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THESIS

THE DIFFERENTIAL DIAGNOSIS OF COMA

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UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE

1935

Introduction

Almost everybody has at some time witnessed some individual in a state of coma or stupor. This state of affairs stimulates almost everyone to do something, but often due to the conditions of excitement or other emotional circumstances the patient, whom all desire to aid, is done much harm due to the unintelligible and haphazard means which are taken.

This state of affairs in my experience, has thrown me into a feeling of helplessness. One desires much to help such a patient in a condition so acute, and if the condition is not understood there is always the fear that an error will push the patient into fatality, while the correct step would be as important and spectacular in helping the patient to recovery. In corroboration with my views R.B.Todd M.D., F.R.S. of London in 1850 said, "Among the most formidable indications of disturbance of the great Central organ of the nervous system, the brain, are those states which are known as coma and delirium. These states are so destructive to the consciousness of the patient, or pervert to so great a degree his intellectual powers that we cannot wonder that the utmost alarm should be excited in the minds of all, whether friend or medical men, who may be in attendance of him.

And perhaps there are few occasions in which the physician stands more in need of all that self-possession which sound knowledge is most likely to impart, than when during his

attendance upon a patient, either delirious or a comatose state, should be suddenly added to his previous symptoms."

William Macewen M.D. of Glasgow, 1879 said, "The Medical Journals and ordinary newspapers contain from time to time paragraphs detailing instances of mistakes having been made by surgeons in determining the cause of insensibility. The consequence of these mistakes have been serious and often fatal."

J.T.Eskridge M.D. 1898 said, "The proper management of cases of coma depends so largely upon our knowledge of the cause of the comatose state in each individual case that it is necessary for us to have a comprehensive knowledge of the various symptoms of the coma that results from each of the numerous causes."

David Forsythe M.D., D.S.C., F.R.C.P. of London 1912 has said, "Given a comatose patient, perhaps a stranger and, for the first time at any rate, with no friend to tell his history, how is this state to be diagnosed? The situation, though familiar to us all, can never abate its claim on our serious attention. The majority of such cases fall within the bounds of our resources."

E.D.Friedman M.D., N.Y. 1933 has said, "The physician frequently encounters patients in coma or stupor. The determination of the causes of the varying degrees of coma is the important factor since treatment is dependent on it."

E.Appelbaum M.D. and L.F.Bishop Jr. M.D. 1929 have said,

"A rapid solution of the problem of people in comatose states is often imperative."

G.S.Young 1934 said, "Since the discovery of insulin and the rapid development of brain surgery have introduced new methods of treatment, an early and accurate diagnosis has assumed new importance."

The above statements made over a period of almost all of the last century go to show that the problem of differential diagnosis of comatose states is not new and is one that causes much concern. For this reason I have chosen to review the methods used by men in the past and present, and from this knowledge formulate a routine whereby a patient found in coma can be handled intelligently, first with only those instruments commonly carried in the physician's ordinary kit, and secondly a routine to be carried out after hospital facilities have been attained.

The Differential Diagnosis of Coma

Dorland defines coma as a state of complete loss of consciousness from which the patient cannot be aroused even by the most powerful stimulation. He defines stupor as partial or nearly complete unconsciousness. As this paper is an attempt to summate and formulate a means of differential diagnosis of coma from a practical standpoint some of the deeper states of stupor must be dealt with. So in this paper in speaking of coma I wish to refer to it not only as true coma, but also to include the deeper states of stupor.

Coma as I wish to refer to it has been an object of interest for as long as history dates. James Breasted's translation of the Edwin Smith Surgical Papyrus brings to us a document which disclosed to us for the first time the human mind peering into the mysteries of the human body, and recognizing conditions and processes there as due to intelligible physical causes. While the copy of this document which has come down to us dates from the seventh century B.C., the original author's first manuscript was produced at least a thousand years earlier. Breasted has conjectured that a surgical treatise of such importance and appearing in the pyramid age, may possibly have been written by the earliest known physician, Imhotep, the great architect-physician who flourished in the thirtieth century B.C.

The seat of consciousness and intelligence was from the

earliest times regarded by the Egyptians as both the heart and bowels or abdomen. These surgeons however have observed the fact that injuries to the brain affected the other parts of the body and especially was experienced in the lower limbs. They noted the drag or shuffle of one foot presumably the partial paralysis resulting from a cranial wound. Here then is the discovery of the fact that the brain is the source of control of the movements of the body.

So the surgeon for the first time in the history of science noted the effect of the cranial injury on the lower limbs with reference to the side of the skull which had received the injury. In other words the orient Egyptian surgeon began observations on the localization of the function of the brain.

Charles C. Thomas on selected readings in pathology from Aegina 625-690 gives us proof of detailed observation of coma at this period. Aegina 625-690 found that when the common origin of the nerves, the brain, was affected, it resulted that all the other parts of the body lost their motion and sensibility. The affection was called apoplexy by which the leading energies were impaired, but if the obstruction was in either side it was called hemiplegi and paralysis, and if the injury was seated in any one part it was called an affection of that part, hence Hippocrates says "His leg was seized with apoplexy." Wherefore in apoplectics the respiration remained small. When it was greatly perverted from its natural energy, it would

induce a strong affection, and when but little a weak one. Respiration was regarded as worst when it intermitted or was performed with great exertion. Aegina thought that the affection arose suddenly from a cold phlegm obstructing the most important cavities of the brain. If the sense of smell was affected, this arose from the anterior cavities of the brain or from the pores of the ethmoid bone being obstructed. Thus apopleptics laid speechless, motionless, and insensible without fever. The precursors of this affection he found was sudden and acute pain in the head, distension of the juglar veins, vertigo, flashes as it were of light in the eyes, an inordinate coldness of the extremities, palpitation and difficult motion of the whole body and grinding of the teeth in sleep. Their urine was small in quantity and of a greenish or black color. The affections occurred in old age in those of a phlegmatic temperament, and those who used a diet of this nature. If it occurred in youth, it was in the season of summer and it indicated a strong exciting cause.

Another interesting article on coma in regard to its physiology was written by J. Mac Donnell M.D. 1835, who made observations with a view of directing attention to the physical condition of the brain for example with reference to the circulation of the blood in it and to the pressure of atmosphere on it. He pointed out that those acquainted with laws of the atmospheric pressure and the manner in which it may be conveyed

by circulating liquids, will know that the brain is as completely under this influence as if the brain was exposed naked. He also observed that in many diseases of the brain, comatose symptoms etc. were almost constantly referred to by practitioners as compression of the brain as their cause, and this he pointed out will no doubt appear absurd from the following considerations together with what we know positively of the affect of increased pressure on the brain, that in the majority of cases compression of the brain had no concern in producing symptoms. For instance animals with a brain similar to man, for example the whale, can stand a pressure on their body equal to two hundred eleven thousand two hundred tons, without any ill effects, also man himself can stand much pressure in a diving bell. Due to transference of the pressure equally to all parts of the body by the blood as proven by laws of liquids; the brain receives equal pressure to that on the outside. In none of these cases was a comatose condition produced and he concluded that simple compression of the brain did not produce such symptoms. So Mac Donnell was convinced that the affect on the circulation is a circumstance of importance in the production of comatose symptoms, if it did not actually produce them.

The above citations represent the relative slow progress in regard to coma until the beginning of the nineteenth century. There was little progress from 3000 B.C. until 600 A.D. The conjectures in 1835 by Mac Donnell are uncertain and rather

crude but do show a fineness in that he based his reasoning on physical laws in proving and disproving statements and beliefs.

Charles Mercier M.B. 1835 was one of the first men to submit an article on coma with reference to observed symptoms. He noted a paralysis more or less complete of all the voluntary muscular system and an incomplete paralysis of the muscular system of the skin, of the arteries, of the bladder, of the pupils, and of the respiratory system, the heart muscles being the only ones not weakened in some degree. In all states of real coma he found the eyes moved independently of one another and even diverged in some cases, this being useful in ruling out malingering and hysteria.

George Johnson M.D., F.R.C.P. mentioned the difficulty in distinguishing the difference between drunkenness, epilepsy and other forms of coma. He recognized that first you must consider what are the possible causes of the symptoms of drunkenness. First the patient may have a clot of blood in his brain as the result of disease or blow. Secondly it may be due to epilepsy, and thirdly it may be due to excess alcohol or opium, and lastly it may be due to uremia. His idea was that if in doubt of diagnosis it is better to err, if at all, on the side of caution and safety.

J.T.Eskridge M.D. 1898 was of an opinion that proper management of cases of coma depended so largely upon our knowledge of the cause of the comatose state in each individual

case that it was necessary to have a comprehensive knowledge of the various symptoms of the coma that result from each of the numerous causes. He referred to coma to designate a more or less acute condition of insensibility from which the patient can be only partially aroused, or not aroused at all, this being practically the meaning I have bestowed upon it. He ruled out coma due to hyperpyrexia, typhoid, cancer other than brain and yellow atrophy of the liver.

He divided the causes of coma into seven groups which are:

I. Transient

1. Syncope in fainting.

II. From lethal doses of medicinal agents.

1. Choral
2. Opium
3. Belladonna
4. Hyoscyamus and its alkaloids
5. Alcohol
6. Lead

III. Coma from poison other than medicinal agents circulating in the blood.

1. Asphyxia from poisonous gases
2. Ptomaines
3. Uremia
4. Diabetes

IV. Convulsive states preceding coma.

1. The preeruptive stage of the exthemata, sometimes preceded by convulsion.
2. Reflex convulsion such as teething, overloading the stomach etc. in children.
3. Epilepsy
4. Hysteria

5. Epileptoid and appoplectoid attacks due to partidementia or other organic brain diseases such as lues and alcoholism.

V. Voluntary coma.

1. Feigning

VI. Coma from profound disturbances of the cerebral circulation, but unattended by organic lesions of the brain substance.

1. Shock
2. Concussion of the brain
3. Conjestion of the brain
4. Anemia of the brain

VII. Coma from organic disease of the brain.

1. Simple apoplexy of the aged
2. Trauma of the brain
3. Syphilis of the brain
4. Cerebral meningitis
5. Abscess of the brain
6. Tumor of the brain
7. Cerebral hemorrhage
8. Cerebral embolism
9. Cerebral thrombosis

Group I. Transient coma.

This he thought due to a sudden weaking or temporary arrest of the heart's action producing anemia of the brain and resulting in unconsciousness, more or less profound.

Symptoms:

1. Paleness of the surface of the body especially lips, gums and face.
2. Dialated pupils.
3. Cold perspiration if severe.
4. Respiration weak.
5. Thready pulse.
6. Face becomes cyanatic and puffing of lips at each respiration.

Negative Symptoms:

1. No convulsions.
2. No muscular rigidity.
3. No paresis or paralysis.

The fact that symptoms are fleeting in character and pass away on return of blood to the head is important in diagnosis.

Differential diagnosis:

Degeneration of the heart, apoplectoid attacks of cerebral origin, hysteria.

Degeneration of the heart will show other heart symptoms such as edema, history of similar attacks, heart enlargement etc., more marked cyanosis. Bishop S.F. and Bishop S.F.Jr. have summarized that:

1. Faintness or syncope is seen by the cardiologist relatively often as a chief complaint.
2. Its frequent occurrence in hypertension is emphasized. It is not often found to be associated with hypotension.
3. Disturbance in cardiac mechanism may often be the underlying cause of faintness or syncope.
4. Faintness or syncope may be a symptom of coronary disease.

Eskridge continues and has found that apoplectoid attacks have less cyanosis, may be twitching of face muscles, stertor for a few seconds, symptoms less fleeting on lowering the head and headaches subsequently.

Hysteria: pulse unchanged, no paleness or cyanosis.

Group II. Coma from lethal doses of medicinal agents.

Choral: Temperature lowered, respiration quiet, may be more full, pulse weak and rather slow pupils generally small, difficult to arouse. Later respiration irregular and rapid.

Opium: Slow pulse, noisy respiration, reduced to 4 to 10 respirations per minute, contracted pupils, capillaries of face dilated before death, pulse fast, eyes dilate.

Differential diagnosis: The contracted pupil, slow pulse, and labored respiration sometimes found from hemorrhage in the base of the brain, especially in the lateral ventricles or into the pons, may at first lead one to suspect poisoning from opium, but the subnormal temperature from hemorrhage into the ventricles, and sudden increase in body heat from pontine lesions, together with localizing symptoms would render the diagnosis quiet easy.

Belladonnae poisoning: Coma preceded by delirium and convulsions.

Skin has scarlet rash and is hot and dry, pupils dilated, temperature raised, pulse rapid, respiration at first deep and slow, later before death it becomes rapid.

Differential diagnosis: Scarlet color of the skin, the delirium and absence of localizing symptoms serve to distinguish coma from belladonna poisoning, from that due to brain lesions. The scarlet skin, the delirium and excitement of the heart, aid in separating from other mydriatics especially hyoscyamus and its alkoids.

Alcoholic coma: A noisy unconscious condition points to alcohol.

The patient may be profoundly unconscious, cannot be aroused, often striking at the one who is disturbing him, conjunctive congested.

One must bear in mind that a person who is intoxicated may be suffering from organic disease of the brain, either acute or chronic. In all cases a careful examination should be made for such.

Lead coma: Preceded usually by vomiting, purging and convulsion. Urine analysis reveals lead. Lead line if taken into stomach for several days.

Differential diagnosis: Sometimes it is extremely difficult to diagnose a condition of encephalopathy from lead unless a careful analysis of the urine is made for this poison, or the history should point to a source by which the metal has found entrance to the system.

Group III. Coma from poisons other than medicinal agents circulating in the blood.

Asphyxia from poison gases: Lividity of the tongue and lips, and embarrassed respiration are commonly found in these cases. Escaping gas, charred wood etc. help in diagnosis.

Ptomaines: The cases of coma that have resulted from the ingestion of putrid food, have usually been preceded by an irritating gastro-intestinal poison, which results in vomiting and diarrhoea for a few hours to days before coma.

Coma from uremia: The onset is often attended by delirium and convulsions, which would exclude alcohol and opium. Albumen and tube casts in urine suggest uremia, but albumen is often found in cerebral hemorrhage.

It must be borne in mind that renal disease especially the senile form, (granular disease of the kidney) occurs in about a third of the cases of cerebral hemorrhage. The peculiar physiognomy of a person suffering from renal disease with edema, equal pupils, often less profound coma than in cerebral hemorrhage with usually nearly normal temperature, the presence of urine odor on the breath, and the absence of hemiplegic symptoms occurring in a young subject are usually sufficient to enable uremia coma diagnosis.

Gower states that, "Rigidity of limbs or local muscular twitching during coma is, if constant in seat, is in favor of cerebral mischief; if variable in position, it is in favor of uremia. Temperature not important, equal in both sides help exclude brain lesions."

Diabetic coma: Usually gradual in onset, sugar urine. In absence of localizing symptoms the diagnosis is justified.

Group IV. Convulsive states. Coma ushered in by a convulsion is occasionally observed in children in the pre-eruptive stage of the exanthematous fevers, especially scarlet fever.

Differential diagnosis: Would be between these, exanthematous, fevers, reflex convulsions from an overloaded stomach etc.

Scarlet fever: Temperature high (104-105), pulse frequent, and generally only one or two convulsions, respiration rapid, and coma stops when rash appears. When convulsions usher in the obtrusive symptoms of meningitis, they may recur every few minutes to an hour until several have occurred. Temperature high 104-106 pulse less rapid. High temperature one convulsion, pulse 140-180 favors S.C. fever.

Coma from teething or an overloaded stomach: Rarely attended by temperature over 103 degrees, pulse less rapid than exanthemata, face flushed, temperature 103, pulse 120, flushed face would indicate overloaded stomach. Repeated convulsions, high temperature and pale face would suggest meningitis. Diagnosis can not always be made during the first twenty four hours or even thirty six.

Coma from epilepsy: If the history of the patient is known and the onset of convulsion witnessed, little difficulty of diagnosis is witnessed. However, persons suffering from epilepsy are usually subject to other causes of coma. If the patient is first seen in the comatose stage following a fit, and no knowledge of history present, the absence of hemiplegic symptoms and the short duration of the coma would serve to distinguish the case from unilateral brain lesion. In the absence of history it may not be possible to differentiate between sudden cerebral

lesion without unilateral symptoms. However in some cases of epilepsy the seizures are followed by a weakness of one arm or leg especially at the distal end of the extremity. Multiple thrombi and emboli may give rise to a condition resembling status epilepticus without being attended by a single unilateral symptom. Brain hemorrhage generally shows unilateral symptoms however.

Coma from hysteria: Subjects are usually females. Comes on suddenly, often preceded by delirium, or may alternate with it. Face flushed and hot, may be much physical exertion. Pupils are normal, and react normally unless the patient is making considerable muscular effort, and then they may be slightly dilated and react irregularly. Pulse normal, urine retained, respiration rapid but not stertorous.

Differential diagnosis: Hysteria, organic brain disease especially cerebral congestion, meningitis, or vascular lesion. Feigning may be excluded by the absence of all symptoms except those that may be produced by physical exertion.

Hysterical coma, and coma due to cerebral exhaustion resulting in passive congestion. When patient is profoundly unconscious and pupils won't respond to light, a diagnosis of hysteria should not be made.

With reference to hysterical coma we may conclude that:

1. The initial symptoms of a case of organic brain disease may apparently be hysterical in character.
2. The case may be characteristic of hysteria at the start and as it progresses, organic changes, or other changes proving equally intractable, may develop from exhaustion, disturbance of circulation, or disturbed metabolic process.

Coma from epileptoid and apoplectoid attacks:
Due to parietic dementia, or other organic brain disease, such as syphilis and chronic alcoholism.

In paretic dementia the history of the patient would be the only guide to the diagnosis, as the coma is similar in every respect to that of vascular lesion. When however the convulsion, with resulting coma is the first symptom of paretic dementia, time only would differentiate the attack from epileptic or apoplectiform in character.

Coma from syphilis and chronic alcoholism: Can only be diagnosed by evidence of certain organic brain changes. Also these persons are liable to other causes of coma.

Group V. Voluntary coma: Feigning. Everthing usually observed in an epileptic fit can easily be simulated by a clever malingerer except the change in the color of the face, the sudden and unexpected irritation.

Color of face: By muscle action redness of face can be produced, but it is very difficult to hold the breath until cyanosis occurs as in genuine convulsions.

Conditions of the pupils: Early in the fit the pupils are said to be contracted, but during most of the tonic stage and all of the clonic they are dilated and do not respond perfectly to light. On regaining consciousness they may dilate and contract every few seconds. However on severe muscular exertion the pupils are dilated and react sluggishly. So it is concluded that unless the irrides fail absolutely to respond the pupils are of no value in diagnosis of genuine epileptic fits.

Insensibility to suddenly inflicted irritation: In unconsciousness from epileptic convulsions, if the cornea is touched the eyelid moves a little, if the supra orbital nerves are firmly pressed, the forehead may corrugate a little. If a pin is thrust into the leg a slight movement may be produced but no purposeful effort. In feigned epilepsy a sudden irritation unexpected will cause an intelligent effort to be

made to avoid it. The best method of differential diagnosis is to give anesthetic.

Group VI. Coma from profound disturbances of the cerebral circulation, but unattended by organic lesion of the brain substance.

Coma from shock and concussion of the brain:
In severe shock consciousness may be completely lost and it immediately follows the depressing cause.

Symptoms: Pupils usually dilated, muscles relaxed, face pale, surface of body cool, temperature lowered, pulse weak and rapid, sometimes slow and irregular, respiration shallow and often frequent. Sphincters sometimes relaxed, and there may be vomiting.

Differential diagnosis of delayed shock which comes on sometime after the injury and shock, followed by delayed or imperfect reaction, both of which are due to concealed hemorrhage of some other complication: When the shock has been caused by a blow on the head, it is often difficult to tell whether the symptoms are due to shock of cerebral concussion, or to those of an inter cranial lesion. The absence of any localizing symptoms, and the presence of slightly subnormal temperature equal in each axilla: weak and rapid pulse, noiseless respiration with evidence soon after injury of beginning reaction, which is continuous after it has become manifest, point toward the functional nature of the trouble. It is sometimes impossible to exclude organic disease of the brain in these cases until days have elapsed. If coma and semi comatose condition lasts for days or weeks, even in the absence of an organic nature, it is probable that laceration of the brain has occurred or slight capillary hemorrhage into the brain substance has taken place.

Coma from cerebral congestion: It is rare for coma to result from cerebral congestion, unless the cause be isolation of the vasomotor disturbance found in parietic dementia. The lighter forms generally pass into stupor.

In this event it comes on gradual and is often preceded by headache and delirium. In the active form there will be history of exposure. The face flushed, carotids throb, pulse rapid, full and strong, temperature elevated, pupils contracted, conjunctive congestion, no localizing symptoms are present, and recovery is soon if inflammation does not result or the patient is not exhausted by high fever.

In the passive form the appearance of venous stasis is indicated, respiration rapid and noisy, pulse rapid and temperature is elevated one to two degrees. If death does not result recovery soon takes place.

In neither form of congestion is there any localizing symptoms of evidence of cranial nerve involvement. Differential diagnosis of meningitis is by stiff neck, and vascular lesion by unilateral symptoms.

Coma from cerebral anemia: Most commonly caused by sudden loss of blood. The appearance of the patient and condition of temperature and pulse would be just opposite to those of cerebral congestion. Delirium and convulsions may occur if loss of blood is rapid and profuse. Diagnosis is generally evident by history and symptoms.

Group VII. Coma from organic disease of the brain.

Coma from simple apoplexy of the aged: In persons advanced in life, a condition sometimes occurs with a train of symptoms that perfectly resemble those of a vascular lesion of the brain, but at autopsy no trace of hemorrhage or other gross lesion can be found. The age of the patient and the history of previous similar attacks would aid in the diagnosis but the first attack of epilepsy might puzzle the best diagnostician especially if unilateral symptoms as sometimes is the case is present.

Coma from traumatism of the brain: If an injury has occurred to the head and coma results the diagnosis will lie between simple concussion, shock, and organic lesion of the brain. Delayed coma, deeping coma, and delayed reaction from a deep comatose state would be almost

positive evidence of organic intra cranial trouble usually vascular in nature. Immediate coma may be due to an organic lesion or a functional disturbance. The sooner that reaction begins to take place and the more complete it is, a few hours after the head injury, the greater the probability that it is functional. Stertorous respiration, congestion of the face, slow full pulse, unilateral symptoms and convulsions would indicate intra cranial hemorrhage.

Coma from meningitis: In the absence of a history, the diagnosis would depend on rigidity of neck, cranial nerve involvement, increased temperature, irregular respiration, slow irregular pulse if pressure symptoms are still present, but rapid pulse, if the stage of exhaustion had been reached. Papilidema may be present. If of long duration, optic atrophy may be present. Optic nerve changes would enable one to exclude all vascular lesions except possibly thrombosis. Choked disc with evidence of meningitis would indicate that the meningeal inflammation was secondary to tumor or abscess. The greater the choked disc the more likely the trouble was tumor primarily. A tuberculous tumor or nodule is about the only growth of the brain that causes general meningitis.

Coma from abscess of the brain: The history of the case, with a probable cause of intra cranial suppuration vacillating temperature, slight swelling of optic discs and unilateral symptoms, if these were present, would be the principle aids to diagnosis. If no history is obtainable and optic neuritis is present and the other cause except tumor excluded, the differential diagnosis of tumor and abscess would be necessary. The greater the choked disc the more likely a tumor. The greater the variation of temperature from normal and the more profuse the perspiration during the coma the more likely that it is abscess. The terminal stage of abscess of the brain is usually attended with high temperature and the body is bathed in perspiration.

Coma from tumor of the brain: It sometimes happens that when a patient with a brain tumor is seen for the first time they are in a coma. This

may be the coma that just precedes death, or may be any time in the progress of the growth, especially if it is in the region of the cerebellum so as to cause pressure on the veins of Galen or cause effusion into the lateral ventricle. Pronounced choking of the discs would settle the diagnosis in favor of tumor provided that renal disease, anemia and lead encephalopathy had been excluded.

Coma from non traumatic vascular lesions: These include hemorrhage and thrombotic and embolic occlusion of vessels. Coma from vascular lesion is as a rule sudden in its onset and more or less profound, depending on the nature of the vascular disturbances. One of the most common symptoms is hemiplegia. Having determined that the coma in a given case is due to hemiplegia or occlusion of a vessel, it is important to determine the nature of the vascular lesion.

Coma from embolic occlusion: It is probable that we are never justified in diagnosing cerebral embolism in the absence of an apparent cause. Endocarditis, chronic in nature, with subacute attacks of endocarditis is the most common cause. Next most frequent cause is a suppurative process especially in the thorax or abdominal cavity. The parturient state and the blood changes often found in chronic syphilis, with exhaustion often favors embolism. Coma from embolism is generally shorter and less profound than in hemorrhage. Cases with more or less hemiplegia in young person without profound coma of several hours duration would be against hemorrhage. The less the primary disturbance of temperature provided the paralysis is extensive the less likely that we have cerebral hemorrhage.

Coma from thrombotic occlusion of a cerebral vessel: Atheroma of the vessels, a potent factor in the causation of thrombosis does not occur before 35-40 years of age except possibly in association with Bright's disease or as a result of it. Syphilitic arteritis is a frequent cause of thrombosis from the twentieth to the fiftieth year and it may occur earlier or later but the frequency diminishes in the two extremes of life.

Some points in diagnosis between coma of thrombotic occlusion and cerebral hemorrhage.

Thrombotic occlusion.

Cerebral hemorrhage.

- | | |
|--|---|
| 1. Prodromes frequent. | 1. Prodromes infrequent. |
| 2. Great degeneration of the arteries of the limbs or a history of syphilis. | 2. Turgid face and strongly beating of the arteries of the neck. |
| 3. Pulse soft and often compressible. | 3. High artery tension regardless of size of pulse. |
| 4. Heart feeble, dilated, and irregular. | 4. Heart hypertrophied and beating strongly. |
| 5. Coma less marked in depth and duration. | 5. Coma more intense and larger. |
| 6. Following depressing influence. | 6. Due to mental excitement. |
| 7. Local convulsion more frequent. | 7. General convulsion more frequent. |
| 8. Slight initial temperature disturbed. | 8. Often great initial temperature disturbance. |
| 9. Slight variation of temperature. | 9. Rise in temperature 12-24 hours after. |
| 10. Secondary inflammatory symptoms frequent and well marked. | 10. Secondary inflammatory symptoms less in frequency and degree. |

The above paper covers the subject fairly thoroughly but other points are stressed more clearly by other men and other systems of discussion have been used.

N.I.Spriggs has briefly considered some of the most distinctive features of each class of coma from a diagnostic point of view.

1. **Apoplexy:** Profound coma, sudden onset in an elderly man, thick arteries and may be hemiplegia.
2. **Compression:** History of injury with lucid interval following succeeded by profound coma, with possibly one pupil dilated or hemiplegic signs.
3. **Concussion:** History of injury immediately succeeded by a less profound coma, with pale face and feeble pulse and respiration.
4. **Epileptic coma:** History of previous fits or of convulsions preceding the coma, most probably a young

subject with possibly a bitten tongue, rapid improvement.

5. Epileptiform attacks of G.P.I., cerebral syphilis, disseminated sclerosis, or cerebral tumor cannot possibly be diagnosed from epilepsy while comatose.
6. Uremic coma: Coma with intermittent convulsions or twitchings, -other signs of Bright's disease.
7. Opium poisoning: Pin point pupils, ashen hue, and history.
8. Heat stroke: History and hyperpyrexia.
9. Alcoholic coma: History of little importance, alcohol will be left when other causes are excluded.

He points out that in different diagnosis between cerebral hemorrhage, embolism and thrombosis that:

1. The more severe and prolonged the coma the more likely it is hemorrhage.
2. Hemiplegia in which coma is absent or is only slightly present favors embolism.
3. Age favors hemiplegia or thrombosis, youth excludes hemiplegia unless there is an antecedent embolism.
4. Heart lesions favor embolism.
5. Gradual onset favors thrombosis.
6. Aphasia is slightly more frequent with embolism as the left carotid is a more easy path for emboli.

E.D.Friedman M.D., 1912 has classified and discussed the causes of coma as follows:

1. General eg. alcohol, uremia, diabetes, opium, and gas poisoning and hypoglycemia.
2. Epilepsy.
3. Intracranial lesions with or without focal signs.
 - a. Apoplexy
 - b. Meningitis
 - c. Abscess of brain
 - d. Tumor of brain
 - e. Encephalitis
 - f. Spontaneous sub-archnoid hemorrhage
4. Trauma
 - a. Concussion of the brain
 - b. Gross hemorrhage with or without fracture

I. General causes:

Alcohol coma is recognized by the odor of alcohol in the breath, the incidence of vomiting, delirium and restlessness. In the uremia we find evidences of nephritis, characteris-

tic changes in the fundi, hypertension, edema of the limbs, high N.P.N., transitory hemiplegias or monoplegias. The onset of coma in these cases is usually gradual and is preceeded by toxic headaches, vomiting, and other signs of toxicity.

Opium poisoning can be identified by the small pupils, relative slow pulse and shallow breathing.

Gas poison: History of exposure, at times one may detect a characteristic odor in the breath, and the spectroscopic test is positive. The patient usually presents marked cyanosis and a peculiar redness of the skin.

In diabetic coma we find the "soft eye ball" (due to low intra-ocular tension), elevation of the blood sugar level and signs of acidosis, including low carbondioxide combining power in the blood. There is usually a preceeding nausea, vomiting and epigastric pain.

Physical examination shows the characteristic Kussmaul breathing, low temperature and dryness of the skin (due to desiccation of tissue).

Hypoglycemic shock is usually the result of hyper insulinism. Friedman recalls two instances of this type of stupor, one was a case of supra sellar cyst with hypoglycemic crisis, the other a tumor of the third ventricle. These states were relieved by glucose.

G.S.Young has found that discoloration of the mouth hands or clothes is characteristic enough for corrosives.

II. Epilepsy: Post-epileptic stupor is identified by the history, evidence of tongue bite, and usually rapid recovery.

III. Intracranial lesions.

a. Apoplexy.

This is one of the most common causes of coma, and is due either to hemiplegia or thrombosis. The latter is more apt to occur slowly, especially during sleep, while the onset of hemiplegia is usually sudden. In the typical case the patient is usually cyanotic, the breathing is stertorous, the blood pressure is elevated, the pulse slow and full, and a general flacidity and more pronounced on the paralyzed, As a rule we find conjugate deviation of the eyes and head, either way, in accordance as to whether oculoogyric centers are irritated or paralyzed, the latter toward the lesion. The pupils are dilated and fixed unless the hemorrhage is pontine in localization. The deep reflexes are first diminished and Babinski positive. The corneal reflex is lost on the hemiplegic side. The temperature rises and in the fatal cases the scene ends with a termin-

al hypostatic pneumonia. In these individuals one usually obtains a history of nephritis and hypertension and frequently a history of preliminary small "insults" with dizziness and defective memory, antedating the major attack.

b. Meningitis.

This may be the cause of stupor or coma. It may be primary (T.B., meningococic or influenzic) or secondary following or accompanying otitis media and sinusitis etc.

In the T.B. type one frequently elicits a history of bone or joint T.B. or there may be evidence of the primary infection in the lungs or kidneys. It may follow Pott's disease, it may follow miliary T.B. of the lungs.

The lesion in the brain is as a rule most marked in the interpeduncular space, hence the frequency of oculo motor palsy. At first the meningeal signs are only suggestive but later on one demonstrates distinct rigid neck and positive kernig.

The spinal fluid is characteristic fairly clear and colorless but may be xanthochromic. There is a definite lymphocytosis which rises as the disease process advances, the protein is increased and the sugar content falls progressively, after a few hours a pellicle forms in which the T.B. bacilli can frequently be demonstrated.

The meningococcus infection are as a rule more fulminating, there is more constitutional disturbance and the meningitic phenomena are more pronounced, herpes is frequently present. The spinal fluid is purulent and usually gram negative cocci can be found, sugar reduced or absent.

The secondary meningitis usually accompanies otitis media and sinusitis and may be sterile or infectious. In the sterile form one finds either a polynucleosis or a lymphocytosis in the spinal fluid but no organisms. Meningeal signs not pronounced nor the sugar altered. The secondary bacterial meningitides prove almost uniformly fatal.

c. Abscess of the brain.

Brain abscess is another cause of stupor and coma. It may be otogenic in which it involves the temporal lobe or cerebellum. It may also accompany the sphenoids. Brain abscess may be metastatic in origin secondary to a primary focus in the lung. In such instances it is usually a terminal event. It is important to remember that brain abscess may also be found as a part of the picture of general sepsis.

As a rule the patient is mentally torpid the pulse slow and the temperature only slightly elevated. In most instances there are focal signs pointing either to the temporal lobe (speech and field defects) or to the cerebellum (nystagamus and cerebellum phenomenon)

d. Tumor of the brain.

Occasionally hemorrhage into a tumor (a latent glioma, especially of the right hemisphere) may be ushered in by an attacks of stuporousness. In such instances the presences of focal signs and changes in the fundus helps to identify the lesion.

e. Encephalitis.

This as a cause of coma may be a primary process when it occurs in epidemic form, or as a part of the syndrome of Heine Medin's disease (Poliomyelitis). It may be secondary when it follows in the wake of measles, vaccination, varicella, or other infectious disease.

It may also be the result of salvarsan intoxication, in such instances it is hemorrhagic in type and is usually fatal. There is nothing in cases of this group to suggest either a vascular accident or an otitic complication and the spinal fluid is usually normal.

There is another type of encephalitis described by Wernicke as polio-encephalitum superior alcoholica. It occurs in chronic alcoholism. The lesions are largely peri-aqueductal in location, as in encephalitis, hence the frequency of nystagamus, ocular muscle palsies, somnolence and bladder retention. The spinal fluid may be xanthochromic. Friedman has seen one individual who presented this syndrome in association with wide spread alcoholic polyneuritis and a typical Korsakoff psychosis.

f. Spontaneous subarachnoid hemorrhage.

This a frequent cause of coma and may occur in all ages. The onset is usually abrupt, with pain at the nape of the neck, headache and vomiting. Stupor rapidly supervenes. Very soon thereafter the patient exhibits fever leucocytosis and slowing of the pulse. Physical examination shows signs of meningitis and the spinal fluid reveals characteristic fluid, uniformly bloody, does not coagulate. If the test tube is allowed to stand, the supernatent fluid is usually xanthochromic. Later on there may be a relative lymphocytosis due to reaction to foreign protein.

The most frequent cause of subarachnoid hemorrhage is rupture of a military aneurysm, involving one of the branches of the circle of Willis. Preceding some of the cranial nerves may be involved.

These aneurysms may be congenital, in which case the patient may exhibit the external evidence of the so-called thymolympathic constitution (horizontal pubic hair, scant beard and body hair in the male, hypoplastic cardiovascular systems). They may also be encountered in the adult with hypertensive disease and in children suffering from subacute infectious endocarditis who develop mycotic aneurysm in cerebral vessels. Many of the patients recover and give a history of episodes of meningeal irritation. Progress depends on the size of the rent

IV. Trauma: This may give rise to either concussion of the brain or gross hemorrhage. Concussion differs only qualitatively from contusion of the brain. In these cases there are local signs of injury and the stupor follows the trauma immediately. Concussion was at one time ascribed to an acute compression of the brain with cerebral anemia. We now believe that it is due to minute perivascular hemorrhage in the cortex, basal ganglia and mid-brain. L.R. Broster O.B.E., M.Ch., F.R.C.S. brings out the fact that there is no satisfactory theory explaining the pathology of concussion.

Friedman finds that gross hemorrhage may be either epidural (from laceration of the middle meningeal artery) subdural, subarachnoid, or intercerebral.

- a. Epidural hemorrhage follows soon after trauma. In most instances there is a brief free interval but not always. At first there are signs of cerebral irritation and later stupor which increases. The diagnosis of the lesion is difficult and many are overlooked and are discovered at autopsy. X-Ray examination of the skull may be of assistance, especially if the fracture involves the temporal bone. In such cases the diagnosis of laceration of the middle meningeal artery can be entertained.
- b. Sub-dural hemorrhage occurs outside the subarachnoid space. It varies only in degree from the condition described as pachymeningitis hemorrhagica interna. It may be encountered in case of general paresis and chronic alcoholism. It is especially apt to occur in the wake of a relatively slight trauma. The bleeding is ascribed to rupture of the veins

which enter the longitudinal sinus almost at a right angle; the oozing continues until a fairly large clot forms on the surface of the brain.

There is as a rule a considerable free interval after the injury (thus differing from middle meningeal bleeding). Often the patient has completely forgotten the trauma. He may continue his work although he appears to be quiet retarded and does not appear to be quiet himself. Later he develops headache, dizziness, vomiting, and signs of increased intracranial pressure (slow pulse and chokedisc). At first there is drowsiness which later deepens to coma. Periodically the patient emerges from and elapses into stupor. Lumbar puncture generally reveals no abnormalities aside from increased intra thecal pressure (the sub-dural sac being distinct from subarachnoid space) at times the fluid may be xanthochromic. In most cases the subdural hematoma there are focal signs pointing to a lesion of one hemisphere and in a number of instances homolateral dilatation of the pupil has been found. In many of our cases the hematoma was looked for over the apparently involved hemisphere but it was not found there. Exploration of the other side was then carried out and the hematoma located. This anomalous situation has been ascribed to a contrecoup mechanism, the hematoma on one side displacing the entire brain to the opposite side, or jamming the brain stem against the tentorial incisure of the opposite side, thus giving rise to ipsilateral pyramidal tract sign.

- c. Subarachnoid bleeding may accompany fracture of the skull but it is frequently non traumatic in origin.
- d. Trauma may also give rise to intra cerebral bleeding with out fracture to the skull or subarachnoid hemorrhage

Friedman observed at least three instances of this syndrome following trauma. They all presented signs of a focal lesion in the brain. Exploration failed to reveal the presence of either epi or subdural bleeding but aspiration of the brain yielded bloody fluid. Two of the cases terminated fatally, the third is still alive but presented clinical evidence of a residual lesion in the right hemisphere. Intra ventricular hemorrhage usually accompanies the apoplexies in which bleeding plows its way through the brain substance and reaches the ventricle. Patients with this condition exhibit convulsions, generalized muscular rigidity, meningeal signs, bradycardia and bloody spinal fluid.

These cases proved rapidly fatal.

Another system of discussion of coma is presented by David Forsythe M.D., F.R.C.P., in which he keeps in mind around a half dozen causes which are:

1. Vascular derangements of the brain.
(hemorrhage, thrombosis, embolism)
2. Injuries to the head.
3. Epilepsy
4. Diabetes
5. Poisons
6. Adams-Stoke's disease.

With a summary for easy reference the author has tabulated a list of pathological causes.

- I. Dearrangement of cerebral circulation as given already.
- II. Comas culminating illnesses
 1. Meningitis
 2. Encephalitis
 3. Cerebral abscess
 4. Cerebral tumor
 5. Fevers
 6. Eclampsia
 7. Cholemia
 8. Epidemic enteritis
 9. General paralysis
 10. Disseminated sclerosis
- III. Pernicious malaria
Muscle exhaustion
Heat stroke

After having come in contact with a case Group III will hardly ever be confounded with the rest. In Group II the cases are much like Group III, as coma in these instances does not appear without warning, but rather as a culmination of an illness already suspected and probably identified. This however is not infallable he points out, for in T.B. meningitis, cerebral tumor, and specific fevers of childhood the first

manifestion noted, under certain circumstances is convulsion passing into coma. This applying also to G.P.I., and disseminating sclerosis.

The kernel of the whole subject he states however is Group I. Here as the first step in the differential diagnosis it is useful as a rule to separate on the one hand, cerebral hemorrhage, thrombosis, embolism, and injury, from on the other hand epilepsy, uremia, diabetes, poison and Adam-Stoke's disease or broadly the unilateral from the bilateral lesions.

David Forsythe 1912 has said if the blood instead of ploughing thru the internal capsule, has torn up the pons the symptoms will be modified and the dammage may be located by a crossed facial paralysis, tightly contracted pupils, a rising temperature, and not infrequently convulsions. In either event if, as is likeliest, Bright's disease lies at the bottom of the mischief, the proof must be sought in the hypertrophied heart and tense degenerated arteries, in the urine and retina, nor if still in doubt, must a chance be overlooked that the cerebro-spinal fluid may be tinged with blood that has trickled down from the head.

Cerebral thrombosis passes through a few hours or days of premonitory symptoms, more especially headaches and dizziness with ill defined sensations of tingling or numbness on one side, while his mental confusion may be apparent to his friends as well as felt by himself. Later as the clot spreads and more

brain is affected he becomes hemiplegic and finally subsided in coma. However not all thrombosis of the brain go to coma depending on the size and the distribution of the symptoms. This must depend on the vessel that has become plugged. With a median artery like the basilar, the effects will be spread over both sides and since the pons bear the brunt, the symptoms will be severe like those of pontine hemorrhage. On the other hand with the thrombosis in the posterior cerebral artery carrying blood to a part of the occipital lobe the symptoms are likely to be a hemianopsia with sensory aphasia; or with obstruction in the middle cerebral artery supplying the motor cortex, hemiplegia, and if the left is affected, aphasia results.

With embolism the patient even at times instantaneously loses consciousness, and if he comes around again finds himself powerless on one side and may be speechless. Ulcerating vegetations are often the seat of the emboli and this is manifested by petechia on the skin.

With the surgical injuries to the head only hemorrhage is the likely result. However not with standing that the history counts for much, but often a history may be lacking. And this is important, as the injury itself may be the result of some other cause of coma that is apopleptics, epipleptus etc.

Commonly however the history of an injury will serve to distinguish the surgical condition, while with a fracture of the bone you will probably see blood coming from the ear or

nose, possibly some cerebral spinal fluid, blood may be beneath the conjunctiva or about the mastoid process and if a cranial nerve is injured a resulting palsy. Again in comparison with meningeal hemorrhage the patient recovers temporarily and after several hours becomes comatose. Though now with his meningeal artery bleeding furiously, he goes under very fast, his symptoms at first being hemiplegic with the corresponding pupil widely open. In compression by a fragment of bone, on the other hand, the coma will follow hard on the injury.

In epilepsy the patient's history of previous attacks, confirmed by possible scars on the head or body, the legacies from earlier seizures, the testimony from an eye witness, the saliva churned up in his mouth, the bitten tongue, voided urine, the symmetry of his nervous symptoms and as a rule the gradual return to consciousness---some or all of these should make the diagnosis relatively easy even while the coma persists.

In contrast to this simple form, we must recognize that uremia is one of the most misleading varieties of coma. No doubt in some cases it is recognized readily enough, but in others especially when it affects a hemiplegic distribution it succeeds in passing off as a unilateral lesion, most probably as a hemorrhage. Perhaps on these occasions the imposition is to be set down to a limited edema in one hemisphere, but whatever the explanation this must not be forgotten---that uremia at one time or another simulates most organic cerebral conditions.

It assumes many guises, and it is almost impossible not to be deceived at times.

In a difficulty you must put your trust mainly in evidence of Bright's disease, supported negatively enough, no doubt, by the absence of any sufficient reason for making another diagnosis. Also the coma is not always very profound, and sometimes the patient if briskly stimulated will stir. G.S.Young said, "A diagnosis of uremia may be made only if the N.P.N. is above 120 milligrams percent."

The coma of diabetes brings us once more on firmer ground, provided that is to say, we remember that not all that reduces Fehling's solution is diabetic sugar. A pontine lesion, cerebral hemorrhage or even epilepsy may cause sugar to be present, but there will not be so much always. However with diabetes proved other causes are possible as they may die by way of apoplexy etc.

Next among the poisons responsible for coma, only three need be considered alcohol, opium, and lead. The alcoholic patient however deep in his cups, can generally be aroused, at any rate to murmur and grumble. Moreover his nervous signs are no more on one side than the other, urine low in specific gravity and probably free from albumen and sugar. The same points hold good for opium but this is differentiated by its cardinal symptoms so often mentioned. As to lead it will be enough to remember that now and again instead of its customary attacks

on the outposts of the nervous system it gathers its forces to storm the citadel itself, and so hot is the assault that the brain, after a few increasingly distressing days marked by headache, vomiting and double vision is pushed to its last extremity of violent but useless exhausting convulsions and soon all is quiet in a fatal coma.

Finally there is the unconsciousness of the Adams-Stoke's disease which beginning suddenly and often preceded by an epilepticform convulsion, comes from anemia of the brain when the ventricle of the heart stops beating in some grave disease of the auricular-ventricular bundle. Diagnosis is by finger on the pulse when with a beat of only thirty or even fewer per minute the nature of the seizure will be apparent.

Then for a last word Forsythe adds that what ever our difficulties in recognizing the various states, we have always in addition to beware of the cunning of the malingerers and the delusions of the hysteric. These patients are not really comatose and do not assume the essential signs of coma. Mistakes can be made in thinking that the patient has been comatose and is now mending. Yet if you feel any uncertainty wait, for whatever happens you must not mistake a genuine coma for one of these. If we are to err we must at all times incline to the side of caution and prudence.

In regard to diabetic coma L.W.Dunlop M.B. has found that as a rule the onset is gradual and the early symptoms are

frequently misleading. The development of dehydration is the outstanding feature. As the acidosis develops, the patient experiences nausea, abdominal pain and may vomit. Later drowsiness begins as a result less and less fluid is taken which is due to, 1. nausea 2. later because the patient is too drowsy to heed, thirst 3. polyuria and glycosuria 4. air hunger increases the evaporation of water from the lungs. As a result, a dry parched tongue, sunken and soft eyeballs (Krause's signs) and loose skin is observed. Drowsiness increases and Kussmaul breathing becomes prominent. The breath has an odor of acetone, the pulse is fast and the blood pressure low. The vomitus may contain blood and the patient becomes unconscious with flaccid muscles and knee jerks disappear early.

Differential diagnosis, Hypoglycemia: Inquire as to whether insulin has been given before hand. Absence of sugar in the urine, quiet breathing, moist tongue and absence of desiccated look make a different picture than diabetic coma.

Cerebral conditions: Sugar and ketones may be present in the urine, but obvious neurological signs as a rule make the diagnosis clear.

G.H.Wolfe has discussed the differential diagnosis of diabetes coma and acidosis from acute abdominal emergency. He has found that in both conditions there is an elevated white count, a certain amount of shock, and fever or subnormal temperature. However if it is remembered that a diabetic

bordering on coma may have pain in any part, elevated or sub-normal temperature, a flushed face, air hunger and nausea and vomiting, soft eye balls and stupor we should on general principles, assume that this patient with sugar in the urine is a potential victim of coma rather than appendicitis, pleurisy, neuritis or rheumatism.

Some points of differentail diagnosis otherwise are:

1. diabetic coma, comes on slowly and insulin shock rapid
2. coma is due to too much food (diet or fever) insulin shock is due to too little food.
3. infection common in coma and rare in shock
4. vision dim in coma and double in shock
5. patient appears sick in coma and weak and faint in
6. shock, respiration increased in coma and normal in shock.
7. onset restless in coma and excited in shock.

R.R.Lawrence has pointed out a case of profound and prolonged coma (diabetic) in an elderly woman, who was almost pulseless and so weak that air hunger was replaced by rapid shallow breathing.

E.P.Ralli and A.M.Waterhouse have reported a case age sixteen years in which coma occurred nineteen times during life. The exit was brought on by a typical onset of thirst and vomiting and death in two days.

In regard to brain lesions Anthony Feiling M.D., F.R.C.P., has found that the vascular lesions of the brain are the most common causes of hemiplegia. The vascular lesions responsible for the great majority of hemiplegia are:

1. Arterial obstruction

- a. Embolism
- b. Thrombosis
- 2. Arterial hemorrhage

The causes of embolism with the relative frequency taken in descending order are :

1. Mitral stenosis with or without auricular fibrillation and auricular fibrillation without valvular disease.
2. Malignant endocarditis (vegetative). The above causes include ninety per cent.
3. Clot in aortic aneurism.
4. Coronary thrombosis (embolic from the lungs).

Symptoms: Loss of consciousness is the rule unless only a small vessel be obstructed and when the middle cerebral artery or one of its main branches is affected, the loss of consciousness is not only sudden but may be prolonged, owing to the relative frequency with which the left middle cerebral artery is affected, aphasia is a common result of cerebral embolism. If loss of consciousness does not occur, an abrupt onset of giddiness or a dazed feeling may accompany the loss of power on the affected side of the body. Convulsions are not the rule.

Cerebral thrombosis: The most important causes of thrombosis of the cerebral arteries is a reduction of their lumen due to a thickening intima. Thrombosis occurs most frequently in 1. atheromatous disease of the arteries, 2. syphilitic endarterites other factors are, 3. diminished blood flow, 4. increased coagulability. Syphilis is caused in younger people 20-40 years and athetoma in later age. Senile changes in the

myocardium are a frequent association. The actual occurrence of the thrombosis appears to be precipitated by any factor which lowers the blood pressure, such as sleep, exhaustion from unusual physical strain, or as the result of some debilitating illness. But there is another type of patient in whom thrombosis also occurs, one in whom the diagnosis of hemorrhage is often erroneously made. Such a patient may be younger 45-55 years and may present those features which are commonly held to be characteristic of cerebral hemorrhage for example arterial hypertrophy with an enlarged heart and high blood pressure. In not a few patients of this type, however a thrombosis is the primary form of stroke which is followed some time later by a hemorrhage often fatal in the same area of the brain. Thrombosis may occur in any part of the brain but generally involves the cortex and subcortical white matter more commonly than does hemorrhage. Hence hemiplegia with aphasia is more often due to thrombosis than hemorrhage.

Convulsions generally of brief duration and limited to the limbs which are the seat of subsequent paralysis, may accompany the onset. Loss of consciousness is less common than in embolism, but may occur if any large vessel in the cerebral hemisphere is affected. It is quite common however for severe paralysis to occur without any loss of consciousness.

Cerebral hemorrhage: arterial hemorrhage into the brain is a less common cause of hemiplegia than is thrombosis in the

arteries, but is undoubtedly a more fatal lesion. The principle factors in production of an intra cerebral hemorrhage are degeneration of the arterial wall and a high blood pressure. The well recognized picture of cerebral hemorrhage generalized arterial hypertrophy, enlarged heart and granular kidneys is familiar and needs no further emphasis, Males between 50-60 years are most frequently affected. A familial tendency is accepted. The corpus striatum and corona radiata are the most frequent point of trouble. In a large proportion of cases the blood breaks into the lateral ventricle. Premonitory symptoms may occur, and are then of the same kind as have been described in cases of thrombosis. On the whole however they are found less commonly than in thrombosis. Except very small hemorrhage, and such are uncommon-loss of consciousness is the rule.

In large hemorrhage the unconsciousness gradually deepens into the most profound coma, an ominous indication of an increasing and dangerous cerebral compression.

Marked disturbances of the body temperature are often seen in severe hemorrhages, and are more common than in cases of thrombosis and embolism. A high temperature immediately after the onset suggests a pontine hemorrhage.

In arriving at a differential diagnosis the following symptoms may be regarded as characteristic of hemorrhage as opposed to thrombosis and embolism.

1. loss of consciousness which deepens steadily till a profound coma with stertorous breathing is reached.

2. Marked congestion of the face and neck.
3. A hard pulse with a blood pressure which rises under observation.
4. The rapid appearance of fever and respiratory paralysis.

If diagnosis is doubtful a lumbar puncture should be made.

In regard to the result of acute head injuries A.A.Berg M.D. has found that in dealing with cranial injuries the diagnostician's main concern is to determine whether a cerebral lesion is present and if so what its nature and localization is. If there are or have been no cerebral symptoms such as headaches, vomiting, stupor, unconsciousness, paralyses, convulsions, slow pulse and slow respiration there is as a rule no complicating cerebral condition. Compression may follow a slight hemorrhage after twenty four or more.

The nature of the cerebral injury is determined from the character of the symptoms. Concussion is always transient and the chief characteristics are unconsciousness and slow pulse. The severe forms of concussion are accompanied by deep coma, pulse slow and irregular, respiration shallow and irregular, insensitive cornea, and the pupils fail to react, urine and stools may be retained or passed. Repeated vomiting occurs especially directly after the injury. The condition may last for hours or days, but reaction usually follows, the pulse becoming stronger, and more rapid, the respiration deeper, the skin warm and consciousness returning. In fatal cases the coma becomes deeper, the pulse slower until just before death, when

it is rapid and feeble and there are convulsions and paralysis. Continued unconsciousness points to some other cerebral lesion.

A.A.Berg M.D. continues with the complications of concussion. Compression of the brain is one of the most common and has already been discussed, Cerebral laceration he has to be accompanied by severe concussion and is followed by a disturbed function of the affected part. If this happens to be one of the silent areas, there will be no localizing unless the laceration is extensive, with considerable extravasation of blood, there will be no evidence of compression.

L.R.Broster O.B.E., M.Ch., F.R.C.S. is of an opinion that it is not possible to dismiss the subject of concussion with out referring to contusions and lacerations, both of which may be regarded as stages in its recovery. These form by far the most common structural lesion of the brain after injury and differ in the degree of hemorrhage. In both of these are focal necrosis of neural elements. In laceration there is considerable bleeding whereas in contusion the hemorrhage is less and only punctate with edema is present. The clinical picture associated with these lesions has for a long time been called "cerebral irritation" (traumatic delirium) and has been ascribed to increased irritability of the nervous elements owing to venous congestion and anoxemia. The symptomatology is well known. After the recovery from concussion the patient is irritable and drowsy during the day and often delirious and maniacal at

night. He resents interference, lies curled up in bed with his eyes turned from the light and complains of throbbing headache. This condition usually reaches its height within a few days and then begins to abate, but an abnormal state of health may persist for an indefinite time. Focal signs of damage are often absent and recovery of mental function is good. The symptoms of cerebral contusion depends on two factors first the effect on the intracranial pressure and secondly the precise situation in the brain. The focal signs of contusion are those of cerebral disease generally, as such focal signs are rarely detected for it is probably the relatively slight cases which recover. The symptoms may follow immediately or weeks after the injury. The three main symptoms are headache, giddiness, and mental disability. Headache is most commonly intermittent and is influenced by changes of posture, worse when lying down, the patient finding the optimum position which gives the most relief. Giddiness also varies with posture. The mental complaints are inability to concentrate, defective memory and indecision.

A.A.Berg M.D. in regard to the localization of cerebral lesion has found that hemiplegia or convulsion of the muscles of one side of the body point respectively to destructive and irritative lesion of the motor area of the opposite side of the brain. Also hemiplegia may follow lesion in the internal capsule, lesion of the crus cerebri, of the pons, the medulla oblongata all of the opposite side.

Lesions of the cortex necessary to produce complete hemiplegia must be more extensive than similar lesion of the capsule, pons, and medulla. From the extent of the cerebral motor area it follows that hemiplegia due to cortical lesions frequently comes in the arm leg or face and spread with more or less rapidity to the entire half of the body, while lesions of the internal capsule, crus cerebri, pons, or medulla the complete hemiplegia is present from the onset.

In hemiplegia due to a lesion of the internal capsule the muscles supplied by the upper branches of the facial nerve and the back muscles are usually not involved. In addition there is also hemianaesthesias and vaso motor disturbances. The lesion is usually located in the posterior portion of the internal capsule.

Hemiplegia due to a lesion of the crus cerebri is always attended by paralysis of the muscles supplied by the third cranial nerve opposite the side of the hemiplegia.

In lesion of the pons there is usually a paralysis of the fifth, sixth, seventh and twelfth cranial nerve on the side opposite the hemiplegia.

Lesions of the bulb of the medulla oblongata is associated with hemianesthesia of the other side of the body and crossed hemiplegia.

Lesions of the type just mentioned may cause the symptoms on the same side due to contre-coup injury or because the motor

tracts do not decussate.

Berg finds also that hemianesthesias and loss of muscle sense, with some ataxia of one half of the body are due to lesions of the cortical sensory center of the opposite side of the brain or to the posterior portion of the internal capsule, crura cerebri, pons, or medulla oblongata. The points mentioned above for the location of the site of the lesion in hemiplegia as given above apply to hemianesthesia.

Hemianopsia or blindness of one half of the visual field is due first to a lesion of the cuneus and upper part of the occipital lobe, and secondly to a lesion of the optic tract behind the chiasm of the same side as the blind half of the retina. However localization is not always possible as A.B. Johnson Ph.B. M.D. has found that certain large areas of the brain may be injured or destroyed and yet no symptoms or only indefinite symptoms will follow.

L.B. Alford M.D. has observed that in twenty-seven out of fifty-five cases of old hemiplegia due to left capsular injury definite and permanent confusion of consciousness resulted. He concluded that an area somewhere in the left lobe is concerned with the maintenance of awareness.

Having considered the signs and symptoms of coma as it is presented by different men I wish to consider a routine for procedure in case a patient in coma is to be diagnosed.

L.F. Bishop Jr. M.D. and E. Appelbaum M.D. have felt that

unless a systematic routine is followed out in the investigation of coma some very important items may be overlooked. As a result they devised the following routine.

History: The history has to be obtained from sources other than the patient. It is important therefore to get as detailed information as possible from the relative or other persons present.

Physical examination: A general thorough physical examination is important. Special attention is paid to examination of the head including the fundus and the ears. A very complete neurological survey is made. The blood pressure is taken as soon after admission as possible and repeated at definite intervals.

Laboratory studies: 1. urine, catheterized soon after entrance. 2. spinal fluid. The great importance of lumbar puncture in coma can not be over estimated. A study of the spinal fluid should include physical appearance, amount, pressure, cytology, smear and culture for search of organism, a chemical analysis for albumen, globulin and sugar, wasserman where indicated and also colloidal gold. Blood analysis may be helpful especially blood chemistry.

Gastric analysis: A careful study of the stomach contents is very important especially where there is a question of some form of poison. In regard to washing out the stomach of an unconscious patient H.L.Mariott advises that a stomach

wash out on an unconscious patient whose cough reflex is absent may be lethal. Stomach contents are always regurgitated around the tube and must inevitably, in the absence of precaution, flow into the defenseless tracheae which results in either immediate suffocation or later bronchial pneumonia. The principle precaution necessary is that the mouth and pharynx should be lower than the larynx. This is achieved either by putting pillows under the shoulders and bending the head right back or by hanging the head and shoulders over the end of the couch. Trendelenburg position on the operating table is the most satisfactory as it can be easily maintained. Suction pumps are very useful also.

E.D.Friedman has formulated a similar routine which is very similar to the above. He also starts with the history which he considers extremely important in every case as to 1. the incidence of previous illness (otitis, sinus disease, diabetes, nephritis). 2. presence of injury, 3. the mode of onset, (sudden in apoplexy and subarachnoid bleeding), gradual in hematoma(subdural), uremia and diabetes, 4. associated symptoms, such as convulsions, headache, dizziness and vomiting. In all cases the head should be examined for evidences of local injury and bleeding from the various orifices. One should be on the lookout for the odor of alcohol on the breath, and blood pressure should be determined (high in uremia and advancing compression of the brain). The urine which should be obtained

by catheterization, must be examined for sugar, acetone, albumen and casts. In this connection it is important to remember that sugar may appear in the urine after apopleptic and epileptic seizures. In such instances the glycosuria rarely persists for more than twenty-four hours, it has been ascribed to disturbances in the Claude Bernard center in the fourth ventricle. The blood must also be examined for N.P.N., and hyperglycemia. In many cases we find conjugate deviation of the eyes, In cases of profound apoplexy the patient, "looks at his lesion" Forester and others have described a number of oculogyric center in the brain frontal parietal and temporal in location. When these centers are irritated they cause cephalo and oculogyric movements to the opposite side. When they are paralyzed the head and eyes are turned toward the side of the destructive lesion.

The facies must be examined for evidence of assymetry, eye grounds looked at after dilation for signs of albumemuric retinitis choked disc or diabetic retinitis, the superficial and deep reflexes must be gone over. In profound coma all the reflexes are abolished but in most cases of apoplexy the superficial reflexes are diminished on the side of the hemiplegia. Friedman has found that the unilateral abolition of the corneal reflex is an important sign of coma due to vascular lesion of the brain. It is frequently associated with loss of abdominal reflexes and diminution of the plantar reflex on the same side.

The deep reflexes are usually diminished or absent in all cases of increased intra cranial pressure and curiously enough this is more often the case with the knee jerk than the achilles jerk.

The deep reflexes may also be diminished as a result of diabetic "pseudo Tabes". A positive Babinski is of great significance. In early apoplexy and subarachnoid hemorrhage this is usually present bilaterally.

The state of muscle tone must be determined. In coma there is as a rule general flaccidity of the limbs except in cases of intra ventricular bleeding in which we find generalized rigidity.

Lumbar puncture must also be carried out. It is extremely important for the detection of the various forms of meningitis and subarachnoid bleeding. In some cases of skull injury the intra thecal pressure is very low. In Friedman's experiences this is a bad sign. X-Ray examination of the skull must be performed in order to determine the presence of fracture of the skull or convolutional markings due to increased intra cranial pressure.

N.I.Spriggs has a similiar routine starting also with the history but takes into consideration that the history is just what we often can not get. Therefore one must proceed without this help. The following points are placed in what seems to be the order of their practical utility.

1. Ascertain the deph of the coma. If a patient cannot be aroused at all, by any stimulus, for an interval of a quarter

of an hour, the probabilities are that he is suffering from an organic lesion i.e. apoplexy, embolism, thrombosis or compression. One of the most efficient ways of getting a reaction is to press on the supra orbital nerve, of course in any of the forms of coma no response may be obtained, but if the coma is so very deep, think first of organic lesion.

2. Look at the patient himself. If young think of epilepsy, if young and thin with dry skin think of diabetes, if more elderly and full blooded think of apoplexy or uremia, if a young woman think of syncope and hysteria. Of course these are only probabilities but the difference between a good and bad diagnosis is that one is more probable than the other. Examine the head for injuries: in the great majority of cases of concussion or compression quite definite signs will be found. In such cases slight injuries of the head favor apoplectic alcoholics more than concussion than depression

3. Examine for hemiplegia or monoplegia or for one sided rigidity. Hemiplegia in a young patient is most like embolism so examine the heart. In older patients suspect apoplexy. In any patient suspect meningeal hemorrhage and look for a lesion, but remember its rarity. Uremia and epilepsy may rarely cause transient hemiplegia.

4. Eyes: Conjunctival, corneal, and light reflexes will simply be of value as showing the depth of coma.

Pupils: If dilated think of concussion, uremia,

alcohol or simple syncope, if pin point, opium, pontine hemorrhage heat stroke, or rarely alcohol, if unequal not much significance but if one is dilated and fixed, think of concussion at once, if both dilated wide and fixed think of organic lesion.

5. Pulse: slow and full in apoplexy and compression, slow and feeble in opium, concussion (early) uremia and embolism

In any of these cases it may be rapid before death. It is usually rapid in syncope, epilepsy, alcohol and diabetes.

6. Respiration: usually definitely stertorous in cerebral hemorrhage or compression, often stertorous in deep alcoholic coma, heat stroke and uremia and in the later it has a hissing quality, it is shallow in opium poison and concussion, in epilepsy irregular and grasping, in diabetic coma it may be slow and deep or rather quick and noisy.

7. Temperature: usually subnormal in coma. It soon begins to rise in a large hemorrhage and with pressure in meningeal hemorrhage. There may be hyperpyrexia in large ventricular hemorrhage, in pontine hemorrhage or in heat stroke after convulsion it is often raised, unequal temperature on the two sides may be valuable when hemiplegia can not be demonstrated. Diagnostic value of temperature is not usually great. A very rapid rise or no rise gives a bad prognosis.

8. Convulsions: Think first of epileptiform attacks. If repeated probably uremia for convulsions or twitchings are rarely associated with any other coma.

9. Uremia: a little albumen is of no value, much albumen with epithelial casts speaks strongly for uremia, sugar in definite quantity makes diabetic coma probable.

10. Tongue: often bitten in epileptics, large red and dry in diabetes, foul in uremia, if protruded to one side it is sign of paralysis of that side.

11. Heart: hypertrophied in apopleptic cases or in uremia, bruits point to embolism.

12. If case can not be diagnosed wash out stomach and examine contents.

13. Sphincters show only depth of coma, full bladder and low specific gravity points to alcohol.

14. The same may be said of reflexes.

Inter diagnosis of organic lesion of the brain rests largely on history, if there is a history of injury causing concussion followed by a lucid interval and then on coming coma, compression is likely. If no history diagnosis can be made only by scalp lesion. Five other points may be noted:

a. In meningeal hemorrhage we ought to be able to trace the course of the effused blood creeping over the motor area, by the associated paralysis.

b. Meningeal hemorrhage is likely to end fatally if untreated. Inter cerebral hemorrhage causing coma often recovers untreated.

c. Hutchinson pupil is more characteristic of meningeal

hemorrhage (the pupil on the side dilated and fixed).

d. Conjugate deviation is much more frequent in cerebral hemorrhage.

e. Cross hemiplegia will not occur in meningeal hemorrhage.

I.S.Wechsler M.D. when contacting a comatose patient first makes inquiry into the history whether the coma was sudden or gradual in onset, preceded by headache, convulsion or trauma to the head (both immediate and less recent), whether the patient was previously suffering from symptoms pointing to general disease such as diabetes, nephritis, cerebral arteriosclerosis, syphili or malaria.

The patient should be examined for gross signs of injury, bleeding from the mouth ears etc. and the breath smelled for acetone, uræmia odor, alcohol or poisons. The heart should be examined, the pulse counted, blood pressure determined, the respiration observed, the pupils investigated, corneal sensation tested, optic discs and fundi examined, the bladder catheterized and the urine tested for sugar and albumen, the temperature taken and in tropical countries a blood smear examined for malarial plasmodia, and a neurological examination made to determine the presence or absence of paralyses, blood chemistry is generally indicated.

Before formulating a routine of examination of coma let us consider the relative frequency of the different causes of

coma. B. Holcomb has reported on 394 autopsied cases entering the Cook County Hospital and the relative frequency of the cases are and percent of correct diagnosis:

Skull fracture	92
Cerebral hemorrhage	88
Uremia	37
Meningitis	23
Cerebral thrombosis	20
Labar and bronchial pneumonia	18
Alcoholism	16

55.2% were correctly diagnosed. On 92 deaths from skull fracture 66.3% were correctly diagnosed. The missed diagnosed were as follows:

Cerebral hemorrhage	11
Meningitis	5
Epilepsy	2
Uremia	1
Epidemic encephalitis	1
Lobar pneumonis	1
Syphilis	1
Alcoholism	1
Multiple lacerations	1
Edema of the lungs	1
No diagnosis	4

39 or 33.7% missed

diagnosis in 88 cases of cerebral hemorrhage, missed diagnoses were as follows:

Skull fracture	7
Uremia	4
Gas poison	4
Cerebral thrombosis	3
Bronchial pneumonia	2
Meningitis	1
Cerebral embolism	1
Lobar pneumonia	1
Epilepsy	2
No diagnosis	7

32 total missed or

36.4

W.W.Bissell M.D. and E.R.Lecount M.D. have reported on the causes of coma in 400 deaths and find the percentages of each cause as follows:

Skull fracture	36
Cerebral hemorrhage	24
Meningitis	6
Lobar pneumonia	5.2
Uremia	4.8
Cardio-vasc	3
Cerebral circulatory condition	3
Delirium tremens	2.8
Atrophic cirrhoses of the liver	2.6
Diabetic coma	2
Brain abscess	1.9
Traumatic intra-cranial hemorrhage	1.5

From the above it is seen that deaths from coma due to skull fracture and cerebral hemorrhage rank high and at the same time missed diagnoses are around one third of the cases that come in. With these facts in mind let us attempt to formulate a method whereby a patient may be handled with the means that a physician commonly has at his command out of the hospital and his office which are 1. ability to take history 2. physical examination, 3. neurological examination, 4. ophthalmoscopic examination, 5. blood pressure, 6. temperature.

In any attempt of diagnosis history is first to be considered and in a condition of coma where bodily injury means so much in ruling out certain causes it is most important. So in taking a history we may proceed 1. was there any injury which brought on the condition, 2. was there any incidence of previous

illness such as diabetes, nephritis, apoplexy, epilepsy, etc.

3. Was the onset sudden or gradual.

4. Was there associated symptoms of convulsion, headache, dizziness, vomiting, blindness, etc.

The physical examination next to be considered is very important especially when it is impossible to take a history.

The first steps to be taken are:

1. Observe the patient grossly for injuries and since causes of coma are often due to head injuries the head should be inspected immediately but it should be understood that a head injury is especially minor may not have any bearing as to the cause of the coma.

2. Age and sex should be automatically be born in mind.

3. The depth of the coma should be examined and if the patient can not be aroused for a quarter of an hour for example by pressure on the supra orbital nerve etc. it is probable that the condition is organic.

4. Presence of hemiplegia or monoplegia point strongly to brain lesions.

5. The character of the pulse should be noticed especially the rate force and rhythm.

6. The respiration if stertorous, irregular, grasping shallow or Kussmaul are significant.

7. A bitten tongue, scars on the cubital fossa of the arm and frequent scars elsewhere are especially significant.

8. The temperature should be taken rectally and recorded at intervals if possible.
9. The blood pressure should also be taken at intervals.
10. A n ophthalmoscopic examination may be helpful in corroborating other evidence.
11. Gastric lavage if poisons are suspected.

With the information gained from this simple examination of the patient the typical condition of vascular lesions of the brain embolism, thrombosis and hemorrhage, concussion, lacerations, and compressions of the brain, diabetes, uremia, epilepsy alcohol, morphine and the types of coma following meningitis, pneumonia etc. can be recognized. Taking into consideration the fact that a large per cent of comas contacted are injuries to the brain it is important to know what measures are to be taken in handling the patient.

A. Feiling M.D., F.R.C.P., is of the opinion that the immediate treatment of the unconscious patient whether the vascular lesion of the brain be due to embolism, thrombosis or hemorrhage is the same. Absolute rest is essential, head and shoulders should be slightly raised, tight clothing removed about the chest and neck. Rectal injection of glucose if the coma lasts over twenty-four hours. When the patient gains consciousness give small liquid feedings. If the case is hemorrhage an aperient may be given 2-3 grains calomel. Give 30 grains Bromides t.i.d. if restless.

With this treatment in mind it is obvious that if the patient is moved without adequate facilities it may prove fatal. If the above routine has been applied in diagnosing the case and the hospital is reached, what further information may be attained.

A catheterized specimen of urine should be examined for sugar, acetone, casts and albumen.

A spinal puncture should be done and the spinal fluid studied as to its physical appearance, pressure, cytology, chemical analysis for globulin albumen and sugar, and a wasserman where indicated.

X-Ray of skull when fracture is suspected.

If poisons are suspected a gastric lavage should be performed with analysis of the contents if it has not already been done.

Blood analysis; N.P.N. and blood sugar are most important.

A complete neurological examination should be done and repeated at intervals if there is a probability of brain injury and if there has been any positive neurological findings.

Conclusion

In the preparation of this paper it has been interesting to me that as far as the description of these comatose states, as to the signs and symptoms, relatively little has been added in contrast to the rapid development of refined laboratory means. Only in 1891 W.E.Wynter reported the first paracentesis of the theca vertebralis for the relief of spinal fluid pressure. The toxicologist is almost indispensable in the detection of many poisons from the analysis of gastric contents such as mushroom poisoning, methyl alcohol or the other alcohols so often found in ethyl alcoholic drinks, certain medicines taken to excess by mistake, etc., while less than half a century ago he played little part in this role.

Another point of interest to be considered is that after a diagnosis of the type of coma has been made, what conditions lend themselves to an immediate and specific treatment. First head injuries without laceration or hemorrhage require no drastic means of treatment. 2. Head injuries with hemorrhage or laceration does require in many cases immediate steps in surgery. 3. The apoplectics aside from venesection or lumbar puncture together with sedatives and other simple measures needs no radical steps. 4. Uremia, uncomplicated, with its bad prognosis, calls mainly for the treatment of Bright's disease. 5. Epileptics require no specific treatment. 6. Diabetics require immediate insulin and relief from

dehydration. 7. Syncope, hysteria, and feigning if the diagnosis is certain do not cause much alarm. 8. Poisons do require immediate steps in treatment. Thus in a large number of instances it is important to make an immediate diagnosis in order that necessary means may be made in treatment.

Finally however considering that there is a large error in diagnosis of coma, even under the best conditions, this subject remains one which will undoubtedly bring forth many anxious moments within the lives of medical men, for as long a time as people become comatose.

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