Neck trauma management

Terry Martin

Abstract

Neck trauma is common, and may result from blunt, penetrating or combined mechanisms. Although much of this trauma is minor, the complex and vulnerable anatomy of the neck predisposes to major life threatening complications from even relatively low energy transfer injury patterns. This article reviews mechanisms of injury along with investigation and management principles.

Keywords EAST; neck trauma; NEXUS; spinal cord

Introduction

The adage '*when you discover injury in one compartment of the body and in another more distant compartment, then you must look for injuries in the intermediate compartment*' is never more likely than when concerning the neck. As a flexible link between the head and the thorax, the neck is the structural support for the head and yet allows it to rotate in multiple directions (flexion and extension, lateral flexion and extension, and rotation). However, the lack of bony protection to the anterior structures and the vital structures within the neck predisposes to devastating injuries to the upper airway, oesophagus, major blood vessels and to nerves emanating from the spinal cord and brachial plexus, the sympathetic chain and also from the brain stem (cranial nerves).

Of the many ways of categorizing injuries to the neck, the clearest way to start is by distinguishing between those due to blunt force and those due to penetrating trauma. Given enough force, blunt trauma to the neck can have consequences on many structures and systems, although these injuries may well be covert and patients are often unconscious and unable to give a history or describe their symptoms. In contrast, penetrating injuries may appear localized and therefore easier to manage, but carry a high risk of concurrent injury to multiple vital structures.

Mechanisms of injury

Penetrating trauma

The neck is described in three anatomical zones (Figure 1), and injuries which penetrate the platysma are traditionally described according to the zone of the injury:

- zone 1: between the sternal notch and the cricoid cartilage
- zone 2: between the angle of the mandible and the cricoid cartilage
- zone 3: between the base of the skull and the angle of the mandible.

Terry Martin *BSc FRCS(Ed) FRCA is an Intensive Care Consultant and Medical Director, Capital Air Ambulance, UK. Conflicts of Interest: none declared.*

As with many other areas of trauma surgery, the role of selective non-operative management, particularly for zone 2 penetrations, is being expanded and clarified. Many penetrating neck injuries which would have been operated upon previously, are now successfully managed without intervention following advanced imaging diagnosis.[1,2] The Eastern Association for the Surgery of Trauma (EAST) have specific management guidelines for penetrating neck injuries. See below for additional details.

Blunt trauma

The physical loads generated within the neck are a complex combination of bending, shear and axial forces, and given sufficient force, any or all of the following three patterns may be seen:

- Fractures of cervical vertebrae and/or damage to the ligaments and soft tissues supporting the vertebral column. The fracture mechanisms range from complete 'burst' fractures under high axial loads to minor avulsion fractures of the vertebrae processes. Some of these injuries are stable, some unstable, and some potentially unstable. Imaging and advanced assessment will help classify these for treatment.
- Injury to the spinal cord may result from impingement by splinters of fractured bone, by bending or shearing forces between cervical vertebrae, and/or by longitudinal distraction of the spinal cord. Any damage to the spinal cord, particularly in the atlas (C1) and axis (C2), can be fatal, or lead to permanent paralysis.
- Impact damage to other vital structures of the neck, may include the trachea and larynx, oesophagus, thyroid and parathyroid glands, peripheral and autonomic nerves, major arteries, veins and lymphatics.

Injuries to the vertebral column and spinal cord

Excessive bending, shearing or axial displacements can be produced by indirect or direct loading on the neck from significant relative displacements between the head and the torso.

Indirect loads can be generated by relative head to torso displacements which are produced by two main mechanisms (Figure 2):

- In a seat with no head rest, a rear impact results in the head being accelerated rapidly backwards, thus producing neck hyperextension ('whiplash'). The resulting (mainly soft tissue) damage is rarely life threatening.
- In a front impact, the head is rapidly decelerated as the torso continues to move forward and is decelerated by linkage with the neck. The higher relative mass of the torso produces potentially the most severe injuries with the possibility of vertebral fractures and dislocations.

Direct loads fall mainly in to two major impact injury mechanisms (Figure 3):

- Compression–flexion: Impact on the head with the head flexed forwards, producing vertebral wedge and burst fractures and dislocations.
- Compression–extension: Impact on face with the head in neutral or extended backwards, producing fractures of the spinous processes, damage to the vertebral load bearing surfaces and dislocations.

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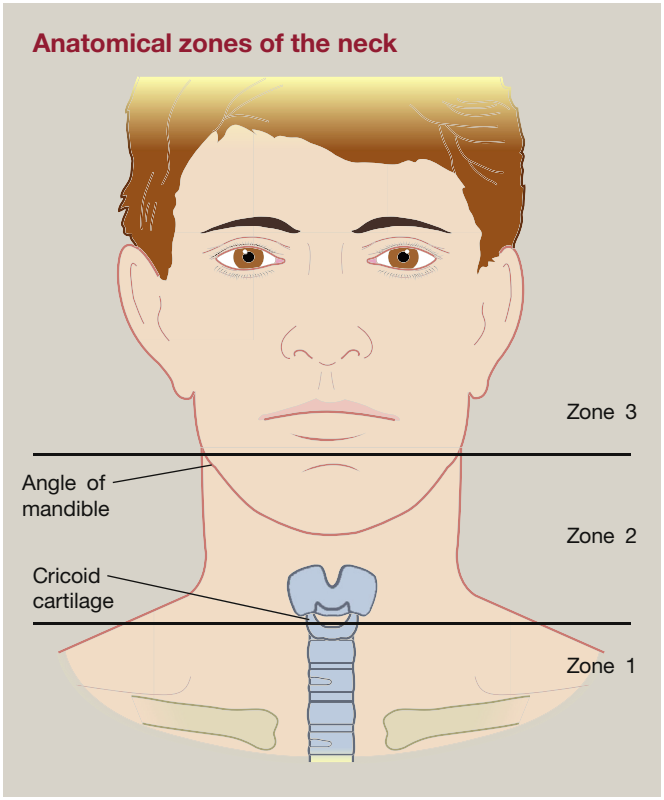

Figure 1

Any lateral bending or lateral rotation of the head prior to, or during the impact will complicate these injuries.

Soft-tissue injuries of the anterior neck

Direct vascular injuries: injuries to the major blood vessels in the neck offer the greatest risk of mortality and morbidity, with the exception of high spinal cord transection and complete airway obstruction. The most serious sequelae of vascular trauma are not just major haemorrhages, but also the complications of airway obstruction from blood, air embolism from sucking wounds, focal cerebral hypoperfusion leading to infarction, and neurologic deficits following global cerebral hypoxia. Although direct pressure will control obvious haemorrhage, covert bleeding may only be identified by discoloration swelling, lack of superficial pulses or bruits.

Laryngeal injury: upper airway obstruction is the second most common cause of death resulting from head and neck trauma but even a minor airway obstruction may suddenly become life threatening as swelling and inflammation develops. This may be delayed for several hours after injury. The anatomy of the larynx is complex (Figure 4), and the area of the front of the neck is relatively exposed and unprotected in frontal impacts. As such, the larynx may be crushed between a blunt object and the anterior cervical spine, leading to cartilaginous fractures, subluxation, and/or dislocation. The most common fracture of the thyroid cartilage is that of a vertical anterior split between the thyroid notch and the cricothyroid membrane producing avulsion of the anterior vocal cord attachments and haematoma.

Laryngeal injury usually produces a loud stridor but this may be absent if the obstruction is severe enough to completely occlude the airway. Other evidence of laryngeal fracture includes oedema with loss of cartilaginous landmarks, dysphonia from paresis or haematoma, pain increased by neck motion, dyspnoea, dysphagia, subcutaneous emphysema and local tenderness.

Cricoid cartilage injuries: since the cricoid cartilage completely encircles the airway, displaced fractures require urgent surgical reduction to prevent the danger of obstruction, especially as post-trauma inflammation will further narrow the lumen. Visualization of the airway by laryngoscopy/bronchoscopy may help to prevent subglottic stenosis which commonly occurs if mucosal lacerations are not properly repaired.

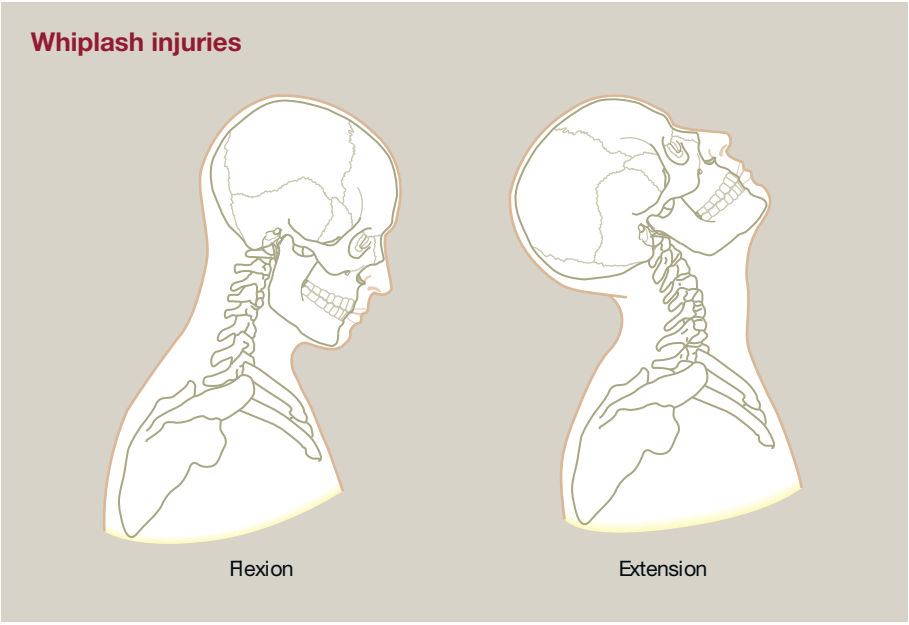

Figure 2

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