Thesis presented by
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Coma, its clinical and practical aspects.
Having had during the last eighteen months two very distinctive cases of myxoedema under my observation, I had almost determined to make this disease the subject of my thesis, when, for no means the first time in my experience, I was confronted with a case of coma occurring under circumstances which rendered the immediate diagnosis of the cause of the coma by no means easy matter.

A man aged 49, single, was admitted into hospital complaining of very severe pain in the right side of the neck, careful examination revealed the presence of a deep dental abscess. It was opened by Mr. B. F. D. and drained. Early the next morning I received an urgent message from the nurse saying that the
Patient was sleeping very heavily and that she was unable to arouse him.

I found the man in a state of stupor. The breathing was slow and stertorous, the pulse 80 and full, the temperature 101.2. The face was somewhat cyanosed with beads of perspiration on the forehead. The pupils constricted seriously contracted. The urine has been examined on the previous day and was found to be normal.

Nurse informed me that the patient was complaining of much pain. She had about half an hour before given him a hypodermic injection of morphia and that the stupor had gradually supervened.

What was the cause of the present condition? Was it an embolism from the abscess or could it possibly be due to the morphia injected? The contracted pupils cutter
inclined me to the latter opinion, though the infection appeared & a perfectly legitimate one. The ordinary candidates were accordingly administered but the stings gradually disappeared and the man died. At the post mortem examination I found a large effusion of blood into the centre of the Pons Varolii. This was under a case of apoplectic coma, the locality of the clot causing the contraction of the pupils which had had so much to do with misleading me.

This case deeply impressed me with the difficulties which a medical man must often experience in arriving at a correct diagnosis and its cause, when brought face to face in practice with a comatose patient, and I finally returned to select this symptom of disease which is common to such various conditions as the
subject of my thesis.

In the first place we are deprived of one of the main elements of diagnosis in ordinary cases, viz. the patient's own story of the onset of his ailment and of his present sensations, and often enough no reliable account can be obtained from friends or relations, and we have to rely entirely on our own observations.

This circumstance alone considerably increases the difficulty we so often meet with in coming to a correct conclusion as to the cause of the unconsciousness. In some cases indeed a correct diagnosis is impossible and we are compelled to wait events, usually however a knowledge of the varying phenomena associated with different forms of coma will suffice for a diagnosis, al-
Phenomena of Coma:

By coma we understand that state in which consciousness is in abeyance; not as in ordinary sleep, a condition from which the individual may be aroused, but a state in which external impressions pass unheeded or without effect; or if they do suffice to arouse the sleeper he soon f lapses again to his

This definition of coma is unsatisfactory when we consider the several causes of coma. In one way however there several causes may be brought into relation with each other, for they all act by affecting the nutrition of the brain, by depriving it of a free supply of healthy blood. Dr. Savory defines coma as
a state of insensibility from which the patient cannot be completely aroused, tether with a tendency to death chiefly by asphyxia: (Medical Times "Gazette", Jan. 10, 1856, p. 41.)

Dr. Héré (see Medical Times "Gazette", Mar. 21, 1858, p. 41) accepts, with one slight modification, this definition of L. Savoy's. The modification being the substitution of the phrase "evidence of defect of consciousness" for the term "insensibility" as being more correctly descriptive of a state in which it is impossible to determine whether or not consciousness is absolutely lost.

The term coma as it is generally used expresses a state of functional inactivity of the brain beginning at the cerebrum and characterized at the outset by loss of consciousness, perception and voluntary motion. In this state Automalic...
and after movements continue afterwards the large paralytic masses at the base of the 
brain become riolover, and the capacity for automatic 
movement becomes more 
and more hinderer--touching 
the conjunctiva or cornea no longer produces writing, 
the pupils may fail to 
contract to light and the 
visual field becomes lost-
the but slight comma iris 
this visual field is often 
obliterated or lost altogether. 
The voice is relaxed or placid. 
The paralyzed muscles are 
usually placid; the latter 
by falls heavily again, the 
bladder contracts slightly, 
the cheeks & lips help with the 
movements of the 
respiratory air and the paralytic 
affection of the muscles about 
the region of the pharynx 
was used to strain on 
Snoring. Dr. Bowles discloses the following variations of 
stenor: (i) Velarine stenor
When the air in rushing through the nose or mouth causes a vibration of the soft palate.

2. Pharyngeal Stridor - which is the most frequent in inspiration, and by far the most dangerous - this variety depends upon the base of the tongue dropping back into the pharynx and obstructing the passage of the air. It occurs when the mouth is wide open and is a harsher and a stronger noise than either of the other varieties.

3. Vocal cords Stridor - depends upon the air bubbling through the vocal cords in the larger bronchial tubes. It may exist alone or in combination with either of the other varieties.

4. Laryngeal Stridor - heard most frequently during the inhalation. Stridor as pointed out by Prof. Letterman was likely due to spasm of the
Thus, or from paralysis of some of the language muscles, Dr. Bowles is not prepared to say.

1. Nasal Stridor. Due to paralysis of the nerves supplying the elevator and dilator of the alae nasi, so that the inflowing air as in sniffling draws the alae nasi towards the septum and obstructs the breathing. This can be remedied by pressing the tip of the nose upwards or by dilating the nares. He considers this a very grave symptom as it is dependent on paralysis of the pons dura and therefore indicates that the mischief is spreading to that part of the brain which governs the functions of organic life, or that it is due to the venous engorgement at the base of the brain in consequence of the
Supportive action damping
the Poplar

b. Nasal stridor - the puffing out and flapping
of the cheeks and nose is dependent on the same
causes as laryngeal stridor.

He very clearly points out
the advantage of the lateral
position in stridor under
every condition. The
tongue in this position
pulling forwards, the mucus
draining freely away, and
the ease in breathing.
Thus re-established causes
great improvement of the
general symptoms.

In apoplectic cases he shows
that the patient should
always be placed with the
paralyzed side downwards
to prevent the circulation
of the fluids from the
mucous covered lungs to
the paralyzed side to the opposite
side, as in its passage
across the trachea the
mucus becomes chronic.
up into foam by the
infectant air, cannot. Nasal
Stridor, Great Hypopnea, and
if not removed, death.
He insists on the importance
of position in all conditions
in which much fluid is
contained in the lungs. So
that - the lung may as far
as possible be left free
for breathing, citing examples
illustrative of its application
in cases of drowning in the
Sanctuary of Dec. 28th, 1880. p. 1010.
Nasal Stridor he says is
unaffected, but may be relieved
by mechanical means.
Palatine Stridor is of least
importance. It obstructs
the breathing only partially;
and cannot always be
removed by changing the
position of the body. It is
affected by the size of the
tongue, length of the
larynx, position of the
clavicle, and other incidental
conditions, all of which may
be obliterated if necessary.
Pharyngeal stenosis is most common in fever cases, especially when the patient is recumbent. Here the patient should be arranged so that the paralyzed tongue may gravitate to one side, rather than against the back of the pharynx.

In coma stenosis, when unconscious with lump enlargement only occurs in very serious cases, depending upon interference with the nutritive processes of the lung tissues, probably resulting from accident or pressure upon the medulla oblongata, and proper attention to the position of the body may remove this.

Another advantage of placing the patient with the paralyzed side forward is that the injured side of the brain is upward and therefore relieved from hydrostatic complication. (Medical Times June 24th 1860, p. 136. Lancet - Dec. 23rd 1860, p. 971.)
June 18th, 1870. P. 876.

In a paper in the "Medical Times," Feb. 16th, 1860, p. 178, also wrote on the importance of position in coma.

In more serious cases the loss of function may extend to the medulla oblongata, and we get the "bulbar symptoms" as they are called. The affection may be in the centres presiding over the functions of circulation, or respiration, or the thermoregulating mechanism that regulates the animal heat. These bulbar symptoms only occur in deep coma and are of great importance as pointing to a fatal issue.

The pulse is probably at first slow, full and hard, though it may be quite unaffected, but as the coma increases the failure of the circulation may be indicated by a rapid, weak and thready pulse.
to 140° or even 160° in the

The respiratory function is
affected in one of two ways,
either by a failure in the
true oxygenation of the blood,
the patient becomingcyanosed
or the respiratory rhythm
becomes irregular—"central
breathing" as it is termed, passing
by regular gradations into
the typical Cheyne-Stokes
type, where periods of complete
apnoea alternate with
series of respiratory move-
ments, each series commencing
with shallow efforts gradually
increasing in depth and
force, and gradually dying
away again to cease in
the next period of apnoea.

Dr. Increase in a letter by scientific
exposition (see Medical Times and
p. 411) states that the failure
of respiratory power is due
to the same condition of
powerlessness and loss of
muscular tone that
penetrate the whole organism.
in the comatose state. He says that the comatose patient is not inert because he is unconscious, but because he is actually paralyzed. The cardiac muscle alone not there in this general paralysis. From the condition of complete coma to that precamotose of the mental confusion are together, weakness, there are all gradations as may be well seen for example in alcoholic poisoning. These various {\underline{\text{temperature}}}$

are met with in the comatose state:

6. There may be considerable general depression, especially in Maligne coma and in some cases of tubercular meningitis, sinking even as low as 80° in the bladder. There is also a slight or but slighter and temporary depression in coma accident on cerebral hemorrhage, and
in some cases of softening, according to Retzius (see Lausen, 32d 1880, pp. 581), a very low temperature, four degrees or more below normal in those usually termed Comatose or grave concussion of the brain. After epilepsy, apoplexy, intoxication, the temperature is more often normal, or even slightly above normal.

(2) This may be of very considerable use, with hyperpyrexia from whatever cause, as it is usually attended by Coma. Coma dependent on lesions about the four is particularly apt to be associated with heat. Elevation of temperature to 108° or 110° is almost the fatal result of hemorrhage into any part of the cerebrum or commissure, even to a considerable elevation. Coma is usually dependent on lesions in the lateral ventricle, is associated with a full, followed by a rather coarse
rise of temperature.

Another variation of temperature met with in the comatose state is that known as pyrexia. While the temperature of the exposed part of the body is very low, there is a great rise of that of the interior e.g. of the stomach. This occurs also in cholera and in other conditions.

Bonaventure and others have made a careful study of the range of the temperatures in cases of cerebral hemorrhage. There is always an initial depression, and in very severe cases death may occur during this period, within an hour or two of the onset. In cases less rapidly fatal, say from 12 to 24 hours, the initial fall is followed by a rapid and continuous rise. In the commoner class of cases where death occurs after—
a few days the initial fall is followed by a rise to the usual level or a little above, where, with slight oscillations, it remains for a time and the fatal event is again preceded by a steady and continuous rise. This course is well exemplified in the accompanying chart.

Dr. Southey remarks on the difficulty of diagnosing in some cases of coma (see Lancet, Dec. 4, 1830, p. 881) says that coma due to effusion into the ventricles is a gradually increasing torpor up to profound coma. Reflex movements may be at first excited, later the face is pale and the temperature falls. The breathing is either Cheyne-Stokes or principal triphasic with a stridorous sound. The pupils are non-characteristic. This is due to depressed posture or to effusion between the brain and membranes.
In severe, the breathing is stertorous. The pupils contracted. The temperature normal or above normal, the skin perspires profusely, no, or only slight reflex responses. In that case is alteration in the molecular state of the brain, local contusion, or apoplectic extravasation. The face is usually pale. The temperature line subnormal, reflex irritability is oft to be greater in one side than the other of the body, than the other. The pupils may be unequal. There is generally some evidence of hemiplegia. Hemiplegia in a case of coma points rather to localized rising of the brain than to general pressure.

In coma due to brain poisoning, significant clinical appearance, other than breath, should be noted. (See Lancer, Dec. 11th, 1880, p. 881.) So much for the phenomena.
observed in a state of coma.

The Pathology I shall touch
but briefly on, confining
myself very nearly to a
bare enumeration of the
conditions which may determine
its onset.

We have every reason to
believe that consciousness
is a function of the
gray matter - the cortex
of the cerebrum; and further
that a momentary suspension
or disturbance of the
innervation of the cortical
cells suffices to produce
a marked change in the
state of consciousness -
either in the direction of
perverted though wasted
action - delirium - or, which
more nearly concerns us
now, its complete abatement,
which is coma.

Johnson has maintained
that all forms of coma
are due to asphixia of
The cortical elements—a representative assimilation of supply of oxygen (see Medical Times-Gazette April 3rd 1869, p.357). Doubtless this is the commonest element affecting their nutrition, but other forms of disturbance are equally potent; it seems, to say the least, an unnecessary far fetched idea to contend that coma is dependent on extensive laceration and destruction of the cortex co-terminally traceable to the deprivation of the vis lifter elements. Oxygen—this seems to partake more of the character of an epiphenomenon. The function of the cortex is maintained by a constant supply of blood of normal quality, and it is to the disturbance of the circulation that we must look for the production of many of the forms of unconsciousness. The disturbance...
may be of the nature of a quantitative change or a qualitative. The changes in quantity of supply are either deficiency or excess. A deficient supply of blood may be a part of a general deficiency in the whole body, as in swooning from hemorrhage, or the deficiency may be local and confined to the brain. This is what occurs in stunning or concussion, which is usually due to some external injury, as a severe blow on the head. The cerebral spinal fluid is displaced and more or less passing to the base of the brain and region of the medulla, affects the vasomotor centers there, which in their turn produce a contraction of the smaller arteries of the brain and so the cortex is deprived of blood.
The contractile arteries shortly relax, and the temporary contraction is followed by a paralytic relaxation. A condition of concussion. All the phenomena of concussion may be produced by a sudden suspension of or a sudden hemorrhage.

Changes in the direction of excess of concretion of the brain may probably in the same way an conditions of Anacemia. Although the vessels are dilated the current is slow and there is still a sufficient supply of Oxygen to the Cellular elements. 

Changes, then, may be a general impoverishment of the blood as to its nutritive elements or to red corpuscles, thus we explain the growing rust with in conditions of profound Anacemia, and the comatose condition that ensues upon a state of profound Anacemia.
of the so-called spurious hydrocephalus in children
the subjects of protracted diarrhoea or other uraemic states.
The qualitative change may also consist in the presence
in the blood of some
one morbid material,
or a group of the same.
Those generated by the internal economy of the
body by unnatural metabolic processes are
four in number, occurring
in the states of uremia, diabetes, malignant jaundice
and asphonia.
Those introduced from
without are either chemical
or vital.
In the former class belong
the various narcotic
poisons - Opium, Chloroform,
and the like. In the
latter the specific poisons
of various infectious
fevers, such as Scarlet Fever
and Typhus, which in
sufficient intensity may produce a state of coma. The coma that occurs in the specific fevers may be perhaps traceable secundary to some of the other recognized agencies - either to the hyperpyrexia, or the accumulation in the blood of excretery products of which the kidneys fail to separate, and which in the febrile state are produced in excess - a state closely allied to true Amaurosis. The very peculiar state of coma, which occurs in typhus and rarely in endemic fever, would seem rather to depend upon the direct action of the specific poison on the nervous tissues.

Kussmaul was the first to describe under the name "diabetic coma" a peculiar mode of termination of diabetes, which he attributed to a "chemical
Decomposition of the blood, an "infection," which depends upon "chemical destruction of the economy in diabetes." The most characteristic symptoms which are (1) dyspnoea with the respiratory movement and unobstructed air passages and generally lower respiration; (2) rapid pulse; (3) excitement with groaning or sighing, vast, labor, severe pains, and finally coma. The temperature is not notably increased, and is sometimes below normal.

Dr. Saunby points out that this diabetic coma may occur as the result of self-infliction by the products of abnormal processes of decomposition within the body, due to the retention of normal excretion, the reabsorption of substances found in the body by abnormal concoction of abnormally large quantities of normal substances.
(see London Medical Record. June 15th 1884
p. 93)

In the Medical Times & Gazette
Jan 21st 1885. P. 139 is printed
a paper by Dr. Samby based on
the two cases of Kussmaul's
tumor, in which he points out
that while this peculiar
condition was originally
described as occurring in
Diabetes it is now
unknown but occurs in many
other diseases, especially
where there is profound
blood disorder e.g. Pernicious
Anemia, Bright's Disease
with Anemia, and in
Echocetic States. He discussed
the etiology of the
condition, rejecting
untenable the various views
which ascribe the symptoms
to anatomical changes, whether
of the nature of Pneumonic
Vitriase or in the shape
of fat Emboli and
maintained that it is more
probably due to some
at present undeterminer form of toxemia.

Apart from agencies acting directly through medium of the circulation, the nutrition of the cortical cells may be affected in various ways.

Of local conditions we may cite cross mechanical affection, such as result from injury or compression by depressed bone, extravasated blood on the products of inflammation, intellectual spasmaneous hemorrhage or true apoplexy, softening from vascular occlusion, irritative inflammation. Whether of the brain itself or its membranes, the growth of neoplastic or granulomatous tumors, and the chronic sclerotic change of seleral paralysis and disseminated or vascular sclerosis.

Of functional conditions epilepsy and apoplectic disease or serous apoplexy. Any of these may be attested
by coma, and their pathology calls for no further comment here.

The last condition which I shall mention is that of hyperpyrexia, where the associate coma is likely due to a change in the cortical cells. Directly 1 point sent out an increased heat, and analogous to the cloudy swelling and granular degeneration met with in granular and muscular tissues, coma from this cause occurs in the course of acute rheumatism, in diphtheria, occasionally in typhus and typhoid, toward the close of rickets, and in chronic cases. It is believed that any case of coma may be referred to one or other of the conditions which I have thus cursorily enumerated and the vol-
is sufficiently long. On the accompanying chart I have arranged these etiological connexions in the form of a table, which will show perhaps more clearly their relation to each other according to the arrangement I have adopted.
When brought face to face with a comatose patient it is often a most difficult matter to arrive at a correct conclusion as to the cause of unconsciousness. I propose to reflect altogether those cases which from their nature and the circumstances under which they occur can give rise to no difficulty in diagnosis and to confine myself to those in which difficulty may arise, illustrating some of those difficulties by cases that have come under my own observation. My friend Dr. Cave, late resident medical officer at the Bath Royal United Hospital, has also kindly favoured me with a few illustrative cases from his case book. We will reflect then those forms of unconsciousness associated with difficulty of blood supply to the brain.
as also those occurring in the course of various specific maladies, such as typhus, when not uncommonly it is a precursor of death.

We may note in passing that nearly all diseases in which delirium or delirium of sepsis are present—at any stage, are apt to pass into coma, which signifies merely a deeper functional disturbance of the same anatomical elements concerned in the production of these symptoms.

Of the other conditions which I have enumerated, and nearly any one may lead to the sudden development of coma, confusion may arise in their distinction. In some cases, however, will nearly always give rise to premonitory symptoms, which in the presence of a correct history will present unmistakable signs.
with abscess of the brain or idiopathic meningitis. In all three the presence of symptoms pointing to intracranial mischief, such as headache, vomiting, optic hemianopia, or convulsions, may fairly be looked for prior to the establishment of coma - still difficult to arrive at.

A girl, aged 21, single, was admitted to the hospital in a state of stupor. She had not previously kept her bed, but beyond this fact and the statement that she had vomited two or three times on the morning of her admission, nothing could be found. None of the limbs were paralyzed.

All the reflex actions were sluggish but equal on the two sides. Temp. 98.8; Pulse 80. Respiration 18, regular. Tone normal. Nothing objective to nature except the stupor. This gradually deepened.
The urine was passed into the bed. This passage of urine at once awakened suspicion that something serious was amiss, for previously a functional condition was suspected. In this case the stupor deepened into profound coma without localizing symptoms whatever, and a normal temperature throughout.

After death a tumour was discovered of a gliomatous nature at the base of the brain, very symmetrically disposed on either side of the middle line, and unimpairing mainly the optic thalamus and posterior parts of the corpora striata.

As regards Abscess take the following case:

G.B. aged 28. Servant. Was admitted to hospital for paralysis of the left
hand good and arm.

She stated that a footnight before she pricked her finger and it blest itself. The same evening she lost the use of the three fingers on the ulnar side of the same hand. There was no abnormal sensation. Four days before the loss of power intended to the rest of the hand and forearm.

This paralysis continued after admission. The electrical reactions were normal, the temperature normal throughout and no signs of the hysteric.

It was a complete semblance of hysterical paralysis. Fourteen days after admission the vomit changed to syrupy and quietly comatose and the same evening died.

At the post mortem a small abscess was discovered the size of a pigeon’s eye, which I will not describe further than to say that it
occupies the area affected by sensory movement of the hand or forearm.

The following will show that meningitis may also complicate—
E. M. act 27. Single servant was admitted to hospital complaining of headache and much malaise of eight days duration.
On admission she was in a state of lethargy, made no complaint and answered very briefly. When addressed she has suffered frequently from hysterical fits—there were no positive objective symptoms. When stimulated by the faradic current—she cried and threw herself about like a hysterical girl. The next day however there was noticed to be slight
drawing up of one angle of the mouth, and twelve hours later distinct right hemiplegia—
The stupor persisted to coma and three days after admission she died.

There had been no vomiting, no photophobia, a marked family predisposition to phthisis.

At the necropsy was found the typical lesion of tubercular meningitis.

This case seems admirably to emphasize the caution that no ailment should be regarded absolutely as functional until its inconsistency with any known organic basis is established.

When there is a possibility of an organic lesion, however improbable it may seem, we should always suspect our judgment and remember that in hysterical subjects the first symptom of some grave organic affection may be a typical so-called hysterical fit.
A still greater difficulty arises in the case of absolutely sudden or temporary unconsciousness, where a man is struck down suddenly unconscious while about his daily business, is often taken in the public street, and found by the police.

Here we have to distinguish between the action of poisons, internally generated, as in asphyxia, or externally foreign to the body, accidentally or maliciously administered or taken, as Opiates and Alcohol. The effects of injury, or impotence, cerebral haemorrhage or softening, convulsive attacks especially, or the so-called Fevers, or Simple or Severe Apoplexy.

The best process of examination, and that usually adopted in hospital on the admission of such a case, is the
absence of any reliable history is:

First to make a thorough examination of the body as to the presence of any wound or other injury, especially of the heart, always remembering that a man may suffer from concussion, or even fracture of the skull from violence, with no external manifestation of injury; and conversely that a man falling in a sudden fit, not infrequently in the very act sustains a bruise or wound of the scalp, note haemorrhage or discharge from the same.

Next as to the colour of the breath—the commonest smell is that of alcohol and that which gives rise to most errors. Book distinctive are the characteristic

odours of Russian Aar.
or Carboxylic Acid, or Opium,
or the sweet breath of
the Diabetic Coma.

The fallacies in connection
with the alcoholic form
are well known.
A man may be simply
drunk and comatose—
he may have in addition
haemorrhage going on in
his brain, or a fracture
of the skull; or a man
falling in a pit
may have previously
had a glass or two of
spirits, and finally
spirits may be poured
down his throat by
bystanders when he is
unconsciousness.

Here again caution is
the wisest course—
far better let a comatose
sleep of his rebirth
in a hospital ward
than for a fractured skull
or apoplectic effusion
to remain in the cells
of a police station.
In any case where a man cannot be fully aroused by tactile senses by a cold effusion or electrical stimulation he should be put to bed and watched by a competent nurse until he recovers.

Scansile in connection with such cases constantly occur.

Note next the presence or absence of dizziness of drop of temperature of renal affections and the possibility of trauma.

There important information may be derived from the eyes. Conjugate deviation a forcible turning of the eye to the side in the comatose state points to a lesion of the cerebrum on the same side as that to which the deviation occurs. As some one says, "the eye seems to be looking for the lesion"—hence the symptom has much the
same significance as hemiplegia - a turning of the head and neck in the same direction is confirmatory. The conjunctival reflex is commonly absent in deep coma; the pupils are sluggish, or fail to react at all. In cerebral haemorrhage they may be dilated, especially when death is near; contractions, irregular and natural, they may be unequal as they the result of injury, or from the effect of old disease as triton contracted in Opium poisoning, in affections of the brain, in poisoning by carbolic acid, in apoplexy, in ordinary sleep dilated by alcohol, chloroform and other drugs.

An ophthalmoscopic examination may reveal the characteristic cervical change of kidney disease, or the presence of other lesions, as in coma from lead...
Impregnation.

The temperature, pulse, and respiration are noted as matters of importance. A considerable rise in temperature occurs in meningitis or encephalitis, and in some cases, whether acute or chronic, 9 hours of the fever occurring becoming in the course of acute inflammation it can present no difficulties. The temperature in cerebral hemorrhage we have already referred to. In uremia it is generally depressed, never elevated, and may still as low as 80.7.

A prolonged depression points generally to uremia. The pulse may be slow and thready, and in renal disease may be associated with either with uremia or cerebral hemorrhage, which occurs most commonly in conjunction with...
Granular vitreous - it is slow in many cases of increased intracranial pressure. The respiration is slow in many forms of coma, especially in opium poisoning and apoplexy. It may be regular as already mentioned.

Next we look for any indication of local paralysis which may be seen even in a slight form.

Often in hemorrhagic conditions the limbs on one side will be a little more placid, or fall more helplessly when raised, than on the other, and especially significant is a difference in corresponding reflex action, when such can be obtained.

Finally, as soon as possible we examine the urine to ascertain the presence or absence of albumin, sugar, red blood cell deposits, etc.—Drawing off
with a soft catheter for this purpose, what may be in the bladder.

Dr. Johnson, in his clinical lecture on diseases of the kidneys, published in the Medical Times and Gazette on March 27th, 1858, wrote about the importance of always examining the urine in cases of illness in which coma exists as a symptom. In citing an illustrative case in which a patient suffering from renal disease was comatose, he remarks on the peculiar muscular twitchings sometimes observed in advanced cases of Bright's disease. This instability of the muscles being probably induced by the impurities circulating in the blood.

In any case of sudden unconsciousness we cannot afford to neglect any one of the data thus obtainable.
they will suffice for a probable, if not a definite diagnosis.

Take for example the following as illustrating the distinction between alcoholic poisoning and severe injury of the brain — the familiar "sensation of a drunk!" of the sensational reporter:

A young man was found by the police some time after midnight, sitting on a door step in a disreputable street. He was removed to the station where he was found to smell strongly of drink. He remained in a cell till 6 a.m. when, as he could not be waked, he was brought to the hospital and at once had a profoundly comatose, with a few slight bruises on his scalp and other parts of the body — no localising symptoms.
of a paralytic nature.

Temp. 98.5. Pulse 10. Urine normal.

Pupils sluggish and of moderate size.

Here the report of the coma and its duration points to some cause other than alcohol for its production.

Twenty-four hours later, his condition being otherwise unaltered, the temperature rose slightly — there were signs of facial paralysis later followed unilateral convulsions, and finally death.

The examination disclosed a fracture of the base of the skull.

Cases of this kind could be multiplied indefinitely.

Dr. Hume, in speaking of the phenomena of convulsions and coma, says that it consists of a long process of degradation, the last point of attack being the heart.
penultimate the breathing, as with the body so with
the mental functions. The
progress of coma is marked
in the one as in the
other by a gradually increasing
incoherence at first—
then difficulty in performing
delicate mental operations,
and then inability for any
coherent thought and, lastly,
evidence of want of
consciousness, in response
to impressions from without.
No evidence of response
to impressions from within.
And this coincides with the
relaxation of muscular tone,
the ataxia passing into
paralysis so that the
power to execute voluntary
movement fails pari passu
with the failing intelligence,
and suggests overwhelmingly
the inference that—both
mental and bodily defects —are
the same.

(Medical Times Journal 1886, p. 411)
I will relate one more case of fracture of the skull as illustrating the complete absence of symptoms in this serious condition:

A middle-aged man, laborer, came to me one morning with a scalp wound of about two days' duration. He said that whilst riding on a mow of hay by night, he fell off, and was half drowned. It was a fine night so he went to the next field, lay down, and slept till morning, when he was all right except for the slight wound of the scalp. This was dressed in the ordinary manner. Two days later, on his way to work in the morning, he suddenly fell unconscious in the street, he was taken to the hospital and put to bed. When I saw him he was prostrated, comatose—He rapidly developed symptoms
of hematomas, and at the post-mortem examination we discovered a large fracture in the posterior fossa of the skull at the base, which had doubtless occurred when he fell.

A week before any symptoms pointing to brain modification occurred.

To illustrate similar difficulties in cases of cerebral haemorrhage, take the following:

I was standing one evening on the platform of the Bath station talking to the station master, when he called my attention to a woman who had just got out of a first-class carriage. She was palpably intoxicated and, after staggering for a few seconds, fell on the platform. On approaching closely, the odour of alcohol was pronounced. I took hold of her hand.
and, more from habit than anything else, felt her pulse - I was immediately struck with its extreme tension. Then observed her more closely - she was about 45 years of age, and rather puffy about the eye lids, she would not, or could not speak intelligibly.

Watching her as she struggled I thought her right limbs were stronger than the left, although by no means paralysed. Finally, he removed her to the hospital, where in a few hours she became hemiplegic and comatose, and died in three days. She had a large clot in the left cortex striatum and its neighbourhood. With advanced pyramidal degeneration of the kidneys - her temperature chart I have referred to above as illustrating the curve characteristics of cerebral
An interesting case of hemorrhaige.

An interesting case of

Semi-natal Apoplexy is recorded
in the Medical Times & Gazette
Nov 2nd 1867. P. 489.

A man, aged 41, was
picked up in the street
and brought to the London
hospital by two policemen.
He was unable to speak,
and did not appear to under-
stand. His speech paralytic;
pupils normal. Movie
albuminorius.
Six hours later he became
comatose. Later, sensation
of the eyes to the right
was observed. He gradually
sank and died next day.

Apoplexy was diagnosed,
but the autopsy showed
the whole subarachnoid par-
mater much inflamed, with
much panulent infiltration.
No villi to the skull could be found, although
most carefully looked for.
There was a history of walking to the beat which had occurred in a railway accident 15 months before, and from that time the wife said he had been a 'different man' and subject to thirst except for severe pain in the head for a few days before his death, he was as well as usual. Eating and sleeping well. No rise of the temperature was taken.

In this case undoubtedly the most natural conclusion to arrive at was that it was a case of apoplexy.

The following case, which my friend Dr. Cave has kindly favoured me with, illustrates conditions of sudden coma with raised temperature.

In the very hot summer of 1850 a London postman was found sitting on the pavement by a police man
vomiting. He was taken to St. Bartholomew's Hospital. On admission he was quite unconscious, very pale, with weak rapid pulse, noisy breathing, and dilated pupils; motions passed involuntarily. His temperature was found to be 109°F.

He was treated with external application of cold rice to the head, and ice and enemata.

His temperature reached a point—between 110° and 111°—but he recovered. An extremely rare event.

The case was one of...

Inoculation

Death from cold is in this country happily rare, and, when it does occur, is probably partly associated with starvation. The fruit effect of prolonged exposure to cold, beyond the mere bodily suffering caused by it, is often a tendency to—
Another illustrative case of sudden coma with raised temperature is the following:

A footman, aged 27, came under my care complaining of vague nervous symptoms apparently of the nature of hypercholinuria.

One morning he was found in bed with an unconscious
with a flushed face, rapid pulse, and a temperature at first 101.7, which two hours later was found to be nearly 104°. The next day the coma had passed off entirely, but there was slight hemiplegia. The temperature fell as rapidly as it rose and the hemiplegia disappeared in a few days. This was one of the so-called convulsive attacks and established the diagnosis of General Paralysis of the Insane.

The main characteristic is the rapid and considerable rise of temperature early in the attack. This distinguishes it from ordinary epilepsy in which a solitary fit causes no pyrexia, a long succession of rapid and recurring epileptic convulsions may be associated with a considerable rise of temperature; but this is easily
So far from the
convulsive attacks we are
now Viscous.
In the course that follows
an ordinary epileptic fit—
we usually derive assistance
from the history of previous
attacks; while the condition
generally manifests itself
rather in a stupid, vacuous
state which is very suggestible
of its nature.
In cases of doubt a few
hours watching will
establish the diagnosis.

Another form of unconscious
ness is that which was
formerly called Simple or
Seron Apoplexy—
a number of such cases
are recovered by liberality,
but doubtless many of
them were cases of
ordinary epilepsy, some perhaps
of mania.
We must remember that
in old people the Brain
atrophies and shrinks, and
The space is filled by an excess of cerebro-spinal fluid - the serum effusion formerly considered characteristic of simple apoplexy.

Still a form of apoplexy unconsciousness does occur now and then in old people. it whose brain the most skilled pathologists have failed to diagnose ought always may be considered cases of simple apoplexy.

I have never seen a characteristic fatal case.

Lastly to illustrate the toxic form of coma - and first those due to the injection of poisonous substances wholly foreign to the body -

the number of persons suffering symptoms of coma is very considerable, but only a few are met with except at rare intervals.
Some of the commonest causes of coma are alcohol, chloroform, chloral and their allies, opium, carbonic acid, formic acid. The diagnosis of coma due to these agencies rarely causes much difficulty, as there are usually circumstantial points of evidence which immediately direct attention to the probable causation.

The danger that alcoholic coma may act as a mask to some grave condition I have already insisted on.

The following cases are fair examples of the many cases of coma:

Coma from puerperal:

I was one morning called hurriedly to see Mr. W. B. aged 37, whom I found lying on her bed in a state of comatose stupor. The pupils...
were much contracted, face pale, no marks of injury, temperature normal to the hand. Respiration 12.
Pulse 100.

There was no obvious recognition table in the breast.

A small bottle containing laevanum, half empty, was
found on her dressing table.

She moved slightly when vigorously stimulated but
could not stand or

sleep.

Her stomach was very
weak and there being
nothing recognizable in the
washings but a little
orange pulp.

Strong coffee was injected
through the tube, and
atropine at moments hyper-

ercally.

In an hour later she
was rather more responsive
to electric stimuli.

Respiration 10, pulse 144.

She was in much the
same condition for two
hours, when she became rather more sensible, complaining of severe itching all over the body and violently rubbing and scratching herself.

Next day, except for severe headache, and inability to evacuate the bladder, she was quite recovered. She confessed that d套cked with nervous pains. She has taken an overdose of Laudanum.

The affection that may be most easily mistaken for opium poisoning is an acute affection of the pain as illustrated in the case mentioned in the beginning of this paper.

The instinctive case in point is carefully related by Dr. Taffe in his Principles & Practice of Medicine Vol 1. 1850. Which received even such an experienced viiiagnostican as
himself.
He also states that two fatal cases of poisoning by Prussic acid which he witnessed could not have been distinguished from asphyxia except by the
over and surrounding circumstances.

There are numerous case
records of cases of trauma
from different narcotic
poisons, but I will content
myself with recounting a
case of Carbolic acid poisoning
which came under my
own observation:—
A. W., aged 45, labourer,
was brought to hospital
in a semi-unconscious state.
There were brownish stains
about the mouth and lips,
the mucous membrane
of which was white and
swollen. He smelt strongly
of Carbolic acid.
He felt cold and clammy.
Respiration 14. Pulse 95, small.
The stomach was washed out with olive oil administered by the tube. However, gradually it became intussuscepted, muscles contracted and intussusceptum tightened.

The battery was used and artificial respiration employed, but the breathing became shallower and shallower.

He died twelve hours after admission.

At 1 P.M. the stomach and intestines were found compressed and considerably thickened.

One poison which we should remember may manifest itself in this way is lead. Besides convulsions and coma, the presence of a blue line on the skin and a blue hue of the gums are fairly constant, as is also the peculiar earthy
Cachexia, neural myxification is common, but not essential.

Dr. Francotide relates the poisoning of a family by lead as interesting cases of the influence of lead upon the nervous system. Selenium v. corna were present, and in the case of the mother a condition alike to catatpsy and ecstasy was also occasionally present. This has also been noticed by San Guerel and Macbers in the instance —

(Handbill's Half-Platy Abstract of the Medical Sciences July 1849. 197)

The conditions in which coma may occur from acute gastric toxic agencies are four in number —

1. Vesicular Ulceration
2. Diabetes
3. Acute yellow atrophy of the liver or malignant jaundice
4. Apoplexia

When these conditions
supervene suddenly in persons of whom we have no previous knowledge, some difficulty may occur in their recognition.

In conditions of malaria the occurrence of convulsions is usual, and between the convulsive attacks there is usually some slight recovery of consciousness. The convulsions are as a rule general, but may be unilateral or local, according to most observers they may occur without
tremor.

The following case will suffice for illustration:—

A sailor, age 45, was admitted to a casualty state to hospital.

This casualty told me that a fortnight before he had taken some
cholera and was much acutely ill and went after well
ward, symptoms, recovered and resumed his work,
As he sat not set up that morning she went to his room and found him in a sleepy condition, which made her suspect a fresh attempt on his life.

Directly after admission he had a convolution which was entirely right sided, succeeded by paralytic deviation to the left. His pulse was 96. Very tense. Respiration 32. Pulse and Verp and 8.7.8. Temperature 94.7.

His color in his health pupils equal and moderately dilated.

After a few hours he became somewhat more conscious, but still very drowsy.

Urines Sp. grav. 1010. Acid, pale albumenous. No froth. Some white spots in the retina near the macula. He has later general convulsions followed by
Deep coma -
At the p.m. we discovered
an advanced degree of
granular degeneration of the
kidneys - hypertrophied heart.
No recognizable lesion of
the brain.
The case was considered
to be one of pure
Uracemia.

Diabetic coma has
more than once brought
trouble on the practitioner
failing to recognize -
I remember reading not
long since the case of
the Metropolitan Police-
surgeon who was missed
two or three times a constable
appealing to him for
release from duty, under
the impression that his
symptoms were due to
syphilitic intoxication -
the man died in the
hospital a few hours
taken from diabetic coma -
and the surgeon lost his
The following is a typical case:—

7. 13. a charwoman went to her work one morning as usual, but was noticed to be stupid and heavy. I saw her and sent her off to the hospital. She could not stand on examination. Temperature 97.8. Respiration 20. Pulse 120. Very heavy and thirsty. She could not be persuaded to put out her tongue. Sweet odour in the breath. Deep sighing respiration, filling her breath to the utmost at each inspiration. ("air-hunger" as she somewhat more or less called)

post were colour with for clo. granular and fatty casts. She remained in the same state and died next morning. Her temperature rising just before death to 104°.

In coma due to yellow abscess of the liver, the presence of jaundice and other concomitant phenomena will prevent mistakes. This is a good instance recorded by Dr. E. T. Galt in the British Med. Journal, Feb 23 1844.

The coma of chlorosis is a very common phenomenon. It is the commonest form cause of the coma of the dying, and in ordinary cases can cause no difficulty of diagnosis. In the following case which I have heard Dr. Littlejohn describe it escaped recognition:

A middle aged man, a tramp,
was seated in a common lodging house with two others eating a platter of a dinner of beef steak and onions. In the midst he fell up and walked into a yard at the back, as his companions thought to say home. In a moment they heard him fall and stout out-forms him on the ground in a fit - he gave two or three suspet respiritory movements and died, as was thought, from central haemorrhage.

At the P.M. was discovered a large piece of very thin tough beef steak water in the back of the pharynx and completely blocking the larynx. There was no other disease and he had died of suffocation.

This case is a warning always to be on the look out for a similar condition. Whenever
there is a possibility of choking the finger should the thumb be thrust to the back of the throat, the teeth being forced open for this purpose if necessary. If this is impossible and the condition clear, tracheotomy should be immediately performed.

In a few of these cases which I have recorded there were errors in diagnosis, and in looking back on the records of these and other cases I am struck with the fact that although in some our accurate diagnosis was with our present method unavoidable, yet in others a greater attention to details of observation, which is often called the most essential point in clinical medicine, would have justified a more accurate judgment.
There was actually former.
Dr. Harcliff writes that cases of coma from supposed apoplexy are often in reality but a transitory coma from cerebral exhaustion, and urges that every case of coma should be seen in this way, and treated accordingly, until the contrary conclusion is forced upon the mind by the unsatisfactory progress of the disorder.

(Stance: April 12th, 1873. Pp. 18)

This concludes what I have to say on the subject of coma.
I have taken it purely from a clinical and practical stand point.
The consideration as a psychical state I have not dwelt upon, although many interesting facts of psychology turn upon its correct interpretation.
On this I am incompetent.
to touch.

Much more might be said from the aesthetic side, e.g. on the coma of the dying. Let it suffice for us to note in how large a proportion it mercifully covers in its sleepy shroud the last hours or moments of the struggle.