Coma and Consciousness

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ALL TOO OFTEN, patients with coma or lesser alterations of consciousness present to the physiciar with an inadequate or an unreliable history. The comatose patient, hence, is a challenge in diagnosis and therapy. Having grappled and reversed potential life-threatening situations, the physician then proceeds with an orderly rational approach to the diagnosis and treatment of the precipitating factors responsible for the comatose state.

The purpose of this discussion is to define coma and discuss briefly its pathophysiology. In addition, we hope to present clinical entities that could produce coma and describe a practical approach from the point of view of physical examination, neurological examination and investigations towards an adequate management of these patients.

In order to understand and approach rationally the problems of coma, one must first comprehend the levels of awareness and the need for categorising the various "levels of consciousness". We cannot, therefore, limit our discussion to coma, but rather must define and discuss impaired "levels of consciousness".

One should not attempt to define consciousness in pure biological terms, as it is that process which utilises sensory perception via exteroceptors, memory, intellect, attention and comprehension, all of which help to produce an appropriate response. Coma is then an extreme lack of consciousness in which a state of vegetative existence prevails. In other "levels of consciousness," superficial reflexes, such as corneal and gag reflexes, may be present. All responses may be absent or often abnormal. The latter may take the form of decerebrate posturing elicited, say with mere touch pain or other exteroceptive stimuli.

Between these two extremes lie arbitrarily delineated categories. If one reviews the literature discussing this subject, one sees that most authors have judicially avoided definitive categories and many terms have been used synonymously. For the purpose of this discussion, the realm between coma and consciousness can be categorised in a manner that is meaningful and can be communicated as follows:— (Table 1)

Stupor or semi-coma is that state of relative unresponsiveness to environment or specific sensory stimuli, in which superficial reflexes are intact, and deep reflexes may or may not be elicited. No response is obtained on questioning alone although not uncommonly an inappropriate or unintelligible grunt may be heard. However, appropriate but

TABLE I

1) Coma
Vegetative state.
In lighter planes, superficial reflexes such as corneal and gag reflexes persist.
No response to verbal stimulation.
No response or abnormal response to painful stimulus such as decerebrate posturing.

2) Semi-coma or Stupor
Superficial reflexes intact.
Appropriate, but primative response to painful stimulus (i.e. flexion withdrawal).
No response to verbal stimuli.
May or may not respond to pain with unintelligible utterings.
Can be of psychogenic origin, such as catatonic stupor.

3) Somnolent
Reflexes intact.
Tends to return to sleep-like state; totally inattentive to environment unless continually subjected to direct sensory stimulation.

4) Lethargy
Reflexes intact.
Disinterested in environment.
Responds appropriately to minimal direct stimulation.

5) Confusional States
Alert or lethargic-responds to minimal stimulation.
Reflexes intact.
Responses are inappropriate; may confabulate or hallucinate.
At times, difficult to distinguish from dysphasia.

6) Conscious
Alert, oriented.
Responds appropriately to environment and specific sensory stimulation.

primitive responses, such as flexor withdrawal, are elicited to painful stimuli. Catatonic stupor, a classical example of psychogenic stupor, should be entertained in the differential in these instances. Sometimes patients are described as somnolent. This is characterised by a persistent sleep-like state in which the patient is totally inattentive to the environment unless stimulated repeatedly.

Lethargy, on the other hand, is best described as a disinterest and lack of attention to the environment. However, in this state the patient responds readily and often correctly to minimal direct stimulation, including questioning. Definition of commonly used terminology will not be complete unless confusional states are also included. This category is used to distinguish the confused, disoriented, and often hallucinating patient from the conscious one, because consciousness implies appropriate responses to sensory stimulation. Whereas the conscious patient is always alert, the confused or delirious patient may be alert lethargic, or somnolent. A dysphasic patient often gives the impression of being confused and unless this is borne in mind, errors in diagnosis are likely to result.

The physiology of consciousness and sleep, as well as the pathophysiology of coma, remains poorly understood despite numerous publications on the subject.[1]

Consciousness is attributed to the integrity and proper functioning of the periaqueductal grey area,[2,3] and the reticular formation. Both these areas, and especially the latter, are physiologic systems that defy precise anatomical delineation. The reticular formation has been shown to extend from the lower medulla to the thalamus with its pathway within the central portions of the brainstem. In 1949, Moruzzi and Magoun identified the ascending reticular activating system (ARAS) which has been shown to extend from the midbrain to the thalamus and to have definite but diffuse cortical connection.[4,5]

Subsequent experimentation of their thesis revealed that interruption of the ascending reticular activating system in any way led to coma. That sleep and coma were similar in that they were both due to a failure of the ARAS to activate the cerebrum, thus found support. However, both electroencephalography and single neuron recordings within the reticular formation, demonstrate an increased activity of these cells rather than electrical silence during sleep. As a result, current opinion that physiological sleep is an active, rather than a passive process,[6] seems plausible. Although the "principal" and "sustaining" projections of the ARAS are diffuse and varied, the reticular formation proper is housed compactly within the brain stem. It is therefore understandable why small mass lesions, hemorrhages, infarctions in the brain stem can compromise its

functional integrity and produce profound coma.[7,8] On the other hand, hemispheric lesions not causing brain stem compression do not result in altered consciousness. However, metabolic disturbances from infection,[9] drugs,[10] endocrinop athies[11,12] or various system dysfunctions may result in coma due to impairment secondarily of the metabolism and thus function of the neurons and supporting glial cells of the cortex.[13,14] However, here again it is difficult not to implicate the reticular formation in some way or even disregard it.

The clinical conditions which can lead to alterations of brain function are varied and include trauma, mass lesions intracranially, endocrine disturbances, exogenous toxins and failure of extracranial organ systems resulting in either primary or secondary metabolic disturbances of the neurones and glia.

The classification and discussion, as well as the approach to diagnosis and therapy, can therefore best be delineated if these disorders are classified into two basic groups. The first category includes primary disorders of the brain and its vasculature. These include trauma, cerebral vascular lesions, neoplasms, infectious disease, metabolic disorders, seizure disorders and psychogenic alterations. (Table II)

These can again be divided into three groups:

1) Those which impair the function of the reticular formation with the brain stem by a disruption of this system by way of compression and or displacement or by impairment of its circulation as a sequelae to thrombosis or hemorrhage.
2) Those which interfere with neuronal and glial cell function, e.g. infections of the brain and meninges, toxins, system electrolyte and nutritional deficiency states, primary metabolic disorders of the brain (see Table III).
3) Those of psychogenic origin notably catatonic states, severe depressive reactions, and hysteria.

Because of the wide variety of entities involved, the clinician faced with the problem of a patient with altered consciousness must approach the problem logically and efficiently. All too often, when one is faced with a deeply comatose patient, the feeling of inadequacy and insecurity results in a dramatic array of assorted laboratory tests and administration of drugs based on illogical diagnosis and without a comprehensive examination. It must be kept in mind that proper diagnosis and treatment of the comatose patient often results in his return to a functional life.

Its recognition and treatment should thus be an emergency in the true sense of the word in view of this potential reversibility. There is, for instance, no

TABLE II
PRIMARY BRAIN DISORDERS AFFECTING CONSCIOUSNESS

1) Trauma
- Concussion
- Contusion
- Epidural hematoma
- Subdural hematoma
- Depressed skull fracture
- Penetrating wound

2) Cerebral Vascular Lesions
- Cerebral artery thrombosis
- Basilar artery thrombosis or embolus
- Cerebral embolus
- Brain stem hemorrhage
- Intracerebral hemorrhage
- Cerebellar hemorrhage
- Subarachnoid hemorrhage
- Ruptured aneurysm
- Arteriovenous malformation

3) Neoplasms
- Gliomas
- Meningiomas
- Neuromas
- Metastatic lesions

4) Infectious Diseases
- Meningitis
- Encephalitis
- Meningoencephalitis
- Abscess
- Cerebral
- Empyema
- Subdural
- Epidural

5) Metabolic Disorders of the CNS
- Jacob-Creutefeldt disease
- Picks disease
- Schilders disease
- Leukodystrophies
- Progressive myoclonic epilepsy
- Huntingtons Chorea

6) Seizure Disorders
- Status epilepticus
- Post-ictal states

7) Psychogenic States
- Hysteria (conversion reactions)
- Depressive reactions (severe)
- Catatonia

TABLE III
SYSTEMIC DISORDERS AFFECTING CONSCIOUSNESS

1) Metabolic Disorders
- Hypoglycemia
- Insulin induced
  - exogenous
  - endogenous
- Alcoholic induced
- Vitamin deficiency
- Thiamine
- Pyridoxine
- Niacin
- B1
- Alkalosis and acidosis
- Hepatic failure
- Porphyria
- Renal failure
- Electrolyte imbalance
- Fever

2) Exogenous Toxins
- Alcohols
- Barbiturates
- Tranquilisers
- Hallucinogenic drugs
- Hypnotics
- Salicylates
- Carbon monoxide
- Cyanide
- Anticholinergics
- Heavy metals

3) Endocrine Disorders
- Hypopituitism
- Hyper & hypothyroidism
- Hyper & hypoparathyroidism
- Adrenal insufficiency
- Pancreatic dysfunction

4) Cardiorespiratory System
- Cardiac dysfunction
- Myocardial infarction
- Congestive heart failure
- Hypersensitive carotid sinus
- Valvular disease
- Arrhythmias
- Pulmonary
- Hypoventilation
- Airway obstruction

TABLE IV
PROBABLE DIAGNOSIS RELATED TO ONSET OF COMA

1) Acute
- Trauma
- Cerebral vascular lesions
- Basilar artery thrombosis or embolus
- Brain stem hemorrhage
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Cerebral hemorrhage
- Neoplasms
- Hemorrhage into pre-existing tumor
- Insulin coma
- Acute adrenal insufficiency
- Carotid sinus sensitivity
- Cardiac arrest
- Hysterical coma

2) Sub-acute
- Trauma
- Subdural hematoma
- Cerebral vascular hematomas
- Cerebral thrombosis
- Cerebral embolus
- Subarachnoid hemorrhage
- Neoplasms
- Hemorrhage into pre-existing tumors
- Infectious disease of the CNS
- Seizure disorders
- Systemic metabolic disorders
- Exogenous toxins
- Endocrine disorders
- Cardio-respiratory system dysfunction

3) Chronic
- Trauma
- Subdural hematoma
- Neoplasm
- Infection of the CNS
- Brain abscess only
- Metabolic disorders of the CNS
- Systemic metabolic disorders
- Pulmonary disorders
- Psychogenic disorders

substitute for an adequate history as the basis of approach to the treatment of the comatose patient. If nothing more, the rate of alteration of consciousness to the level of coma manifested should be ascertained. This is because the rate of transformation from consciousness to coma provides a clue to a

rational appraoch to diagnosis and treatment. Table IV indicates how these rates of alterations from consciousness to coma help predict the line of aetiologic probabilities in the genesis of the coma, and thus help to both prognosticate and treat these patients meaningfully.

It is an accepted fact that trauma can result in instantaneous coma. However, it must be remembered that particularly subdural hematomas may not cause neurological deficits or altered sensoria for days to months after the injury. In this context, it may be stated that concussion is usually not a significant therapeutic problem since, by definition, it is a trauma-induced transient coma lasting no more than a few minutes with full recovery and no structural damage.[15,16] However, these patients should be observed for possible development of subdural hematomas. Hospital observation is advised anywhere from 24 to 48 hours.

Cerebral vascular lesions of the brain stem,[17] whether they be infarctions or hemorrhages, are capable of producing instantaneous coma. It is indeed rare for a cerebral thrombosis or an embolus to produce acute coma. Coma from a cerebral thrombosis, when it occurs, takes hours to days to develop because it is the resultant cerebral edema that leads to secondary compression of the reticular system. Subarachnoid hemorrhage, on the other hand, will often produce coma in seconds to hours, especially if it is accompanied by ventricular extension of the hemorrhage. Massive intracerebral hemorrhage must be included in the acute category, but smaller hemorrhages usually produce neurological deficits with lesser states of altered consciousness.

A pre-existing tumor may result in rapid or sudden coma when hemorrhage occurs into it. This is unusual but we have seen it associated with glioblastomas and oligodendrogliomas. Injudicious use of spinal puncture in a patient with an intracranial mass lesion, be it tumor, intracerebral hemorrhage or a subdural hematoma, may result in precipitous coma as a result of transtentorial herniation with resultant brain stem compression.[18]

Infections of the central nervous system usually result in altered levels of consciousness accompanied by clinical evidence of an infectious process and coma may take hours to develop in untreated cases.

Seizure disorders rarely produce coma except during the actual ictal period. This transient period is due to the bombardment of the reticular system by an abnormal large volley of electrical impulses. The post-ictal state is most often one of lethargy and somnolence accompanied by confusion and amnesia. Occasionally, however, the post-ictal state may be one of stupor.

Primary metabolic and psychogenic origins of profoundly altered levels of consciousness usually have a compatible history of a chronic nature. The systemic disorders produce coma with appropriate prodromes. However, rarely an acute onset of coma may be seen following cardiac arrest, adrenal insufficiency, or hypoglycemia. However, it should be kept in mind that hysterical coma may be precipitated suddenly in an appropriate situation from which the patient needs complete escape.

The clinical evaluation of the patient centers around four essential parameters in addition to the history. These are:

1) Type and character of the coma;
2) Pupillary responses;
3) Oculomotor signs; and
4) Patterns of respiration.

These evaluations are only of relative value and in profound coma, there are usually no eye signs of value. Such parameters are of greatest value when the patient has a limited alteration of consciousness.

The degree of alteration of consciousness as alluded to previously can be summed up as follows: The lighter stages are usually of supratentorial origin and may be (1) primary brain dysfunction, (2) secondary to toxic substances, or (3) as a result of disruption in cerebral metabolism from extracranial system failure. These situations are of a subacute and chronic nature. Profound alterations of consciousness are either due to primary organic brain stem pathology, which is of an acute nature, or are secondary to diffuse cerebral impairment, with subsequent brain stem compression representing an end stage in a subacute or chronic process.

Pupillary responses provide clues as to the origin and depth of central nervous system dysfunction. The pupils are evaluated as to size, and response to light and accommodation. The ciliospinal reflex characterised by a dilatation of the pupil as a result of painful stimulation to the ipsilateral side of the neck should also be elicited. When it is intact, it mitigates against the likelihood of a severe brain stem destruction.

Pupillary signs in coma are of value in terms of providing clues as to its possible origin. Cortical anoxia, atropine and scopolamine produce widely dilated pupils unresponsive to light stimulation. Posterior fossa hemorrhages, pontine hemorrhage, and

opiates produce pinpoint pupils. Pupillary responses are also of value in localising brain stem lesions. Midbrain (tectal) lesions obviate the light reflex and the pupils are round and 5-6 mm. in diameter. They tend to fluctuate in size spontaneously and the ciliospinal reflex remains intact.

With midbrain lesions involving oculomotor nuclei, the pupils are in midposition and unresponsive to all stimuli. Pontine lesions and compressive lesions of the posterior fossa tend to produce pinpoint pupils. One should be aware that as a general rule, pupils are reactive in metabolic coma. Although about 12 per cent of the normal population exhibit unequal pupils, anisocoria should always arouse suspicion of localised central nervous system pathology.

The third parameter is that of the oculomotor responses which result from vestibular stimulation.[19] Stimulation of the semicircular canals induces the vestibulo-ocular reflexes. This can be accomplished by two simple methods: 1) Doll's eye maneuver; 2) Caloric stimulation.[20]

The doll's eye maneuver is described as such because it is reminiscent of the mechanical movement of the eyes of a doll, i.e. when the reflex is intact, sudden rotatory movement of the head stimulates the semicircular canal initiating the reflex and induces conjugate deviation of the eyes in the opposite direction. Caloric response is most readily elicited by irrigating the external auditory canal with ice water for about 30 seconds. The intact reflex system produces nystagmus with the fast component away from the irrigated ear.

The caloric test is of significance in evaluating the integrity of the brain stem and is of exceptional value in helping to separate the organic from the psychogenic coma. In the deeper stages of cortical depression, the reflex from caloric stimulation and the doll's eye response become exceptionally brisk. With brain stem involvement, the reflex becomes perverted and the response depends upon the level of involvement of the brain stem.

Although several forms of respiration associated with central nervous system pathology have been described,[21,22] there are three basic types which are of value in the evaluation of the comatose patient. These are: 1) Cheyne-Stokes Respirations (CSR): 2) Central Neurogenic Hyperventilation (CNH); and 3) Ataxic Breathing.

Cheyne-Stokes Respiration (CSR) is a regular pattern of alternating periods of respiration and apnea. The respirations increase in depth to a point, then decrease in depth in a regular pattern. This type of respiratory pattern is most commonly seen with deep lesions of the cerebral hemispheres or diencephalon which are often bilateral.

Central Neurogenic Hyperventilation (CNH) is characterised by deep, regular, rapid respirations without alteration in depth or rate and there are no periods of apnea. This respiratory pattern denotes dysfunction at the ponto-mesencephalic levels. Unlike in metabolic acidosis, (Knussmaul breathing), these respirations have a forced expiratory component to them.

The third form of respiratory pattern is that which has been labelled as ataxic breathing. This type of respiration is irregular in rate and depth; there is, in addition, no pattern to the rhythm or periods of apnea. Ataxic breathing indicates medullary dysfunction and is a sign of impending respiratory arrest. Reversible coma, associated with ataxic respirations, demands respiratory assistance and a tracheostomy is therefore recommended.

Laboratory Evaluation

There are several laboratory examinations which are of value in determining the origin of coma and are of invaluable assistance to the physician in reaching an accurate diagnosis. These are presented here only in terms of indications and relative value of such examinations.

Basic blood values to be drawn immediately when one is faced with a comatose patient and the etiology is obscure include:

1) BUN;
2) Blood Sugar;
3) Serum Electrolytes;
4) CBC; and
5) Alcohol and barbiturate levels.

These basic studies, as well as a routine urinalysis, serve as a guide to possible extracranial system dysfunction and certain exogenous intoxicants as the precipitating factor of the coma.

Other useful studies to be utilised include:

1) ECG;
2) Echoencephalogram;
3) Brain scan;
4) EEG; and
5) Spinal puncture.

ECG

The electrocardiogram should always be performed in patients with alterations of consciousness since failure of cardiac output induced by myocardial infarction, arrhythmia, chronic heart failure, and valvular disease is a common precipitating factor of alterations of consciousness, especially in patients

over the age of 50. It must be kept in mind, however, that intracranial pathology, especially trauma and subarachnoid hemorrhage, produce alterations in the ECG, varying from arrhythmias to ischemic patterns.[23]

Echoencephalogram

The echoencephalogram utilises ultrasound to detect shifts in midline structures and is of particular value in cases of traumatic origin. However, it must be kept in mind that this study is only as good as the technician performing the procedure and negative results never rule out intracranial mass lesions.

Brain Scan

The use of isotopes to ascertain intracranial pathology is of relative value in terms of definitve diagnosis. Positive scans are most commonly seen with tumors, hematomas and ischemic lesions. Again, a negative study does not rule out an intracranial pathology. One must also be cautioned that a positive scan will be seen from scalp trauma and it often remains positive for six to eight weeks[24] after the injury.

EEG

The electroencephalogram is invaluable in detection of intracranial pathology, especially with diffuse cortical depression. The electroencephalogram is often able to separate cortical dysfunction due to primary[25] brain pathology from systemic disorders and drug ingestion. The EEG is also of great value in separating the organic from the psychogenic origins of alterations of consciousness.

Spinal Puncture

Lumbar puncture of the subarachnoid space, when indicated and if carefully performed, has a definite place in the diagnosis and management of comatose patients. The usual precautions will have to be undertaken. Fundoscopy is of paramount importance to rule out papilloedema. In those cases where intracranial mass lesion are suspected, be they tumor, hematoma or cerebral edema from any cause including infarction, the risk of herniation is always present. It is our experience that rises in intracranial pressure that have occurred slowly over a period of weeks to months, e.g. a neoplasm is more dangerous from this point of view than cases, say, of subarachnoid hemorrhage. This may be a function of the moulding that occurs in the region of the brain stem from a chronic increase in pressure above the region of the brain stem.

Further examination of the cerebrospinal fluid is indeed of great value in the diagnosis of primary demyelinating disorders and other metabolic disorders of the central nervous system as well as in central nervous system syphilis. However, in these situations, the puncture can be performed selectively after one is sure that the patient is stable and mass lesions have been ruled out, thus avoiding the risk of acute herniation of the brain stem. In all cases with papilloedema, with suspected mass lesions, it is recommended that mass lesions be ruled out by means of EEG, brain scan, or contrast studies prior to the examination of the cerebrospinal fluid.

When spinal puncture is performed, it is necessary to obtain all available information at the time, as indicated by the patient's clinical state. This includes:

1) Carefully measured opening and closing pressures when the patient is relaxed, and after care has been taken to be sure that the patient's knees or a pillow are not compressing the abdomen, since this will give falsely high readings.
2) Specimen of cerebrospinal fluid is to be examined for: a) Cell count and morphology; b) Protein; c) Seriology; d) Colloidal gold and e) Sugar concentrations.
3) In addition, in cases of suspected infection, CSF should be examined for: a) Smear; b) Routine culture and sensitivity; c) India ink preparations; d) TB and fungal cultures and also Giemsa stains for possible malignant cells, as the latter not uncommonly produce a carcinomatous meningitis with altered sensorium.

In those cases where cerebrospinal fluid sugar content becomes crucial in an infection, a blood sugar estimation done at the time of the lumbar puncture could help to interpret the results.

4) In cases of suspected primary central nervous system metabolic disorders, a specimen should be sent for gamma globulin estimation where this study is available.
5) The Queckenstedt test (jugular compression) should not be done as a routine during lumbar puncture. It only serves to increase the venostasis within the brain and aggravates the patient's condition further. Its use is not recommended in the investigation of patients in coma.

In the initial management of the comatose patient, the function of the physician in the face of coma must be to prevent a deterioration of an already serious situation. This requires primary attention to the airway. The insertion of a plastic oral airway is recommended in all patients in coma even if they

exhibit a stable respiratory pattern associated with adequate pulmonary ventilation and oxygenation. When poor ventilation, cyanosis or ataxic bleeding is present, nasal tracheal or endotracheal intubation is indicated, followed by tracheostomy when the patient has stabilised. In deep coma with poor ventilation, respiratory assistance may be indicated. Often in these cases, external ventilatory assistance is advisable with a manual mask resuscitator prior to the intubation. Also, prior to intubation, 0.4 to 0.8 mgms. of Atropine sulphate administered intravenously is recommended to minimise vagal reflexes during intubation. After an airway has been established, pharyngeal and tracheal suction should be undertaken and repeated as frequently as necessary to obtain patency or the tracheobronchial tree. In addition, the patient should be maintained on his side or prone unless a cuffed endotracheal or tracheostomy tube is in place. This ensures drainage of the respiratory tract, and offsets the chances of an aspiration.

In addition to pulmonary ventilation, hemorrhage and shock are the other problems which can produce death and irreversible brain damage most readily in these patients.

Hemorrhage outside the nervous system, needless to say, must be controlled and blood replacement instituted. A patient himself in shock with spontaneous respiration is always a suspect for an extracranial cause for the shock. If hemorrhage is not overt, then four quadrant abdominal taps are recommended to exclude an intra-abdominal bleed. Hypotension accompanying alterations of consciousness can be due to drug ingestion, cardiac dysfunction, dehydration, or anoxia. When necessary, vasopressors are indicated and it is recommended that systolic pressure not be taken over 100 to 110 mm. of mercury initially.

The remainder of the management can be divided into two groups: 1) Traumatic and 2) Non-traumatic induced coma. (Tables V and VI)

Coma of traumatic origin requires initially supportive management with attention directed to the airway and hemorrhage, as outlined above. The necessity for a general physical evaluation cannot be overstressed. Blood sugar, electrolytes, CBC, and urinalysis should be obtained, whenever able in these patients, in order to have a base line prior to anesthesia and surgery. Intravenous fluids should be started with Ringer's Lactate (whole blood when indicated). An echoencephalogram is of value and may delineate the site of pathology.

When otorrhea or rhinorrhea is suspected, the

TABLE V
CLINICAL MANAGEMENT OF TRAUMA INDUCED COMA

Primary evaluation and management

1. Maintain airway
2. Control hemorrhage
3. Maintain patient on side to prevent aspiration
4. History
5. General physical evaluation
6. Neurological evaluation
7. Laboratory studies as indicated
8. IV fluids
9. Foley catheter to drainage
10. Type and cross-match for whole blood
11. Echoencephalography
12. Skull and cervical spine X-rays

Secondary evaluation and management

1. Neurosurgical evaluation
2. Radiographic examination
3. Radiographic contrast studies as indicated
4. Conservative management or surgery as indicated

TABLE VI
CLINICAL MANAGEMENT OF COMA NOT OF TRAUMATIC ORIGIN

Primary evaluation and management

1. Maintain airway
2. Reverse shock
3. Maintain patient on side to prevent aspiration
4. History
5. General physical evaluation
6. Neurological evaluation
7. ECG
8. Laboratory studies as indicated by history – BUN, CBC, blood sugar, urinalysis, ETOH and barbiturate levels, electrolytes, liver profile, blood gases.
9. Spinal puncture when **infection is suspected**
10. Initiation of therapy directed at systemic disorders affecting brain function

Secondary evaluation and management

1. Skull X-ray
2. EEG
3. Brain scan
4. Echoencephalogram
5. Radiographic contrast studies when indicated
6. Spinal puncture when indicated (see text)

drainage should be tested with "Dextrostix", for the presence of sugar. A positive test would indicate a cerebrospinal fluid leak. Neurological evaluation should be performed to ascertain the degree of injury and level of function. When the patient is stable, skull and cervical spine X-rays are recommended in all cases of coma of traumatic origin. These studies should be accomplished with the least amount of movement possible. Supportive measures should be maintained, vital signs checked frequently and neurosurgical evaluation obtained as soon as possible.

Coma of non-traumatic origin requires the same initial supportive measures, as well as a general and neurological evaluation. History is the most pertinent guide to the initial "work-up". When only a limited history is available, it is wise to obtain a urinalysis and blood for CBC, electrolytes, glucose, BUN, and levels of common exogenous toxins, e.g. barbiturates and alcohol. Blood gases and a liver profile are often of value.

After blood is drawn, it is recommended that intravenous fluids be started with 5% dextrose and water and that 50 c.c. of 50% dextrose solution be given to reverse hypoglycemia if present. The intravenous fluids should be changed in accordance with the results of the electrolyte determinations. Gastric lavage is indicated only if ingestion has occurred within four hours. In patients with no gag reflex, lavage should be done only after a cuffed endotracheal or tracheostomy tube has been inserted and the cuff inflated.

As outlined earlier in this discussion, spinal fluid examination at this time is recommended only when infection is strongly suspected or when the history and examination findings point to a subarachnoid hemorrhage without focal findings. Otherwise, lumbar puncture should be postponed until the patient is stable and intracranial mass lesions have been definitely ruled out by means of EEG, brain scan, echoencephalogram and contrast studies.

In summary, this discussion has been directed at: 1) Defining the terms used to describe the alterations of consciousness in a manner that can be communicated meaningfully; 2) Presenting a brief discussion as to the pathophysiology and clinical conditions responsible for the problem; 3) Providing a useful, rational guide to the initial evaluation and management of the alterations of consciousness.

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