lost & the respiratory movement ceases. But here both these factors are working. The case of Epilepsy is somewhat like the Case of Cerebral Constriction. Do it they Epilepsy? How are we to ascertain this? Think over the initial stages of the disease, Enquire fully into the patient history, ask a friend out if the patient has suffered from previous attacks, visit upon seeing the evidence of old lesions produced on the tongue by biting during the paroxysm. If the tongue is found to be unscarred, if the physician is deprived of information relating to the antecedents.
if this is a probability of delirium tremens, and the following phenomena which is present will go in favor of establishing the diagnosis that your patient is suffering from alcohol poisoning. These are:

1. The retardation & uncertainty of the words which he speaks.
2. The tremulousness of the lips and hands.

But if it Satinane intoxication? — Examine the causes aloft at the gums. In the case of lead poisoning there will be a blue line on the gums.

In Satinane intoxication a blue line appears along the gums, near to the teeth, after lead in small quantity has found its way into the system for any considerable time. It is caused by the metal deposited in the tissues of the gums, being converted into a sulphide by the action of Sulphuretted hydrogen degenerated from decomposition of fragments of food remaining between the teeth. It is best marked over the region of the incisors, it is absent or indistinct where the teeth are away.

The difference between Syncope & Epilepsy

In Syncope, the arterial pulsation is lost & the expiratory movement ceases. But here both these factors are working. The coma of Epilepsy is somewhat like the coma of Central Congestion. Do it then Epilepsy? How are we to ascertain this? Think over the initial stages of the disease, Enquire fully into the patient's history, and refresh out if the patient has suffered from previous attacks, insist upon seeing the evidence of old lesion produced on the tongue by biting during the paroxysm. If the tongue is found to be unscarred, if the physician is deprived of information relating to the antecedent-
of the patient, the immediate diagnosis will then be an impos-
ible task. The coma of Epilepsy could last as long as that of congestion, therefore we have to fall back upon the con-
sideration of the greater frequency on one hand, or the pre-
cence or absence of Cardiac lesion, Arterial lesion or Pulmonary lesion, which are often determining factors in a very severe kind of cerebral hypostenmia.

**Vertigo of Gastric disorders**

Slight forms of Cerebral Congestion may be mistaken for Vertigo arising from gastric disorders. But in such cases the patient will be found to have suffered from dyspepsia or gastralgia of more or less old standing, so that in these cases the symptoms abate or cease immediately after the ingestion of food. It is not increased by lowering the head, it is accompanied by pain or nausea during nausea. These symptoms are not found in Vertigo which is due to Congestion. Then when the patient awakes the patient never loses his Con-
sciousness, he has always a clear idea of what is going on around him. The gastric Vertigo when treated with tonics, alkalines, bitter will disappear; whereas the Vertigo of true Cerebral Congestion will be but augmented in its intensity by any such treatment.

**The Kind of Congestion**

Once settled that it is Cerebral Congestion, it is important to know what kind of Congestion it is. Is it an increased flow or is it a Stasis? Is it an essential Condition or is it only symptomatic? Careful examination of all the organs, principally of blood vessels, heart, lungs, will settle the first point. If the patient does not show any organic condition which can serve.
chemically bring about the Stasis, one will be obliged to ad-
mit of active & spontaneous Congestion, the true or Sympathetic
Character of which will be revealed by the knowledge of the
Causes which have produced it, by the pathological anat.
omy of the individual. The Sunstroke, the intestinal
fatigue & the prolonged watching, the excess of food, are
the most ordinary cases of true or essential Congestion which
is due to excessive influx of blood. Cerebral lesion, gout,
rheumatism, haemorrhoids, disordered menstruation are
the most Common Conditions which produce active Congestion
which may be described as Sympathetic.

**Prognosis**

Cerebral Congestion is a serious matter. The statistics of death
rates prove this clearly. But it is not the immediate danger
which should be so much taken into account. The remote con-
sequences & Complications of this disease are really alarming.
The repeated attacks are also dangerous. The Complications are
phlegmasia, eczema, scrofulous haemorrhage &c. The
gravest of all the forms of Cerebral Congestion is the delirious
form. It is to this Variety that the fearful death Rate is due
(Androl Messiye). This form often terminates in haemorrhage, or
by pulmonary hyperaemia with aculi secura. This Coin-
cidence of pulmonary Conflagration is frequent with Cerebral Con-
gestion. The Gravity of the case is exceptional. With the old, the
Congestion is generally a very Grave Condition, because of the
alteration of the blood Vessels which predisposes them to haemorrhage.
The initial Congestion which comes on in explosive form, in typhoic
fever, are also very dangerous. In the alternated or in individuals,
who are already affected with Cerebral lesions, the Congestion is a formidable phenomenon which hurries on the disease & which aggravates materially pre-existing accidents.

Result of Congestion on Pre-existing lesions

The secondary congestions are often partial. They are those which in the Course of Cerebral diseases of Chronic character suddenly temporarily change the series of symptoms. A tumor of the brain may have remained for a time without showing any apparent indications. Congestion comes on & in its track serious signs are revealed which tell the tale of an existence of the lesion. Thus again, the occurrence & the relapse of congestion gives a key to many obscure facts of Cerebral pathology. It explains that symptoms of excitation coincide with the old lesion which have destroyed the brain matter. It explains the Cause of Disappearance of Certain phenomena of depression in paralysis, in spite of the persistence of the first change: it explains even how a lesion of the same extent & of the same locality could present in different individuals different groups of symptoms.

  Thrombic fever or Sunstroke — Wood. Philadelphia 1872.
  Particulars of a Case of Sunstroke — Clapham. The Lancet 1872.
  Étude Sur le Coup de Chaleur — Horties Paris 1873.
  Two Cases of Sunstroke with unusually high temperature & death — Smith. The Lancet 1876.


Treatment

Blood letting general or local. Poultices and the application of cold are the only therapeutic means. But still certain judgment must be used in selecting the plan of treatment. For the sake of clearness, treatment will be divided into several headings, as has been done in describing the disease.

Active Congestion

In active congestion when the blood flow seems to be under the influence of an exaggerated action of the heart, provided that there does not exist valvular lesion, or probably mechanical dilatation of the vessels of the brain, the general treatment is indicated. But the case must be treated according to its symptoms, the blood letting will be followed by the administration of saline purgatives which should be kept up for several days. If the cerebral arteries have been engorged for reason of an obstacle, pressure to narrowing the blood channel of the areas, thus sending excessive blood to cerebral arteries, try to simplify the blood vessels in order to diminish the influence of such obstacles.

Compensatory Engorgement

In the Compensatory Engorgement, which follows the suppression of the menae, that is bleeding from the uterine, etc. the first
indications is to re-establish the flow, therefore apply leeches to the arms or at the upper aspect of the thigh. Give repeated doses of purgatives. If there is no reason to believe that the condition of the intestinal canal will not allow of its act. minimisation, take drastic purgatives, such as preparation of aloes. When these means do not produce the desired result or when cerebral symptoms persist or become menacing in approach, it is necessary to follow a general bleeding. It is clear that it will not bring physiological or habitual homoeopathy, the arrest of which has caused congestion, but it will directly attack the phenomena of congestion, it will allay the immediate danger until time to take proceedings against the cause of dyspepsia.

**Sunstroke & Excessive Fatigue**

In Congestions caused by sunstroke, excessive fatigue or general blood letting is advised. The application of leeches on the head, vesicles of the inferior extremities, purging are the best methods of treatment. If however one thinks that some blood must be taken away, it is better to perform local blood letting.

Take away blood by applying 5, 6 or 8 leeches or even 10 or 20 according to the constitution of the patient. Now when one has to put leeches to the thighs or to the arms, it is necessary to follow this rule, but it is also important to bear that in mind in case of application of leeches behind the ear, one could in these cases obtain the desired for result by two different ways:

1. In applying together at the same time a number of leeches.
2. Putting only 3 or 4 replacing new ones as the older ones fall off, thus obtaining a flow nearly continuous during
10 to 12 hours as the Case requires.

The indication of bleeding is more rare in irritative enervation which comes on at the advent of serious fevers.

In such cases cold with ordinary solids, or solids agitated with aromatic vinegars, would be of greatest service.

**Congestion due to nervous Causes**

The circumstances are not different when in cases of enervation caused by nervous condition. Do not allow general blood letting.

The local blood letting is only useful in some particular cases. Subcutaneous, viscera, at the base of the neck or to the inferior extremities are the means which are adopted.

In one case, in one case only evacuation is indicated. It is the cerebral Congestion caused by indigestion, rapid evacuation of the stomach will check the disease, but this emetic treatment ought not to be done to the old, or to individuals whose arterial systems are abnormally rigid. To produce vomiting in these cases will be to expose the patient to the danger of cerebral haemorrhage.

**Passive Congestion or Stasis**

In passive congestion the blood letting, be it local, as application of leeches behind the ear, best be it general, is ordinarily indicated. But the reasons are quite different to those mentioned above. Observe for a moment the influence of arterial blood on the Venous System. When the Venous system of the brain is dilated with the blood which hastily circulates through these same channels, it is clear if that circulation continues, red blood could not arrive in sufficient quantity, from this cause the
Cerebral Congestion with Heart Complication

The most complete type of Cerebral Congestion by Stasis is found in the heart disease, especially in lesions at the Mitral orifice. The Digitals will act admirably upon Cerebral upon Cardiac lesion. But bear in mind that the remedy does not produce instantaneous results. The pulp is most pressing, therefore practice bleeding or thin afterwards exhibit Digitals. In other cases the Stasis is caused by weakness of Circulation, by a kind of inactivity of the Heart vessels or by general debility. The indication is very different, give tonics and stimulating or proper diuretics.

Precaution to patients who are Susceptible to Cerebral Congestion

In individuals who are predisposed or subject to active Cerebral Congestion ought to take great hygiene precautions by which they will often be able to ward off this formidable
accident. The dietetic ought to be of simple nature. It should consist of vegetables and fruit, the wine of the simplest kind, but no stimulating drinks, tea or black coffee should be precluded. Frequent visiting of the places of amusement, theatres or concerts which lead to excitement ought to be enjoined. Prolonged watching, occupation involving intellectual fatigue or excessive excitement should be forbidden. The life must not be led in a sedentary way, the bed room ought to have even temperature and ought to be fresh; the bed made up should be made of hair and not of feather. Habitual constipation must be prevented by taking aloes pill at bed time at intervals of 2 or 3 days or by means of the various waters which will produce the same effect.

Conclusion
It is necessary to understand that the treatment of cerebral congestion is variable. The rational methods of treatment are only to be found by bearing in mind various physiological and pathological conditions which produce the disease.
**Cerebral Anaemia**

The most common causes of cerebral anaemia are haemorrhages of various kinds, but most frequent obvious of all these is intercranial haemorrhage. Diseases of long duration, exhausting nature, e.g., coryza, bone suppuration, the last stage of phthisis, are frequent causes of cerebral anaemia. Excessive organic exhaustion or organic apoplexy such as found in women who bear children in quick succession and suckle them themselves, may also lead to cerebral anaemia.

There is a point of practical importance both as to these cases. It is this — The anaemia does not often come on spontaneously. Thus women do not show the least sign of the disease in their ordinary way of life. But at the slightest approach of some acute disease, the signs of cerebral anaemia are at once manifest. Chlorosis is also regarded as one of the most powerful causes of the bloodlessness of the brain.

**Systematic Enquiry into the Causes of Cerebral Anaemia**

An examination of the above facts will enable us to divide the causes of cerebral anaemia into two great classes, viz —

1. Anaemia may be the result of excessive loss of organic materials.
2. It may be due to defective acquisition of nutrient materials or insufficiency of assimilation.

**The Defective Assimilation**

The defective assimilation may be brought about by two ways, namely:

1. By defective alimentation.
2. By defective Elaboration

Such is the case in the anaemia of children who are dullish. In them the organic work is limited with insufficiency of tissue gain (insufficient food etc.), or in them also the anaemia is preceded by Cephalo-stomach diarrhoea; the disease is not only found at the period of weaning: children may even be exposed to the attack of this malady at the time of their adolescence. At this period children are more subject to gastric, intestinal catarrh: to this in many cases more than to improper nourishment that the infantile anaemia takes its origin.

In 1825 Marshall Hall for the first time described cerebral anaemia under the name of "Hydrocephaloidea Anaemia" wishing to associate it with Hydrocephalus because of the analogy which it presents with that disease.


Anaemia associated with Fevers.

In fevers of specific origin there is a variety of slow cerebral anaemia. Do not lay the blame upon the deficiencies. True, a state of Pyrexia leads to defective nutrition. But it is evident it is not a simple anaemia of the brain; the blood itself has undergone certain changes. The fact is, it is an anaemia with dyscrasia.

* Cases of delirium probably dependant on impoverishment of the blood. St. George’s Hospital. Reports 1868. Barclay.

Anaemia Caused by Vaso-Motor disturbances.

The Anaemia may also originate from disturbances of vaso-motor innervation. The blood vessels of the encephalon may suffer
from wide spread or general contraction. The Cerebral Anæmia due to various kinds of emotions, belongs to this; it is characterized by palor of the cheeks, loss of consciousness. The old writers have described it under the name of neurotic apoplectic. See on the Physical Health of Nervous System. New York 1871.

Reflex Contraction

That there may also be a reflex contraction of the vessels of the cerebral has been demonstrated once for all by the researches of Nau der Becke, Callenfels, and by the recent researches of Rentinagel. The exhaustive enquiries of these two authors show there may be reflex anæmia—what constitutes certain forms of eclampsia & anaemia.


The Opinion of Gallo

The last variety of anæmia is purely mechanical, it is caused either by constricting of the organs or by the retention of blood in them. It may be produced by various kinds of operations: for example the rapid emptying of the bladder, ovarian cysts, or of arteries: in these cases the pressure is taken away from the abdominal vessels, they now admit a large quantity of blood into them. Gallo maintains that the venous walls become distended because long compression has weakened their power of resistance. They yield to the excess of pressure, their dilated walls contain a larger quantity of blood.
Sufficient quantity of blood does not reach the head, therefore sufficient quantity does not enter into the arterial channels, & such a state of 

...as can be easily seen, will produce cerebral anemia. Of the same order belongs syncope, dizziness, fainting which are suddenly seen in patients who suddenly rise from the recumbent into standing position. Two Causes may simultaneously produce this effect.

1. Accumulation of blood in the inferior extremity or
2. Weakness of the cardiac impulse.

For this reason the faulty degeneration of the heart & non-compensating lesion which diminishes the blood wave, are most fruitful Causes of anemia. It is the arterial insufficiency which frequently brings on this condition. The diminution of the capacity of the cranial cavity by exudation, by effusions & tumors are regarded by Niemeyer as some of the Causes. But this is a point of contention. We have often seen in the frontal bone room pronounced hyperaemia of the brain in tumors or in effusions. In other cases it is true that anemia is present. The Cause of the insufficiency may be due to the time when the effusion took place or the length of time it has since been growing. Therefore, this could not be formulated as absolute conditions in the production of anemia.

* Consider: *Sur l'ischemie et l'hypoxémie centrales, *Daussetville.


The atheromatous arteries

Atheroma of the arteries of the brain is another Cause of cerebral Anæmia. It destroys the elasticity and contractibility of the blood vessels. This leads to the slowing of the blood flow, it reduces the Calibres of arteries, it modifies the process of exanæmia in those by their joint effects produce anæmia which is grave and permanent. Very often in the old the injurious action of the artery is aided by the slowing action of the weakened heart.

* Recherche sur l'anæmie crânale. Étienne Strasbourg, 1888
Des effets produits sur l'encéphale par l'oblitération des vaisseaux artériels qui s'y rendent. Paris 1860.
De l'anæmie et de l'œdème crâne. Petronii. Thèse delens. 1894

Experiments of Schiff

Schiff compressed the common carotid of one side at the level of the larynx, the half of the face of the same side became pale and the sight was affected; on the opposite side sensation of heat and numbness was developed; in some cases slight muscular tremblings and convulsive movements were found. after 8 or 10 minutes the phenomena disappeared. The collateral circulation rapidly compensated the effect of compression. The ligatures of both the carotids which is less dangerous in the mammal than with human being.

The tying of both the carotid arteries in the mammal is followed by disorders such as those which have just been mentioned— with notable acceleration of the heart, of movements of inspiration, it developed characteristic uncertainty of locomotion. The effects generally last for a little while, but they may be only transient. The intra-cranial anæmatomous of the carotid & vertebral arteries
Supply the defect which the exclusion of the Carotid involves.

If after both the Carotids have been tied, the Vertebrals are Compressed, Convulsions quickly come on, the pupils dilate, and the

contraction of the animals falls in coma. This state of apparent death can be prolonged without bringing on real death, if artificial respiration is kept up, and taking away the pressure from the Vertebral arteries these phenomena slowly gradually disappear.

Effect of tying the Carotids + Vertebrals.

When one tie successively both the Carotids + Vertebrals the animal falls senseless + shows no signs of voluntary motion yet dies in a state of coma which makes its appearance between a general or partial convulsion. In such a time it is not unusual to find secretions of large quantities of saliva & involuntary evacuation of urine & feces. The excitability does not disappear at the same time. Vulpian tells us that after the extinction of cerebral function, the function of the spinal cord may still persist for some few minutes, respiration may still nearly be normal & the reflex movements may still be elicited from the trunk + the extremities.

Reflex excitability of Cornea.

After death the reflex excitability of Cornea persists longer than that of the Scleral; and Kussmaul & tenner believe that the right side of the heart may continue beating even when the muscles of the extremities are rigid. The above result may also be seen when an animal is bled for a very considerable extent.

The value of the experiments.

The absence of arterial blood modifies in a marked degree the
excellency of nerve tissue, its effects thus produced may be temporary or persistent. This modification produces its effect upon the brain substance hence we have sudden loss of function of the brain as manifester by
Coma = Complete Revolution. In certain cases this modification may original signs of excitation & depression at the same time. When the
Cause which destroys nervous excitability is not sufficiently strong to
do its action at the very first shot, the "nerve paralysis" is always far
called by a period in which excitability remains in a state of exaggeration.
In other words when one of the Conditions of normal excitability is sub. 
present, the loss of that excitability is constant; but it may be primary or secondary. It will be secondary if the activity of the Cause which pro-
duces it is less strong & more gradualist. In this case there is a stage of
Super excitation before the loss of function. The time of this Super Excita-
tion is in inverse ratio to the violence of the Symptoms; after which depression most certainly comes on slowly but surely provided the Cause
is still present. The Cause may be primary if the pathogenic changes are
prompt & sudden. This law has no exception. It is the very key of a
great number of phenomena.

Inference from above experiment

These experimental evidences are of great value. Compare them
with pathological Conditions which produce Cerebral anaemia; &
a well balanced comparison will bring into you mind a clear
Conception of the disease which we are now discussing. These experi-
ments at once demonstrate, that when the arteries channels of the
brain are obstructed, or when from any cause the total quantity
of blood in the body is diminished, Cerebral anaemia is produced.
But this is not all. Examine for a moment the function of Oxygen.
for intermediary haematosis oxygen is necessary; this haematosis
maintains the normal nutritive processes. But the red blood Cells
which are the moving agents in peripheral respiration are oxygen and carbonic acid. The insufficient, or deficient, blood in the brain means the same thing, as far as this question is concerned, as the insufficient, or deficient, blood itself. Therefore it may be concluded that the brain anemia may be produced by anemia or by oligocyanemia.

Pathology

In removing the brain from the cranium sometimes it is found that the brain is distended with cerebrospinal fluid or blood, but sometimes anemia may be found with a large amount of cerebrospinal fluid. How should we regard these two opposed conditions? In the former case it has been presumed that the anemia has preceded a cerebral hemorrhage, in the latter the cerebral anemia has been produced by defective or insufficient alimentation or from an organic affection which looks at the brain substance. The meninges or the brain substance are anemic, but sometimes this may be a state of hypoxemia of the membranes, which, but strongly contracts with the state of the nervous tissue. Pale, discolored is the gray substance; a Slight number of punctiform hemorrhages are seen on the cut surface of the brain. In children the color is whitish blue, in the adult it is rather yellowish. The nucleus of the pineal body is atrophied, the ventricles contain often a large quantity of impure cerebrospinal fluid, but this change which is believed to be constant a priori, does not exist in reality.

# Manual of Pathology - Coats

Symptoms

The symptoms vary according to whether the disease is

1. Sudden & rapid
2. Gradual or slow
In the slow-gradual anaemia, symptoms are seen in slight cases, which has been briefly called the irritability of weakness. In very severe cases, the depressing symptoms predominate; sometimes it may be seen even in slight cases. The patient suffers from continual headache, the organs of memory impressions are all in a state of alert; light troubles him, there is a great dislike even of the slightest noise; sometimes he suffers from nausea, the pulse is small & complicate, the heart beat is quick & slow; Vertigo, fainting & syncope are common, there is muscular trembling & intellectual weakness is general.

The above phenomena explained

Under the influence of anaemia & insufficient nutrition, the excitability of the nervous elements is weakened, that is to say, the effects are less energetic & the weakening is more rapid; because the vitality of the cells is lowered & the excitability is put into play by a very slight stimulus, a stimulus which in normal physiological condition will have no effect upon the nervous tissue. Observe then there is a reaction which is quick & of long duration, that it is produced by impressions which ought not to arouse its dormant excitability. As has been stated, the condition such as this has been designated as the irritability or excitability of weakness.


Encephalos Synoria - Marchand. The lancet. 1864.

The symptoms of sudden & rapid form of anaemia:

It is commonly seen after haemorrhage, the primary symptoms are diminished sensibility, noises in the Ear & dizziness. The pupil is at first in a state of contraction, then it dilates because it is no longer able to respond to external stimulus; this state is followed by loss of consciousness & voluntary movements. The skin is cold & pale,
The patient enters into a state of coma. The respiratory movement which has been rapid and accelerated begins to get slower.

The explanation of general Convulsion in Anæmia

The Theory of Stendel & of Kussmaul & Tenner.

The Convulsions are under the Control of Mass. Cephalon. That being so, two explanations have been given to us why this should be Convulsive movements in Central Anæmia. The opinion of Stendel is that this Convulsion is due to hypersæmia. Kussmaul & Tenner maintain that it is simple anæmia. They say that although there is absolute Anæmia of the brain, it is not of the same intensity in the mass. Cephalon. Now which of these two theories is correct? The experimental evidence which are gathered from the life line of the four arteries of the encephalon are perhaps in favour of the view expressed by the last two authors. The Anæmia produced by the interposition of the blood flow in the Carotids may be mentioned as a fact supporting their theory. But what about the rapid Anæmia which comes on after a great haemorrhage? In such a case none of the above theories can be accepted & why? The total quantity of blood in the body is diminished. Therefore what reason could be advanced for the fact that there should be also pronounced Anæmia in the mass. Cephalon than in the hemispheres? The Convulsions or the phenomena which are seen under the circumstances are the consequences of pathogenic law — a law that teaches us that functional or stimulation is a free sum of intention — the coming cessation of Central Function; the reflex involuntary mechanism of the spinal cord loses its controlling influence — and thus its physiological influence is disturbed. This is the explanation of the convulsive movements which are seen in these cases.

  Gazete des Hopitaux — 1866 & 1867.
When cerebral anemia and spinal anemia are associated together, it is known under the name of spinal irritation.

The symptoms may be enumerated as follows:—

1. Psychical disturbances due to weakness or disorder of intellect.
2. Disorders of sensibility, which result in pain, anesthesia, or hyperesthesia.
3. Locomotor disturbances, which vary according to circumstances.

Either general or partial, or this may be paralysis which in many cases may take the form of paraplegia.

4. Functional disturbance of heart.

The symptoms are numerous, and the various pathological Complains tend to give us clinical pictures which are infinitely multiform, and no wonder, many of the Continental authorities have designated the existence of such a disease—1828. Brown of Glasgow called the disease “spinal irritation,” since that time various names have tried to clarify and arrange its symptoms. But Hammond has observed 112 cases and Kristeller has given it the name of “Cerebro-Cardiac Neurosis.” They have also very judiciously differentiated it from a vague ill-defined nervous condition with which it has often been mistaken.

Hammond's View

The Cerebro-cerebral anemia is an anatomical condition which is very difficult to define, but at the same time its pathological conditions...
there a distinct form of phenomenon. Hammond believes it is simply an anaemia of the spinal cord. But against this view may be cited a constant disorder of cerebral commissuration. The transatlantic authorities very judiciously maintain that various clinical conditions are due to localisation of anaemia in different parts of some circuits. This of course may possibly give predominance of one or one form of symptoms in a patient, in another patient another series of symptoms may be seen in their prominent aspect.

**The Anaemia Caused by atheroma of the Arteries**

The anaemia caused by atheroma of the arteries must now be considered. The blood vessels may also undergo rupture or obliteration. This obliteration may be in the arteries, in the capillaries, in the venous sinuses of the cranial cavity.

**Obliteration of the arteries of the Encaphalon**

Authors describe this condition under different names. They may call it thrombotic or embolism, partial anaemia or cerebral ischaemia, or softening necrosis, or simply softening or necrosis of the Encaphalon, but this profusion of synonyms is not of any great use. Thrombotic embolism is a special condition which may produce obliteration; partial anaemia or ischaemia necrotic softening is properly a state of changes produced in the nervous tissue by the obliteration of the arteries. But we shall not consider here anaemia which is produced by alterations of the blood vessels, other than that which is caused by atheroma or atheroma; therefore partial anaemia which embolism or thrombosis of the blood vessels may bring on, will not be considered in this essay.

*Recherches sur le ramollissement du cerveau... Robert Paris 1820*
Symptoms of arterial aneurisma

The most common symptom is fainting. This time is vertigo which lasts during the whole course of the disease. This vertigo is accompanied by disturbance of vision, the patient falls if he cannot grasp at a support. A few minutes pass he regains his equilibrium and feels better. Either by series of occurrences or in a frequent repetition of slight attacks. But not only when he is erect or sitting - the patient may be struck even when he is in bed. Here is a picture! The patient is speaking to you without betraying any sign of disease, apparently everything is going on well with him, suddenly he turns pale, he stops speaking, his head becomes fixed, then in an instant he recovers, with an expression of astonishment which is characteristic. In other cases the accident is grave. The attack lasts longer. To the preceding symptoms are added other phenomena which denote a serious state. The patient is drowsy, he looks stupified, his words are long drawn, hesitating and slow. He has numbness of the extremities, either one or both sides, or of one side more than the other. There is nausea and vomiting. How long will this state last? It may last 24 hours or more. In another form, grace than these two which I have just mentioned, a true apoplectic attack may take place.
In such cases, the symptoms may pass off, but the paralysis remains. The physician ought to bear in mind, if he has compared the physiological & anatomical facts to pathological changes, that the central anaemia produced by the atheromatous blood vessels must necessarily involve cerebral degeneration bringing with it in many cases a partial disturbance of memory.

The Symptoms in Infants

In infants there is found alternate periods of excitation & depressions, the symptoms which they show offer to us a kind of analogy between this disease & meningitis. But our knowledge of the feeble state of health of the patient previous to the attack, the absence of temperature which is present in fever, the astounding rapidity with which often a cure is effected when proper treatment is adopted will save us from making blunders.

Several important points in

regards to Symptoms in the adult

In all cases the phenomena which have been described above are the least marked when the patient is in the horizontal position. For obvious reasons it becomes more marked when he stands up. To this, is due the Vertigo + Syncope in these cases. A person who quickly get up from his bed, dizziness is general in these. Those who suffer from central anaemia. In patients in whom anaemia has been produced by inanition, delirium is not uncommon. In an advanced period of the acute variety, the delirious form of anaemia is observed in which several paroxysmal conditions are united together to produce the irritability of weakness.

The anaemia is much more marked as it comes on sooner.
if the patient has suffered from prolonged thymus excisions, or she has been subjected to a severe antiphlogistic treatment.

**Prognosis**

The prognosis is a matter of great difficulty, it can only be decided after a course of treatment. Children often present alarming symptoms, but a correct diagnosis of proper treatment will very soon bring the symptoms under your control.

Kussmaul & Jenner teach us that the state of the pupil of your patient must be watched. They believe that the situation becomes grave when the persistent dilatation dilatations of the pupil give way to its continued contraction.

**Prognosis of Anaemia produced by Atheroma**

Anaemia produced by atheroma is extremely dangerous. Because pathlogical conditions of the blood vessels make its course perilous, thus it gradually increases in its intensity & it creates very powerful participation to the softening of the brain; often this fearful eventuality does not make its appearance for years. The patient may be afflicted with this ischaemia, without showing symptoms of a serious disease.

**Diagnosis**

This is a remarkable analogy between the symptoms of Anaemia & Atheroma. This analogy is distinct & defined in the delirious form of anaemia, but it is of the very first importance to the physician to clear up his diagnosis. In both there is habitual pain in the head, in both there is trouble of the ears, in both there is faintness & vertigo, in both there is physical & intellectual disturbance & sleeplessness & fatigue on exertion. Make your diagnosis
clear. But I am going to say more than this. Here is a
man before you; he is strong & robust; examine his heart
& you find no cardiac disorder; his pulse is full &
strong. Take another patient, a woman, for example—
a woman sickly, weak—her pulse is feeble as the slightest pressure
makes it indistinguishable. She has a soft murmur at the base of
the heart, & an intermittent, but persistent noise in the vessels of
the neck. Put your first patient, under lowering treatment; you
will cure him. Proceed for the woman stimulating treatment;
the disease will soon yield to your influence.

Evidently then, the one suffered from a very common form of
Cerebral hyperemia; the other presented a common form of
Cerebral anemia. Now, how can an accurate diagnosis be
made? Can it be made by the indications of Cerebral phenomena?
No. The Cerebral phenomena are identical in both cases. Here
you have to make your diagnosis from collateral evidences &
from a knowledge of the general health of your patient before
the attack came on. This is the only means of diagnosis in these
cases which present to us so much analogy.

The Cerebral disorders have nothing in themselves, the delirium,
however, may be as violent as in the most acute Cerebral hyperemia.
The color of the eye & of the face is not always a faithful indication
of the actual state of the brain. Therefore you must look in some
other direction to clear up your doubts. The state of the constitution,
the pathological & therapeutic circumstances which have preceded
the development of the accident, must first of all be taken into
consideration in order to arrive at a rational conclusion. Then
examine carefully the effect produced upon your patient by
standing & also by horizontal position. Mark whether this is an
aggravation or diminution of the phenomena. Watch the effect on
the constitution of various kinds by food and drink. Then inquire into the
state of the patient's heart. If the heart be healthy, the weakness
of Cardiac function, the diminution or disappearance of the first
normal beat is a strong presumption in favour of anaemia.
Such are the various kinds of information, which will surely lead
us to a point from where the difficulty of diagnosing will gradually
disappear.

The Vertigo of Anaemia & Epilepsy.

The Vertigo produced by the cerebral disorders Anaemia may be mis-
taken for Epileptic Vertigo. If the patient has suffered from the
Vertigo after an epileptic attack, then the diagnosis is well-nigh
settled, but if not then the diagnosis may be a matter of great dif-
ficulty. In such a Case examine first of all the character of the
feeling. In ischaemia total loss of consciousness is exceptional,
but it is very common in Epilepsy. But the time of the appearance
of the attack is very significant. The epileptic Vertigo belongs to the
age of infancy, to youth. The ischaemicic Vertigo is proper to
advanced age, or at least when the arteries assume the condition
in which it is found in old age, or by some special pathological
condition, such as alcoholism, Scurvy, Intoxication &c., produces
structural changes, which will of course remove all difficulty from
the just conclusion of a differential diagnosis.

Treatment

The treatment of habitual Anaemia is often confounded with
general Anaemia. The main rationale is the stimulating treatment.
Exhibition of peptogenic preparations or the recoveries which the
hydro-therapeutic offers to a physician are the means which he
will speedily employ. In all Cases of Central Anaemia just the
patient in a horizontal position which the head lightly elevated above the trunk. If a tendency to fainting or syncope is present, place always beside the patient some kind of strong stimulating medicine, preferably some strong ammonia preparation.

**Chapman’s Treatment**

Chapman has proposed a new method of treatment. He advises the continual application of ice to the cervical-dorsal region. But he includes only those cases in which Acasia has not been produced by haemorrhage, but when it has been produced by the Constriction of the Spinal Cord. The cold acts on the Spinal Constriction. The blood vessels contracted, the blood is driven into the vessels of the Encephalon. Various authors have spoken in praise of this treatment. But Hallophile claims that there are patients who cannot tolerate this mode of procedure. In them the application of ice produces respiratory disturbance, Cyanosis and intense pain.

**Treatment of Sudden Haemorrhage.**

In cases of Acasia caused by great Haemorrhage, there is no time to lose. The treatment must be rapid and effective. There are two lines of treatment:

1. To increase the blood flow into the brain
2. To maintain the failing excitability of the nervous tissue.

The first step can be secured by prompt compression of the abdominal aorta or the arteries of upper extremities.

The second object can be secured by administration of stimulants such as Brandy preparations of Acetals of Ammonia. What else should be done? Wrap the patient in warm clothing whose want is repeated anesthetic on the cutaneous nerves. Keep up the excitability of the Encephalon. If the bleeding has been stopped, or if it has not been abundant, these means will
generally be found to be sufficient; but if the loss of blood has been in such great proportion that these methods are unable to stimulate the functional activity of the nerves, then your efforts are fruitless. The Transfusion of Blood now is necessary, but it must be admitted that this is an extreme measure which unhappily is not always convenient to employ.

**Anemia in Children**

We have indicated that Anemia in children may be mistaken for acute hydrocephalus. But it is better to be on one's guard when one is called upon to treat such a case. The lowliness and all debilitating methods for the time being should be put aside. In children the most ordinary cause of Anemia is diarrhoea. Try to arrest this diarrhoea by appropriate means; if it is the weaning which has originated their disease, give to the little patient as much milk as you can as food; if it is found in a child a little older who cannot take or in which ordinary diet disappears, do not hesitate to give him very finely minced meat; after this give wine + quinine. Marshall Hall has recommended small doses of brandy + quinine in the form of a mixture or as an injection from 10 to 20 centigrammes according to age has often proved efficacious.

**The Secondary Anemia**

The Secondary Anemia which is the result of some acute disease, is com-
battled by wine + regulation of diet + by use of wine + quinine. In all cases other than convalescence is established do not let the patient get up for some time. Let him lie first sitting in his bed—when he finds that the holding up of his head or the sitting up in bed does not produce Vertigo or fainting, then he can—but very cautiously + very moderately acclimatize himself to stand up by degrees. Too much elation cannot be laid upon this point—the neglect of it has often brought on grave and fatal Syncope.