Head Trauma

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Mechanisms

Primary injury is a direct result of the initial insult, is complete at the time of presentation and cannot be altered. **Secondary** brain injury is an alteration of brain tissue, either anatomical or physiological, which occurs after the primary injury, and can be prevented or ameliorated with optimal supportive care. Secondary injury includes bleeds, cerebral edema, vasospasms, and elevations in intracranial pressure (ICP). The intracranial contents are enclosed in a non-distensible protective bone and consist of brain tissue (80%), cerebrospinal fluid (CSF) (10%), and blood (10 %). An increase in one component will result in a decrease in volume, increase in pressure. Initially, CSF fluid is displaced into the subarachnoid space, then cerebral blood flow is displaced into the jugular vessels. Subsequent very small increases in intracranial volume results in marked increase in ICP. A brain shift or herniation can result. Contusions, hematomas, intracranial bleeds, edema, and tumors are the most common findings that lead to pressure-volume decompensation. A slow, progressive rise is better tolerated than a small acute increase in ICP.

During hypoxia or ischemia, the brain cannot meet the energy demands causing a mismatching of oxygen supply and tissue demand. Cerebral blood flow (CBF) is regulated by: neuronal stimulation, PaO_2, $PaCO_2$, and pressure autoregulation. The brain is very sensitive to changes in $PaCO_2$, with a 1 mmHg change resulting in a 3-4% change in CBF and cerebral blood volume. CBF is maintained over a range of mean arterial pressures of 50-150 mmHg. The true driving force of CBF is cerebral perfusion pressure (CPP), and when ICP is elevated, CPP equals MAP - ICP. Injured areas of the brain lose the ability to autoregulate, with regional blood flow becoming ldependent upon mean arterial pressure (MAP) and ICP.

There is a flow-metabolism coupling, with the cerebral metabolic rate ($CMRO_2$) dependent upon the CBF. Local $CMRO_2$ increases with neuronal activity, such as seizures or fever, and decreases with decreased activity as with hypothermia and anesthetic agents. When CBF decreases below autoregulation, oxygen extraction increases to maintain $CMRO_2$ until a further decline in CBF causes compensatory mechanisms to be exhausted.

Two of the most dramatic causes of secondary injury to the brain is hypotension and hypoxemia, capable of downgrading the outcome. In man, elevation in ICP is a critical secondary insult and the single major contributor to the mortality rate encountered in TBI in humans. Any cause of increased ICP must be sought out and effectively treated.

Physical and neurologic examination

It is important to look for external and internal evidence of trauma. Evidence of hypoxia or cyanosis, echymosis or petechiation, or cardiac or respiratory insufficiency warrants investigation for metabolic etiologies. Retinal exam findings of hemorrhage or distended vessels suggests hypertension or coagulopathy; papilledema suggests cerebral edema; retinal detachment suggests infectious, neoplastic or hypertensive causes. Bradycardia reflects midbrain, pontine or medullary pathology. Evidence of blood around the ears, eyes and nose can reflect trauma severe enough to cause intracranial bleeding. Palpation of the skull can reveal compression fractures that require surgical decompression. When trauma is the cause, careful examination is necessary to detect life-threatening pneumothorax, hemothorax, pulmonary contusions, cardiac arrhythmias, internal bleeding, external bleeding, fractures, and shock. Rapid, irregular breathing patterns with or without cyanosis can suggest respiratory compromise or thromboembolic disease and hypoxemia.

The clinical signs of head injury are related to the degree of secondary brain injury. Neurologic signs localized to a focal area are more typical of intracranial bleeds, herniation or focal vasospasms. Generalized neurologic deficits are more likely to be due to cerebral edema . Secondary brain injury is often initiated at the scene of the trauma when there is hypotension and hypoxia associated with blood loss and shock. Trauma induced brain injury can worsen dramatically during resuscitative efforts due to hypertension and intracranial bleeds. The brain has high oxygen and glucose requirements, minimal storage of oxygen, few recruitable capillaries, and consumes oxygen at a constant rate - setting the stage for hypoxic injury. Fever, seizures or thrashing and afferent stimuli increase the cerebral metabolic rate of oxygen consumption. These conditions should be avoided.

A decline in the level of consciousness implies progression of secondary brain injury due to intracranial bleeding, herniation or cerebral edema.

Whether or not the animal had seizure activity aids in localizing the brain lesion to the cerebral cortex or diencephalon. Traumatic etiologies are associated with secondary brain injury from either bleeding or cerebral edema.

Brain injury affecting any portion of the ascending reticular activating system (ARAS: reticular formation with pathways to the thalamus and cerebral cortex), can alter the level of consciousness in the small animal patient. The levels of consciousness range from awake to mental depression, then delirium, to stupor, and then coma. Stupor is defined as unconscious but responsive to noxious stimuli. Coma implies unresponsive loss of consciousness.

The goal of the neurologic exam is to detect the level of severity and guide the intensity of therapeutics by determining whether the lesion is focal or diffuse and localizing the lesion [to either the cerebral cortical or subcortical area (better prognosis), the midbrain, or the brainstem (grave prognosis)]. The neurolotic examination should determine the level of consciousness and whether or not animal is arousable..

Abnormal respiratory patterns can help localize CNS lesions. Cerebral and diencephalic lesions may produce Cheyne Stokes respiratory patterns, typically seen as a rhythmic waxing and waning in ventilatory rate and depth. In the dog and cat, there has to be diffuse, severe cortical damage for this to be seen. Hyperventilation can be seen with lesions of the midbrain or pons, though metabolic acidosis, respiratory alkalosis, and pain must be ruled out. Irregular respirations (apneustic breathing) can be associated with toxicity or suppression of the brain stem respiratory centers.Detection of severe bradycardia or arrhythmias can be suggestive of brainstem problems.

Localization of brain injury can be done by assessing the function of the cranial nerves: normal cranial nerve exam with cerebral and diencephalic pathology; CNIII deficits reflect midbrain pathology; any deficit of CN V-XII reflects pontine and medullary pathology. Pupillary light reflexes are evaluated: normal responsive pupils or small but reactive pupils indicate cerebral or diencephalic lesion; dilated unresponsive pupils (unilateral or bilateral) or midpoint fixed unresponsive pupils reflect midbrain lesions; mid position unresponsive suggests pons; mid position or normal suggests medulla oblongata. The eye position can reflect regions of the brain that are affected: ventrolateral strabismus reflects midbrain pathology. The oculocephalic reflex is done(if etiology allows cervical manipulation): loss of vestibular nystagmus reflects midbrain or brainstem pathology.

Postural changes are imortant: decerebrate rigidity is a result of midbrain pathology. Midbrain and brainstem signs can result from bleeds, thrombosis, trauma, progression of cerebral edema, and brain herniation, most commonly. Brain edema, herniation, hemorrhage, laceration, contusion, hematomas, or skull fracture are some of the possibilities.

Diagnostic approach

Initial diagnostic tests should include hemogram, serum chemistry profile and urinalysis. Blood glucose values are termined to detect hypoglycemia or significant hyperglycemia which affect the level of consciousness. Arterial blood gases are evaluated for evidence of hypoxemia, severe pH changes or hypercarbia.

When trauma is severe with focal neurologic signs, intracranial bleeding or thrombosis may be responsible for the brain pathology and a coagulogram (i.e. PT, PTT, fibrinogen, FDPs, platelet count, antithrombin III, buccal bleeding time) is performed. Electrocardiographic evaluation aids in determining cardiac dysfunction which may be contributing to the etiology of the brain injury, or a result of the brain pathology. Blood pressure measurements detect hypotension and guide resuscitative efforts to bring the systolic arterial blood pressure >90 mmHg while avoiding hypertension.

Survey radiographs of the chest and abdomen are done looking for evidence of organ changes secondary to trauma or ischemia. Abdominal ultrasound may detect hemorrhage not seen on survey radiographs. Skull radiographs are evaluated for fractures in trauma. Computed tomography scanning is the best imaging modality for detection of acute blood within the cranial vault or brain. It is also the method of choice for imaging the skull bones, looking for depressed skull fractures or intracranial foreign bodies.

Intracranial pressure measurements can be evaluated to determine the severity of ICP elevation and the response of ICP to therapy. Brain stem auditory evoked potentials help determine brain stem function but disappear only after brain death is imminent.

Therapeutics

Goals are: a) support of mean arterial pressure and b) control of intracranial hypertension by i) reduction of cerebral tissue volume, ii) reduction of cerebral blood volume, and iii) reduction of cerebral spinal fluid volume.

Support of mean arterial pressure

Intravascular volume expansion is a requirement of resuscitation of MAP and perfusion in hypovolemic, hemorrhagic, and distributive shock. Crystalloid fluids will extravasate from the vessel to the intercellular space. Hetastarch has been shown to increase CPP compared to lactated ringers and hypertonic saline-dextran combination. The use and selection of colloids is controversial with experimental evidence available for all arguments. If hypotension persists after volume resuscitation, the author recommends the following agents be tried in the order listed: stroma-free hemoglobin (Oxyglobin®) – 1-5 ml/kg IV slowly; dopamine – 5-10 ug/kg/min. Systemic hypertension can be present or result from fluid resuscitation. When the BP is sustained above 150 mmHg MAP, it may have to be controlled utilizing vasodilators such as nitroprusside. This has been shown, however, to increase cerebral blood volume. Other vasodilators such as calcium entry blockers and beta and alpha antagonists have not been shown to reduce cerebral blood flow.

Control of intracranial hypertension

This depends upon effective control of cerebral tissue volume, cerebral blood volume, and CSF volume.

Reduction of cerebral tissue volume is accomplished medically through the osmotic diuretic, mannitol., which works by changing the brain blood rheology The osmolality should be kept within normal limits. Smaller doses (0.1-0.5 g/kg) of mannitol have been used

effectively for maintenance of reduction in closed head injured human patients. Its positive effects depend upon the presence of an intact blood-brain barrier. Furosemide is administered first and has been found to reduce the mannitol induced elevation in ICP. Without an MR or CT to make this assessment, the decision to use mannitol should be based upon the neurologic examination of the patient (Figure 1). Glucocorticosteroids appear to be ineffective in reducing cerebral edema after closed head injury and may increase morbidity.

Hypernatremia can cause changes in osmolality that is damaging to the neurons. Rapid correction of hypernatremia is equally as detrimental. Hypernatremia is treated with sodium containing fluids and lowered very slowly over several days. The use of sodium wasting diuretics and ADH may be warranted. Serum magnesium concentrations should be maintained within a normal range for the species.

iReduction of cerebral blood volume is done by reducing CBF or by facilitating cerebral venous drainage. Endotracheal intubation and mechanical ventilation ensure that hypoxemia and hypercarbia are not contributing to an increase in CBF. The PaO_2 is maintained above 60 mmHg. Cerebral vasodilation is minimized keeping the PaC02 between 30-40 mmHg. Intubation with coughing has been shown to increase the ICP. To offset this, IV lidocaine is suggested at 0.75 mg/kg (decreases CBF). Adequate sedation and analgesia will reduce CMRO2 and CBF. Narcotics are required for analgesia and for long term intubation, narcotics and pentobarbital can be used or narcotic and neuromuscular blockade. Extubation with subsequent coughing will elevate ICP. Prior to extubation, lidocaine should be repeated IV and 1/4 of the narcotic antagonist dosage given. Once the tube is out, the rest of the antagonist can be given. Head position, head elevation and ensuring patency of the jugular veins are important contibuting factors.

Animals and human with hyperglycemia and brain injury have a worsened prognosis for neurologic recovery. The blood glucose should be maintained between 120-180 mg/dl. There is evidence that a glucose concentration of > 200 mg/dl can be detrimental to recovery. If hyperglycemia is present after fluid and blood pressure resuscitation, a CRI of regular insulin (0.25 – 1.0 unit/kg/day) may be required to keep the glucose within the optimal range. iii. *Reduction of cerebral spinal fluid volume* can be done mechanically through dehydration or CSF withdrawal through intraventricular catheters. Dehydration is not recommended. Drugs such as furosemide, acetazolamide and digoxin reduce CSF production. Furosemide at 0.75 mg/kg tid IV has been used.

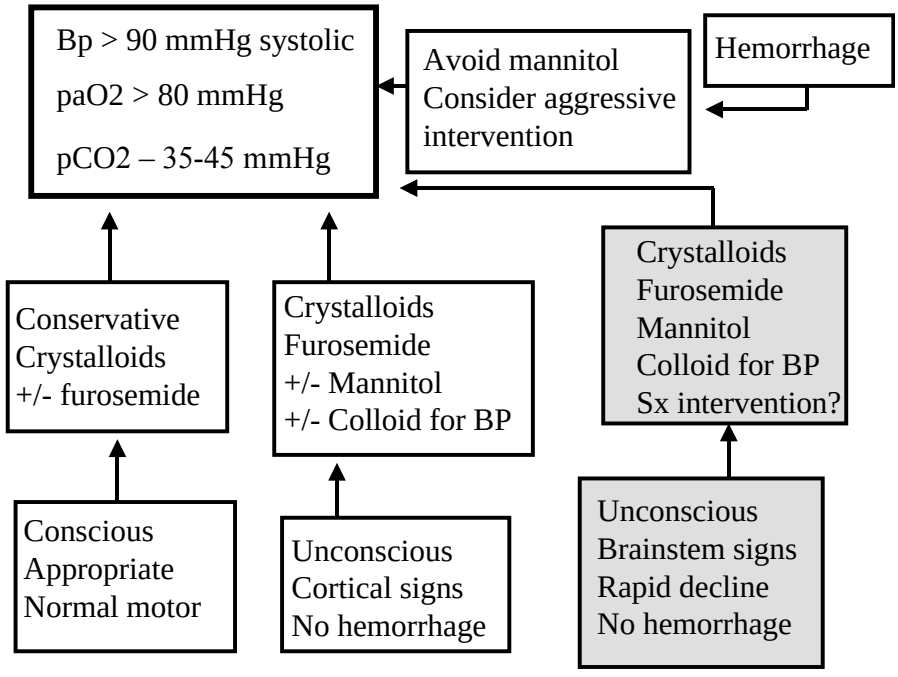

Figure 1. Algorithm for making therapeutic decisions based solely upon neurologic findings or changes in neurologic status.