

***Table 1 – Injury Classification***

Primary / Mechanical CNS Injury	Secondary / Resulting CNS Injury
<ul style="list-style-type: none"><li>• Parenchymal contusions / lacerations</li><li>• Direct neuronal-axonal disruption</li><li>• Penetrating objects (ie: bullets, bone fragments)</li><li>• Diffuse vascular damage</li><li>• Hemorrhage<ul style="list-style-type: none"><li>▪ Epidural</li><li>▪ Subdural</li><li>▪ Subarachnoid</li><li>▪ Parenchymal</li></ul></li></ul>	<ul style="list-style-type: none"><li>• Ischemia</li><li>• Inflammation</li><li>• Excitotoxicity</li><li>• Metabolic insults</li><li>• ATP depletion</li><li>• Intracellular sodium and calcium accumulation</li><li>• Oxygen-derived free radical production / Lipid peroxidation</li><li>• Increased cytokine production</li><li>• Nitric oxide accumulation</li><li>• Lactic acidosis</li><li>• Activation of arachidonic acid, complement, and coagulation cascades</li></ul>

**Table 2: Modified Glasgow Coma Scale**

	<u>Score</u>
<u>Motor Activity</u>	
• Normal gait, normal reflexes	6
• Hemiparesis, tetraparesis, or decerebrate activity	5
• Recumbent, intermittent extensor rigidity	4
• Recumbent, constant extensor rigidity	3
• Recumbent, constant extensor rigidity + opisthotonus	2
• Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
<u>Brain Stem Reflexes</u>	
• Normal PLR and oculocephalic reflexes	6
• Slow PLR + normal to reduced oculocephalic reflexes	5
• Bilateral unresponsive miosis + normal to reduced oculocephalic reflexes	4
• Pinpoint pupils + reduced to absent oculocephalic reflexes	3
• Unilateral, unresponsive mydriasis + reduced to absent oculocephalic reflexes	2
• Bilateral unresponsive mydriasis + reduced to absent oculocephalic reflexes	1
<u>Level of Consciousness</u>	
• Occasional periods of alertness and responsive to environment	6
• Depression or delirium, capable of responding, but response inappropriate	5
• Semicomatose, responsive to visual stimuli	4
• Semicomatose, responsive to auditory stimuli	3
• Semicomatose, responsive only to noxious stimuli	2
• Comatose, unresponsive to noxious stimuli	1
<u>Suggested Prognosis</u>	
• Grave	3-8
• Guarded	9-14
• Good	15-18

**Table 3: Address condition**

1) Secure airway	
2) Elevate head 30 degrees above heart	
3) Oxygen delivery:	<ul style="list-style-type: none"><li>• Maintain SpO<sub>2</sub> at 99% and PaO<sub>2</sub> at 90 mmHg with lowest amount O<sub>2</sub> necessary to avoid O<sub>2</sub> toxicity</li></ul>
4) Ventilation:	<ul style="list-style-type: none"><li>• Monitor PaCO<sub>2</sub>, maintain arterial CO<sub>2</sub> at 30-35 mmHg</li><li>• If inappropriate (apneic/hypoventilating), mechanically ventilate</li><li>• If sedation required or hyperventilating – oxymorphone 0.1mg/kg + Diazepam 0.25 –0.5mg/kg bolus (or to effect)</li><li>• If needed, continue Diazepam at 0.25-0.3mg/kg/h CRI</li></ul>
5) Fluid therapy:	<ul style="list-style-type: none"><li>• Isotonic crystalloid fluid <u>to effect</u></li><li>• If in shock – hypertonic saline (7.5%) at 4 ml/kg IV over 1-2 minutes followed by isotonic crystalloid or colloid</li><li>• If hemorrhage or SIRS – Colloids (hetastarch) at 20-30 ml/kg dogs, 10-15 ml/kg cats over 20-30 min. Then 20 ml/kg/day as needed.</li><li>• If disseminated intravascular coagulation, loss of clotting factors, marked hypoalbuminemia = Fresh-Frozen Plasma</li><li>• If anemia – blood transfusion / oxyglobin</li></ul>
6) Maintain blood glucose between 100 to 200 mg/dl:	<ul style="list-style-type: none"><li>• Insulin constant rate infusion to effect (frequent monitoring)</li><li>• Do not supplement Dextrose unless BG &lt; 70mg/dl</li></ul>
7) Hypothermia:	<ul style="list-style-type: none"><li>• Cool body to 31-35<sup>0</sup> C (88-95<sup>0</sup> F)</li><li>• External ice packs</li><li>• Cold water enema</li><li>• Cold water infusion into urinary bladder</li></ul>
8) Avoid ↑ ICP:	<ul style="list-style-type: none"><li>• Mannitol: 0.5 to 1.0 g/kg over 20-30 minutes q 6-8 hours as needed based on ICP</li><li>• Furosemide: 0.7 mg/kg IV 15 minutes after mannitol</li></ul>
9) If evidence of seizure activity:	<ul style="list-style-type: none"><li>• Diazepam: 0.2-0.5 mg/kg IV 3x q10-15 min as needed</li><li>• Midazolam: 0.1-0.25 mg/kg IV 3x q10-15 min as needed</li><li>• Phenobarbital: 5-10 mg/kg PO/IV divided BID-TID for minimum 6 months (check blood levels and adjust dose after initial 2-3 weeks)</li><li>• Last resort - Barbiturates:<ul style="list-style-type: none"><li>• Pentobarbital IV bolus 4-16 mg/kg as necessary to induce</li><li>• Follow with CRI 0.2-1 mg/kg/hr to maintain coma</li></ul></li><li>• Last resort - Propofol:<ul style="list-style-type: none"><li>• 6 mg/kg IV then 0.1-0.2 mg/kg/min constant rate infusion</li></ul></li></ul>
10) If evidence of open head wounds or sinus fracture:	<ul style="list-style-type: none"><li>• 3<sup>rd</sup> generation cephalosporins (Cefixime PO, Cefotaxime 25-50 mg/kg IM/IV TID, Ceftiofur 4.4-5.5 mg/kg SQ SID dogs&lt;caution&gt;)</li><li>• Metronidazole (25 mg/kg PO BID)</li><li>• Trimethoprim/Sulfa (15 mg/kg PO/SQ BID-TID)</li><li>• Clindamycin (5-10 mg/kg IM/SQ/PO BID)</li><li>• Enrofloxacin (10 mg/kg PO/IV diluted SID)</li><li>• Note: PO dose only to be used after full mentation returns</li></ul>
11) If possible skull fracture, ongoing CNS hemorrhage, or progressive deterioration	<ul style="list-style-type: none"><li>• CT or MRI</li><li>• Surgery to correct depressed fracture, hemorrhage, and/or treat open wounds</li></ul>
12) Other considerations	<ul style="list-style-type: none"><li>• Frequent turning (atelectasis, pressure sores)</li><li>• Nutritional management</li><li>• Bladder and bowel function / general hygiene</li></ul>

***Table 4: AVOID***

Airway obstruction	Hypotonic Crystalloids
Cardiac Arrhythmias	Hypoventilation
CSF tap	Infection
Dehydration	Jugular vein obstruction
Dextrose Fluids	Narcotics
Glucocorticoids	Overhydration
Halothane	Pneumothorax
Hemorrhage	Pressure Sores
Hyperthermia	Shock
Hyperventilation	Vomiting

PREMISE:

*The treatment of head trauma is to be directed towards cerebral protection, the minimization of secondary brain injury, and the provision of an optimal physiologic environment for the recovery of neural function via the control of intracranial hypertension using measures that support cerebral perfusion.*

