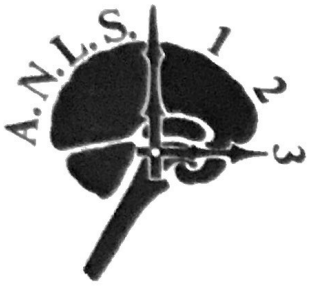

ADVANCED

NEUROLOGICAL

LIFE

SUPPORT

STUDENT MANUAL

This course is dedicated to the memory of

John Wilson Crosby

&

James 'Sparkey' Copeland

This course has been accredited for 15 hours of continuing medical education by the College of Family Physicians of Canada. A certificate will be issued on the successful completion of lectures, workshops and examinations.

Although due care has been taken to make sure all treatments are current and accurate, the physician is advised to consult local formularies and specialists for advice on treatment.

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ADVANCED NEUROLOGICAL LIFE SUPPORT COURSE MANUAL

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ADVANCED NEUROLOGICAL LIFE SUPPORT

INTRODUCTION

General practitioners, surgeons, emergency physicians, internists and paramedics are often reponsible for the initial management of neurological and neurosurgical emergencies. Many feel uncomfortable in these situations as neurology is perceived as being difficult and much of the pathology is devastating and irreversible. Neurological emergencies can be managed confidently by following the priniciples of ANLS' resuscitation.

For neurons to function they must have energy (glucose and oxygen) delivered by a transport mechanism (circulatory system). Exogenous toxins (drugs, metabolic wastes) or mechanical pressure from injury or disease can deprive neurons of these essentials.

The Advanced Neurological Life Support Course teaches resuscitation and rapid neurological assessment as they apply to trauma, coma, status epilepticus, dizziness, headache, infection and stroke through the use of a pre and post test, a standardized manual, straight forward lectures, "hands on" workshops and simulated patient exams. It is hoped that the student will adopt a uniform, methodical approach to all neurological emergencies.

CHAPTER 1:
RESUSCITATION AND NEUROLOGICAL ASSESSMENT

Objectives:

1. To provide an organized approach to and resusctiationtof the patient with a neuro-emergency.

2. To conduct rapid neuro-assessment of a simulated patient.

Case Presentation

You are working in a community emergency department with CT and neurological back-up one hour away. A 55 year old unconscious male is admitted. The ambulance officers state they were called by a passerby who saw him lying in an alley surrounded by empty whiskey bottles. On examination he responds only to deep pain and is filthy, smelling strongly of alcohol. How do you manage this patient?

We will return to this case and ask for your assessment and treatment after we review resuscitation and neurological assessment.

THE ANLS APPROACH - A B C D E

A - Airway (with C-spine control)

As in all resuscitation, airway maintenance is paramount. The brain requires oxygen for normal function. Patients suffering neuro-emergencies are at special risk for aspiration of vomitus, teeth or foreign objects. They often have depressed or absent gag reflexes and may have C-spine injuries. In status epilepticus, jaw clenching is often a problem. Workshops will review positioning and basic and advanced airway maintenance. Management of airways in combative patients and in semi-conscious overdose patients will be addressed.

All head injuries have an injured C-spine until proven otherwise. Techniques of C-spine immobilization will be reviewed in the workshop. A workshop on C-spine x-ray interpretation will also be presented.

B - Breathing

Patients with neuro-emergencies may have compromised breathing due to coma, chest wall or cord injuries. The physician must assess and assist breathing not only initially but also subsequently as respiratory effort may decrease with time. This may not be noticed in the confusion of caring for other problems. The airway workshop will review how to assess adequacy of breathing. All of these patients should be given high concentration oxygen. Blood gases should be done early.

C - Circulation

Internal and external bleeding must be recognized and treated and large bore IV's of normal saline should be started and run as fast as possible if the patient is hypotensive (while preparing for definitive surgical management). A common mistake in head injured patients is to restrict fluids to avoid cerebral edema. Hypotension must be treated vigorously and fluid restriction is undertaken **only** in patients with a **stable** circulation. It is also important to note that hypotension is rarely due to neurogenic shock. Other, more common, and correctable causes such as thoracic (i.e.hemothorax) or abdominal pathology (i.e. ruptured viscera) should be ruled out first.

D - Drugs

In patients with an altered level of consciousness consider hypoglycemia and narcotic overdose. Do a chemstix glucose measurement and give glucose if low. Consider giving Narcan if appropriate. Neurons can be rapidly and irreversibly damaged if hypoxia or hypoglycemia are not immediately reversed. More will be said about this in the metabolic coma lecture.

E - Evaluation - Rapid Neuro Assessment

The rapid neuro assessment includes:

History

Check with ambulance officers, police, next of kin, friends, and witnesses to find out the length of time the patient was in coma, possibility of injury or drug ingestion, past medical history and allergies. Check the patient's wallet and Medic Alert bracelet. Call the family doctor for information. Request an old chart if available.

Level of Consciouness

The Glasgow Coma Scale will be taught in the workshops. See Chapter 1. Page 10.

Head and Neck Assessment

Inspect and feel the head for bumps and bruises. In non traumatic situations check for neck stiffness (may be caused by pus or blood).

ENT Assessment

Ear examination may show red ear drums or a CSF leak. Blood may be present in the ear canal with a basal skull fracture. The **nose** may have blood and/or CSF. The **throat** may reveal odors of exogenous toxins (ethanol) or endogenour odors (ketones, fetor hepaticus).

Cranial Nerves

The following are important cranial nerves in the emergency setting:

II - Optic nerve - check pupils for symmetry, reaction to light (includes III); check for hemorrhages and papilloedema.
V - Trigeminal and
VII - Facial - test corneal reflexes bilaterally for symmetry.
IX - Glossopharyngeal and X-Vagus - check for presence and symmetry of gag reflex.

Sensation and Power

See if the patient moves all limbs equally, either spontaneously or in response to painful stimuli. Check the patient's ability to feel pin prick and light touch in awake patients in all limbs and trunk.

Reflexes

Check biceps, triceps, patella and ankle reflexes for symmetry. Check for symmetry and direction of plantar reflexes.

Returning to our case presentation, we start with **ABCDE.** The patient is intubated with c-spine control before x-rays are available. 100 % O2 is started with good spontaneous ventilation. Lungs are clear and chest x-ray is normal. Circulation shows a normal blood pressure and pulse and abdominal examination. Drugs, stat. alcohol, drug screen, glucose, metabolic profile are normal and Narcan has no effect. Head palpation reveals a large hematoma hidden by long hair in the occipital area. Skull x-ray shows an occipital fracture. ENT, cranial II, III,V,VII,IX,X reflexes and response to pain are normal.

The danger in this case was to assume that his coma was due to Ethanol. The ABCDE's of ANLS made sure he was stable and we didn't miss hypoglycemia (often seen in alcoholics) and the rapid neuro assessment led us to palpate his head revealing the hidden pathology. All comas must be treated as undifferentiated to avoid misdiagnosis.

Summary

Airway with C-Spine control
Breathing - 100% O2
Circulation - IV normal saline
Drugs - Narcan/Glucose and Thiamine when appropriate
Evaluation with rapid neuroassessment of level of consciousness, feel head check ENT, cranial nerves II, III, V, VII, IX, and X, sensation, power and reflexes.

CHAPTER 2:
HEAD INJURIES

Objectives:

1. To recognize the symptoms and signs of head injury.
2. To be able to resuscitate the acute head injury patient.
3. To recognize and treat raised intracranial pressure.

THE ANLS APPROACH - A B C D E

Airway with c-spine control	Clear and intubate if oral airway tolerated.
Breathing	Rule out and treat pneumothorax-give 100% 02 continually. Reassess for decreasing respiratory effort.
Circulation	Use IV normal saline +/- blood to restore BP to normal then keep vein open. Assess rest of body for sources of blood loss.
Drugs	Consider glucose (+/-thiamine), Narcan, drug screen.
Evaluation	Rapid neuro assessment (L.O.C.=G.C.S., feel head, ENT, cranials II,III,V,VII,IX, reflexes, pain). Total head to toe assessment in stripped patient.

Head injury affects 300 out of 100,000 people yearly.

Anatomy and Physiology of the Skull

The shape of the skull and the varying thickness of the bones composing it predispose to characteristic injuries. In younger patients blunt impact may separate the interdigitations of suture lines. In patients of all ages blunt trauma to the face and frontal regions may produce fractures through the anterior cranial fossa so that there may be communication between the paranasal air sinuses (ethmoid, sphenoid and frontal) and the subarachnoid space. Such an occurrence may be detected by air fluid levels in a sinus on a brow-up lateral skull x-ray, or by pneumocephalus. Linear fractures, in the relatively thin squamous temporal bone, may be caused by direct trauma or by outbending of the bone from a blunt impact to the occiput. Blunt impact to the occiput may result in disruption of the frontal or temporal lobe opposite to the site of

impact - a "contre-coup" injury.

MENINGES

The dura mater, or tough covering of the brain, is adherent to the inner table of the skull. The major draining veins of the brain, i.e., the superior sagittal, transverse and sigmoid sinuses, are located between its two layers and may be injured by depressed fractures. The middle meningeal artery, which emerges from the foramen spinosum and traverses the temporal bone in a groove, may be torn when the bone is fractured with blood released that strips the dura from the inner table of the skull. The arterial grooves are visible on a lateral skull x-ray; a fracture that crosses an arterial groove may signal an epidural hematoma.

Inside the dura are the leptomeninges, the arachnoid layer which contains the cerebrospinal fluid, and the pia on the surface of the brain. Draining veins bridge from the surface of the brain to the venous sinuses and may be torn when angular acceleration of the cranial cavity and its contents causes movement of the brain relative to the skull. Characteristically blood from torn bridging veins is released into the potential space between the dura and the arachnoid with the production of a subdural hematoma.

BRAIN

The cerebrum is divided into two hemispheres with one side receiving sensation from and governing muscle movement in the contralateral side of the body. Vegetative functions, such as the control of body temperature, blood pressure, heart rate and respiration, are controlled by central portions of the brain including the hypothalamus and brainstem. The "brainstem" comprises the mid-brain, pons and medulla. The reticular activiating system, which subserves consciousness, is located in the mid-brain. The cranial nerve nuclei and cranial nerves controlling eye movement are located in the mid-brain and pons. Their assessment may aid in the diagnosis of stupor and coma.

CEREBROSPINAL FLUID (CSF)

Cerebrospinal fluid is produced by the choroid plexus within the ventricular system. The normal circulation of CSF is from the lateral ventricles, via the foramina of Monro to the 3rd ventricle, thence via the aqueduct of Sylvius to the 4th ventricle at the level of the pons. From there, the CSF emerges principally by the formen of Magendie and circulates in the subarachnoid space upward through the tentorial hiatus to be reabsorbed via the arachnoid villi into the superior sagittal sinus. The flow of cerebrospinal fluid may be interrupted by distortions of the brain, or impeded by blood released into the cerebrospinal fluid, when cerebral tissue is disrupted. The

obstruction of cerebrospinal fluid circulation may further compound an already serious brain injury. Fractures, resulting in communication of the subarachnoid space which contains the cerebrospinal fluid with paranasal air sinuses or the exterior, may be diagnosed by the observation of CSF rhinorrhea (a leak via the nose) or otorrhea (a leak of CSF via the ear).

CRANIAL NERVES

Inferences regarding the site and severity of craniocerebral trauma may be made from the examination of the cranial nerves. In the management of acutely injured patients the examination of the cranial nerves should be abridged to include only those observations that can be made rapidly and accurately and which serve as a guide to therapy.

OPTIC (II)

Funduscopic examination may show subhyaloid (preretinal) heamorrhages that result from a rapid, severe rise of intracranial pressure transmitted to the optic nerve sheath. Papilledema takes hours to develop and is not seen early after an injury. Most patients with multiple injuries will not co-operate for a detailed examination of visual acuity, so that presence or absence of vision in an eye may be the only assessment possible. Severely reduced visual acuity in an eye reduces or abolishes the direct pupillary (constriction) response to light. The consensual response (constriction of the pupil when the light is shone in the contralateral eye) is preserved.

OCULOMOTOR (III)

The 3rd nerve, the nucleus of which is in the midbrain and which passes along the medial edge of the temporal lobe, subserves constriction of the pupil of the ipsilateral eye. Distortion of the nerve by displacement of the temporal lobe (e.g., transtentorial herniation) produces a dilated pupil that does not react to light (fixed) on the same side (94 % of the time) as the displaced temporal lobe. In addition, there is ptosis or drooping of the eyelid and inability to adduct the eye. A unilateral 3rd nerve palsy may be an important lateralizing sign in planning an emergency operation for an intracranial clot. An ocular injury may also cause fixed pupillary dilation (traumatic mydriasis). When there is orbital bruising, traumatic mydriasis may be suspected but should not allay concern regarding transtentorial herniation.

TRIGEMINAL (V)

The trigeminal nerve is the sensory nerve of the face. An injury to the nerve or its nucleus may render part of the face anesthetic, with the result that there may be abolition of corneal sensation and absence of the corneal reflex, that is, closing of the

eye when the cornea is touched.

FACIAL (VII)

The facial nerve may be injured in (basal) skull fractures of the temporal bone. Asymmetrical grimace to painful stimuli or inability to close an eye may indicate a facial nerve injury on the side of the palsy.

TENTORIUM

The tentorium separates the anterior and middle cranial fossae from the posterior fossa of the skull. The tentorial hiatus transmits the midbrain. The 3rd cranial (oculomotor) nerve passes near the edge of the tentorial hiatus in relation to the medial edge of the temporal lobe. It may be distorted by transtentorial herniation of the uncus, a hook like medial gyrus of the temporal lobe. Pressure on the cerebral peduncle containing the corticospinal tracts characteristically produces a contralateral hemiparesis. As the entire brain stem may be displaced to impinge on the sharp contralateral edge of the tentorium with the production of a hemiparesis ipsilateral to the uncal herniation, **the hemiparesis ipsilateral is a less reliable guide to the site of a probable intracranial clot than is the dilated pupil.**

UNCONSCIOUNESS

In blunt trauma, unconsciousness is usually the product of diffuse injury throughout the brain produced by shear strains that tend to separate one layer of cerebral tissue from another. Relatively minor stresses, producing only physiological abnormalities without structural damage, cause short lived concussion, whereas ruptured axons, cerebral petechiae and intracerebral hematomas produce permanent injury and possibly prolonged or permanent coma. Unconsciousness may also result from direct injury to the reticular activating system in the midbrain.

INTRACRANIAL PRESSURE

Diffuse injury to the brain, with damage to small blood vessels or to axons, may result in diffuse cerebral edema or hematomas within the substance of the brain or on the surface when blood vessels are torn by energy imparted to the brain by acceleration. Accommodation to an expanding intracranial mass is made by displacement of cerbrospinal fluid through the foramen magnum and, to a lesser extent, by compression of cerebral veins. If ventilation is inadequate and the partial pressure of carbon dioxide in the blood rises, there will be venous dilation and a further rise in intracranial pressure. As the cerebral blood flow to injured regions of the brain varies directly with cerebral perfusion pressure (the difference between systemic arterial pressure and intracranial pressure), failure to adequately treat shock and maintain a

good systemic arterial pressure may lead to worse cerebral damage or even death. For these reasons, adherence to the resuscitation priorities is an essential first step in the management of raised intracranial pressure. **This means do not restrict fluids to head injured patients until their blood pressure is restored.**

HISTORY

When management of unconscious patients must be based on clinical grounds alone one must distinguish, if possible, between mechanical damage caused by angular acceleration and expanding intracranial hematomas. Nothing can be done about the former, but successful treatment of the latter makes the difference between survival and death. The characteristic story of a patient unconscious because of direct mechanical injury is: immediate unconsciousness and/or focal neurological deficit followed either by no change or by improvement. This history may be obtained from bystanders who accompany the patient to hospital or from ambulance attendants/paramedics.

In contrast, falling level of consciousness and/or increasing focal neurological deficit are the characteristic indications of an expanding intracranial hematoma. Once the patient comes under continuous observation in the emergency department, a changing Glasgow Coma Scale is sufficient to decide whether the patient is improving, staying the same or getting worse. It is not always possible to make the distinction between mechanical damage and an expanding intracranial hematoma on clinical grounds alone, as the two may co-exist, or the intracranial pressure may be rising and causing worsening coma as the effects of the initial concussion recede.

LEVEL OF CONSCIOUSNESS

The Glasgow Coma Scale characterizes thhe patient's level of consciousness by observation of eye opening, best motor response and best verbal response. It should be repeated and charted every 1/2 hour of the initial resuscitation.

GLASGOW COMA SCALE

Eyes Open	Spontaneously	4
	To Speech	3
	To Pain	2
	None	1
Best Verbal Response	Oriented	5
	Confused	4
	Inapprop. words	3
	Incompre. Sounds	2
	None	1
Best Motor Response	Obey commands	6
	Localize pain	5
	Withdrawal to pain	4
	Flexion to pain	3
	Extension to pain	2
	None	1

SPECIAL BRAIN STEM RESPONSES

The function of cranial nerves III, IV, and VI may be assessed by turning the head, oculocephalic response of "doll's eyes" and/or the oculovestibular response (ice water irrigation of the ear canals). These tests contribute little to the emergency management of the unconscious patient and should be reserved for the neurosurgical unit, as the former may aggravate a spinal cord injury and the latter may introduce bacteria via a basal skull fracture.

SKULL X-RAYS

With the exception of penetrating injuries where metallic foreign bodies may be identified, skull x-rays contribute relatively little to decision making. Skull x-rays must never be obtained at the expense of continuing efforts at resuscitation.

The presence of a linear skull fracture increases the probability of an intracranial hematoma, especially when it crosses vascular skull markings, for example, the middle meningeal artery as it runs in the squamous temporal bone. Fractures of the

petrous temporal bone may not show on skull radiographs but can be diagnosed by examination of the external ear canal. Otorrhagia (blood running from the external ear canal) and hemotympanum (blood behind the tympanic membrane) are reliable signs of basal skull fracture and may also signal the presence of an epidural heamatoma.

Closed (intact scalp) depressed skull fractures do not constitute an emergency but may be significant if the outer table of the depressed fragment is depressed as far as the inner table of the adjacent skull. Compound (open) depressed skull fractures require early operative intervention with elevation or removal of the fragments. Elevation of the fragments should only be undertaken in an operating room setting by a physician prepared to effect hemostasis, as the fragments sometimes tamponade torn blood vessels. If a decision is made to transport a patient with a compound depressed skull fracture to a neurosurgical centre, any superficial bleeders should be secured with a hemostat, sutures or metallic clips and voluminous sterile dressing applied.

Basal skull fractures that produce a communication between the subarachnoid space and paranasal air sinuses or the exterior may result in air fluid levels within those sinuses on brow up lateral radiographs, or in pneumocephalus (air inside the head). Dripping of fluid from the nose (CSF rhinorrhea) or from the ear (otorrhea) may signal a basal skull fracture and alert the physician to the danger of subsequent meningitis. As the spinal fluid of patients with significant head injuries is often mixed with blood, the characteristic "ring" or "target" sign may be observed. If a drop of spinal fluid is allowed to fall on filter paper or the patient's sheet, the liquid phase of the mixture migrates farther than the solid components that form a circular central "bulls-eye".

Fracture of orbital bones release blood that is constrained by periosteal attachments to a crisp periocular distribution - the "raccoons-eye" sign. Contusion of orbital contents may produce ecchymoses with more diffuse boundaries.

Frontal basal skull fractures may permit the inadvertent intracranial passage of a nasogastric tube. A decline in the level of consciousness or an increase in neurological deficit may be the result of inadequate ventilation or systemic arterial pressure. These causes of neurological decline should be considered first. If the neurological condition is worsening, despite adequate ventilation and perfusion pressure, then an expanding intracranial clot as the cause is likely.

CONCUSSION

Angular acceleration, if not too severe, may produce a short and apparently completely reversible loss of consciousness. If mild enough, angular acceleration may

not alter the patient's apparent level of consciousness but will temporarily interrupt the continous recording of memory. Conscious patients should be questioned regarding the details of their accident to establish whether or not there is retrograde post-traumatic amnesia. If the neurological examination is completely normal and if the patient's home is not too remote from the hospital, he may be released in the care of a responsible person with instructions (see Appendix 2, page 18, head injury routine) to return if drowsiness or neurological deficit develop. Recent evidence is that, despite an apparent return to a normal mental status, there may be a measurable injury caused by concussion that accounts for the "post-concussive syndrome" of headache, irritability and impaired concentration.

CONTUSIONS

A prolonged episode of unconsciousness or persistent drowsiness in the wake of a concussion may signal petechiae or contusions within the cerebral substance. A focal neurological deficit may be present. These patients should be observed in hospital until they are alert, usually a period of 48 hours or more. These patients may harbour surprisingly large intracranial hematomas that may only be detected in the first instance by CT scanning.

INTRACRANIAL HEMATOMAS

Hematomas requiring surgical evacuation may be classified as extracerebral or intracerebral.

Extracerebral

Epidural Hemorrhage

Epidural hematomas are less common than subdural hematomas. The commonest cause of epidural bleeding is interruption of the middle meningeal artery with release of blood between the dura and the inner table of the skull. Venous sinuses or diploic veins (venous channels between the inner and outer table of the skull) may also be the source of epidural bleeding. The so called "lucid interval" between a period of unconsciousness from concussion and the onset of coma from raised intracranial pressure is the exception rather than the rule. Acute epidural hematomas are indistinguishable on clinical grounds from acute subdural hematomas. An epidural heamatoma will most likely be found in relation to a fracture diagnosed by an overlying scalp hematoma, a skull radiograph or by hemotypanum and/or otorrhagia. The pupil on the side of the hematoma may be dilated.

Acute Subdural Hematoma

In contrast to acute epidural hematomas the outcome, after evacuation of subdural

hematomas, is not as likely to be good. Blood is released into the subdural space by torn cortical or bridging veins or from cortical lacerations. As for rapidly falling level of consciousness and increasing neurological deficit due to epidural hematoma, hyperventilation should be commenced and 350 cc.'s of 20% Mannitol should be given and the hematoma evacuated promptly.

Traumatic Intracerebral Hematomas

Severe angular acceleration and strong shearing forces may tear blood vessels within the substance of the brain. Focal neurological deficit and coma may result. Intracerebral hematomas are best diagnosed by CT scanning as they will be missed by exploratory burr holes or craniotomies performed in the expectation of finding a surface hematoma.

Patients who present with a foreign body protruding from the skull should be transferred for definitive neurosurgical treatment with the object in place, as it may be tamponading torn blood vessels. Skull x-rays are necessary to demonstrate the extent of penetration. In missile injuries skull x-rays are necessary to demonstrate the distribution of metallic fragments so that an effective surgical procedure may be planned.

CEREBRAL EDEMA, VASOSPASM AND/OR LOSS OF CEREBRAL AUTOREGULATION

Cerebral edema may develop as a consequence of diffuse shearing injury to the brain which disrupts axons and small blood vessels. Severe, diffuse cerebral edema is more likely to occur in younger patients with severe head injury. The control of diffuse cerebral edema may be difficult and therapeutic manipulations, such as relative fluid restriction and Mannitol, may be the opposite of those recommended for non-neurological injuries. Consult a neurosurgeon for advice.

Vasospasm caused by direct injury to cerebral blood vessels or by the release of vaso-active substances into the subarachnoid cerebrospinal fluid may be sufficiently severe to cause cerebral infarction. Cerebral autoregulation refers to the phenomenon by which a constant blood flow per volume of cerebral tissue is maintained despite fluctuation in systemic arterial blood pressure. With a brain injury, cerebral autoregulation is lost and the regional cerebral blood flow to the traumatized portion of the brain varies directly with cerebral perfusion pressure, that is, the difference between systemic arterial and intracranial pressure. Inadequate systemic arterial pressure or uncontrolled intracranial pressure, both of which reduce the cerebral perfusion pressure, will lead to further cerebral infarction. With the loss of vascular integrity from such an episode and the re-establishment of high cerebral perfusion pressure, cerebral edema and intracranial hypertension are made worse. The continous maintenance of adequate cerebral perfusion pressure, ie., the difference

between mean systemic arterial pressure and intracranial pressure, (60 torr) is essential. **Shock must be treated.**

Treatment

A Airway with C-Spine Control
B Breathing (will be discussed in the workshops) remember to give 100% O2 and rule out pneumothorax. Reassess continously.
C Circulation

It is the rule to treat severe systemic injuries with large volumes of balanced salt solutions. The physician caring for a patient with multiple injuries must use judgement in the administration of intravenous fluids. Over hydration, which makes cerebral edema worse should be avoided. Try to attain a normal blood pressure. Rule out thoracic and abdominal trauma.

D Drugs in head injury - check glucose, consider Narcan, screen for alcohol and drug overdose.

Steroids (glucocorticoids) in high or low doses are of no benefit in the treatment of patients with severe brain injuries and are not recommended.

Diuretics: Mannitol as a 20% solution, given as a rapid intravenous infusion over 15 minutes in an initial dose of 1 gm/kg (350 cc. for a 70 kg. person) creates an osmotic gradient between intact brain and the blood stream. This results in the movement of brain water into the blood stream with reduction in brain bulk and relief of intracranial hypertension. When the amount of intact brain is relatively small, the drug is less effective. With an expanding intracranial clot, the duration of action of Mannitol may be relatively short. Subsequent doses are progressively less effective. The drug is most useful in lowering intracranial pressure while preparations for evacuation of an intracranial hematoma are being completed. A urinary catheter should be in place when Mannitol is given, as there is usually brisk diuresis; the diuresis is sometimes of sufficient volume to nullify the effects of fluid adminstered for vascular support in systemic injuries. Judgement in its administration must therefore be used . In general, the drug should be given if a decision is made to evacuate an intracranial hematoma prior to ransfer to a neurosurgical centre in consultation with a neurosurgeon who has agreed to accept the patient in transfer. The drug should not be given unless one has reason to believe that there is raised ICP.
Hypocapania: carbon dioxide is an extremely potent vasodilator. When mechanial ventilation is indicated for other reasons, intracranial pressure seems best controlled with the partial pressure of carbon dioxide adjusted to between 25 and 30 torr. With respect to the head injury alone, intubation and mechanical ventilation are indicated if the patient is unconscious and, therefore, unable to maintain an adequate airway or if arterial blood gases indicate hypercarbia.

E Evaluate - rapid neuro assessment. LOC = GCS, Head, ENT, Cranial Nerves II, III, V, VII, IX, X, pain, reflexes.

OTHER SIGNS AND SYMPTOMS OF HEAD INJURY

Restlessness

Patients with multiple injuries, with or without significant brain injury, are often restless. A diligent search for a cause of restlessness, such as hypoxia, hypotension, distended urinary bladder, undiscovered fractures, excessively tight plasters and bandages, etc. should be made. Morphine, which significantly reduces the level of consciousness, or Chlorpromazine, which may cause hypotension and drowsiness are not indicated.

Scalp Wounds

The scalp has a luxurious blood supply. Unless tissue loss makes apposition of the edges of scalp wounds difficult they are usually easy to repair and they heal well.

Blood Loss

Although blood loss from scalp wounds can be particularly severe, hypotension should not be attributed to bleeding from the scalp until other causes are ruled out. As with any wound, large vesesls should be clamped and litigated. The smaller ones may be cauterized. As the blood vessels of the scalp run between the skin and the galea (fibrous aponeurotic inner layer of the scalp), temporary tamponade of the vessels may be obtained by placing hemostats on the galea and everting the wound.

If the wound gapes, then the galea is torn. The wound should be carefully inspected and palpated using sterile technique. Bone fragments and foreign bodies may be identified in this way. CSF or brain tissue in the wound indicate a compound depressed skull fracture with laceration of the dura and arachnoid.

Repairing the Scalp Wound

The region of the wound should be shaved, prepared and draped in a sterile field. Hair and foreign bodies should be removed and the wound should be irrigated with a large volume of saline. Loose bone fragments may be removed, but an operating room setting is recommended as impacted fragments may sometimes tamponade torn blood vessels which will bleed as the pressure is released.

If a good cleansing of the wound has been possible, it is best closed in layers. The galea is reapproximated first and then the skin. If there has been gross contamination of the wound or, if a definitive repair is not possible and a prolonged transfer time is anticipated, the wound may be closed with vertical mattress sutures. A bulky dressing that applies mild pressure to the wound is preferred.

COMPUTED TOMOGRAPHY (CT SCAN)

A CT scan is recommended at the first opportunity permitted by ongoing resuscitation, especially when pharmacological paralysis and ventilation are required for the treatment of other injuries (lung contusion, flail chest, etc.,) or when the patient will be anaesthetized for a long surgical procedure, such as open reduction and internal fixation of fractures. If a laparotomy is required for managment of the patient in a centre that has no CT scanner, and there are clinical grounds to suspect an intracranial hematoma, exploratory burr holes should be considered in lieu of a CT scan.

OTHER TESTS

Electroencephalography and isotope scanning have no role in the acute management of patients with head injuries. Lumbar puncture, which may exaggerate pressure gradients across the tentorium and the foramen magnum, predisposing to herniations of the brain, **is not only useless but potentially lethal as well.**

Surgical Management

In the early stages as the intracranial clot expands, displacement of cerebrospinal fluid and, to a lesser extent, compression of cerebral veins mitigate rising intracranial pressure and slow the decline of the level of consciousness. As these mechanisms of compensation are exhausted the deepening of coma accelerates. If there is any decline in the patient's neurological status, as the Glasgow Coma Scale is repeated, then transfer may be advisable. The advice and consent of the neurosurgeon who is to receive the patient should be obtained. In deciding whether exploratory burr holes and possibly a craniotomy should be done in the primary hospital, the patient's ABCD(E)'s and other injuries, the neurological status of the patient, his rate of decline, the expected transport time and the skill and experience of the sending physician must all be considered.

The development of a unilateral fixed, dilated pupil during resuscitation of a patient who initially localized painful stimuli but who begins to exhibit spastic flexion or extension, signals the need for an immediate evacuation of his intracranial hematoma. The dilation of a second pupil when one has been dilated is equally ominous. Mannitol should be given. The entire scalp should shaved, prepared and draped in a sterile field, but the first burr hole should be placed on the side of the initial pupil.

With the pressure relieved, a craniotomy may be completed with less urgency or the burr hole may be enlarged to a craniectomy to permit better access to the cranial cavity. Craniotomy is the preferred method of evacuation of acute intracranial hematomas.

If a hematoma on the surface of the brain is not discovered but intracranial hypertension is identified, the prognosis is poor as the cause may be an intracerebral hematoma or diffuse brain swelling. The patient should then be transferred to a neurosurgical centre with a CT scanner for definitive diagnosis and treatment. **The advice of the receiving neurosurgeon should alway be obtained early in the case.**

Patients with spastic extensor posturing and bilateral fixed dilated pupils from the outset may harbour intracranial hematomas but the likelihood of a good outcome is very small. Neurosurgical advice should be obtained regarding management.

SUMMARY

- Ensure ABCD(E)'s, especially good ventilation and good systemic arterial blood pressure.
- Establish a baseline neurological examination, using the Glasgow Coma Scale
- Discover and treat associated injuries.
- Re-examine for deterioration in neurological status.
- Obtain a neurosurgical consultation regarding transfer early.
- Treat life threatening surgical and neurosurgical emergencies that preclude transfer.

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APPENDIX II

HEAD INJURY ROUTINE

This routine is NOT to be issued to the patient, but to a responsible third patry.
Patients should return to Emergency Department at once, day or night, if there is:

1. Increasing drowsiness and/or confusion.
2. Persistent headache (avoid giving aspirin).
3. Persistent vomiting (more than two times).
4. Altered vision or unequal pupil size.
5. Neck stiffness.
6. Difficulty rousing (wake patient every two hours during the first night).
7. Seizures (convulsions), twitching or abnormal movements of limbs.
8. Bleeding or clear fluid dripping from ears or nose.
9. Weakness of either leg or arm.

CHAPTER 3:
SPINAL CORD EMERGENCIES

Objectives:

1. To recognize the symptoms, signs and radiological manifestations of spinal cord emergencies.

2. To be able to resuscitate the acute spinal cord injured patient.

Airway	-	With c-spine control
Breathing	-	100% 02 Rule out pneumothorax Assist if high paralysis
Circulation	-	IV to restore blood pressure Assume shock is due to treatable cause (ruptured abdominal viscus)
Drugs	-	Steroids may help in cord damage Check with your local neurosurgeon

INTRODUCTION

In the field of acute spinal cord injury, the ultimate outcome of an individual case depends upon the accuracy, adequacy and speed of the first aid management, diagnosis and treatment offered by all medical, nursing or paramedical personnel the victim encounters within the first few hours. Irreparable damage or long term benefit may follow incorrect management, respectively. Fortunately, cord injuries are not nearly as common as other injuries, such as head injuries, but unfortunately, the emergency diagnosis is occasionally missed or inaccurate, and the initial treatment is often incorrect, insufficient or too late. With injury, mistakes may be very costly. During the past ten years some major changes have occurred in the management of cord injuries.

The purposes of this course are to review the difficult aspects of first aid, diagnosis and treatment of these injuries, and to offer clues to overcome the difficulties.

FIRST AID MANAGEMENT

The priorities of the first aid management of spinal cord injuries are similar to the priorities of management of any trauma victim - **airway, breathing, circulation and drugs** with the addition of special attention to extrication, positing and immobilization. A workshop will cover these topics.

Airway and Breathing

With respect to emergency management of the airway and breathing, there are some specific features of cord injury that require attention. In the cervical region, if the injury is above C4, apnea may occur because of failure of both the intercostal muscles and the diaphragm. These patients may be conscious and alert intially, but have complete or almost complete respiratory paralysis depending on the severity of the cord injury. Only artificial ventilation will save the patient. In the field, this may be accomplished by mouth to mouth, or ventilation with a mask and bag. In the hospital, intubation and mechanical ventilation are preferable. Endotracheal intubation must be carefully performed lest any neck manipulation drive bone or disk fragments into the cord. This will be covered in the workshops. If the injury is below C4, diaphragmatic breathing is usually sufficient to maintain life in most individuals, although older patients, especially those with pre-existing pulmonary or cardiac disease, may rapidly develop respiratory failure, especially with upper thoracic injuries. In lower thoracic injuries, persisting intercostal muscle and diaphragmatic function will usually be sufficient to allow adequate ventilation. In lumbar injuries, airway and respiration are usually not affected, unless there are other injuries, (e.g. head inuury).

Circulation

Of course, bleeding must be stopped and systemic shock counteracted. Hypotension, due to neurogenic shock (an extrememly rare condition) or bleeding, may worsen the post traumatic ischemia of the cord which follows major cord injury. The blood pressure should be restored to normal levels, preferably by the administration of normal saline and blood transfusions.

Drugs

A recently published study from the National Acute Spinal Cord Injury Study suggests that massive doses of corticosteroids (methylprednisolone) given within eight hours of injury may improve the outcome in initially complete as well as initially incomplete spinal cord lesions. The treatment regimen for methylprednisolone involves a bolus of 30 mg./kg. body weight, followed by an infusion of 5.4 mg./kg./hour for the

next 23 hours. The treatment is complete at 24 hours.

While this is a recent study, which has been subjected to some criticism from experts, it does offer the first glimmer of hope that a therapeutic intervention may improve the outcome in these neurologically devastated patients. Pending further studies, which may confirm or refute these results it would seem reasonable to initiate this type of treatment when patients are first seen, because the benefit demonstrated in the pilot study required commencing treatment within eight hours.

Problems with Extrication

The problem of extrication is extremely important in certain accidents such as motor vehicle accidents or diving accidents. During extrication a moderate amount of traction should be applied to the neck, but torsional or angulation stresses on any segment of the spine must be avoided then and during any transfers either at the accident scene or hospital. K.E.D.'s, short and long spine boards, semi rigid collars and sandbags are standard equipment in all emergency vehicles.

Positioning

The positioning of spinal cord injured patients is extremely important. In general, the normal anatomical position is the most favourable for restoration or maintenance of spinal alignment and spine-spinal cord relationships. Excessive traction, and all torsion or angulation may worsen spinal cord or nerve root compression. To lift or transfer a cord injured patient, three or preferably four people are required. One applies approximately five pounds traction to the head, one lifts the shoulder, one the buttocks and one the thighs. Rolling a supine patient into the semiprone position to improve the airway or to prevent aspiration of vomitus can be safely done by the "log rolling" technique. Workshops will deal with this issue.

Reduction of Deformity

The question of when to reduce a spinal deformity is a difficult one. In general, spinal deformities in the cervical, thoracic or lumbar regions are best reduced under controlled circumstances in the hospital with radiological monitoring. With neck injuries, the purpose of applying traction during extrication or transport is not to reduce a spinal deformity but to prevent further deformity. However, with cervical injuries, if the airway is obstructed and breathing is compromised, it may be necessary to attempt to restore more normal anatomical alignment by exerting a moderate amount of traction to reduce the deformity.

Immobilization

Immobilization is the next problem to be considered. During trasnport to the hospital or transfer within the hospital, patients with cord injury must be adequately immobilized with sandbags alongside the neck, and with straps or belts around the trunk. Semi rigid collars are advised. As the ambulance rounds a corner in the road or the stretcher negotiates the corridors between the emergency and radiology departments, the patient must be prevented from moving on the stretcher or spine board or indeed from falling off it.

DIAGNOSTIC DIFFICULTIES

The diagnosis may be missed because of an inadequate or incomplete history or physical examination. In terms of the history, keep in mind that most spinal cord injuries occur in one of the four following situations: motor vehicle accidents, work accidents, sport-recreation activities and falls at home. Thus, when managing accident victims in any of these categories, think of the possibility of spinal cord injury. Unfortunately, in each of these situations, difficulties arise because multiple factors may obscure diagnosis. An example is the patient who was not wearing a seat belt and had too much to drink, who drove off the road and was catapulted from his car after the doors flew open upon impact. The combination of the accompanying head injury and the inebriation made it extremely difficult to examine the patient. Indeed, the presence of multiple trauma diverts the examiner's attention towards the more obvious, but less important limb fractures. Other difficult situations arise when the patient is psychologically upset by the injury or is hypoxic, and as a result is restless, uncooperative or agitated. In all of these instances the practitioner's diagnostic acumen is severely taxed. Indeed, the patient's reactions may be so bizarre that the diagnosis of hysteria may be mistakenly made. This diagnosis is extremely dangerous in situations involving trauma. Thus, in the presence of alcohol, multiple trauma or head injury, one must make the "safe assumption" that there is an accompanying spinal injury. With major trauma to the abdomen or chest, always suspect that the trauma may have been sufficient to dislocate the spine.

History Clues

Clues from the history of an accident victim are exceedingly helpful. Was the patient moving his legs at the scene of the accident only to have the function disappear later, or were the limbs motionless at the scene of the accident with subsequent gradual improvement? The former situation would indicate an unstable spine with severe pressure on the cord from progressive dislocation while the latter may indicate a period of spinal shock which is now passing.

Examination Clues

Inability to move one or more limbs, or relative lack of movement of one or more limbs is highly suspicious, and thus, the diagnosis can often be made on the evidence from inspection alone. Complaints of weakness, tingling, or loss of sensation must be regarded as of extreme importance. Post traumatic urinary retention is a danger sign.

Clues from the examination are gained from testing the strength, sensation and reflexes of all four limbs. The spine must be palpated in its entirety, paying special attention to tenderness, swelling, step deformity and crepitus. The examiner's hand can be passed safely between the patient and the mattress or stretcher, and the spine palpated all the way from the cervical region to the sacrum. This must be done in every instance. Recognize that hypotension, bradycardia and warm extremeties are due to cervical cord injury and not systemic shock which usually causes hypotension, tachycardia and cold extremities. Recognize that cervical cord injuries cause paradoxical respiration in which the chest cage is drawn in and the abdomen expands because of intercostal muscle paralysis and preserved diaphragmatic contraction. Don't misinterpret reflex withdrawal of the limbs in response to "painful" stimulation of the extremeties as being due to voluntary movement. Rule out treatable causes of shock first.

RADIOLOGICAL DIFFICULTIES

The diagnosis of spinal or spinal cord injury may be missed due to inadequate or incomplete x-rays. In all multiple trauma victims, <u>the entire spine must be x-rayed</u> because in about 7% of patients with spinal injuries more than one level of the spinal cord is injured. Injuries to the cervical region present the most frequent pitfalls. Indeed, the most common radiological deficiency is the failure to include the lower cervical spine on the lateral view. Often the x-rays extends only as far down as C5 or C6 on the lateral view, or the vertebrae below that are obscured by the bones and musculature of the upper arms and shoulder. This difficulty commonly occurs in young muscular males, precisely the group comprising about 60% of all patients with cord injuries. Added to the conspriacy against the medical practitioner is the fact that fracture/dislocations of C5-6, C6-7, or C7-T1 are extremely common. They will be missed unless a good lateral view is obtained all the way down to the top of T1. Radiology technicians, radiologists, emergency physicians, GP's and specialists all share the responsibility of ensuring that a patient with a neck injury must never leave the radiology or emergency departments without a clear view of the entire cervical spine, on both anteroposterior and lateral views, all the way down to the top of T1. If complete visualization cannot be obtained with the plain films, then there are numerous "tricks" that the radiology technician or radiologist can use to show the lower cervical spine, including swimmer's views, oblique views, pillar views, tomography and pulling down on both arms. Computed tomography has been an extremely helpful method of

diagnosing acute spinal injury. If in doubt immobilize the spine and get specialist help.

Radiological Clues

Soft tissue swelling between the spinal column and the air shadows of the respiratory and digestive tracts indicates trauma. Loss of the normal curves, such as reversal of the cervical lordotic curve indicates trauma. Remember that in older people significant injury to the cord may occur as a result of pinching of the cord by osteophytes and ligamenta flava, in which case the x-rays may show no signs of trauma. The C1-2 region is also a frequent area of difficulty where lesions may be missed. Don't forget than an open mouth view is essential for adequate visualization of the odontoid in the anteroposterior direction. Workshops will review C-spine interpretation.

TREATMENT DIFFICULTIES

The patient has been transported supine and has just vomited because his stomach is full of beer, and his associated head injury has made him drowsy and nauseated. Rapid, accurate treatment will be life saving, and will prevent further cord damage from his unstable spinal fracture. As stated above, proper respiratory management remains the first priority during the entire acute phase. Suction his **airway** and for **breathing** give hims oxygen by nasal prongs, mask or endotracheal tube as required. **Circulation**, treat any hypotension with plasma or blood to improve the impaired post traumatic spinal cord blood flow. **Drugs**, steroids may help spinal cord lesions. Empty the stomach with a NG tube and treat the urinary retention due to neurogenic bladder with an indwelling catheter initially. Keep the patient in the normal anatomical position and "log roll" him when necessary. Don't forget the "two hour rule" regarding pressure sores. In cord injured patients, pressure sores occuring on the firth day, or any time thereafter, may persist for the rest of thier lives. Indeed, they may be fatal. If the injury is in the cervical region and x-rays who the dislocation or fracture, institute cervical immobilization to be demonstrated in the work shop.

CONSIDER SPINE INJURY IN:

1. Every patient with a head injury
2. Every patient with multiple trauma.
3. Every motor vehicle accident victim.
4. Every diving or drowning victim.
5. Every severe work accident case or fall at home.
6. Any movement of the spinal column after trauma will cause irreparable damage to the spinal cord.

SUMMARY

Principles of the acute treatment of the injured spinal cord or spine:

1. Establish **Airway** to ensure adequate oxygen and support **Breathing.**

2. **Circulation** - to ensure adequate blood pressure. Rule out treatable causes of shock.

3. **Drugs** - steroids may help, check with your local neurosurgeon.

4. Immobilize spine with back board, semi rigid collar, tape and sandbags, to:
   - provide best milieu for root and cord recovery.
   - to decrease pain.
   - to allow rapid mobilization

5. Detect and treat persisting cord compression in:
   - selected incomplete cases, compression should be relieved immediately.
   - in most immediate complete cervical or thoracic cases decompression is useless, except in selected cases with complete conus or cauda equina lesions.
   - consider subacute decompression in complete cervical cases for nerve root recovery.

6. Operative fusion may be required for selected unstable fractures or dislocations.

7. Open wounds generally require debridement and closure.

8. Prevent pressure sores by "log rolling" every 2 hours.

9. Prevent vomiting and aspiration by nasogastric tube.

10. Prevent bladder distention by using an indwelling catheter and then intermittent catheterization.

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CHAPTER 4 : SUBARACHNOID HEMORRHAGE

Objectives:

1. To recognize the symptoms and signs of subarachnoid hemorrhage and to stabilize and refer patients early for definitive neurosurgical care.

2. To know the indications and contraindications for lumbar puncture in suspected cases of subarachnoid hemorrhage.

3. To recognize and treat serious acute sequelae of subarachnoid hemorrhage, including raised intracranial pressure, arterial hypertension, seizures and depressed level of consciousness.

- The diagnosis of subarachnoid hemorrhage must be considered in all patients presenting with the spontaneous onset of sudden severe headache.

- Failure to recognize and initiate neurosurgical referral for patients with subarachnoid hemorrhage may result in re-bleeding and other catastrophic sequelae which have a high risk of death and severe disability.

- CT scanning is the confirmatory assessment of choice but may be negative in mild cases particularly those presenting after a delay of several days following subarachnoid hemorrhage.

- The judicious use of lumbar puncture may confirm the diagnosis of subarachnoid hemorrhage in uncertain cases, even if the CT scan has been negative.

INTRODUCTION

The rupture of an intracranial aneurysm is the commonest cause of spontaneous subarachnoid hemorrhage (SAH), accounting for 80% of cases. The other causes (see below) are less common and generally less lethal than the rupture of an intracranial aneurysm.

Incidence

There are estimated to be 28,000 cases of aneurysmal SAH per year in North America (3000 in Canada) and, of those, only 8500 undergo neurosurgical repair of the aneurysm and only 5000 cases are returned to good health. While the devastation of the first recognizable hemorrhage may be overwhelming, there is considerable evidence that many patients suffer milder initial episodes ("sentinel" or "warning leak") followed, within days or a few weeks, with a more severe hemorrhage. Failure to recognize the mild initial episode may occur when patients do not seek medical attention, but is more frequently the result of lack of prompt recognition by the physician when help is sought. Aneurysmal SAH carries a much higher mortality and morbidity than myocardial infarction and the cause is potentially surgically curable so that discharging a patient who comes to the emergency room with a mild SAH is a graver error than discharging a patient whose chest pain heralds a myocardial infarction.

Demographics

Aneurysmal SAH affects woman more frequently than men (2:1 incidence) and the average age at presentation is about 50 years. No age group is immune, but aneurysmal SAH is relatively infrequent in children. Rarely, predisposing conditions are associated, such as aortic coarctation or polycystic kidney disease. In the latter condition there may be a family history, but cerebral aneurysm is usually a sporadic condition without clear genetic predisposition. Autopsy series suggest that about one percent of the population harbour an intracranial aneurysm, but some of these lesions remain asymptomatic.

ANLS APPROACH TO SUBARACHNOID HEMORRHAGE

PRESENTATION

Patients complain of the sudden onset of excruciatingly severe headache, often associated with orgasm, heavy lifting or straining at stool. Whereas the initial pain may be anywhere in the head, occipital and upper cervical discomfort soon follow and often are the predominant site of pain. Vomiting is very frequent and may lead to

aspiration in patients whose level of consciousness is depressed. Though it may wax and wane in severeity and be partially eased by analgesics the headache rarely subsides completely for days after the ictus (stroke/attack, hemorrhage). Photophobia is frequently reported. Migraine, a frequent misdiagnosis after SAH, also produces headache with vomiting and photophobia, and the patient may appear to be quite ill. This ubiquitous condition does not exclude the patient from the risk of aneurysmal SAH and the physician must retain a high level of suspicion in all but the most stereotyped attacks of headache. Why did the chronic migraine patient seek medical attention for this attack? Was the onset more sudden and severe? Was there transient collapse at the onset? A negative investigation for SAH will not harm a migraine patient, while failure to correctly diagnose an aneurysmal subarachmoid hemorrhage may have catastrophic consequences.

In the most severe cases, the patient may collapse clutching the head, and rapidly die or lapse into irreversible coma. Other patients may have an initial loss of consciousness and then improve, sometimes regaining consciousness in minutes. Seizures may occur, especially at the onset. Patients may exhibit focal signs, such as aphasia, hemiparesis or paralysis of eye movements. Disorientation and confusion are frequent.

Given the history of an acute onset, the physician should examine the patient for meningeal irritation, especially neck stiffness to passive flexion. Meningism may take minutes to hours to develop after the ictus, so this assessment may need to be repeated as the patient is observed in the emergency room. In deeply comatose patients meningeal irritation may not be demonstrable. Conversely, "false meningism" may result from endotracheal intubation in patients with depressed consciousness who are not in deep coma.

OCULAR HEMORRHAGES

Examination of the ocular fundus may reveal subhyaloid (or preretinal) hemorrhages, expecially in more severe cases. These large dark hemorrhages may alter with position due to gravitational effect, and occasionally rupture into the vitreous, obscuring vision for the patient and visualization of the fundus by the physician.

LEVEL OF CONSCIOUSNESS

The most important feature of the examination for prognosis in this, as well as other acute intracranial processes, is the level of consciousness. This is best observed and recorded along the lines of the Glasgow Coma Scale, as outlined in Chapter 1. A rapid neurological assessment will reveal confusion or speech disturbances, such as dysarthria or dysphasia, or cranial nerve palsy. Evidence of third nerve (oculomotor) palsy, such as mydriasis, ptosis or paralysis of adduction and vertical eye movements, with good preservation of consciousness, may indicate that the aneurysm is adjacent

to the third nerve.

DIAGNOSIS

When clinical suspicion of SAH occurs, prompt confirmation of the diagnosis is essential to allow definitive treatment before rebleeding occurs.

CT SCANNING

If CT scanning is readily available, a plain (unenhanced) CT of the head should be obtained as soon as possible. This may demonstrate blood in the subarachnoid space, in the basal cisterns or the sulci of the cerebral or cerebellar hemispheres. The site of the subarachnoid blood shown may suggest the likely source of the bleeding. For example, SAH in one sylvian fissure suggests a middle cerebral artery aneurysm on that side. The amount of SAH seems to correlate with the risk of subsequent symptomatic vasospam developing. Hydrocephalus may be seen, especially after severe bleeding. Intraventricular or even subdural hematoma may occur as the result of aneurysmal rupture. Intracerebral hematoma may also be demonstrated by the CT scan. The site of the intracerebral clot and the other associated features shown by the scan usually permit an experienced observer to anticipate a ruptured aneurysm as the cause of the bleeding. Occasionally, particularly in large aneurysms, the aneurysm itself may be seen. If the CT scan is positive, lumbar puncture (LP) is unnecessary and frequently unwise.

NEGATIVE CT SCAN

The CT scan may be entirely normal ("false negative result") in three important situations. In each case, LP is required to make a definitive diagnosis in these situations and it should be performed without hesitation.

1. A very mild "sentinel" hemorrhage, in which the layer of SAH is not thick enough in any site to be shown on CT.

2. When there has been a delay of several days or more from the ictus. Subarachnoid blood undergoes hemolysis and its density decreases steadily in the days after the bleeding episode. Naturally, this is especially apt to result in a falsely negative scan after a mild hemorrhage and this is the very circumstance in which there may be delay in seeking medical attention.

3. Pyogenic meningitis or aseptic meningitis. Meningitis may have a fulminating onset and, of course, produces headache, meningism, altered consciousness and neurological signs. Expecially if the onset of the illness was unwitnessed, and the patient is too obtunded to describe it,

the differential diagnosis of SAH and meningitis may be difficult until the LP is done. When the history and signs suggest meningitis, with an onset over hours, perhaps after a prodomal illness, and when significant fever is present, LP should be done at once before CT scanning, even if CT scanning is readily available.

If CT scanning is not available on site, and the clinical diagnosis of SAH is made, there is a question of performing an LP primarily, or transferring the patient to a neurosurgical centre for diagnosis. This requires some judgement, but clear guidelines are available. An LP should be done at the primary hospital if:

1. Pyogenic meningitis is considered a likely possibility.
2. The patient is in good neurological condition, complaining of headache, with or without meningism, but with no depresssion of consciousness or focal signs.

LUMBAR PUNCTURE

Using a small LP needle (#22) when possible, three small specimens of CSF should be obtained. If there is a question of iatrogenic trauma, the red cell count in the first and last tubes is compared. A constant value suggests spontaneous SAH, while a precipitous drop from tube one to tube three suggests a traumatic tap. If more than 12 hours has elapsed since the ictus, xanthrochromia will be present in the supernatant, and one tube should be sent for prompt centrifugation. Several days after SAH, the specimen may be entirely xanthrochromic when obtained, rather than pink or bloody looking. If the fluid is clear and colourless, SAH is substantially excluded, but a cell count should still be obtained. Pleocytosis may indicate aseptic meningitis, explaining the patient's symptoms. Neurologically intact patients may be discharged home after a negative LP. They should be advised to recline in the car as they are driven home, and they should spend 24 hours resting in bed. Fluids are encouraged. The occasional post LP headache is a small price to pay to exclude a life threatening illness. If a hospital "holding area" is available, the patient may stay for 12-24 hours to recover from a negative LP, but this is not essential.

Contraindications to Lumbar Puncture

It is best to transfer the patient to a neurosurgical centre prior to LP under certain circumstances:

1. When the history and signs are unequivocal, with instantaneous onset, definite meningism, and perhaps subhyaloid fundal hemorrhages, a partial third nerve palsy, or a history of unconsciousness or seizure at the onset. In this setting, an LP simply is not necessary and the diagnosis can be confirmed by CT scan on arrival at the neurosurgical centre.

2. When there is a serious question of raised intracranial pressure (ICP) and a mass lesion, as suggested clinically by depression of consciousness or focal signs, such as pupillary abnormality or hemiparesis. The absence of papilloedema does not exclude raised ICP, especially in acute events such as SAH.

In doubtful situations, telephone consultation with a neurosurgeon should be sought, prior to LP or transfer. Then too, advice about intial resuscitative measures and precautions for transfer can be discussed.

TRANSFER

After your assessment and discussion with a neurosurgical centre, fully conscious patients should be given an injectable analgesic (eg. Codeine 60 mgm., IM) and sent by ambulance, calmly but promptly, to a neurosurgical centre. Ideally, a nurse should accompany the patient in transfer.

Hypertension

An injectable antihypertensive should be given if the systolic blood pressure exceeds 150 torr. Hydralazine 20 mgm., IM, is usually quite satisfactory and can be repeated every 1-2 hours as needed.

Airway

Drowsy and confused patients, still capable of intelligible speech, should be transferred in a semiprone ("park bench") position, and must be accompanied by a nurse with suction equipment available during transport. Unconscious patients should be intubated. Muscle relaxants and Pentathol may be used to minimize the technical difficulty and the stress to the patient inherent to intubation. Ideally a physician should accompany such patients in transfer.

Management of Raised ICP

Patients with markedly depressed consciousness and especially those who progressively deteriorate under observation should be intubated and transferred to a neurosurgical centre as quickly as possible. Hyperventilation to lower PC02 to 25-30 torr is recommended. If pupillary dilatation occurs, or focal paralysis steadily progresses, Mannitol should be administered. The 20% solution is preferred; the dose is 5 ml/kgm. or 350 cc.'s for a 70 kgm. patient. A urinary catheter is advisable.

Seizures

Seizures should be treated as in any other acute illness unassociated with SAH (see

Chapter 6). As always, airway protection and ventilatory support are crucial in uncontrolled seizures, especially if large quantities of depressant drugs are administered.

SUMMARY

The treatment of ruptured intracranial aneurysm has improved dramatically in recent years with advances in radiological diagnosis, anaesthetic and operative technique and perioperative management. If these techinical advances are to impact at all on the overall mortality and morbidity of this devastating condition, awareness of the diagnostic features must be increased through education of both the public at large and primary care physicians. Aneurysmal SAH frequently affects young productive members of society who are otherwise well, and its toll on society is, therefore, particularly costly. SAH is now universally accepted as a surgical illness and prompt recognition and referral to a neurosurgical centre is appropriate.

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CHAPTER 5:
COMA - AN OVERVIEW

Objectives:

1. To know how to resuscitate and assess, using the ANLS ABCDE, approach the unconscious patient.

2. To know how to distinguish metabolic/toxic causes of coma (medical) from structural causes (surgical) using bedside examination, laboratory and imaging procedures.

3. To know how to recognize and treat increased intracranial pressure.

THE ANLS APPROACH - A B C D E

Airway with c-spine control	-	clear and secure it, assess/stabilize (if trauma)
Breathing	-	assess and assist, give high contentration O2
Circulation	-	IV line and fluids
Drugs	-	Glucose and Thiamine, Narcan, when appropriate drug screen
Evaluate (and Expose)	-	rapid neuro assessment

Introduction

Consciousness is the product of the reticular activating system (RAS), a network of neurons extending from the brainstem up to the cerebral cortex. The brainstem portion of the RAS collects incoming neural impulses from the internal and external environments, and transmits them to the cerebral cortex, which it activates; the cortex interprets this neural data. Roughly the brainstrem RAS is the fact of consciousness; the cerebral RAS is the content of consciousness. In general therefore it requires an extensive lesion in the cerebrum to ablate consciousness, but only a small strategically placed one in the brainstem.

The neurons of the RAS may be damaged by three major mechanisms;

Structural lesions 1. an expanding **supratentorial** lesion which increases intracranial pressure and progressively displaces the brainstem downwards, telescoping and warping the brainstem.

2. a structural **infratentorial** (posterior fossa) lesion that directly compresses and/or damages the brainstem.

Metabolic Encephalopathies

3. **toxic or deficiency states** which impair the biochemical/electric functions of the RAS neurons.

Structural lesions account for 1/3 of all cases of coma and often call for the recognition and treatment of increased intracranial pressure and consideration of neurosurgical intervention. Metabolic encephalopathies cause 2/3 of all cases of coma, and involve detection and correction of underlying toxic and deficiency states.

THE ANLS APPROACH

A - Airway (with C-Spine control)

-clear it, inspect, finger sweep, suction.
-secure it, oropharyngeal airway until evident that coma may be prolonged - then intubation
-if any suspician of trauma, institute c-spine stabilization procedures - avoid hyperextension while intubating, stabilize with collar or sandbags, transfer precautions, radiology of c-spine when patient stable.

B - Breathing

-if cyanosis or respiratory distress persists after airway cleared, give ventilatory support (ambubag) + 100% O2.
-if "first pass" lab work (see below) shows hypoxia or hypercarbia, give ventilatory support + 100% O2.

C - Circulation

-start a secure IV with a large bore needle or cannula and keep open with NS drip (draw sample now for "first pass" lab - see below).
-if patient hypotensive, bolus immediately with 250 ml. NS and continue infusion until BP is stable and normal, while seeking blood/fluid loss or cardiac insufficiency.

D - Drugs

-unless blood glucose level is known immediately (Glucostat) to be normal or high, give 50 ml. of 50% glucose IV; for an adult and 25% glucose 2 ml/kg for a child. Be sure blood sample for glucose determination has been drawn first.

-if glucose given (or if patient is malnourished, suspected alcoholic) give Thiamine 100 mgm. by slow IV push to avert or treat Wernicke's encephalopathy.
-Narcan 2.0 mgm. IV, if no response within 5 minutes, repeat. Child's dose is 0.01 mgm/kg. Remember children may develop narcotic overdose from too much cough syrup.

Note: If patient fails to arouse following glucose, Thiamine and Narcan, consider the possiblity that no rapidly reversible cause of coma exists, and consider endotracheal or nasotracheal intubation now if not already done.

E - Evaluation

-"first pass" laboratory work is done on blood sample drawn when starting IV line (hemoglobin, WBC, differential, smear, ESR, BUN, creatinine, lytes, magnesium, calcium, glucose, PT/PTT, ethanol, osmolality), plus arterial blood (ph + gases) and urinalysis (including ketones).
-establish the level of consciousness by performing a rapid neurological exam; quantitate using Glasgow Coma Scale (Table I), monitor by repeated brief examinations (physician or nurse) the course of the coma (Table II).
-on the basis of the examinations, triage the comatose patient into one of three groups (see introduction):
1. an expanding supratentorial lesion (neurosurgical consult)
2. a structural infratentorial lesion (neurosurgical consult)
3. a toxic or deficiency state (medical management)

-further investigation and treatment is determined by the category into which the patient has been triaged.

NEUROLOGICAL EXAMINATION

1. Level of Consciousness

Drowsy - patient awake, eyes open, communicating without persistent stimulation.

Stuporous - patient unresponsive, eyes closed, but will rouse and communicate during (usually noxious) stimulation only to lapse back into unresponsiveness on cessation of stimulation.

Comatose - Lightly comatose - responds to stimuli by semipurposeful withdrawal movements of limbs +/- unformed vocalizations

Comatose-decorticate	responds to stimuli by flexion of upper limb(s) and extension of lower limb(s).
Comatose-decerebrate	responds to stimuli by extension of upper and lower limb(s)
Comatose-unresponsive	no response to noxious stimuli

GLASGOW COMA SCALE - TABLE I

Eyes Open	Spontaneously	4
	To Speech	3
	To Pain	2
	None	1
Best Verbal Response	Oriented	5
	Confused	4
	Inapprop. words	3
	Incompre. sounds	2
	None	1
Best Motor Response	Obey Commands	6
	Localize pain	5
	Withdrawal to pain	4
	Flexion to pain	3
	Extension to pain	2
	None	1

The sum of the patient's response in each of the three categories is the GCS (Glasgow Coma Scale) score.

Best possible score = 15
Worst possible score = 3

2. Pupils

-are they equal in size?
-do both pupils respond to light?

General Rules - constriction of pupils in response to light is preserved in metabolic encephalopathies with only the following exceptions: -extremely severe (usually lethal) encephalopathies

Specific drugs - Atropine + Atropinic substances, Glutethimide

- constriction of pupils in response to light is impaired then abolished in coma caused by expanding supratentorial lesions. If only one pupil is "fixed" (ie. unresponsive to light) the lesion is on the same side as the "fixed" pupil
- small (1 mm. diameter or less) pupils are seen in brainstem lesions ("pinpoint pontine pupils"). . . and in narcotic overdose. Therefore, pinpoint pupils demand Narcan.

3. Respiratory Pattern

General Rule - respiratory distress in a comatose patient is more often due to airway obstruction than to neurologic disease.

Cheyne-Stokes (periodic) respirations: waxing and waning of respirations in regular fashion due to lack of cortical and basal ganglia regulation of respiratory centres.

Centreal neurogenic hyperventilation: very rapid shallow (and therefore inefficient) respirations caused by loss of upper brainstem regulation of respiratory centres.

Shallow, slow, irregular respirations: caused by direct involvement of the respiratory centres in the medulla. Usually fatal.

4. Pulse and Blood Pressure

General Rules

- HYPOTENSION in a comatose patient should always prompt immediate administration of IV saline (250 ml. as a bolus, followed by continous infusion) and a search for blood loss. Hypotension due to brain disease is rare; when present it is an ominous prognostic sign.
- HYPERTENSION and BRADYCARDIA, the "Cushing response" to increased intracranial pressure, are uncommon and therefore not to be depended upon in terms of their absence ruling out increased intracranial pressure (ICP). When present they signify a severe increase in ICP.

Please see Table II.

5. Head

- examine for evidence of injury, including ear drums (blood behind drums signifies basal skull fracture) and retroauricular areas (blood behind ear, "Battle's sign", signifies basal skull fracture).
- if any clear fluid dripping from nose, check with Dextrostix. CSF contains sugar, nasal mucosa does not.
- check fundi for papilloedema.

6. Neck

General Rules

- EVERY HEAD INJURY is a neck injury until proven otherwise.
- ALWAYS CHECK for meningeal irritation by slowly flexing the neck.

COMA SYNDROMES

On the basis of the examination and on the changes in the findings while the patient is being observed, the patient is triaged into of three coma syndromes.

1. AN EXPANDING SUPRATENTORIAL LESION - such as subdural hematoma, extradural hematoma, intracerebral hemorrhage, massive cerebral infarct with edema, etc.

2. A PRIMARY SUBTENTORIAL LESION - such as brainstem infarct, a pontine hemorrhage, or lateral compression of the brainstem by a rapidly expanding cerebellar lesion such as cerebellar hemorrhage.

3. A METABOLIC ENCEPHALOPATHY - with dysfunction of reticular activating system caused by exogenous toxins (eg. drugs) endogenous toxins (eg. renal failure) or deprivation of essential substrate (eg. hypoxia, hypoglycemia). See Chapter 6 - Toxic/Metabolic Coma.

Expanding Supratentorial Lesions

These are characterized by a fairly orderly progressive and usually accelerating loss of function in a rostral caudal (north south) direction (see Table II) as the supratentorial lesion progressively telescopes the RAS (reticular activating system). The pupils usually undergo progressive loss of ability to constrict in response to light, and they enlarge, ultimately becoming fixed and dilated. A unilateral fixed dilated pupil means that the temporal lobe is being pushed through the tentorial notch, stretching the underlying oculomotor nerve and compressing the adjacent brainstem. Increased intracranial pressure (ICP) is the major pathophysiology.

Management -

1. A B C D E
2. Notify neurosurgeon
3. Control ICP as follows:
   - **1st** - intubate and ventilate at 20 ventilations per minutes (every 3 seconds); aim is to reduce PCO2 to 25-30mm. of mercury.
   - **2nd** - while above in progress, start IV Mannitol in a dose of 5 ML/Kgm. (eg. 350 ml. of 20% Mannitol, infused IV over 15-30 minutes for a 70 kg. patient) Catheterize patient and measure output to be sure that diureseis is occurring.
4. While above in progress, obtain CT scan of head. If CT not available, transfer immediately to appropriate facility while measures to reduce ICP are in progress.

Primary Subtentorial Lesions

These present with rapid or abrupt onset of coma, simultaneously with very small, but reactive, pupils ("pinpoint pontine pupils"), and with brainstem signs (such as skew deviation of eye). The prognosis is poor, since most primary subtentorial lesions are intrinsic brainstem lesions (eg. infarcts, hemorrhages) which destroy the RAS. However, a cerebellar hemorrhage may be correctable if neurosurgically treated without delay. The prototypical picture of a cerebellar hemorrhage is 1. sudden occipital headache, 2. vertigo, 3. rapidly deepening coma, 4. pinpoint pupils, 5. deviation of eyes away from lesion. Cerebellar hemorrhage cannot be excluded without a CT scan; therefore, treat all patients with the "primary subtentorial syndrome" aggressively.

Management -

1. A B C D E
2. Notify neurosurgeon
3. Control ICP with hyperventilation and Mannitol.
4. CT scan.

METABOLIC COMA

Please see Chapter 6 for details. This section only deals with recognition and life saving early treatment of metabolic coma. The hallmark of metabolic come is normal pupils even with deep coma. Exceptions to this rule are few (small pupils from Glutethimide and from severe overdose of Atropinic substances such as tricyclics, and from very severe hypoxia and hypoglycemia). Also typical of metabolic coma are, as the patient sinks into coma, the "optional extras" of delirium, tremor, asterixis, myoclonus, and/or seizures. Note that meningitis and subarachnoid hemorrhage

may present as metabolic coma, so always look for a stiff neck.

Management - 1. **A B C D E**
2. Further investigation and treatment as in Chapter 6.

SUMMARY

THE COMATOSE PATIENT

First: Save the life and save the brain

Airway - clear and secure it
with C-Spine Control

Breathing - assess and assist it use high concentration O2
Circulation - also, if trauma check c-spine
Drugs - glucose, thiamine and Narcan

Second: Triage the patient

- supratentorial mass lesion
- primary subtentorial lesion
- metabolic coma, by doing an

Evaluation of: -level of consciousness
-pupils
-respirations, pulse and BP

Third: Further investigation and treatment

Table 2 - The Course of Neurological Deterioration

GCS Score	Level of Dysfunction	Level of Consciousness	Motor Response	Pupils (in Supratentorial Expanding Lesions)	Respiration	Pulse/BP
15-7	Cortex and Cerebrum	Drowsiness ↓ Stupor ↓ Light Coma	Localizess pain ↓ Withdrawal ↓ Decortication	Normal ↓ Sluggish and Mid-size	Normal ↓ Cheyne-Stokes	Stable
7-4	Upper Brainstem	Deepening Coma	Decortication ↓ Decerebration	Unreactive + Enlarging	Cheyne-Stokes ↓ Central Neurogenic Hyperventil-ation	Stable
4-3	Lower Brainstem	Profound Coma	Decerebration ↓ Unresponsive	Fixed + Dilated	Failing	Unstable ↓ Failing

CHAPTER 6:
TOXIC/METABOLIC COMA

Objectives:

1. To know how to recognize and treat causes of metabolic coma.

2. To be able to resuscitate a patient with metabolic coma using the ANLS approach.

3. To be able to use appropriate laboratory investigations to diagnosie the causes of metabolic coma.

THE ANLS APPROACH - A B C D E

Airway	-	Obtain and Maintain
Breathing	-	Ensure adequate oxygenation and ventilation.
Circulation	-	Ensure adequate cardiac output to deliver oxygen and substrates.
Drugs	-	1. Check blood glucose (with sticks), if low administer 50 cc. of D50W. 2. If malnourished/alcoholic give Thiamine 100 mg., IV. 3. Give Naloxone (Narcan) 2.0 mgm., IV. If no or partial response consider repeating IV. May give more depending on clinical situation.

EVALUATION
CASE PRESENTATION

A 42 year old female presents unconscious with a Glasgow Coma Score of 6. She was last seen by friends 24 hours before, and although intoxicated at that time seemed otherwise well. She was found at home by friends unconscious, lying in bed with no evidence of trauma. She is brought to your emergency department by ambulance and on arrival the following is noted:

Airway	-	the patient's airway is partially obstructed until the insertion of an oropharyngeal airway.
Breathing	-	the patient has Kussmaul type breathing with a respiratory rate of 38. The patient appears pink well oxygenated on room air.

Circulation - the patient's pulse is 130 per minute and the blood pressure is 100/60. The patient appears well perfused clinically.

There is no evidence of trauma and the remainder of the physical examination is unremarkable. What would be your approach? What should be the initial treatment? What investigations should be performed?

Introduction

Unconscious patients frequently present to the emergency department. Often the patient has a life threatening complication of coma, such as an inadequate airway or hypotension. In order to deal correctly with the unconcsious patient, a systemic approach to treatment and diganosis must be employed.

Causes of Coma

Coma is divided into structural and non structural causes. Structural causes are those due to physical lesions and are associated either with increased intracranial pressure or disruption of the RAS. Metabolic coma is produced by neuronal dysfunction caused by either a deficiency in substrate or an excess of a toxin (which may be endogenous or exogenous). Metabolic causes are twice as common a structural causes of coma. (Refer to Table 1) Metabolic causes of coma can be classifed as follows:

1. Deprivation of substrated - ie. glucose, oxygen, thiamine.
2. Endogenous toxins - ie. liver, kidneys, endocrine.
3. Exogenous toxins - ie. sedatives, TCA's, alcohols, anticholinergics.
4. Metabolic abnormalities - ie. Na, K, Ca, Mg, P04.
5. CNS infection/inflammation - ie. meningitis, encephalitis, vasculitis.
6. Others - ie. post ictal, hypothermia, hyperthermia, substance withdrawal.

ANLS APPROACH

An unconscious patient presenting in the emergency department should be assessed and treated in the following sequence:

Airway with C-spine Control

Assessment of the patency of the airway by visual and tactile (finger sweep/laryngoscopic) examination is the first priority. If obstruction is present, correct it by either clearing away foreign material or with simple airway maneuvers (ie. jaw thrust, oral airway). Do not forget the possibility of cervical psine trauma in the unconscious patient - protect the cervical spine when appropriate. A patient

tolerates an oral or nasopharyngeal airway without gagging probably has insufficient hypopharygeal (gag) and laryngeal reflexes to protect the airway. Consider early endotracheal intubation in these patients.

Breathing

The rate and pattern of breathing should be observed. Hypoventilation may be seen and require ventilatory support. Regularly irregular or irregularly irregular breathing may give a clue as to the origin of coma or site of the lesion. It is important to support ventilation to prevent secondary brain injury from hypoxia and/or hypercarbia. Supplemental oxygen should be administered.

Circulation

Assess the state of the patient's circulation by determining the blood pressure, pulse rate and attaching an ECG monitor. Two intravenous infusions of normal saline should be initiated. If the patient is hypotensive a fluid bolus should be administered. Venous blood should be drawn for analysis (CBC, BUN, creatinine, electrolytes, Mg, Ca, blood glucose, prothrombin and partial thromboplastin time, osmolality, ethanol and a toxicologic screen - if appropriate). Arterial blood gases should be drawn. A urine specimen should be obtained (by catheter if necessary). Other laboratory investigations (ie. liver function tests) will be guided by the clinical assessment.

Drugs

Coma may be caused by hypoglycemia. Check the blood glucose using a chemical reagent strip (ie. Visidex, Chemstrip, Dextrostrip) with a drop of the patient's blood at the bedside. If the blood glucose is low, administer D50W 50 cc. IV. Also, give Naloxone 2.0 mgm., IV to assess for narcotic induced coma. In patients who are suspected of being alcohol abusers or malnourished, thiamine 100 mgm., IV should b administered.

Evaluation

A head to toe examination should be conducted. At the completion of the ANLS evaluation the following questions should be addressed:

1. What is the major category of dysfunction? (supratentorial, subtentorial, diffuse/metabolic).

2. If metabolic/diffuse, what is the abnormality?

To answer these question, a problem oriented examination is appropriated. This

complete exam will therefore emphasize the following:

A) Palpate head and check ears, nose and throat.
B) Level of consciousness - Glasgow Coma Scale
C) Rapid neuroassessment - motor response to voice or pain: evidence of lateralizing signs, tremor, asterixis, myoclonus.
D) Skin and oropharynx - dry or diaphoretic, flushed or pale, jaundiced or anemic, needle tracks, breath odor, (acetone, alcohol, pesticides)
E) Respiratory pattern
F) Cardiovascular status - blood pressure, heart rate and rhythm (ie. arrhythmias produced by TCA), a 12 lead EKG should be done.
G) Abdomen - bowel sounds absent or present, abdominal distention, ascites, hepatomegaly.
H) Muscoloskeletal - trauma, fasciculation (ie. organophosphates).

History

Unconscious patients may not be able to provide a useful history. However, much information can be gained from "Medic Alert" bracelets, wallet information cards, medication bottles, old hospital charts and information from friends, relatives, police, and ambulance personnel.

Special Investigations

At this point, you should have an indication as to whether the patient has structural or metabolic cause for coma. If a structural cause is postulated, then computed tomography (CT) of the head is appropriate. The interpretation of laboratory results should always include consideration of the three gaps - anion, osmolar and saturation. See Table III. An abnormality in any gap may point towards a specific metabolic aberrancy. If all metabolic indices from the simple investigations are normal and a toxicologic ingestion, inhalation or contamination is not suspected (based on the history and physical examination), a lumbar puncture may be considered to assess for CNS infection or inflammation. If the history and examination are non specific the patient should have a CT scan prior to the LP to rule out intracranial mass lesions and/or increased intracranial pressure. A stat laboratory toxicology screen should be available in most hospitals, consisting of ASA, acetaminophen, ethanol, carboxyhemoglobin and serum iron and TIBC levels. Some toxicologic tests will need to be required for toxicologic testing. Note that these results will not be helpful for at least 6-24 hours. If a toxic cause for coma is suspected, treatment to decrease toxin absorption, increase toxin elimination and administration of a specific antidote if available should be initiated. (Always check with Regional Poison Control for current methods of treatment).

Definitive Management

If a structural cause for coma has been diagnosed, urgent neurosurgical consultation is necessary. The treatment of diffuse/metabolic causes of coma is dependent on the etiology of the coma.

Summary

All patients presenting with coma require an urgent assessment and initiation of the ABCD(E) approach. The etiology of coma may be structural or metabolic. Diagnosis of the specific cause(s) of metabolic coma is necessary because treatment is etiology dependent. If a structural cause for coma is suspected, neurosurgical consultation must be obtained as soon as possible.

SUGGESTED FURTHER READINGS

1. Plum F., Posner, J., The Diagnosis of Stupor and Coma. Ed., 3, Philadelphia, 1980. F.A. Davis Co.

2. Huff, J.S., Coma Rosen, P.,ed.,Emergency Medicine: Concepts and Clinical Practice, Ed, 2, St. Louis, 1987. C.V. Mosby Co., pp. 249-271.

TABLE I

CAUSES OF COMA

STRUCTURAL (1/3 of Cases)

1. Supratentorial - (60%) Example: tumour, vascular accident, traumatic hematoma, contusion.

2. Subtentorial - (40%) Example: pontine hemorrhage/ infarct, basilar artery occlusion, cerebellar hemorrhage, tumour, traumatic hematoma.

DIFFUSE/METABOLIC (2/3 of Cases)

1. Endogenous - Example: uremia, heaptic encephalopathy, post seizure, meningitis, encephalitis, cofactor/vitamin deficiency, endocrine (thyroid, adrenal, pituitary).

2. Exogenous - Example: exogenous toxins.

TABLE II

PUPILLARY PATTERNS IN COMA

1. Small, equal reactive - metabolic coma.
2. Midposition fixed - midbrain lesion.
3. Dilated, Fixed - herniation with brainstem compression; NB; may be reversible.
4. Large, fixed, hippus/rhythm contraction - tectal lesion.
5. Pinpoint, reactive - pontine lesions, opiate overdose.

TABLE III

THE THREE GAPS

1. ANION GAP = NA - (CL + HC03)

   Normal = 12 - 15

   Abnormal > 15

   CAUSES OF INCREASED ANION GAP = A MUDPILE

A	ASA
M	Methanol
U	Uremia
D	Diabetic Ketoacidosis
P	Paraldehyde
I	Iron, INH
L	Lactic Acidosis
E	Ethylene glycol, Ethanol (if associated with a metabolic acidosis)

2. OSMOLAR GAP = measured serum osmolality - calculated serum osmolality

   = measured osmolality - [2(Na) + Blood Glucose + BUN]
   (equation correct for SI units only)
   Normal 10mOsm or less

   Causes of Increased Osmolar Gap

   ethanol
   methanol
   ethylene glycol
   isopropanol
   mannitol
   ketones

3. SATURATION GAP = measured Sa02 - Calculated Sa02

02 satuaration may be measured directly by the laboratoy or calculated (the

usual method) using Pa02 and the oxyheamoglobin dissociation curve.

Any saturation gap, at all, suggests that something is preventing the normal association of 02 and Hb. Causes: Carbon Monoxide, methemoglobinemia, sulfhemoglobinemia.

TABLE IV

TOXICOLOGIC SCREEN

NOT IMMEDIATELY HELPFUL - SIX TO TWENTY FOUR HOUR LAG MINIMUM BETWEEN SENDING SAMPLE AND RECEIVING RESULTS

Requires blood and urine.

Urine needed for:

- narcotics
- amphetamines
- cocaine
- TCA's
- phenothiazines
- barbituates
- chloral hydrate

Most tests are qualitative only, and many drugs are detected days after being taken (ie. results may be irrelevant).

CHAPTER 7:
STATUS EPILEPTICUS

OBJECTIVES:

1. To recognize the clinical syndrome of status epilepticus.
2. To understand the need for immediate aggressive treatment.
3. To know how to treat status epilepticus within 30 minutes of onset.

THE ANLS APPROACH - A B C D E

A - Airway	-clear it, secure it, position patient semiprone, try nasopharyngeal airway
B - Breathing	-assess and assist. Give high concentration 02
C - Circulation	-2 IV lines, normal saline
D - Drugs	-Diazepam 10 mg. IV over 2-3 minutes (repeat once PRN), Phenytoin 18 mgm. mgm/kg, IV, max 50 mgm/ minute, Glucose 50%, 50 ml. IV by slow push, Phenobarb 300 mgm., IV over 2-3 minutes; repeat once PRN, if still seizuring.
	-Drug levels (Dilantin). Draw blood for biochemistry.
E - Evaluate	-rapid neuro assessment
	-respiratory depression from above drugs
	-presence of continuing seizure activity

INTRODUCTION

About 0.5% of the general population are epileptic (ie. susceptible to recurrent seizures). Five per cent of all epileptics at least once in their lives suffer status epilepticus. The mortality rate of status epilepticus is 5-50%, but this can be reduced by prompt, aggressive and appropriate treatment.

Status epilepticus is defined as "a continuing seizure state of at least 30 minutes duration, produced either by one continuous seizure or by seizures repeated at so short an interval that normalcy is not regained between them.

The most common occurrence is in a known epileptic who has discontinued anticonvulsant medication. The next most common cause is an epileptic, on

medication, who has become subject to factors which lower seizure threshold such as fever, alcohol (intoxication or withdrawal) or stress. Less commonly, a cerebral lesion such as an infarct or a tumour may present de novo as status epilepticus.

A - Airway

It can be difficult to clear, secure, and maintain the airway in a convulsing patient. Start by turning the patient on his side; remove dentures, food, etc., from mouth (don't get bitten!), suction and insert orpharyngeal airway. If jaws are clamped shut, insert a nasopharyngeal airway (you will be taught this technique in the "hands on" sessions).

B - Breathing

Convulsive movements and spasm of the truncal muscles impair ventilation and increase the hypoxia (and hypercarbia) already present from upper airway obstruction. Give high flow 100% O2. Be alert for possibility of aspiration having occurred during or after seizure.

C - Circulation

Hypotension may occur with prolonged seizure activity in an individual with pre-existent cardiovascular disease; IV fluids may be necessary. All patients in status epilepticus require an IV line through which to give medication. The ideal is 2 lines, one in each arm. When starting IV, draw 25 ml. of venous blood; hold 10 ml. for subsequent anticonvulsant levels (send immediately of course, if stat levels available in your lab), and send the rest for stat glucose, electrolytes, calcium and creatinine. If there has been difficulty in getting the airway, or if patient is or has been in respiratory distress, send arterial blood for gases, etc., - but do this after the "stat drugs", Diazepam, Phenytoin, and Glucose have been given.

D - Drugs

Into one IV line, give by slow push, Diazepam 10 mgm., (adult dose) over 2-3 minutes (this dose is unlikely to depress respiratory drive, but watch out for it anyway). If seizure does not cease within 2 minutes of completing this injection, give another 10 mgm. by slow push over 5 minutes (watching for respiratory depression). If the status breaks after the Diazepam, as it will in 80% of cases, intubate the patient unless: 1. already intubated. 2. patient awakens promptly to point where a tube will not be tolerated.

Simultaneously with the administration of Diazepam and regardless of whether or not the status breaks with the Diazepam, give Phenytoin by repeated slow IV pushes, at a rate not exceeding 50 mgm./minute, to a full loading dose (18 mgm/kg - 1000 mgm. for a stevedore). Monitor EKG, if possible, during Phenytoin infusion to detect AV block which may be precipitated or intensified by high Phenytoin levels.

While Phenytoin is being given, flush the original "Diazepam line" with saline, give 50 ml. of 50% glucose IV, reflush line with saline, then keep line open with saline drip. Give Thiamine 100 mgm. IV with glucose if any suspicion of malnutrition or alcohol abuse.

Phenobarbital 300 mgm. IV at rate of 100 mgm/minute. If seizure has not stopped in 10 minutes, repeat the dose. Watch respirations and blood pressure and be prepared to support both, since the combination of a benzodiazepine and a barbiturate may depress both.

If Phenobarbital has not broken the status epilepticus proceed immediately to general anaesthetic.

Traditionally Halothane is used, but likely a deep barbiturate anaesthetic is preferable. Load with Pentobarbital 5 mgm./kg. and maintain with 1-3 mgm/kg/hr, to keep EEG monitor in "burst suppression" stage. While patient is anaesthetized, stabilize all metabolic parameters (body temperature, acid-base, fluid and electrolyte balance) and allow patient to wake up. Extubate when tube is no longer tolerated, but leave IV in. Give maintenance anticonvulsants, eg. Phenytoin 200-400 mgm/day PO or IV **or**
Phenobarb 200-600 mgm/day PO or IM.

E - Evaluate

About 80% of the "statuses" are broken by the time the Diazepam, Phenytoin and Glucose have been given; these patients should be transferred to an area that will permit close but quiet observation. Upon recovering consciousness, they are started (or re-started) on maintenance doses of anticonvulsant medication (which can be determined, in part using the anticonvulsant levels drawn on admission).

If status is not broken -

1. Patient is in serious trouble; best to alert your anaesthetist now
2. An endotracheal tube must be inserted even if it entails transiently paralyzing the patient with a short acting neuromuscular blocking agent such as Pancuronium (further medication necessary may depress respirations to the patient where an endotrachea tube will become essential).
3. Further drug must be given in an attempt to stop seizure

SPECIAL CONSIDERATIONS

1. Use of Lorazepam rather than Diazepam

   -No problem. Lorazepam is said to have a longer duration of action and a lesser incidence of respiratory depression. The dose is 0.05 mgm/kg given IV over 2 minutes as a slow push, to a maximum of 4 mgm. If the status fails to break, this dose may be repeated in 10-15 minutes. Maximum dose is 8 mgm. in 12 hours

2. The patient with "no veins" in whom an IV line cannot be established

   -Clearly, a cut down is necessary and should be started as soon as one of the following medications is given intramuscularly in an attempt to stop the seizure:
   -Paraldehyde 5 ml. in each buttock (total = 10 ml.) for an average sized adult - use glass syringe, and optimal IM technique.
   OR -Phenobarbital 300 mgm IM for an average sized adult.

3. Children in status epilepticus

   -IV line available: Diazepam 0.1-0.2 mgm/kg, then Phenytoin 15 mgm/kg.
   -IV not available: rectal Paraldehyde 2-5 ml. "Cut" 50/50 oil, OR Phenobarb 5-10 mgm/kg IM, OR rectal Diazepam 0.1-0.2 mg/kgm.

SUMMARY

The morbidity and mortality of status epilepticus can be reduced by prompt, aggressive and appropriate treatment.

CHAPTER 8:
THE DIZZY PATIENT

Objectives:

1. To be able to discern whether the totally non specific symptom of "dizziness" is due to vestibulolabyrinthine disease (vertigo) or to global insufficiency of cerebral blood flow (presyncope/syncope).

2. To know when vertigo or presyncope/syncope is due to life threatening disease.

3. To know how to intervene to reduce morbidity and mortality of dangerous diseaseswhich present as "dizziness" (vertigo or syncope).

THE ANLS APPROACH - A B C D E

A - Airway - clear it, secure it
B - Breathing - assess and assist, give 100% O2
C - Circulation - IV line (fluid and medication)
treat hypotension
stabilize any cardiac dysrthythmia (monitor)
D - Drugs - antivertigo agents (Dimenhydrinate suppository or IM Trifluoperazine)
E - Evaluate - specific cause of vertigo or syncope
1. vertigo - inner ear (benign)
brainstem (cerebellar hemorrhage, vertibro-basilar insufficiency etc.)
2. syncope - seek ominous causes
heart, hypovolemia, severe anemia

INTRODUCTION

Dizziness - is a totally non specific and therfore unhelpful term used by most patients to denote a sense of unsteadiness and insecurity. Some dizzy patients have vertigo or syncope; most do not. Dizziness is seldom dangerous, but occasionally is a symptom of life threatening disease.

Vertigo - is a false sense of movement of the self or the surroundings produced when a malfunctioning vestibulolabyrinthine system (inner ear, eighth cranial nerve, vestibular nucleus in brainstem)

feeds false information to the cerebral cortex (temporal lobe). Most vertigo comes from inner ear disease and, although unpleasant and frightening to the patient , is benign. Occasionally vertigo comes from brainstem disease, which almost always is ominous. Distinction between brainstem (central) and inner ear (peripheral)vertigo is a crucial diagnostic consideration.

Syncope - is a transient loss of consciousness casued by a transient decrease in global brain blood flow (perfusion). "Presyncope" is a state of faintness or light headedness caused by a decrease in global brain blood flow insufficient to produce unconsciousness. Most people with presyncope and syncope have benign disorders of circulatory regulation which lead to intermittent peripheral systemic vasodilatation, pooling of blood in the periphery, transiently decreased venous return, and transiently diminished cardiac output. Since such peripheral vasodilatation is usually induced by neurogenic impulses, this benign syncope is called "vasovagal syncope". Ominous causes of syncope are heart disease with primary impairment of cardiac output, severe volume depletion(shock) and very severe anemia.

DIAGNOSIS

Vertigo - the essence of vertigo is a sense of movement of self or surroundings. Autonomic features such as nausea, vomiting, sweating and tachy or bradycardia may accompany both central (brainstem) and peripheral (inner ear) vertigo. Peripheral vertigo, which is much the more common type, may be associated with cochlear symptoms such as hearing loss or tinnitus. Central vertigo may be associated with CNS symptoms such as double vision, paralysis, limb ataxia or sensory loss. If nystagmus is present, its characteristics may tell whether the accompanying vertigo is central or peripheral: peripheral nystagmus is always either horizontal or rotatory and is "direction fixed"; that is, the quick phase or the "beat", is always to the same side regardless of the direction of gaze. Central nystagmus may be horizontal, rotatory, vertical, or disconjugate (ie. eyes not lined up), and is "gaze directed"; that is, the quick phase is in the direction of gaze and changes with the director of gaze. Please see Table I.

Syncope - the key features of syncope are: 1. alteration of consciousness,

either at present or in the past, 2. hypotension, either overt or compensated. Important points regarding hypotension are: a) it may be relative; a patient whose customary BP is 220/120 may faint at 120/80; b) if supine BP is normal, check for postural hypotension; a drop of 20 mm. Hg systolic suggests cardiovascular instability, with many possible causes, some of which may be ominous.

The heart may be the cause of hypotension. Look for evidence of disease which may critically impair cardiac output: 1. cardiac arrhythmias; 2. acute myocardial infarction; 3. severe valvular stenosis, especially aortic; 4. cardiac tamponade; 5. massive pulmonary embolism. Hypovolemia (shock) may present with syncope. (Unless there is compelling evidence of primary heart disease, a bolus of 250 ml. of normal saline is an excellent diagnostic challenge test). Very severe anemia may present as syncope despite a normal (or above normal) blood volume and blood pressure; anemia of this degree is usually clinically obvious if looked for, and certainly will be evident on the initial lab work.

VERTIGO

Benign Causes -
- Meniere's Syndrome
- Vestibular Neuronitis
- Positional Vertigo

Ominous Causes -
- Cerebellar Hemorrhage
- Brainstem Ischemia
- Post fossa Tumour
- Multiple Sclerosis

SYNCOPE

Benign Causes -
- Vasovagal Syncope
- Reflex Syncope

Ominous Causes -
- Heart -
  - Arrhythmias
  - M.I.
  - Valve Stenosis
  - Tamponade
  - Pulmonary Embolism
- Hypovolemia
- Severe Anemia

A - Airway

Patients with syncope seldom have much difficulty with their airway. Acutely vertiginous patients often are nauseated and frequenly vomit; they tolerate poorly any oropharyngeal or endotracheal airway - and yet, the vomitus with either central or peripheral vertigo, and the bulbar palsy that may accompany some forms of central vertigo, may make clearing, securing and maintaining the airway a life saving step.

B - Breathing

Patients with syncope, unless in severe heart failure, seldom have much difficulty with their ventilation. Vertiginous patients with brainstem lesions frequently have impaired respiratory drive due to damage to the respiratory centres. This damage may not be apparent at the time of presentation and may progress, thus, careful monitoring if respiratory function is essential. Oxygen is seldom indicated in vertiginous patients (which is just as well since they tolerate masks and nasal catheters poorly); but O2 is indicated in patients whose syncope is due to heart failure and anemia. If in doubt, run it until your arterial blood gases return (ABG's should be done in any syncopal patient).

C - Circulation

Patients with vertigo who have been nauseated (with poor fluid intake) or have vomited may be dehydrated, and thus require an IV line. Syncopal patients are by definition hypotensive, either actually or potentially and require an IV line.

Blood pressure, if normal in the supine position, should be repeated with the patient in the sitting and (if sitting normal) standing position; postural hypotension may indicate barely compenstated circulation due to volume depletion or heart disease. In a syncopal patient, close monitoring of BP is essential, as is monitoring of cardiac rate and rhythm. Unless there is clear evidence of heart failure, the finding of supine or postural hypotension calls for a bolus of 250 ml. of normal saline, followed by continuous infusion of saline. Urinary output should be followed, if necessary with an indwelling catheter.

D - Drugs

It is permissable and will allow better access to the patient to give during the ABC stages, an anti-vertigo agent. The mildest drug is a Dimenhydrinate (Gravol) suppository 100 mgm., a stronger one is Trifluoperazine (Stelazine) 2 mgm. IM. If extrapyramidal side effects occur from the Trifuluoperazine, control with either Diphenhydramine (Benadryl) 25-50 mgm. IV or Benztropine (Congentin)1 mgm. IV.

DISTINGUISH BETWEEN VERTIGO AND SYNCOPE

To this point, the basic resuscitative treatment, the ABCD's, have been the same, regardless of whether the patient is "dizzy" because of vertigo or because of syncope or presyncope. Beyond this point, however, investigation and treatment are different for vertigo than they are for syncope, and it becomes essential now to make the distinctions.

This is usually easy. The nauseated, sometimes vomiting patient, complaining bitterly of "the room going round", and often with evident nystagmus, clearly has vertigo. The lethargic, sallow patient with low supine or sitting blood pressure, complaining of "faintness" likely has had presyncope or syncope; historical or physical evidence of heart disease, blood or fluid loss, or anemia will support the suspicion of syncope.

E - Evaluate (Specific Cause)

For the vertiginous patient who is deemed to have brainstem disease (on the basis of other neurological findings and of vertical or disconjugate nystagmus), do an unenhanced CT scan of the head with "post fossa cuts" to demonstrate or rule out cerebellar or brainstem hemorrhage. Cerebellar hemorrhage is a neurosurgical emergency (see Chapter 4). Brainstem hemorrhage has no successful specific therapy. A normal CT scan implies a diagnosis of either brainstem ischemia or MS; other data, including age of the patient, may assist in narrowing the diagnosis. If CT scanning is unavailable the patient with suspected brainstem vertigo should be stabilized and moved as quickly as possible to a facility with a CT scan. All other diagnostic procedures (except MRI) are a poor second best in this clinical situation.

For the syncopal patient, investigation is aimed at demonstrating or ruling out cardiac disease, shock and severe anemia. This investigation includes: 12 lead EKG; continuous cardiac monitoring; chest x-ray; echocardiogram stat if there is any suspicion of tamponade; arterial blood gases; and venous blood for CBC, enzymes, 'lytes, calcium, BUN and creatinine, stool for OB and sometimes NG tube to check for blood.

SUMMARY

While vertigo and syncope are usually benign, they may uncommonly be produced by serious disease: brainstem infarct, brainstem hemorrhage, and cerebellar hemorrhage may cause vertigo; and heart disease, hypovolemia, and severe anemia may cause syncope. The problem is rendered uncertain by the fact that many vertiginous or syncopal people know only one word to describe their symptoms ..... "dizzy".

Manage the dizzy patient, as all sick patients, by **ABCD**.

Then, using history and examination, distinguish between syncope and vertigo. Patients who clearly are vertiginous may benefit from antivertigo drugs such as Gravol or Stelazine.

Evaluate other parameters in history and examination to decide, in the vertiginous patient, whether the underlying disease is benign (inner ear) or ominous (brainstem); and in the syncopal patient, whether the cause is benign (vasovagal) or ominous (heart disease, hypovolemia, anemia).

Follow with specific management.

TABLE 1

CENTRAL VS. PERIPHERAL VERTIGO

	Cochlear Symptoms	CNS Symptoms	Nystagmus
Central	-	+	Any direction Gaze-directed
Peripheral	+	-	Horizontal or rotatory Fixed direction

CHAPTER 9:
ACUTE CEREBROVASCULAR DISEASE

Objectives:

1. To recognize the diagnostic implications of "the stroke syndrome".
2. To know how to stabilize the patient with an acute "stroke".
3. To know the specific management of cerebral infarction and cerebral hemorrhage.

THE ANLS APPROACH - A B C D E

A - Airway	-clear it, secure it
B - Breathing	-assess and assist, high concentration O2
C - Circulation	-IV line, correct hypotension and serious arrhythmias assess hypertension
Control	-increased intracranial pressure, seizures and metabolic disturbances.
CT Scan	-to help
D - Decide	-whether stroke (cerebrovascular) or pseudostroke (tumour, SDH etc.,) and if stroke, whether hemorrhagic or ischemic
E - Evaluate	-need for early medical (eg. Heparin) or surgical (eg. cerebellar heamatoma) treatment

INTRODUCTION

Stroke - is a non specific term that denotes the rapid or acute onset of a focal neurological deficit (such as a hemiplegia, or an aphasia) caused either by ischemia of brain tissue or hemorrhage into brain tissue. Rapidly evolving focal neurological deficits are not always "strokes" (though too many physicians automatically assume that they are); sometimes a space taking lesion such as a neoplasm or a subdural hematoma may have an "apoplectiform onset". Stroke is a syndrome, and not a disease.

Ischemia - of the brain may produce a variety of clinical pictures depending on the location of the ischemia (Table A) and the stage of the ischemia (Table B).

Ischemia - of the brain may have many causes (Table C). It is important to know the cause as precisely as possible, because different causes may demand different treatment.

Hemorrhage into the brain may be related to hypertension, but may occur in people with normal blood pressure. Uncommon causes of hemorrhage include ruptured berry aneurysm, arteriovenous malformation, angiopathies, and bleeding disorders. Contrary to popular misconceptions, not all hemorrhages are large, and not all hemorrhages spread into the subarachnoid space. Thus, in some hemorrhages, there may be no alteration of consciousness and no headache, and the situation may mimic exactly a cerebral infarct. The only way to distinguish with confidence between hemorrhage and infarct is a CT scan.

The immediate management of the patient with "the stroke syndrome" (acute onset of focal neurological deficit) is the same whether stroke or pseudostroke, hemorrhage or ischemia.

A - Airway

-this is particularly important where:
1. consciousness is obtunded eg. brainstem lesion, large hemorrhages
2. there is dyphagia eg. brainstem ischemia (risk of aspiration)
3. there might be increased intracranial pressure eg. large hemorrhage

B - Breathing

-this is particularly important where:
1. consciousness obtunded
2. possibility of depressed respiratory drive as in brainstem lesions or increased intracranial pressure

-100%oxygen given if patient is in respiratory distress after airway cleared and secured and ventilation deemed adequate.

C - Circulation

-intravenous line; keep open with NS; when starting IV draw blood for CBC (incl. ESR, lytes, BUN, creatinine, glucose, cardiac enzymes)

-assess effectiveness of cardiac output
1. if hypotension, bolus with 250 ml. NS and check for causes including blood loss and silent MI

2. stabilize cardiac arrhythmias: eg. Digoxin for atrial fibrillation with rapid ventricular response.

-defer lowering high blood pressure (unless extreme, eg. >220/120) until sure that situation is not a cerebral infarct with marginally perfused brain tissue (loss of autoregulation in ischemic zone leads to a fall in regional cerebral blood flow that parallels fall in perfusion pressure, or put simply, lowering the blood pressure may enlarge the infarct and worsen the stroke)

C - Control

-the following, which may complicate strokes or pseudostrokes:

1. increased intracranial pressure: likeliest in large intracerebral hemorrhages; earliest evidence usually impaired consciousness; check for pupillary changes (See Chapter 4)
2. seizures: occur in about 4% of the stroke syndrome; in true strokes seen with cortical infarcts; in pseudostrokes seen with tumors.
3. metabolic disturbances: eg. acute neurological events may cause hyponatremia.

CT Scan

-every patient with "the stroke syndrome" needs a CT scan in order to:

1. differentiate pseudostrokes (tumours, subdurals) from true strokes
2. distinguish between hemorrhage and infarction

- hemorrhage readily apparent on CT from time of stroke onwards; ask for an unenhanced CT so that contrast will not be mistaken for blood.
- infarcts usually do not show up on CT scan until they mature, usually a matter of 48-72 hours. However, always do a CT early since a normal CT in a "stroke patient" is good evidence of infarct

-demonstrate cerebral edema around an infarct, a hematoma, or a tumour (as in pseudostroke), thus alerting the physician to the probable need, at present or in the near future, to control increased intracranial pressure;
-show "surgical hemorrhages" such as a cerebellar heamatoma.

D - Decide

-whether stroke or pseudostroke and, if stroke whether hemorrhage or infarction/ischemia. This is the key dividing point in the "decision tree", since from this point, treatment, heretofore common to hemorrhage, infarction/ischemia and pseudostroke, now diverges widely into specific therapy for each entity. Note, especially when contemplating the use of anticoagulants (Heparin or Coumadin), that the only certain way to distinguish between hemorrhage and infarction in many patients is a CT (or MRI) scan.

E - Evaluate

-need for early specific therapy of ischemia/ infarction or heamorrhage.

SPECIFIC MANAGEMENT

ISCHEMIA/INFARCTION

1. What is the role of **anticoagulants** in a patient with acute ischemia/infarction of the brain?

-since the action of both platelet antiaggregators eg. ASA and anticoagulants eg. Coumadin is very slow in onset, it is the role of **intravenous Heparin** that is being considered.

-there is no evidence that Heparin will lyse clots that have already formed, thus, the action of Heparin is to prevent the formation of further clots, thereby reducing the risk of further stroke.

-Indications for Heparin	-	cardiogenic cerebral embolism stroke-in-evolution (perhaps)
-Contraindications to Heparin	-	hemorrhage in the brain (CT) or elsewhere failure to have a CT scan of brain a very large infarct (with a large area of ischemic vessels and therefore increased chances of bleeding into infarct)
-Heparin dose	-	bolus with 5,000 units, and give continuous drip of 800-1000 units per hour, following PTT's daily.

2. What measures are useful in reducing peri-infarctional cerebral edema causing increased intracranial pressure?

-corticosteroids are not useful because this is a cytotoxic edema

-hyperventilation and Mannitol are useful.

3. What is the role of cerebral vasodilators in ischemia/infarction?

-contraindicated, because they cause generalized cerebrovasodilatation, stealing blood from the ischemic area.

4. What is the role of surgical or medical (eg. TPA) disobliteration of occluded cervical or cerebral blood vessels?

-presently felt to be contraindicated because of risk of causing bleeding into infarct, though some small recent TPA series suggest that this risk is not as great as once believed.

5. What is the role of surgical decompression of the brain in ischemia/infarction?

-very limited, though a few patients with an infarcted cerebellar hemisphere who, through post-infarct edema are compressing their brainstem, may benefit (see section below on cerebellar hemorrhage).

Cerebral Hemorrhage

1. What are the guidelines for reducing blood pressure in acute cerebral hemorrhage?

-when blood pressure is so high that it is causing cardiac decompensation it should be lowered rapidly regardless of any other consideration.
-in other situations, elevated blood pressure should be lowered slowly since it may be produced at least in part by a "Cushing effect" - ie. reflex increase of BP in the face of increased intracranial pressure in an attempt to maintain brain perfusion.

2. What measures are useful for reducing intracranial pressure in brain hemorrhage?

-corticosteroids are useful because the edema is vasogenic; note, however, that onset of action will be delayed for many hours
-hyperventilation and/or Mannitol are useful
-surgical removal of some hematomas is occasionally life saving (see below)

3. When is surgery indicated?

-the major indication is for a relatively uncommon entity (< 10% of all brain hemorrhages), cerebellar hemorrhage. Bleeding into the cerebellar hemisphere causes direct pressure on the brainstem (subtentorial coma - see Chapter 5) with obstructive hydrocephalus and increased intracranial pressure. Clinical picture: usually a hypertensive individual with rapid onset of vertigo, nausea and/or vomiting, headache, unsteadiness and deteriorating level of consciousness; abnormal signs include, often, deviation of eyes, small pupils, facial weakness, stiff neck, and long tract signs such as upgoing toe(s). Surgical removal of the cerebellar hematoma relieves the brainstem compression and the hydrocephalus and can be life saving.
-a very few "lobar hemorrhages" - eg. hemorrhages into the anterior portion of the temporal lobe, or frontopolar or occipitopolar hemorrhages may respond to surgical extripation if they are causing serious increase in intracranial pressure.

4. Is there presently a role for "pro-coagulants"?

-no, <u>but</u> there is a demonstrated bleeding tendency such as may occur in hamophilia, leukemias, or Coumadin therapy, appropriate specific measures should be instituted.

SUMMARY

The early management essential for <u>all</u> patients with "the stroke syndrome" (acute onset of a focal neurological deficit) is Airway, Breathing, Circulation, control increased intracranial pressure, seizures, and metabolic disturbances; and perform a CT scan.

The clinical aspects, and more particularly the CT scan, will permit distinction between strokes (acute focal deficits due to cerebrovascular disease) and pseudostrokes (acute

focal deficits due to tumours, subdural hematoma, etc.,) in the case of strokes, the CT will distinguish between hemorrhage and infarction/ischemia.

Two varieties of ischemic strokes (cardiogenic embolism and stroke-in-evolution) warrant intravenous Heparin therapy. Cerebellar hemorrhages may respond to surgical removal of the hematoma.

ACUTE CEREBROVASCULAR DISEASE

TABLE C

SOURCE OF ISCHEMIA	FREQUENCY AS STROKE CAUSE	USUAL CLINICAL PRESENTATIONS	TREATMENT IMPLICATIONS
EMBOLIC			
- ARTERY TO ARTERY	COMMON	T.I.A./INFARCT	ASA
-CARDIOGENIC	COMMON		COUMADIN
ARRHYTHMIA (ESP.A.FIB.)		MOSTLY INFARCTS	
VALVULAR (MITRAL,AORTIC)		SOMETIMES PRESENT	
CARDIOMYOPATHY (INCL.AMI)		WITH T.I.A.'S	
HEMODYNAMIC			
-ARTHEROSCLEROTIC OCCLUSION	UNCOMMON	INFARCT	?SURGERY
-ANGIITIS, DISSECTION ETC.	RARE	INFARCT	SPECIFIC TREATMENT

CHAPTER 10: MENINGITIS

Objectives:

1. To recognize the symptoms and signs of meningitis in the pediatric and adult populations.

2. To be able to resuscitate the acutely ill patient with acute bacterial meningitis, including instituting appropriate antibiotic therapy within 1/2 hour.

3. To know the indications and contraindications for a lumbar puncture.

THE ANLS APPROACH - A B C D E

Airway - Assess/ secure

Breathing - Ensure adequate oxygenation and ventilation

Circulation -
1. Ensure adequate cardiac output
2. Resuscitate with IV fluid and pressure agents if in septic shock.

Drugs -
If unconscious:
1. Check blood glucose (Dextrostix). If low administer 50 cc.'s D50W.
2. If malnourished/alcoholic give Thiamine 100 mg. IV
3. Administer Narcan as appropriate 2 mgm.,I.V.

-
Specific for meningitis:
1. Emergency antibiotic therapy for bacterial meningitis guided by age and history/physical.
2. LP to be done first if appropriate.

INTRODUCTION

Central nervous system infections include a spectrum from acute bacterial meningitis, aseptic meningitis, and encephalitis to brain abscess. Individuals with encephalitis or brain abscess can present with an altered level of consciousness and/or focal neurological symptoms and signs; emergencies arising from these conditions (ie. status epilepticus, coma), are covered in other sections. Acute bacterial meningitis represents a true medical emergency and requires both a rapid diagnosis and the

appropriate institution of treatment without delay.

ACUTE BACTERIAL MENINGITIS

Acute bacterial meningitis is a medical emergency; the prompt recognition of the symptoms and signs of meningitis, coupled with the appropriate resuscitation, will reduce the morbidity and mortality of this serious disease. The presentation and management of a patient with meningitis is markedly different for the paediatric and adult population; these two groups of patients will therefore be discussed separately.

THE PAEDIATRIC PATIENT

Neonatal Meningitis

Neonatal meningitis is defined as occurring within 30 days of birth. The diagnosis is frequently difficult, as the initial signs and symptoms are vague and non specific, consisting of poor feeding, poor activity, apneic spells, respiratory distress or jaundice and at later stages, convulsions, coma and cardiovascular collapse. Neck stiffness, fever, and bulging of the anterior fontanelle are usually absent, and a strong index of suspicion is necessary in the neonate who appears "unwell" and who demonstrates the signs outlined above. The differential diagnosis of the neonate presenting with such vague signs and symptoms includes sepsis, cytomegalovirus infection, toxoplasmosis, systemic herpes, hepatitis, and cardiovascular and pulmonary disease (congenital and acquired). Neonates may be considered to be at higher risk of contracting meningitis following prolonged rupture of membranes, maternal systemic infection or a premature or traumatic delivery; birth history may therefore, be of benefit not only with respect to behaviour of the neonate but also with respect to the perinatal period.

The majority of cases of bacterial meningitis in this age group are caused by group B streptococci and gram negative organisms. The initial treatment of the neonate with meningitis is resuscitation; this will include the administration of oxygen, starting of an intravenous line, and monitoring of the neonate with an ECG monitor. The acutely ill neonate, with meningitis, or other suspected systemic sepsis, should receive antibiotics emergently and excessive delay should not be taken in performing a lumbar puncture or pursuing other investigations. When appropriate in the stable patient, especially when the diagnosis is uncertain, a lumbar puncture should be performed prior to the administration of antibiotics, but excessive delay is always inappropriate.

Figure 1 outlines the appropriate management of the acutely ill patient with meningitis. The neonate in septic shock should receive oxygen, intubation and ventilation as

appropriate, crystalloid resuscitation (bolus of 10 ml/kg of normal saline repeated times three as necessary) followed by pressor agents and colloid as necessary, and emergent antibiotic therapy. The initial antibiotic therapy for the neonate with meningitis is outlined in Tables I and II. Always consult early on with a paediatrician and neurologist when these illnesses are suspected. Remember that hypoglycemia and hypothermia may complicate the disease state.

Childhood Meningitis

Childhood purulent meningitis (over 1 month of age), is primarily caused (95% of cases) by Haemophyllus influenzae type B, meningococcus, pneumococcus, and less frequently streptococcus A. The peak age incidence is between 6 and 12 months of age. In infants and children the signs and symptoms will depend primarily on the age of the child; the younger the child the less specific the clinical picture will be. The hallmark of CNS infection in the older child and adult is the presence of fever, headache, meningismus and an altered level of consciousness. History may reveal preceding evidence of an upper respiratory tract infection, headache in the older child or irritability in the infant. Seizures, vomiting or in later cases ocular motor paralysis may provide a diagnostic clue. Meningismus is typically absent under the age of 1 year. In older infants meningismus may be diagnosed by the presence of neck stiffness. The most common organisms according to patient age are outlined in Table I. In acute situations when an organism is not yet identified, appropriate antibiotic Cefotaxime therapy may be Ampicillin plus Cefotaxime. It is important to add Chloramphenicol to this regimen of treatment as the incidence of Ampicillin resistent Haemophyllus influenzae is increasing and presently greater than 25%. If a lumbar puncture is performed and gram stain reveals evidence of meningococcus the drug of choice is Penicillin G; individuals allergic to Penicillin may receive Chloramphenicol. An alternative antibiotic in this age group is a third generation Cehpalosporin (Cefuroxime sodium, Cefotaxime sodium, Ceftriaxone sodium); although first and second generation cephalsporins penetrate the CSF poorly, third generation cephalosporins diffuse well across the inflammed blood brain barrier, and have been proven to be efficacious for childhood meningitis. The patient presenting with acute meningococcal septicemia, will be discussed below in the section on adult meningitis.

ACUTE BACTERIAL MENINGITIS IN THE ADULT

The diagnosis of acute bacterial meningitis in the adult is usually simpler compared to the paediatric patient, with the important exceptions of the elderly, the alcoholic and immunosuppressed ,and the partially treated patient. Approximately 30-90% of individuals with bacterial meningitis will have an altered level of consciousness, compared to 25-50% of patients presenting with aseptic meningitis; this will of course depend on the time of presentation. Distinguishing features between bacterial and viral meningitis are outlined in Table III; this distinction will frequently be impossible

clinically and the definitive diagnosis will be made by analysis of spinal fluid. The most common complaints with bacterial meningitis are headache, meningeal irritation (stiff neck) and fever. Headache is usually a prominent symptom. A thorough history should focus on predisposing conditions (splenectomy, shunt, neurosurgical procedure, previous head trauma with basilar skull fracture), contact with individuals with meningitis (especially important for meningococcus) preceding infections (searching for a potential source of infection) and present antibiotic treatment (to identify partially treated patients). It should be noted that no single symptom or sign is considered classic and patients will present with various permutations and combinations. Note should again be made of the elderly, immunosuppressed, alcoholic and partially treated patients who may present atypically and have fewer "classic" symptoms and signs; a high index of suspicion is necessary for the patients.

The examination of a patient with suspected meningitis attention should also be focused on the presence of a skin eruption. A petechial or purpuric eruption is suggestive of meningococcemia, although similar eruptions may also be seen with Haemophyllus influenzae, Staphylococcus, or Streptococcus peneumoniae.

In an individual with acute presentation (Figure 1), therapy should be undertaken within 30 minutes. If there is evidence of raised intracranial pressure, with papilloedema or focal neurological deficit, a CT scan should be performed prior to a lumbar puncture. For these paitents antibiotics should be started prior to time consuming investigations. If there is no contraindication to performing a lumbar puncture, it should be performed rapidly and antibiotics started on the basis of suspected organism based on age and presentation. In an individual with a subacute presentation antibiotic therapy should never be delayed beyond 2 hours following the patient's presentation to the emergency department.

Note: A recent review of 135 cases of acute community acquired bacterial meningitis revealed a mean duration 4.9 hours (adult: n = 14) and 2.1 hours (paediatric: n = 121) between arrival of patients in the emergency department and the administration of antibiotics. Overall mortality was 5% for the childhood cases and 43% for the adult cases. "Prompt institution of antimicrobial therapy for acute meningitis. . . remains a major challenge for Emergency Physicians." (Reference: Promptness of Antibiotic Therapy in Acute Bacterial Meningitis. Ann Emerg Med, 1986; 15: 544-547).

INVESTIGATIONS

The most critical investigation for the patient presenting with suspicion of acute bacterial or viral meningitis, is a lumbar puncture. A neurological examination prior to performing a lumbar puncture will reasonably exclude the possibility of raised intracranial pressure. One should be certain prior to performing the procedure that neither a bleeding diathesis or local infection at the site of lumbar puncture are

present.

When performing a lumbar puncture, there are a number of measurements which should be made; these include the opening pressure, the protein content, the glucose content, cell count, gram stain and culture and sensitivity. Special stains and cultures may be performed as indicated. The specific findings in the CSF in the setting of bacterial, viral, and tuberculous meningitis are outlined in Table IV. Two further investigations which may be undertaken on the spinal fluid are Counter Current Immuno-electrophoresis, or more commonly Latex Agglutination. These two immunological procedures are most useful in the setting of a partially treated meningitis, and will be further discussed during the lecture.

MENINGOCOCCEMIA

In either the paediatric or adult population, patients with meningococcal meningitis can present with fulminant disease. These individuals may present with septic shock and frequently the presence of a petechial or purpuric rash. It is important to note that this rash is not specific for meningococcemia and may also be present with other organisms. It is critical that resuscitation be started early for these patients. All of these individuals will receive supplemental oxygen and two large bore intravenous lines should be started. All of these individuals will receive crystalloid resuscitation, as necessary, and the administration of antibiotics. The intial antibiotic therapy for adult patients should be Penicillin; children may receive Ampicillin and Cefotaxime because of a similar presentation with the other childhood organisms. Controversy exists about the relative contribution of steroids. Stress replacement doses (100 mg. Hydrocortisone, q8h) should be given if one suspects Waterhouse-Friderichsen Syndrome*. A lumbar puncture should not be performed until the patient is hemodynamically stable and thrombocytopenia and a consumptive coagulopathy are excluded. Organisms can be identified in these patients by direct smear of the skin lesions, by microscopic examination of the buffy coat (gram stain) or by microscopic or indirect immunological testing done on urine.

*Waterhouse-Friederichsen Syndrome is characterized by vomiting, diarrhea, extensive purpura and circulatory collapse caused by bilateral adrenal hemorrhage and secondary adrenal crisis.

VIRAL MENINGITIS

The clinical distinction between viral and bacterial meningitis can at times be difficult or impossible. This should be clarified with a lumbar puncture. Some of the clinical features distinguishing the two entities are discussed in Tabe III. The CSF findings in individuals with viral meningitis are summarized in Table IV. Individuals with partially treated meningitis may have a CSF profile similar to that of viral meningitis; the Latex

Agglutination and or CCI may be useful to distinguish these two groups.

SUMMARY

The focus of this chapter has been on the recognition and management of the individual with acute bacterial meningitis. These patients require a prompt diagnosis and the institution of appropriate treatment. A high index of suspicion is necessary in peadiatric, immunosuppressed, alcoholic, elderly and partially treated patients. Acutely ill patients with purulent meningitis, require rapid aggressive resuscitation in order to reduce both mortality and long term morbidity.

TABLE I

MOST COMMON ETIOLOGIC AGENTS OF ACUTE BACTERIAL MENINGITIS

AGE	ORGANISM	TREATMENT
0 - 4 weeks (Neonate)	Group B strep E. Coli	Ampicillin 200 mg/kg/day Q6H IV +
	Other Gram Neg.	Gentamycin 7.5 mg/kg/day
	listeria	Q 12h IV
4 years	S. Pneumonia	Ceftriaxone* 250 mgm
4 years - Adult	N. Meningitis	and Ampicillin 1 gm for Listeria

*Check with Regional Infectious Diseaess Unit as dose and type change often.

SPECIAL SITUATIONS

Splenectomy	S. pneumonia H. influenzae
CNS Shunt	S. aureus
Alcoholic/Immunosuppressed	S. pneumonia L. monocytogenes
Post Neurosurgery/CSF Leak	S. pneumonia S. aureus/epidermitis Gram negatives

TABLE II

UNKNOWN ORGANISM

4 WEEKS- 3 Months	Ampilcillin 200 mg/kg/day Q6H IV + Cefotaxime*
Beyond 3 months	Ceftriaxone* (based on suspicion may wish to add Ampicillin to cover Listeria)
4 WEEKS- 3 Months	Cefotaxime 200 mg /kg/day Q6H
Beyond 3 months	Ceftriaxone 100 mg /kg/day Q12H

DOSAGES

AMPICILLIN FOR NEONATES

< 2 kg / 0-7 days old
100 mg/kg/day - Q12H

2 kg / > 7 days old
150 mg/kg/day - Q8H

≥ 2 kg / 0-7 days
150 mg/kg/day - Q8H

> 7 days
200 mg/kg/day - Q6H

GENTAMYCIN FOR NEONATES

≤ 1 kg
3.5 mg/kg/day - once daily
< 37 weeks & > 1 kg
5 mg/kg/day - Q12H
≥ 37 weeks & ≤ 7 days old
5 mg/kg/day - Q12H
≥ 37 weeks & 7 days old
7.5 mg/kg/day - Q8H

INFANTS AND OLDER CHILDREN

AMPICILLIN	200-300 mg/kg/ day - Q6H
1 MOS. - 3 MOS. CEFTRIAXONE	200 mg/kg/day - Q6H
> 3 MOS. CEFTRIAXONE	100 mg/kg/day - Q12H

ADULTS

CEFTRIAXONE	FIRST DOSE - 250 mg.
AMPICILLIN	FIRST DOSE - 1 gm.

TABLE III

DISTINGUISHING CLINICAL FEATURES OF BACTERIAL AND VIRAL MENINGITIS (ADULT)

	BACTERIAL	VIRAL
Acute Presentation (symptoms < 24 hrs)	25%	5%
Headache	Prominent	Prominent
Meningeal signs	80%	60 - 70%
Fever	Common 80%>38.9 C	Common 30-40%>38.9 C
Alteration Mental Status	80 - 90%	25 - 50%
Seizures	30%	5%
Focal Neurological Deficit	50%	<10%

FIGURE 1

APPROACH TO THE ACUTELY ILL PATIENT WITH SUSPECTED MENINGITIS

ACUTELY ILL PATIENT WITH:

Meningococcemia or Septecemia

Resuscitate
1. 02 large bore IV's crystalloid/colloid/ pressor agents.
2. antibiotics appropriate for age
3. consider stress dose of Solucortef (100 mgm. IV)

No lumbar puncture until coagulation parameters and platelet count adequate and patient hemodynamically stable

Resuscitate (ABCDE's) as appropriate

Antibiotics to be started within 30 minutes

No clinical evidence of ICP and hemodynamically stable

LP/treatment as appropriate (treatment not to be delayed beyond 30 minutes)

Clinical evidence raised intracranial pressure

1. treat raised ICP as appropriate
2. start antibiotic as appropriate for age group
3. arrange for CT scan prior to LP

CT not suggestive of ICP

Lumbar puncture-if no contraindication

CT suggestive of ICP

Treat for ICP
continue antibiotic - Rx
Neurosurgical -
Neurological referral

TABLE IV

CSF FINDINGS WITH MENINGITIS

PARAMETER MEASURED	NORMAL ADULT	NORMAL NEONATE/ INFANT<6 M	BACTERIAL	VIRAL	TB
Opening Pressure (mg/1)	15+/-3	15	mean > 30	slightly elevated	
Protein (mg/1)	250-550	<900	>1500	<1000	<1000
CSF/serum glucose	0.6	0.8	<0.4	0.6	<0.4
Cell count (cells x 10 / 1)	<3 mononuclear	<30 (60%:PMN 40% Mono)	>500 (pred PMN)	#<100 (mono pred)	<200 (mono pred)
Gram stain	neg	neg	(+)70%-90%	neg	(-) posi AF stain
CIE/Latex Agglutination	neg	neg	(+)*	neg	neg

*Dependent on causative organism

#Initially PMNS - (Later Lymphocytes)

Note: If blood is present in CSF, subtract one white blood cell and subtract 10 mg/l. protein from totals for each 1000 RBC's.

ADDENDUM
UPPER AIRWAY MANAGEMENT WITH C-SPINE CONTROL

Airway: Demonstrate, then ensure that everyone successfully completes all aspects of the station. Hook an Ambu-bag to the right mainstem bronchus to simulate breath sounds for blind intubation.

GOALS

1. Recognize upper airway obstruction.
2. Outline the methods of treating upper airway obstruction.
3. Demonstrate C-spine immobilization.
4. Demonstrate methods of intubation.

AIRWAY OBSTRUCTION - THE RECOGNITION METHODS AND MANAGEMENT.

Step 1: Determine if the patient can speak. If the patient can speak, this guarantees upper airway patency.

Step 2: Look to see if there are obvious signs of upper airway obstruction - these may be respiratory effort, moving the patient's hands towards his neck, or agitation.

Step 3: Listen for sounds of upper airway obstruction. These are usually respiratory noises - i.e., stridor, snoring.

Step 4: With your ear on your hand, listen/feel around the nose and mouth for air movement (in the deeply comatose patient, it is often difficult to tell on looking at the patient whether there is actually air moving).

TREATMENT:

If you have identified signs of upper airway obstruction, before proceeding any further with the examination, these must be treated. If there is a question, or any suspicion, of C-spine injury, the patient must be protected during the efforts to maintain the airway. The patient has usually been brought and deposited in a supine position. It is best to maintain this supine position during the initial evaluation. The main principle of C-spine immobilization is not to extend the neck while opening the upper airway.

Step 0: This is preparing for patients who are going to need treatment for airway obstruction. Before the arrival of patients who are going to need emergency management of the upper airway, it is imperative to check the emergency cart for vital tools. Oral pharyngeal airways of different sizes should be available. Tonsil suction should be within easy grasp. Oxygen masks for 100%

oxygen ready to go. Endotracheal tubes of different size, Laryngoscope with a light that works and blades of different sizes should be prepared. Ensure that O2 is connected and have a back-up suction in case the original one blocks.

If the size and sex of the patient is known, it is prudent to choose your oral pharyngeal airways and endotracheal tubes, ie., open the appropriate endotracheal tube ahead of time, check the cuff, lubricate the tube and insert the stylet if you are going to use one. Position yourself at the head of the bed so that with all the confusion and excitement on arrival of the patient, you are in position to check airway, maintain C-spine and monitor pulse. The emergency cart should be within easy reach, the suction and oxygen beside you, and the monitor, ie., cardiac monitor usually beside or behind you. With the proper placement of this cluster of instruments, you will still have sufficient room to manoeuvre and to manage the airway.

Step 1: Move the mandible forward, pulling the tongue from the posterior pharynx. This can be done with a chin lift or a jaw thrust. If you have your hands on either side of the patient's head, in and around the patient's ear, the jaw can be moved easily forward while maintaining C-spine control. The chin can also be pulled forward taking care not to extend the head.

Step 2: Suction any secretion, vomitus and remove any foreign bodies, i.e., dentures, etc. from the oral pharyngeal airway.

Step 3: Insert the oral pharyngeal airway to maintain the upper airway patency. This is also a test of the gag reflex. Note: If it has been obvious, to this point, that the patient's level of consciousness will not tolerate an oral pharyngeal airway, i.e., as there is clenching of the jaw, incomprehensible sounds, verbal sounds from the patient, or any other clues to suggest that this patient is not comatose without a gag reflex, do not force the oral pharyngeal airway. Look and listen to determine if there is patency of the airway. An option here, used by some individuals, is the nasal pharyngeal airway. It is not commonly used, but is useful in status epilepticus.

Step 4: Listen with your stethoscope to the anterior chest while looking for chest movement. Determine if air is moving in and out of the chest. If not, you are dealing with an apneic patient and you must proceed to step 5.

Step 5: Endotracheal intubation: In the apneic patient, while you are thinking and preparing for endotracheal intubation in the apneic patient, use your Ambu Bag and mask. Obtain a good seal in and around the mouth. Again, guard against the extension of the head that could compromise the C-spine. and ventilate the patient with **100%** oxygen and then proceed with endotracheal intubation. The difficult part is that you must maintain some C-spine control. Therefore, you may need to delegate some help in maintaining this. Instruct your helper to stand a little to one side of you with his/her hands gripping the head and neck, in and around both ears, pulling gently in a longitudinal fashion, so that the head is not extended on the spine when you intubate. Proceed then with oral endotracheal intubation. Give 10 squirts of xylocaine spray to the pharynx to prevent raised ICP. This is a good time to use the stylet as it is more difficult to perform this kind of intubation with the head extended. Remember that the objective is to obtain the airway in the apneic patient, but that an intact spine in a dead patient is of no use. It may be necessary to sacrifice some extension in order to obtain the airway. This is not a place for nasal tracheal intubation (in an apneic patient) and there is not time for needle cricothyrotomy or surgical cricothyrotomy.

NASOTRACHEAL INTUBATION IN THE BREATHING PATIENT

a) Check the length of the tube that will be required before insertion.

b) Lubricate the nostril (remember one is often bigger than the other).

c) Place some lubricant and/or xylocaine jelly on the end of the endotracheal tube).

d) If you are going to use a stylet, insert it at a right angle to help pass through the hypopharynx (don't let it protrude beyond the ET tube.

e) Proceed with nasal tracheal intubation, remembering again that C-spine position cannot be compromised. The tube is inserted using air movement in the tube as a guide for placement.

Check position by auscultating chest and stomach and do chest x-ray.

SUMMARY:

A brief summary will be given at the end of the workshop.

1. Prepare yourself for airway-compromised patients.

2. Perform the basic manoeuvres to determine patency of the airway.

3. Use a chin left and protect the C-spine.

4. Use an oral pharyngeal airway to maintain the upper airway.

5. Remember in the apneic patient that oral endotracheal intubation is a preferred method.

6. Nasal tracheal intubation is the preferred method in a patient who is breathing and is in need of airway maintenance (unless the C-spine has been cleared) to C7-T1.

7. Check the position always of the endotracheal tube, by auscultating the chest and stomach and re-x-ray the chest, (particularly in children) to check the position. (In children and thin, small patients, auscultation of the chest is not a reliable method of determining whether one has intubated a mainstem bronchus).

8. Remember, vomiting is always a possibility in the comatose patient (especially on intubation) and be prepared to handle this on an emergency basis.

9. Maintain the C-spine at all times. Initially, with some in-line traction, then use sand bags and tape and use the preferred method of obtaining the lateral C-spine x-ray.

EQUIPMENT:

2 Adult Intubation heads, 1 infant
2 ET tubes
1 Stylet
2 Ambu Bags
5 Oropharyngeal Airways of Varying Size
5 Nasopharyngeal Airways of Varying Size
Tonsil Suction
Stethoscope
2 - 10 cc Syringes
2 Laryngoscopes (Adult)
1 Laryngoscope (Infant)