

45 Functional Disability

45.1 Assessment of consciousness; secondary survey

The Secondary Survey means that you have done a primary survey of the patient (41.2). *Proceed only to the secondary survey when the patient is haemodynamically stable* and you have organized the tests you want done (41.3).

CAUTION! *If at any point, the patient deteriorates, return to the primary survey (ABC)!*

Complete the medical history getting as much detail as you can, using the patient, witnesses or family members if present. *You must perform a complete physical examination looking for other, non-life-threatening injuries. Don't rush this!* You may not need to put a finger or tube in every hole the patient has – but consider if such intervention will give you useful information (e.g. a rectal examination to determine anal tone).

Repeat this examination in 6 & 12h, or as needed: remember patients' conditions rarely stay static, and may deteriorate alarmingly quickly.

After you have treated or stabilized the main problems, the patient may now report others, especially neurologic and orthopaedic injuries.

Assess the **disability (neurologic status)**: at the very start of your initial assessment, you should have used the AVPU scale:

A – Awake

V – Verbal Response (to questions)

P – Pain Response (reacts to pain: press over the supraorbital nerve, sternum, or the nail beds)

U – Unresponsive

Now make a more specific neurologic evaluation assessing the level of consciousness, pupillary size and reaction, lateralizing signs and spinal cord injury level.

Remember that 5% of brain-injured patients have an associated spine injury, so *always protect the spine* (54.1).

Vital signs (hypertension with bradycardia) present only late with significantly raised intracranial pressure. *Except in children, bleeding in the head does not cause hypotension, although bleeding from the scalp may do so!*

Observe for spontaneous movements and look for one-sided weakness in the limbs or face: an abnormality suggests an intracranial mass lesion.

Check direct and consensual pupillary reflexes: response to light should be brisk. Pupils should be equal within 1mm in size.

Evaluate the conscious level using the Glasgow Coma Scale (45-1), and observe the patient closely for changes in this score. This means you must get your nurses to record the values carefully over time. If the score drops by >2 points, this indicates a neurological deterioration.

N.B. Use the score from the *best* side, if the patient's examination is different from one side to the other.

GLASGOW COMA SCALE			
Best motor response	Obeys		M 6
	Localizes		M 5
	Withdraws		M 4
	Abnormal flexion - decorticate posture		M 3
	Extensor response - <u>decerebrate</u> posture		M 2
	No movement		M 1
Best Verbal response	Adults	Paediatric	
	Oriented	Appropriate words or social smile, fixes and follows	V 5
	Confused	Cries, but consolable	V 4
	Inappropriate words	Persistently irritable	V 3
	Incomprehensible sounds	Restless, agitated	V 2
	None	None	V 1
Eye opening	Spontaneous		E 4
	To speech		E 3
	To pain		E 2
	None		E 1

Fig. 45-1 THE GLASGOW COMA SCALE (GCS). Though not initially described for trauma, this is the most widely used scale. Absolute values give an idea of the neurological status, but it is changes in scores that are important, and show if a patient is improving or deteriorating.

A minor head injury scores 13-15, a moderate injury 9-12 and a severe injury ≤ 8 . *These latter patients cannot protect their own airway.* The lowest possible score is 3. The lower the score, the worse the expected outcome: *only 7% of those with GCS 3-4 have a good recovery or only moderate disability; about 2/3 of those with GCS 8-10, and 80% of those with GCS >11.*

Most pre-hospital trauma-related deaths are the result of brain injury. Of those who arrive at your hospital, some 75% will have minor injuries, 15% moderate and 10% severe. These are the primary injuries.

Most head injuries deteriorate because of poor oxygenation; it is therefore vital that you always clear a patient's airway, and make sure his breathing is unimpaired. *You must try to prevent secondary brain injury* by allowing enough oxygen to reach the brain through patent air passages, good lung function, and adequate blood pressure.

Remember to look beyond a head injury. Don't assume alcohol or drugs are the result of drowsiness in a head injury patient. Record GCS scores every 15 minutes, and if the patient deteriorates, exclude treatable causes.

You should also try to identify an extradural or subdural haemorrhage that requires evacuation (51.9): typical signs are unequal or dilated pupils. The Cushing response (bradycardia, increased BP and decreased respiratory rate) is a late sign with poor prognosis.

A generalized convulsion is not necessarily a bad sign, but will require anticonvulsant therapy. A focal (localized) convulsion is sign of a more sinister lesion.

N.B. Non-barbiturate anticonvulsants don't alter the GCS score.

Maintain a moderate IV fluid input with diuretics (don't overload pure head injury patients, but make sure the BP is adequate if there is bleeding elsewhere). Cerebral oedema may respond to mannitol 0.25mg/kg IV over 30mins, *but you must exclude an intracranial haematoma first!*

Nurse the patient with the head propped up 20° to the length of the body, lying supine.

Prevent hyperthermia (45.3).

If you need to transport a patient with a significant head injury (GCS <12) without full medical attention & capacities, perform a tracheostomy (42.3) beforehand.

ULTRASOUND

A simple way of checking intracranial pressure is to measure the optic nerve sheath diameter by placing an ultrasound probe gently on the globe of the closed eye, *provided there is no direct eye injury.*

At a distance of 3mm from the posterior border of the globe, a measure >5.2mm is significant indication of intracranial pressure raised >20mmHg.

ULTRASOUND SCAN OF OPTIC NERVE SHEATH DIAMETER

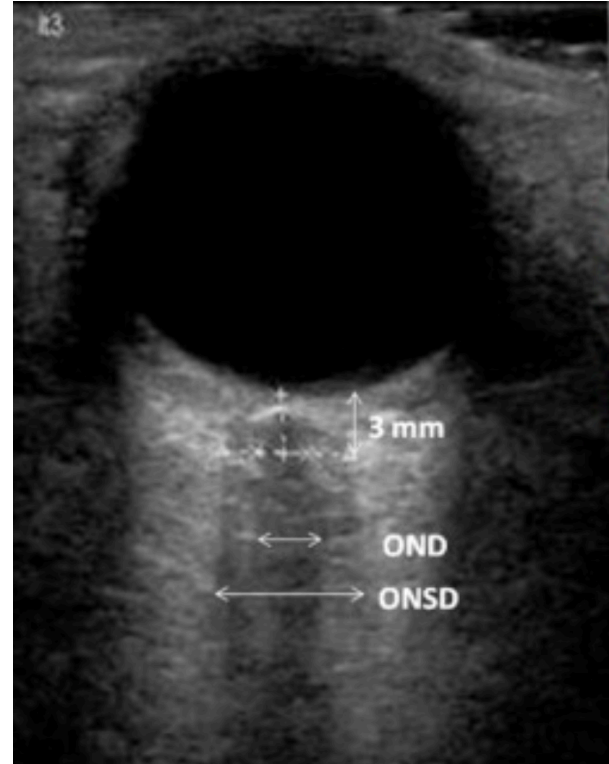


Fig.45-2 ULTRASOUND OF OPTIC NERVE SHEATH DIAMETER can give you early indication of increased intracranial pressure. Measure it 3mm behind the vitreous.

You can carry out this examination repeatedly as it is non-invasive. *Don't press hard or take a long time* or else you may damage the eye.

45.2 Hypothermia

Accidental hypothermia occurs more frequently than you might suspect, even in the heat of the tropics, and certainly in colder climates, or higher up, or at night, and in any case of drowning. It can even occur inside the hospital.

Ironically, such 'indoor hypothermia' is more likely to occur in patients with significant medical comorbidity (alcoholism, sepsis, hypothyroidism, and hypopituitarism) and tends to carry worse outcomes than exposure hypothermia.

You need to be alert to the possibility of hypothermia in any patient that you encounter.

When conditions are dry, radiation causes the majority (55-65%) of heat loss; conduction and convection account for 15%, and respiration and evaporation the remainder.

Conductive and convective heat loss, or direct transfer of heat to another object or circulating air, respectively, are the most common causes of accidental hypothermia.

Patients lose heat up to 30x faster when wet than when dry.

CAUTION! Remove all wet clothing and keep a trauma patient covered with warmed blankets. Always use warmed intravenous fluids if possible.

CAUTION! Make sure that your clinical thermometers can actually measure significantly lower than normal temperatures (as low as 25°C).

Measure core temperatures using a low-reading oesophageal, rectal, or bladder thermometer. Tympanic thermometers are unreliable in profound hypothermia. A rectal probe inserted into stool may yield falsely low readings.

Normal hospital thermometers will not be useful, so order one specially and guard it carefully, not allowing its routine use on the wards.

Hypothermia is classed as mild (32–35°C), moderate (28–32°C), or severe (<28°C). Systemic hypothermia may also be accompanied by localized cold injury (50.13).

Accidental hypothermia increases oxygen consumption (through shivering), blood loss (through coagulopathy). Mortality from traumatic brain injury is increased, especially in the aged, those presenting late, those severely injured, those hypovolaemic and with alcohol (or drug) intoxication.

MILD HYPOTHERMIA (32–35°C)

Signs and symptoms between 34–35°C include shivering, tachycardia, tachypnoea, and vasoconstriction. Mental changes may predominate, with mood change, irritability, poor judgment, and lassitude. Slurred speech and ataxia may mimic a stroke, alcohol intoxication, or high-altitude cerebral oedema.

As the temperature drops below 34°C, there is altered judgment, amnesia, and slurred speech. The respiratory rate may increase.

At c.33°C, ataxia and apathy may develop.

In hypothermia, CNS metabolism decreases in a linear fashion as the core temperature drops. At core temperatures <33°C, brain electrical activity becomes abnormal; between 19–20°C, an electroencephalogram (EEG) may appear consistent with brain death.

Patients are generally stable haemodynamically and able to compensate. You may observe hyperventilation, tachypnoea, tachycardia, and cold diuresis as renal concentrating ability is compromised.

MODERATE HYPOTHERMIA (28–32°C)

Shivering disappears ($\leq 31^\circ\text{C}$) and there may be dysrhythmias, bradycardia, and an altered level of consciousness, even stupor. Oxygen consumption decreases, and the CNS is further depressed: hypoventilation, hyporeflexia, decreased renal flow, and you may notice paradoxical undressing (severely hypothermic persons remove their clothing in response to prolonged cold stress) or rhythmic or repeated motion such as rocking. Reduced muscle perfusion leads to tissue ischaemia, cell death & rhabdomyolysis (49.7).

Between 28–30°C, the pupils may dilate markedly and become minimally responsive to light, a condition that can mimic brain death. Mortality approaches 40%.

At 30°C, risks of dysrhythmias, especially atrial fibrillation and other ventricular dysrhythmias become more likely. The pulse continues to slow progressively, and cardiac output is reduced. You may see a J wave (45-2) on an ECG.

J (or OSBORN) WAVE

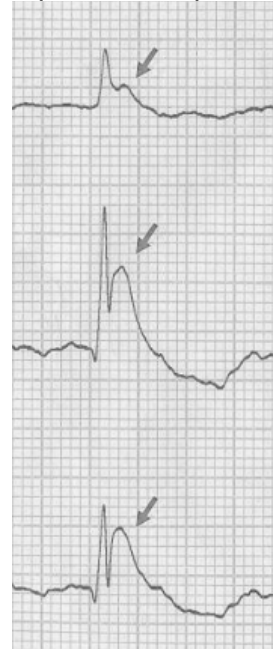


Fig. 45-3 J WAVE IN HYPOTHERMIA. A similar wave may appear in hypercalcaemia.

SEVERE HYPOTHERMIA (<28°C)

At 28°C, the body becomes very susceptible to ventricular fibrillation and further myocardial depression. Coma or cardiac arrest may follow.

Pulmonary oedema, oliguria, hypotension, pulselessness, rigidity, apnoea, areflexia, unresponsiveness, fixed pupils, and decreased or absent brain activity (as seen on EEG) may result.

Many patients are hypovolaemic because of 'cold diuresis'. A haematocrit levels may be deceptively high. Haematocrit levels may increase 2% for each 1°C drop in core temperature. Electrolyte levels can fluctuate wildly and unpredictably. However, a hyperkalaemia ≥ 12 mm is associated with a very low likelihood of recovery.

CAUTION! The usual classic changes on ECG seen with hyperkalaemia may be diminished or absent.

Acute hypothermia may result in hyperglycaemia, while chronic or secondary hypothermia usually presents with hypoglycaemia.

Coagulation studies (performed at 37°C) may be near normal, and will not reflect the serious coagulopathy seen clinically and which is caused by many factors.

In similar fashion, arterial blood gas examinations done at 37°C may report falsely elevated oxygen and carbon dioxide levels and lower pH values. Aspiration pneumonia and pulmonary oedema are common findings.

CPR IN HYPOTHERMIA

N.B. Remember:

"You are not dead until you are warm and dead". Hypothermia may be the only cause of a GCS < 3 .

Obviously, you have to apply some practical tests. **If a patient presents with a frozen chest**, he is dead. From a practical standpoint, there must be some way to rewarm the patient actively while CPR continues; without warming, CPR is a waste of time. *Don't attempt CPR when the patient's temperature is $< 30^{\circ}\text{C}$!*

If there are no signs of life and the core temperature is $> 32^{\circ}\text{C}$ after a period of CPR, further attempts are likely to be futile.

Furthermore, the principles of triage must apply: do you have the resources to dedicate to this patient at the expense of all the others you must take care of?

Ventricular fibrillation in a very cold patient is a desperate event. Try defibrillation x3, despite the fact that it is generally ineffective at very low temperatures.

Intubate any patient whose breathing is failing.

GENERAL TREATMENT PRINCIPLES

- (1) Prevent further heat loss,
- (2) Rewarm the body core temperature
- (3) Avoid precipitating ventricular fibrillation or other malignant dysrhythmia.

The risk of ventricular fibrillation is greatest if the core temperature is $< 22^{\circ}\text{C}$, and inadvertent jerky movement of the patient may provoke this.

Most other dysrhythmias will correct with warming alone. *Don't use any anti-dysrhythmic medication till the temperature is $> 30^{\circ}\text{C}$.* Amiodarone is probably the most useful.

Remove wet clothing, and replace it with dry blankets or sleeping bags.

Start administering warmed, humidified, oxygen; provide heated intravenous saline; and place warmed blankets or heat lamps around the patient. Add external heat packs (e.g. hot water bottles, chemical packs) placed in the axillae, on the groin, and on the abdomen. Avoid causing a burn.

N.B. Surface rewarming is ineffective in very low body temperatures and carries an additional risk of peripheral vasodilation and 'core temperature afterdrop'.

Administer warm fluid IV in those patients who have been hypothermic for $> 45-60$ mins. *Don't use inotropic agents, such as dopamine.*

N.B. In the field, IV fluids freeze in 10mins! Use intra-osseous boluses. A warmed ampoule of adrenaline refreezes in 1min!

N.B. Rapid rewarming, e.g. by inserting heated fluids through nasogastric, chest or peritoneal tubes, may cause nerve damage. Aim to raise the temperature by $\leq 2^{\circ}\text{C}/\text{h}$.

If you need to intubate a victim, don't use relaxants!

Probes for pulse oximetry are better placed on the ears or the forehead than the fingers.

Look out for local cold-induced injuries. Frostbite may result in deep tissue damage. Surgical exploration and debridement may be necessary. Amputate affected parts if gangrene with a clear line of demarcation develops.

Treat rhabdomyolysis (49.6) early.

N.B. An avalanche of snow 50m wide & 150m long weighs 100-1000 tons and can move at $> 50\text{km}/\text{h}$. Mortality is 50% if someone is fully buried, usually through asphyxia. Secondary phase injury occurs 30-90mins after the event.

45.3 Hyperthermia

Hot weather, excessive exercise, lack of hydration, wearing excess clothing (including protective gear), and being locked in a hot space may all lead to temperatures that the body can no longer regulate properly. When the temperature reaches 40°C, there is significant mortality. Such 'heat stroke' occurs more swiftly in children; classically, if left in a locked vehicle in the sun, temperatures may rise above 70°C inside, and a child may die within 1h because of their greater body surface area and less efficient thermoregulation.

Hyperthermia is common amongst improperly prepared marathon runners; consumption of alcohol beforehand makes matters worse!

PRESENTATION

Apart from the high temperature, those at risk have dry hot skin (having used up their capacity for sweating), may have nausea and diarrhea, and become confused or aggressive.

TREATMENT

Remove the clothes, and cool the body surface down by sponging with cold water; if you can, put the person in a bathtub of cold water. Encourage drinking of cold fluids. Put on fans to cool the room air.

N.B. The notion that very cold water causes vasoconstriction does not reverse the effects of cooling!

If the temperature is >39°C, or the patient is losing consciousness, infuse cold saline IV, perform a gastric lavage with cold water, and consider haemodialysis (if possible).

N.B. Beware rhabdomyolysis (49.9) in severe cases!

MALIGNANT HYPERTHERMIA, which is associated with muscle rigidity and tachycardia, may also occur:

- (1) As a reaction to succinyl choline or halothane during anaesthesia,
- (2) In the 'malignant neuroleptic syndrome' with psychotropic drugs,
- (3) After a stroke,
- (4) With anticholinergic drugs,
- (5) In malignant hyperthyroidism.

HYPERTHERMIA



Fig. 45-4 HYPERTHERMIA. Temperatures >39°C are dangerous.

45.4 Monitoring

During any resuscitation, monitor the heart, oxygen tension and blood pressure frequently. An oximeter is essential.

When the patient is stabilized, after the secondary survey, consider if naso- or oro-gastric tubes, bladder catheter and CVP lines are necessary.

N.B. Beware if there has been serious nasal or urethral injury, because passing a tube may further damage these fine structures.

Now get the necessary scan or radiographs done (45.5).

Take a break to see other patients or have a cup of tea and come back to re-assess the trauma victim thoroughly at definite intervals in order to observe any changes in his condition. Such changes will be more obvious than if you had stayed with the patient, yet they often precede a catastrophic fall in blood pressure.

If you have even a simple intensive care unit, this is the place for a patient who is seriously ill, before and after his operation.

Get a specific nurse to record the respiratory rate, pulse, blood pressure, CVP reading, GCS level, fluid input and output.

Make sure that the nurses who complete these charts know that their role is life-saving.

CAUTION! Watch out carefully for the development of a silent pneumothorax. Percuss the lungs twice daily.

Consider monitoring the optic nerve sheath diameter by ultrasound (45.1)

