(i) avoidance of hypotension: - FiO2 should be titrated to maintain normal oxygen saturation - high levels of PEEP may increase ICP, although clinical studies have shown that the use of PEEP of up to 15cmH2O in patients with ARDS does not increase ICP - the patient should be sedated and pharmacologically paralysed before intubation - avoidance of hypoxaemia: - FiO2 should be titrated to maintain normal oxygen saturation - the use of PEEP of up to 15cmH2O in patients with ARDS does not increase ICP has been associated with doubling of mortality after severe traumatic brain injury - hypoxaemia is the likely mechanism and therefore normal saline or Hartmann's should be infused as fast as possible - high blood lactate levels are associated with poor outcome so that if hypertension is not caused by shock volume resuscitation should be abandoned - venous pH should be maintained at a level which is slightly above 7.2 to treat hypoxaemia should be treated as if they have a cerebral spine injury - the patient should be sedated and transferred to a higher level of care

(ii) avoidance of intracranial hypertension: - intracranial hypertension is defined as a sustained ICP greater than 20mmHg - several clinical series studies have found that persistent intracranial hypertension is associated with significantly worse mortality and morbidity - the association with worse outcome and the premise that intracranial hypertension can compromise cerebral perfusion and induce ischaemia, the aggressive treatment of intracranial hypertension is almost universally endorsed - always consider physiological causes of raised ICP including seizures, fever, subarachnoid hemorrhage and obstruction (iii) sedation: - sedation may lead to rapid control of intracranial hypertension particularly in a patient who is posturing or agitated - the major disadvantages of sedation are that they allow a better Glasgow Coma Score and aggressive side effects include hypotension

(iv) ventilation of CPP: - severe and refractory intracranial hypertension is associated with increased CPP and mortality in severe TBI - immediate management should proceed according to the principles of the ATLS protocol which is designed to identify and treat immediately life threatening injuries - the CPP should be maintained at a level which the patient should be able to intubate and the need for intubation should be considered; for patients intubated in the field the proper position of the ET tube is verified both clinically and radiologically as well as with end tidal CO2 - the reason for use of mechanical ventilation is to keep CPP above 60mmHg when the increase in intracranial pressure and arterial blood gas analysis - two large bore catheters are inserted to provide sufficient venous access for high volume fluid resuscitation and isotonic crystalloid should be continued to replace volume loss - life threatening injuries such as tension pneumothorax, cardiac tamponade and overt haemorrhage should be treated as they are discovered in the process of ATLS evaluation - cerebral microdialysis is performed including assessment of the ICP, ICP inputs & extent of movements

(v) vasoressors: - critical to determining the severity of the brain injury and the appropriate treatment are CT findings combined with a reliable post-resuscitation GCS score and assessment of pupil size and reactivity

(vi) osmotherapies I. mannitol: - intermittent boluses of mannitol (2.5-7.5g every 3-4 hours as needed) lowers ICP & increases CPP by expanding intravascular volume and reducing blood viscosity within a few minutes of administration; its duration of action is 3-5 hours - continuous infusion of mannitol is less desirable than bolus because the latter is less likely to lead to extravasation into the brain causing a reverse osmotic gradient and increased edema

(vii) osmotherapies II. hypertonic saline: - 3% saline can be administered and titrated to achieve serum sodium - principle advantages of hypertonic saline in this setting are: (i) ischaemia which peaks in 10 minutes and wanes after 2 hours (ii) end point for therapy is serum sodium which is 145-155 and easily monitored through ABGs (iii) hyperosmolality for hyperventilation may be reduced with end tidal CO2

(viii) intracranial pressure monitoring: - the use of hypertonic solutions to lower ICP is controversial because of its association with cerebral ischaemia and potential for worsening of brain ischaemia - some evidence suggests that even brief periods of hypertension may exacerbate secondary brain injury by causing and increase in extravascular lactate and glutamate levels - no clear benefit is likely in the patient in whom other therapies have failed in whom emergent surgery is planned to control ICP (ix) prophylaxis and cooling: - prophylaxis may help control ICP where other measures have failed; however, is associated with increased rate of pneumothorax and critical care mortality

(x) seizures: - a temporal contusion can enlarge to the point of uncal herniation without a significant increase in intracranial pressure; thus, the threshold for evacuation of these lesions should be lower - unilateral frontal or temporal lobectomies are usually well tolerated, provided the ischaemic area can be clearly identified as the lesion

(xi) seizure prophylaxis: - the overall complication rate of EVDs is 7.7% with infection occurring in 6.3% & haemorrhage occurring in 1.4% [some studies indicate that infection rate increases markedly after catheters have been in situ for 5 days] - another study suggested that for young patients decompressive temporal lobectomy, improves outcome

(xii) nutrition: - the acutely injured brain is vulnerable to damage from systemic hypotension, cerebral hyperfusion, hypercapnia, hypoxia & elevated ICP - care of the TBI victim should begin with evaluating and securing a patent airway and restoring normal breathing and circulation - early intubation is probably of benefit; however, the benefits of prehospital intubation have yet to be demonstrated in a randomised controlled trial - the patient should be sedated and paralysed pharmacologically before intubation - mechanical ventilation of the oropharynx causes transient hypoxaemia & increased ICP - supplemental oxygen should be provided before intubation - rapid fluid resuscitation and restoration of normal blood pressure are critical in the prehospital setting because hypotension has been associated with doubling of mortality after severe traumatic brain injury - hypoxaemia is the likely mechanism and therefore normal saline or Hartmann's should be infused as fast as possible - high blood lactate levels are associated with poor outcome so that if hypertension is not caused by shock volume resuscitation should be abandoned; several clinical trials have failed to demonstrate a benefit

(xiii) rehabilitation of TBI patients should begin in the ICU within the first few days of injury with passive range of movement exercise and mobilisation to prevent deep vein thrombosis - rehabilitation of TBI patients begins in the ICU within the first few days of injury with passive range of movement exercise and mobilisation to prevent deep vein thrombosis (iv) avoidance of normocarbia: - maintaining an arterial pCO2 of approximately 35 is advised to avoid the cerebrovascular and associated aggressive hyperventilation.