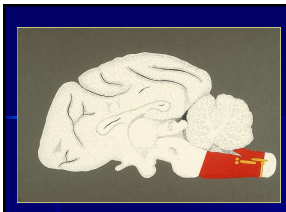
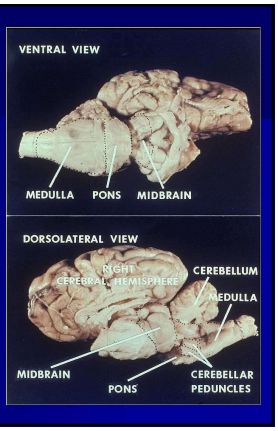
"Teaching is like brain surgery without breaking the skin. It should not be entered into lightly nor performed by amateurs".





Neurologic Emergencies

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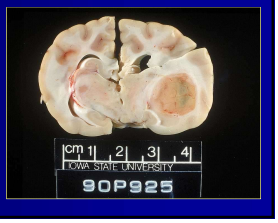
Types of Neurologic Emergencies

- Intracranial
  - Traumatic - HBC
  - Metabolic - Hypoglycemia / HE
  - Neoplastic
  - Vascular
  - Seizures
- Spinal Cord - IVDD,FCE,Trauma
- Neuromuscular - Myasthenia gravis

Neurologic Emergencies What are they ?

- Any structural or functional disturbance of the CNS which has an acute onset
- Can result in reversible neuronal impairment
- Must be treated aggressively to avoid irreversible changes or death



Neurologic Assessment

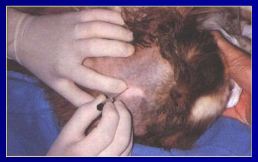
- Mentation
  - Level and content of consciousness
- Cranial nerves
- Gait
- Postural reactions
- Motor
- Sensory

Traumatic Brain Injury

- HBC / Missile injuries / Falls / Bite wounds
- Progressive increase in intracranial pressure (ICP) most deadly
- Remarkable compensatory powers
- Patience is a virtue

Importance of the Neurologic Exam

- Neurolocalization
- Prognostication
- Very important to do serial neurologic examinations to look for progression



Primary brain injury

- Physical disruption of intracranial structures that occurs immediately post-trauma
- Direct damage to brain parenchyma
  - Contusions / lacerations / diffuse axonal injury
- Intracranial hemorrhage / vasogenic edema

Primary brain injury

- Extent is a function of force of impact
- Acceleration / deceleration forces of impacting objects / intracranial contents
- Direct parenchymal damage ☹

Secondary brain injury

- Continued hemorrhage and edema
- Chain of events lead to brain-tissue damage / increased ICP
- ATP depletion – disruption of cellular ionic homeostasis
- Na and Ca influx – leads to cellular swelling (cytotoxic edema) / depolarization

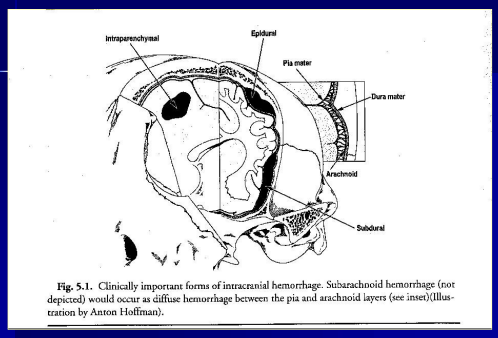


Fig. 5.1. Clinically important forms of intracranial hemorrhage. Subarachnoid hemorrhage (not depicted) would occur as diffuse hemorrhage between the pia and arachnoid layers (see inset)(Illustration by Anton Hoffman).

Secondary brain injury

- Uncontrolled depolarization >> glutamate released extracellularly
- Leads to continued increases in intracellular calcium levels
- Leads to activation of tissue-damaging pathways
  - Arachidonic acid cascade (phospholipase A2 activation)
  - Xanthine oxidase pathway (free-radical production)

Secondary brain injury

- Iron – vital cofactor in xanthine oxidase pathway >> free radical *spp*
  - Hydroxyl and superoxide radicals
- These radicals damage cell membranes high in polyunsaturated fatty acids (PUFA's) and cholesterol
  - Brain tissue high in Fe & membranes with high PUFA's and cholesterol

Secondary brain injury

- Free radicals
  - Very damaging to neuronal membranes
  - Production induced by:
    - Ischemia
    - Arachidonic acid metabolites
    - Catecholamine oxidation
    - Activated neutrophils

Intracranial pressure dynamics

- Normal contents of cranial cavity:
  - Brain tissue (80%)
  - Blood (10%)
  - CSF (10%)
  - ICP remains constant if above in equilibrium
  - MABP 50-150 mm Hg – ICP remains constant – *Pressure autoregulation*
    - Links systemic BP changes to blood vasculature tone
  - If MABP rises >> brain vasoconstriction / if MABP falls >> brain vasodilation
  - In normal animal, the former prevents ICP from rising / the latter prevents ICP from falling

Secondary brain injury

- Other autolytic processes induce the following cascades:
  - Complement
  - Kinin
  - Coagulation / fibrinolytic
- Elevated NO (nitric oxide) / cytokines (TNF / interleukines)
- Self-perpetuating .. Can lead to herniation
- Lactic acid accumulation (anaerobic glycolysis)
- Above results in increased ICP >> clinician has some control over this ☺

Intracranial pressure dynamics

- Chemical autoregulation
  - Direct responsiveness of brain to partial pressure of CO2 in arterial blood
    - Increased PaCO2 >> vasodilation
    - Decreased PaCO2 >> vasoconstriction

Intracranial pressure (ICP) dynamics

- ICP = pressure exerted by tissues/fluids within the cranial vault
  - Normal ICP = 5-12 mm Hg
  - CPP (cerebral perfusion pressure) is a primary determinant of CBF (cerebral blood flow), thus brain oxygenation/nutritional support
  - CPP = MABP - ICP

Intracranial pressure dynamics

- Monro-Kellie principle (intracranial compliance)
  - Expansion of one component requires = decrease in other compartments to maintain normal ICP
  - Following head trauma >> IC hemorrhage / edema increase volume in the skull
  - Initially, CSF / blood flow altered to accommodate increased volume
  - IC compliance overwhelmed >> ICP increases exponentially (low IC compliance)

Intracranial pressure dynamics

- If hypotension (MABP) / hypoxia / hypercapnea develops >> brain vasculature dilates to preserve CBF
  - Serves to increase ICP, but CPP / CBP remain inadequate
- Increasing ICP >> further ischemia / edema >> herniation

Brain herniation

- Caudal transtentorial
- Rostral transtentorial
- Foramen magnum
- Subfacial
- Through a fracture site

Brain herniation

- Secondary effects accumulate
- Intracranial compliance (change in volume per unit change in pressure) decreases as ICP increases
- CPP compromised … herniation (sound familiar ?)

Brain herniation

- Caudal transtentorial
  - Temporal / occipital cortex protrudes under the tentorium cerebelli causing compression and caudal displacement of the midbrain
- Rostral transtentorial
  - Cerebellum displaced rostrally under the tentorium cerebelli compressing and displacing the temporal cortex and brainstem
- Watch for bilateral CNN III compression

Brain herniation

- Brain volume / ICP increase beyond limits of compensation
- Parenchyma shifts causing brain herniation

Brain herniation

- Foramen magnum herniation
  - Caudal cerebellar vermis is displaced through the foramen magnum resulting in malacia and hemorrhage to the cerebellum and medulla oblongata
  - Respiratory arrest >> respiratory centers in the medulla oblongata

Brain herniation

- Subfacial
  - Caudal cerebellar vermis is displaced under the falx cerebri
  - The opposite cingulate gyrus is compressed with minimal neurologic dysfunction apparent

Initial Assessment and Therapy

- ABC's of trauma management
- Quick assessment tests
- Immediate correction of hypoxemia and hypovolemia to avoid increased ICP
- Act calmly but quickly

Other brain injury

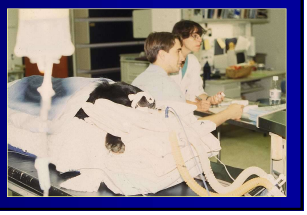
- Coup injury refers to direct injury by external force at the site of impact
- Contrecoup injury is common when the head is in motion or after the initial coup
- Closed head injury refers to brain injury without any penetrating injury to the brain
- Open head injury – bone fragments and foreign material being driven into the brain which could lead to infection

Initial assessment

- Glasgow Coma Scale
  - Human GCS is graded from 3-15
  - Scores below 8 indicate a severe brain injury
  - CT scan allows accurate assessment of skull / brain injuries – epidural hematoma
- Modified GCS (MGCS)
  - Assesses level of consciousness / brainstem reflexes / motor activity on a scale of 3-18
  - Each of these 3 examination categories is assigned a score from 1-6
  - ARAS

Parameters that should be monitored in brain injury

- Blood pressure
- Body temperature
- HR / Rhythm
- RR / character
- Serum glucose / osmolality
- Electrolytes
- Lactate
- SpO2 / PaO2
- PaCO2
- ICP – intracranial pressure monitor



Initial assessment

- Hypotension and hypoxia must be identified ….. and avoided
- Blood glucose concentration – euglycemia should be maintained
- Hyperglycamia has been correlated with severity of head trauma in dogs and cats

Fluid therapy

- Hypertonic saline (7 %): 4-5 ml/kg over 3-5 minutes for shock
- Be careful of sodium >> implicated as a major osmotic agent contributing to brain edema
- Focal areas of damaged parenchyma ?

Fluid Therapy

- Fluid therapy is indicated to support CPP by volume replacement / maintenance of MABP
- Limitation of fluid replacement is CONTRAINDICATED
- Hypotension has been repeatedly shown to be a reliable predictor of sustained elevations of ICP and increased mortality in human head trauma victims

Fluid therapy

- Dextran 70: 10-20 ml/kg (up to 40-50 ml/kg/hr) for shock in dogs
- Cats: 5 ml/kg boluses given over 5-10 minutes – max of 20 ml/kg
- Less than beneficial compared to above
- Potential adjunctive therapy choice

Fluid therapy

- Maintain blood pressure at 90 mm Hg systolic pressure
- Avoid hypo / hypervolemia
- Volume replacement fluids
  - Hypertonic saline can improve MABP and thus CPP w/o exacerbating brain edema
  - Very useful in cases of traumatic brain injury

Fluid therapy

- Crystalloids (LRS, 0.9% saline): 90 ml/kg/hr (dogs), 60 ml/kg /hr (cats)for shock
- Watch for overhydration / brain edema / increased ICP
- Fluid administration should be tapered when physiologic goals met
- Blood product administration …… anemia
  - 4-10 ml/kg/hr (over 4-6 hr) in the stable patient

Oxygenation and hyperventilation

- Hyperoxygenation recommended for acute brain injury
- Maintain arterial blood gas at PaO2 of or above 90 mm Hg for dogs (100 for cats)
- Pulse oximetry

Mannitol (20-25%)

- Osmotic diuretic
  - Decreases cerebral edema and ICP while maintaining CPP
  - Reduces cerebral edema through osmotic mechanisms
  - Decreases CSF production
  - ***Reflex vasoconstriction*** of brain vasculature by decreasing blood viscosity and free radical scavenging
    - Brain's pressure autoregulation mechanism (allows for improved CPP at a lower brain-blood volume – decreased ICP) – occurs in minutes

Oxygenation and ventilation

- O2 supplemention
  - Face mask / nasal O2 / transtracheal / O2 cage
- Loss of consciousness and ventilation
- Maintain PCO2 levels between 25-35 mm Hg
- Avoid overventilation and vasoconstriction

Mannitol

- Dose: 0.5 – 1.5 g/kg IV over 10-20 minutes
- Reaches maximal osmotic effect 10-20 minutes after administration with effects lasting 2-5 hours
- Limit to 3 boluses in a 24 hour period
- Do not use in the face of hypovolemia
- Can repeat every 6-8 hours / monitor serum osmolality / electrolytes

Other therapies

- Head elevation: Maximize cerebral venous drainage >> 30 degrees
- Address cerebral edema >> peaks at 24-48 hours post-injury and can persist for almost 96 hours
- Physical therapy/ Nutritional support

Other Therapeutics

- Lasix (Furosemide)
  - Loop diuretic
  - Dose prior to mannitol administration at 2-5 mg/kg IV to decrease rebound of ICP
- Glucocorticoids - use is questioned
  - "High-dose Methylprednisolone" protocol
    - 30 mg/kg at time 0, then 15 mg/kg at 2 and 4 hours IV
    - Thought to scavange free radicals vs. activating steroid receptors
    - Brain trauma Foundation guidelines state that glucocorticoids are not recommended for improving outcome or reducing ICP in human head trauma patients

Other therapies

- Lazaroids
  - 21-aminosteroids
  - Methylprednisolone analogues
  - Do not activate glucocorticoid receptors
  - Benefits of free radical scavenging
- Avoid hyperglycemia
  - Fuels anaerobic metabolism >> brain lactic acid
  - Associated with increased mortality in humans
  - Recent veterinary study found it to be correlated with severity of neurologic dysfunction, but association with outcome not found

Secondary assessment and diagnostic procedures

- Thorough neurologic examination once the patient is stabilized
- CT scan is the standard of care in human head trauma patients
  - Availability
  - Rapid scan times
  - Better visualization of peracute hemorrhage / bony abnormalities / fractures
- Specific guidelines for therapeutic intervention exist for open/depressed skull fractures

Other therapies

- Free-radical scavengers
  - Dimethylsulfoxide (DMSO)
  - Allopurinol
  - Deferoxamine mesylate
  - Liposome-encapsulated forms of superoxide dismutase/catalase
  - Lack of clinical evidence to support their use currently
- Erythropoietin
- Antagonists of opiate / glutamate receptors
- Hypothermia – human studies (89.6-93.2 degrees)
  - Thought to inhibit inflammatory cytokine/glutamate release

Diagnostic Imaging

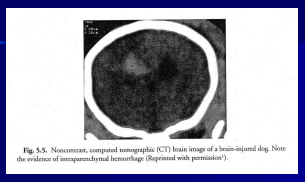


Fig. 5.5. Noncontrast, computed tomographic (CT) brain image of a brain-injured dog. Note the evidence of intraparenchymal hemorrhage (Reprinted with permission[1]).

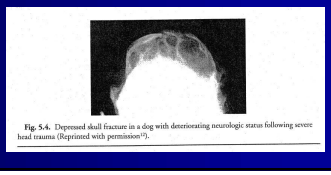
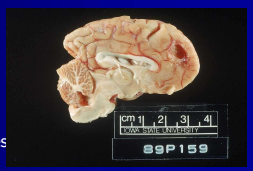


Fig. 5.4. Depressed skull fracture in a dog with deteriorating neurologic status following severe head trauma (Reprinted with permission[1]).

Brain Neoplasia

- Signs observed due to vasogenic edema
- Extremely steroid responsive
- Hypertonic saline 3 ml/kg IV over 20 minutes
- Mannitol
  - Dose: 0.5-1 g/kg IV slow over 10-20 minutes
- Dex SP 0.15 mg/kg IV once
- Anti-convulsants



Evaluation of the Neurologic Patient

- Serial neurologic exams are a must !!
- Look for trends in consciousness, breathing patterns, pain perception and cranial nerve function
- Can indicate worsening of condition or progression to brain herniation
- Take nothing for granted !!

Neurologic Exam

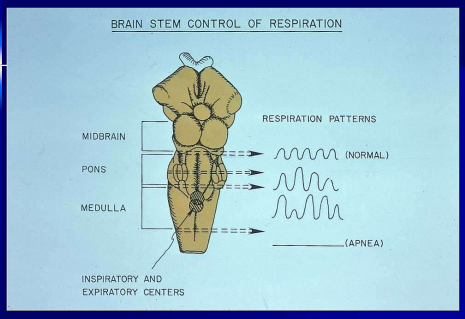
- Loss of consciousness
- PLR
- Vision
- Vestibuloocular movements
- Motor function
- Respiratory pattern

Respiratory Patterns

- Cheyne-Stokes
  - Implies bilateral dysfunction
  - Deep cerebral/diencephalon
- Central neurogenic hyperventilation
  - Mesencephalic lesion
- Irregular / Ataxic breathing
  - Caudal brainstem/medullary lesion

Pupil size and Symmetry

- Normal direct and indirect response
- Unilateral oculomotor lesion
- Midbrain compression
- Bilateral oculomotor lesion
- Diffuse forebrain lesion



Vestibuloocular Movements

- Strabismus
- Presence or absence of ocular movements
- Presence of nystagmus
  - Sustained or resting
  - Positional or changing

Motor Function

- Upper Motor Neuron reflexes
  - Fore > Rear : Deep cerebral
  - Extensor rigid fore > rear : Midbrain
  - Extensor rigid fore/opisthotonus: Cerebellum
  - Fore/Rear : Pons/rostral medulla
  - Absent : Caudal medulla

Post-Traumatic Syndrome

- Hemispheric damage
- Brainstem injury / compression
- Cerebellovestibular dysfunction
- Epilepsy
- Diabetes insipidus
- Effects of : hypovolemia/hypoxia/hypotension

Surgical intervention

- Value of craniotomy solely as a decompressive surgery is unknown in dogs and cats
- In normal dogs and cats, combined craniotomy / durotomy results in dramatic decreases in ICP
- Surgery should be considered if deterioration in neurologic status despite aggressive medical therapy
- ICP pressure monitoring
  - Humans – ICP below 20 mm Hg Cpp at a minimum of 70 mm Hg
  - Fiberoptic epidural ICP monitoring >>> future directions

Surgical intervention - Humans

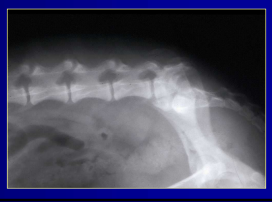
- Indications for intervention are well defined in human head trauma treatment
- Include presence and extent of focal intracranial hemorrhage
- Size, location of focal hemorrhage / presence of a midline shift combined with ICP measurement and GCS – factors affecting surgical decisions in humans

Spinal Cord Emergencies

- Luxations / Subluxations
- Intervertebral Disc rupture
- Fibrocartilagenous embolism (FCE)
- Meningitis
- Remember that aortic thromboembolism can present with similar signs

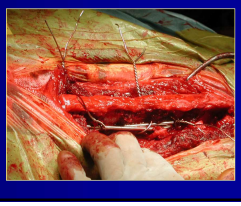
Surgical intervention – veterinary medicine

- Indications for surgical intervention
  - Open or depressed skull fractures
  - Ongoing skull / scalp hemorrhage
  - Intracranial hemorrhage causing a mass effect
- Recent retrospective veterinary study describing CT findings in head trauma highlights the limitations of CT compared to MRI, necropsy and surgical findings
- Study did not identify any characteristic CT findings that correlated with survival
- Authors cautioned that CT findings should not be the only determinant in prognosis



Spinal Cord Emergencies

- The value of neurolocalization
- C1 - C5
- C6 - T2
- T3 - L3
- L4 - S2
- Prognostication
- Keep patient as quiet as possible

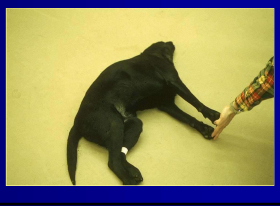


Spinal Cord Emergencies

- Type of injury
- Duration of clinical signs
- Presence or absence of voluntary motor activity
- Presence or absence of conscious pain perception

Spinal Cord Emergencies

- If conscious pain perception is lost (both superficial and deep pain) for > 6 hours, prognosis drops considerably
- If present (depending on cause of injury), > 75-80 % chance of regaining function
- Conscious proprioception > voluntary motor > superficial pain > deep pain



Treatment for Spinal cord Emergencies

- Stabilize the patient - The ABC's of critical care
- Neurologic Assessment
- **Corticosteroids** – Use is controversial
  - Not advised
- **Polyethylene glycol** – membrane repair
- **Oscillating Field Stimulation** – Application of weak electrical field of oscillating polarity to damaged SC region – neuronal / axonal repair

Spinal Cord Injury

- Surgical intervention is indicated where appropriate
- Immediate referral is indicated when animal has lost voluntary motor or is deteriorating
- Beware of mistaking the withdrawal reflex for conscious pain perception

Key Points to Remember

- Complete history
- Thorough physical and neurologic exams
- ABC's of critical care
- Do not be too hasty on prognostication - look for trends
- Treat the animal, not its blood work
- Patience is a virtue