# Management of head and neck injuries A review

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## Abstract:

Head and neck injuries are the most common type of injuries that result in death or comatose in a patient. The management of head and neck injuries in pre hospital and hospital settings have evolved in the past century. For effective management of head and neck injuries a clear knowledge of various causes and mechanisms of head and neck injuries is essential. Timely diagnosis and surgical intervention can prevent catastrophical results. This review focuses on the causes and management of head and neck injuries and how to prevent them.

Keywords: Head, neck, injury, management

# I. Introduction:

Head and neck injuries are associated with high mortality and morbidity rates not only in India, but all over the world. One percent of all deaths in are attributed to head injury; up to 85% of all severely head-injured patients remain disabled after 1 year. Even after apparently mild head injury, nearly 50% of patients have moderate or severe disability 1 year later and of which only 45% return to full functional activity. Therefore for both economic and individual reasons small improvements in the management of head and neck injured patients may have a great effect on outcome. [1]

The neck is protected posteriorly by the spine, the head superiorly, anteriorly by the larynx and trachea, and inferiorly by the chest, but the most exposed parts are the lateral sides of the neck. Neck injuries or cervical spine injuries are of many types ranging from mild to severe. An injury to the neck can be caused due to a trauma to the area,, an accident, or even due to the degenerative changes that occurs in the spine. [2]. Since all the parts of the neck are connected, few emergencies pose as greater threat as a single penetrating neck injury can cause a multi organ system failure. A neck injury can cause laceration of the major vessels like the carotids, leading to haemorrhagic shock.

Approximately one million patients present to the hospital suffering from a head injury in a year. The vast majority of these patients have minor (GCS 13--15) or moderate injuries (GCS 9-12). In adults, young

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people (15--29 yrs) involved in road traffic accidents (responsible for approximately 50% of head injuries) and elderly people involved in domestic accidents. [3]. Overall, males are 2--3 times more susceptible to have a head injury than females. This article mainly focuses on the sign and symptoms of head and neck injury and the methods by which they can be diagnosed and managed. [4]. The management of patients with multiple trauma has not being discussed.

## **Classification of head injury:**

Head injuries may be classified in different ways for example, according to the nature of the insult the injury can be a penetrating injury or a blunt injury. Head injuries can also be closed or open. Its called a closed injury when the skull is intact and the injury hasn't reached the brain. An open head injury or a penetrating head injury is when the skull is open or damaged and is not intact resulting in injury to the brain. Head injuries can also be isolated or can also present with multiple trauma. Although there are many methods present in evaluating the severity of a head injury the Glasgow Coma Score[GCS] is the most common method.[1,9].

Head injuries can be primary which occurs at the site of accident or trauma. Secondary Brain Injury refers to the cascade of physiological and biochemical events that occur after primary injury and worsen outcome. [10].

By mechanism of injury

- blunt and penetrating
- Blunt trauma can be of high or low velocity (eg, motor vehicle crashes, falls, and blunt assault.)

## By morphology: [11]

• Basal fractures have an associated risk of CSF leak.

• Clinical symptoms (eg, raccoon eyes, Battle's sign, otorrhea, and rhinorrhea) should increase the index of suspicion in identifying basal skull fractures.

- Extradural
- Subdural
- Intracerebral

# The Glasgow Coma Scale [GCS]: [1,2,12]

The Glasgow Coma Scale is based on a 15 point scale for estimating and categorizing the outcomes of head injuries; the test measures motor response, verbal response, eye opening with the following values:

	I.	Motor			Response			
6	_	Obeys		commands	fully			
5	_	Localizes	to	noxious	stimuli			
4	_	Withdraws	from	noxious	stimuli			

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3	-	– A	Abnormal	flexio	1,	i.e.	decorticate		posturing
2	-	– E:	xtensor	response	2,	i.e.	decerebrate		posturing
1 - 1	No respo	nse							
	II.			Verbal				Response	
5		_		Aler	Alert and			Oriented	
4		_	Confus	ed,	yet	t coherent,			speech
3	_	Inappropriate	e words	and	jumbled	phrases	consisting	of	words
2			_	Incomprehensible			sounds		
1 - 1	No sound	ls							

Opening	Eye			III.	Ι
opening	eye		Spontan	_	4
speech	to	open	Eyes	_	3
pain	to	open	Eyes	_	2
					1 17

1 – No eye opening

The final score is determined by adding the values of I+II+III.

This number helps medical practitioners categorise the four possible levels for survival, with a lower number indicating a more severe injury and a poorer prognosis:

## Mild (13-15):

Mild brain injuries can result in temporary or permanent neurological symptoms and a neuroimaging tests such as CT and MRI may or may not show evidence of any damage.

## Moderate Disability (9-12):

- Loss of consciousness greater than 30 minutes
- Physical or cognitive impairments which may or may resolve
- Benefit from Rehabilitation

#### Severe Disability (3-8):

• Coma: unconscious state. No meaningful response, no voluntary activities

#### Vegetative State (Less Than 3):

- Sleep wake cycles
- Arousal b, but no interaction with environment
- No localized response to pain

## **Persistent Vegetative State:**

• Vegetative state lasting longer than one month

# **Brain Death:**

- No brain function
- Specific criteria needed for making this diagnosis

The main limitation of Glasgow Coma Scale is that there are possibilities that a patient is intoxicated, or may be under influence of drugs, or simply has low glucose level. Such factors leads to inaccurate scores on GCS.

## Classification of neck injuries: [13].

Zone 1: from the level of the clavicles and sternal notch to the cricoid cartilage

Important structures include the aortic arch, proximal carotid arteries, vertebral arteries, subclavian vessels, innominate vessels, lung apices, oesophagus, trachea, brachial plexus and thoracic duct

Zone 2: from the cricoid cartilage to the angle of the mandible

Important structures include the common, internal and external carotid arteries, the jugular veins, larynx, hypopharynx and proximal oesophagus

Zone 3: from the angle of the mandible to base of skull

Important structures include the internal carotid artery, vertebral artery, external carotid artery, jugular veins, prevertebral venous plexus and facial nerve trunk

Zone I is treated like thoracic injury. Anterior neck area classification anterior to posterior border of sterno cledo mastoid; posterior neck is not further divided. Often patients have multiple wounds or gun shot wound (GSW) tract can involve multiple zones. Superficial wound does not correspond well to deeper structures injured.

## Signs and symptoms of neck injuries: [14].

## Signs of laryngeal or tracheal injury:

- Voice alteration
- Haemoptysis
- Stridor
- Drooling
- Sucking, hissing, or air frothing or bubbling through the neck wound
- Subcutaneous emphysema and/or crepitus
- Hoarseness
- Dyspnoea
- Distortion of the normal anatomic appearance

- Pain on palpation or with coughing or swallowing
- Pain with tongue movement
- Crepitus: Noteworthy in only one third of cases. [14,15].

# Signs of pharyngeal and oesophageal injury: [14].

- Dysphasia
- Bloody saliva
- Sucking neck wound
- Bloody nasogastric aspirate
- Pain and tenderness in the neck
- Resistance of neck with passive motion testing
- Crepitus
- Bleeding from the mouth or nasogastric tube

## Signs of carotid artery injury:

- Decreased level of consciousness
- Contra lateral hemi paresis
- Haemorrhage
- Hematoma
- Dyspnoea secondary to compression of the trachea
- Thrill
- Bruit
- Pulse deficit

# Signs of jugular vein injury:

- Haematoma
- External haemorrhage
- Hypotension

## Signs of spinal cord or brachial plexus injury:

- Diminished upper arm capacity
- Quadriplegia

- Pathologic reflexes
- Brown-Séquard syndrome
- Priapism and loss of the bulbocavernous reflex
- Poor rectal tone
- Urinary retention, fecal incontinence, and paralytic ileus
- Horner syndrome
- Neurogenic shock
- Hypoxia and hypoventilation

# Signs of cranial nerve injury:

- Facial nerve (cranial nerve VII): Drooping of the corner of the mouth
- Glossopharyngeal nerve (cranial nerve IX): Dysphagia (altered gag reflex)
- Vagus nerve (cranial nerve X, recurrent laryngeal): Hoarseness (weak voice)

• Spinal accessory nerve (cranial nerve XI): Inability to shrug a shoulder and to laterally rotate the chin to the opposite shoulder

• Hypoglossal nerve (cranial nerve XII): Deviation of the tongue with protrusion. [14,15,16].

# Signs and symptoms of head injury:

Common symptoms of a minor head injury include:

- a headache
- light headedness
- a spinning sensation
- mild confusion
- nausea
- temporary ringing in the ears. [17].

The symptoms of a severe head injury include many of the symptoms of minor head injuries. They can also include:

- a loss of consciousness
- seizures
- vomiting

- balance or coordination problems
- serious disorientation
- an inability to focus the eyes
- abnormal eye movements
- a loss of muscle control
- a persistent or worsening headache
- memory loss
- changes in mood
- Leaking of clear fluid from the ear or the nose.

## Actiology of head injury:

Generally, head injuries can be caused by two methods, namely, blows to the head in case of trauma or an accident or by shaking. Head injuries that occur due to shaking are common in children but they can also occur in other scenarios where there is violent shaking.

Head injuries caused by a blow to the head are usually associated with:

- motor vehicle accidents
- falls
- physical assaults
- Sports-related accidents. [18].

# Types of head injury:

## Hematoma

A hematoma is a collection, or clotting, of blood outside the blood vessels. It can be very serious if a hematoma occurs in the brain. The clotting can cause pressure to build inside your skull, which can cause you to lose consciousness or result in permanent brain damage.

## Haemorrhage

A haemorrhage is uncontrolled bleeding. There can be bleeding in the space around your brain, which is a subarachnoid haemorrhage, or bleeding within your brain tissue, which is an intracerebral haemorrhage.

Subarachnoid bleeds often cause headaches and vomiting. The severity of intracerebral haemorrhages depends on how much bleeding there is, but over time any amount of blood can cause pressure to build.

## Concussion

A concussion is a brain injury that occurs when your brain bounces against the hard walls of your skull. Generally speaking, the loss of function associated with concussions is temporary. However, repeated concussions can eventually lead to permanent damage.

## Oedema

Any brain injury can lead to oedema, or swelling. Many injuries cause swelling of the surrounding tissues, but it's more serious when it occurs in your brain. Your skull can't stretch to accommodate the swelling, which leads to a build up of pressure in your brain. This can cause your brain to press against your skull.

#### **Skull fracture**

Unlike most bones in your body, your skull doesn't have bone marrow. This makes the skull very strong and difficult to break. A broken skull is unable to absorb the impact of a blow, making it more likely that there will also be damage to your brain.

## Diffuse axonal injury

A diffuse axonal injury, or sheer injury, is an injury to the brain that doesn't cause bleeding but does damage your brain cells. The damage to the brain cells results in them not being able to function and can also result in swelling, causing more damage. Though it isn't as outwardly visible as other forms of brain injury, diffuse axonal injury is one of the most dangerous types of head injuries and can lead to permanent brain damage and even death. [19]

# **II.** Pathophysiology of head, neck injury:

Mechanism of injury:

Head injury:

--Blunt injury

- Motor vehicle collisions
- Assaults
- Falls

## --Penetrating injury:

- Gun shot wounds
- Stabbing
- Explosions

#### Neck injury:

**Blood vessel trauma:** 

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## --Blunt trauma:

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Serious haematoma

## --Laceration:

- Serious exsanguinations
- Entraining of air embolism

#### Airway trauma:

Tracheal rupture or dissection from larynx

Airway swelling and compromise.

## **Cervical spine trauma:**

## --Vertebral fracture:

- Paresthesia, anaesthesia, paresis, or paralysis below the level of injury.
- Neurogenic shock may occur

## Other neck trauma:

## --Subcutaneous emphysema:

- Tension pneumothorax
- Traumatic asphyxia

## --Penetrating Trauma:

- Oesophagus or trachea
- Vagus nerve disruption
- Thyroid and parathyroid glands. [20].

## Monitoring of a severe head injury in an adult patient:

In patients with a severe head injury, additional monitoring may be helpful in management, particularly to guide the timing of repeat scans and neurosurgical intervention. The benefit of the additional monitoring modalities in terms of mortality or morbidity is unclear at the present time.

# ICP (Intra Cranial Pressure) :

The BTF guidelines suggest that there are inadequate data to make ICP monitoring a treatment standard. However, they suggest the following guideline: Intracranial pressure monitoring is appropriate in patients with severe head injury with an abnormal admission CT scan. An abnormal CT scan of the head is one that reveals haematomas, contusions, oedema or compressed basal cisterns. ICP monitoring may be used to guide therapies to limit the increase in ICP or to allow calculation and maintenance of a cerebral perfusion pressure (CPP). Patients with severe head injury and a high ICP have a poorer prognosis than those with a normal ICP. The critical ICP at which action is taken to limit further increase is not clear and varies between 15 and 30 mm Hg. However, 20 mm Hg seems to be accepted as the treatment threshold by a large number of authorities. However, it is possible that the combination of ICP and MAP is more important than the ICP

alone. Thus, CPP may be a more appropriate measure (and target) than ICP, where CPP is taken as MAP – ICP. ICP measurement has never been subjected to a randomized double-blind study, and to do so would be extremely difficult. However, there is a substantial body of evidence that suggests that it helps in early detection of mass lesions (e.g. epidural or subdural haematomata), may limit the indiscriminate use of therapies to control ICP (which in themselves may be harmful) and may be helpful in determining prognosis. ICP may be monitored from various sites using a variety of devices. A solid-state intra parenchymal monitor is associated with a reduced risk of intracranial infections. This is not true of an intraventricular catheter; however, this will allow withdrawal of cerebrospinal fluid (CSF) and thereby provide an alternative method of ICP control. Subdural or epidural catheters have also been used but carry the risk of infection without the potential benefits of CSF aspiration. The preferred site for the ICP monitoring device is the right frontal lobe (non-dominant hemisphere, minimal essential brain tissue). However, this may or may not be the tissue involved in the head injury, and interpretation of pressure readings may be difficult if the monitor is sited in the middle of an expanding contusion.

# III. Managementof patients with higher ICP:

## Medical:

#### **Positioning:**

The patient should be nursed in a head-up (30) position to improve venous drainage and reduce ICP. In order to do this, it is essential to be certain about the integrity of the spine; good working protocols for early clearance should be in place.

#### Sedation and neuromuscular block:

Deep sedation (to Ramsay score of 6 or equivalent) is used to reduce cerebral metabolism. In some units, neuromuscular blockers are used as standard; in others, they are used when ICP remains difficult to control, all other medical measures are in place and there is a concern that muscle activity may be contributing to the pressure. If the ICP remains difficult to control, a thiopental infusion may be used in conjunction with electroencephalographic (EEG) monitoring to bring about burst suppression. Once this is achieved, other sedation agents can be withdrawn.

# Ventilation and carbon di oxide control:

The objective of mechanical ventilation is to maintain PaCO2 at 4--4.5 kPa. If ICP becomes dramatically increased, short-term hyperventilation may be used to gain control while other measures (e.g. mannitol) take effect.

## Mannitol and osmolality management:

An increase in serum osmolality will result in a tendency to decrease brain tissue water and hence decrease ICP. A serum osmolality of 300--310 mosm is targeted in our unit, achieved by incremental 100 ml doses of mannitol 20%

#### Seizure control:

Both clinical and sub clinical seizures may have dramatic effects on cerebral metabolism and ICP; they should be prevented. In patients receiving neuromuscular blocking drugs or in whom sub clinical seizures are suspected, EEG monitoring may aid detection of the fits.

### Temperature control and induced hypothermia:

For reasons given above, an increase in body temperature to more than 37 C should be actively avoided. Induced hypothermia remains contentious and there is conflicting evidence as to whether it affects outcome. There is some evidence that below 35 C brain tissue oxygenation may be impaired, but generally there is agreement that cooling will result in a decrease in ICP.

#### Surgical:

#### **CSF drainage:**

If hydrocephalus is demonstrated on CT scan in a patient with increased ICP, CSF drainage will usually decrease this pressure. In situations in which hydrocephalus is not demonstrated, great care must be exercised. In many patients, the ventricles will be flattened and further supratentorial CSF drainage is not possible. Lumbar drainage of CSF may be dangerous and should only be performed following neurosurgical advice.

#### **Crainectomy:**

A bifrontal decompressive craniectomy may be performed to allow the brain tissue to expand and decrease the ICP. This technique has not been studied in a randomized trial, although scattered reports in the literature suggest that it may be beneficial.

#### Labectomy/removal of contusion:

Either lobectomy or removal of contusion may be possible surgically, depending on the nature and location of the brain injury and whether is midline shift may be exacerbated by removing non-dominant tissue. Again, there is little evidence in terms of improved outcome to support this.

#### **Diagnosis for neck injury:**

## Arteriography:

Gold standard for vascular injury

Diagnostic & therapeutic Zones I & III difficult to assess clinically Zones I & III often involve complex surgery

No arterial injuries on arteriogram if normal exam Cost-effective for zones I & III Decreased surgery rates to 5% in zone I & 13% in zone III.

## Management of neck injury:

Initial orderly assessment, using the Advanced Trauma Life Support protocol as developed by the American College of Surgeons, is appropriate in any trauma. This protocol includes rapid assessment of the "A, B, Cs" of trauma. Accordingly, airway management is the first priority in penetrating neck trauma.

# a. Airway Management:

• Approximately 10 percent of patients present with airway compromise, with larynx or trachea injury. While endotracheal intubation may be performed in these patients, nasotracheal intubation, cricothyroidotomy, or tracheostomy may be required in the presence of spinal instability.

To avoid air embolism, the patient should be supine or in Trendelenburg's position.

• Direct pressure without indiscriminate clamping should be used to control active hemorrhage in the neck.

• Deeply probing open neck wounds below the platysma muscle should be avoided in the emergency room, as this may lead to clot dislodgement and subsequent hemorrhage.

• Two large-bore intravenous lines should be placed to establish access for fluid resuscitation. Subclavian vein injuries should be suspected in Zone I injuries, and intravenous access should be placed on the contralateral side of the penetrating injury to avoid extravasation of fluids.

• Spinal stabilization should be maintained until cleared clinically and/ or radiographically.

• Tetanus toxoid should be administered if the status is unknown or outdated.

• If possible, initial radiographic survey in the trauma bay should include chest x-ray and cervical spine x-rays.

• Prophylactic antibiotics and nasogastric tube suction placement should also be considered.

## b. Muscular Landmarks:

Muscular landmarks are also important:

• Platysma muscle—Penetration of the platysma muscle defines a deep injury in contrast to a superficial injury.

• Sternocleidomastoid muscle—The sternocleidomastoid muscle also serves as a valuable landmark, since this large, obliquely oriented muscle divides each side of the neck into anterior and posterior triangles.

• Anterior triangle—The anterior triangle contains airway, major vasculature, nerves, and gastrointestinal structures, while the posterior triangle contains the spine and muscle.

## Initial management:

Intubation required

• if ventilation or airway is inadequate.

- Unconsciouss patient.
- If the patient requires sedation for diagnostic purposes.
- In case a C- spine injury is considered intubation must be performed by the most Trained professional present.

## Breathing

- Maintain Pco2 levels(>35mmHg)
- Chest X ray to be obtained as quick as possible
- ABG values or end tidal co2 must be monitored regularly.

## **Circulation:**

- Shock may occur due to bleeding from other sites
- Control bleeding as quick as possible.
- Fluid resuscitation by isotonic solutions
- Shock will worsen the head injury hence systolic BP must be maintained at

>90mmHg.

## II) Initial neuro assessment:

- Check GCS
- Note symmetry of motor response. Asymmetry in motor response should increase suspicion of a intra cranial mass which requires immediate surgical intervention.

CT scan to be obtained if,

- Patient is comatose
- GCS>13
- Unequal pupils
- Focal weakness.

A neuro consult to be obtained if the above conditions are present or if the CT is abnormal.

III) Recognise and treat herniation:

- Dilation of pupil
- Deteriorating levels of consciousness.
- Deteriorating levels of motor response.

In case of these conditions a neurosurgeon must be contacted and must be taken to the operating room for intra cranial operation.

1. In the event of an neck or a head injury always suspect a spine injury unless proven to be absent.

2. Use an orogastric tube instead of an nasogastric tube if an antero basciallar facture is suspected.

3. Hyperextension of a head or a neck injury can cause injury to the carotid arteries.

- 4. Systolic BP <90mm Hg can cause secondary brain injury.
- 5. Neurological attributes must never be attributed for the presence of drugs or alcohol.

Standard management of head injuries:

• Assess and stabilise ABCDEs

• Commence at least hourly clinical observations of vital signs, GCS, pupils, PTA and clinical symptoms.

• The initial assessment should be followed by a period clinical observation to detect risk factors for significant intracranial injury. The patient should be risk stratified into "low" or "high" risk groups based on the presence or absence of identified clinical risk factors.

• CT scan not routinely indicated unless one or more high risk factors are present.

• Discharge for home observation with head injury advice sheet at 4 hours post injury if clinically improving with either no risk factors indicating need for CT scan or normal CT scan if performed.

• Consider hospital admission and consult regional neurosurgical service if abnormal CT scan.

• Consider hospital admission for observation if clinically not improving at 4 hours post injury irrespective of CT scan result.

• Consider hospital admission for observation if elderly, known coagulopathy or socially isolated.

• Advise patients to see their local doctor if they do not return to normal within 48 hours so they can be reassessed and monitored for post concussion symptoms.

# **IV.** Conclusion:

The local physician's duty at the accident scene where a serious head or a neck injury is a component of the injury is firstly to maintain the airway by turning the patient onto his/her side. Prior to turning the patient, a collar should be fixed to secure the neck. If this is not available, the patient is turned with head and shoulders moving as one and with the head supported in the lateral position. The next priority is, if possible, to set up an intravenous line. Early an quick management of head and neck injuries can reduce the complications of the injury, and might even save the patient's life.

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