Appendix 2: Protocol for the Management of Stupor and Coma

Philip R Judge

Animal Emergency Centre, 37 Blackburn Rd, Mt Waverley, VIC 3149

Definitions

Stupor and coma are pathological abnormalities caused by an interruption in the structural, metabolic, and/or physiological integrity of the cerebrum or brainstem.
Coma is characterized by an unconscious state from which the animal cannot be aroused by any external stimuli, including those that are noxious.
Stupor is clinically similar to coma, except that the animal can be aroused by external stimuli, but may quickly relapse into its sleep-like state as soon as the stimuli are withdrawn.

Management

- **Airway**
  - Ensure the patient has a patent airway.
  - Provide oxygen by flow-past, mask, or endotracheal tube or catheter.
  - **Avoid nasal oxygen** - sneezing increases intracranial pressure.
  - Intubation reduces the chances of aspiration of gastric and oral secretions and should be performed if the patient has depressed gag reflexes.

- **Breathing**
  - Ensure the patient has a patent airway.
  - Provide in comatose patients, intubate, and provide supplemental oxygen.
  - If patient is semi-comatose, anesthetize, intubate, and ventilate; provide supplemental oxygen.
  - If patient is conscious, provide oxygen if ventilating adequately; if not, consider anesthetizing and ventilating.
  - Ventilate to achieve P_aCO_2 of 30-37 mmHg.

- **Circulation -**
  - Place a peripheral intravenous catheter - avoid struggling and stress.
  - Do not occlude jugular veins.
  - Begin administration of isotonic crystalloid solution (LRS initially) until blood test results available, at rate of 40-60 ml/kg/hr for patients that are hypovolemic.
  - Elevate the head no more than 30 degrees from horizontal to aid in increasing venous drainage from the brain, and reduce intracranial pressure

- **Data Collection** - draw blood for the following tests - do not occlude the jugular veins - **use peripheral vein for blood collection**
  - PCV/TP/Glucose – test immediately
  - Electrolyte levels, full biochemical profile, CBC.
  - Determine serum osmolality

- **Treatment -** begin therapy for specific abnormalities as indicated by blood test results, for example hypoglycemia, hyperglycemia, hypocalcemia. If the patient is not hypernatremic, administration of hypertonic saline and pentaspan or dextran 70 as intravascular replacement fluids may improve blood flow through microvascular beds, and reduce extravasation of administered fluids.

- **Management of Cerebral Edema -** Conduct a neurologic examination, and determine the following:
  - Level of consciousness, pupil responses, pupil position
  - Cranial nerve assessment
  -
  - Respiratory pattern
  - Motor responses
  - Response to noxious stimuli
  - Oculocephalic reflex
  - Localize lesion and determine severity
  - **Record the results**

- Following intravascular volume replacement therapy, treat cerebral edema using the following
- Furosemide at 1-2 mg/kg IV followed in 10 minutes by
- Mannitol 0.5 g/kg IV given over 5-10 minutes**. Contraindication to mannitol administration is hyper-osmolality. Indications include a declining level of consciousness, evidence of brainstem lesion, and craniotomy.**
- If poisoning is a suspected cause, perform a gastric lavage, +/- activated charcoal administration (1-2g/kg) PO and colonic irrigation, and provide specific antidotes as indicated.

- **Perform Coma Scale q 30 minutes during stabilization**
- **Patient Monitoring**
  - Turn the patient every 2-4 hours
  - Eye lubricant
  - Soft bedding
  - Insert urinary catheter and connect to closed collection system
  - Elevate head no more than 30 degrees above horizontal
  - Maintain blood pressure at 100 - 120 mmHg
  - Monitor LOC every 2 hours, perform coma score
  - Control seizures with diazepam at 0.5-2 mg/kg IV - (caution in hepatic encephalopathy, as these patients are more sensitive to benzodiazepines)
  - Control body temperature in low normal range
  - Monitor renal, hepatic, and gastrointestinal function
  - Monitor PCV/TP/ACT
  - Nutritional support is indicated if patient comatose for >12 hours
  - Avoid tight cervical, thoracic, abdominal dressings

Differential Diagnosis of Stupor and Coma

Primary Brain Disease	Secondary Encephalopathy	Abnormal Osmotic States
1. Neoplasia – primary or secondary Abscessation 2. Hemorrhage 3. Concussion, hematoma 4. Cerebral edema 5. Contusion - brain stem 6. Infarction - cerebral, brainstem 7. Degenerative disease 8. Hydrocephalus 9. Lysosomal Storage Diseases 10. Lissencephalopathy 11. Status Epilepticus 12. Canine distemper virus 13. Rabies 14. Feline infectious peritonitis 15. Fungal, protozoal and bacterial infections 16. Granulomatous meningoencephalitis	1. Renal disease (uremia, acidosis) 2. Liver disease (hypoglycemia, hyperammonemia) 3. Pancreatic disease - Insulinoma, diabetes mellitus, hypoglycemia 4. Myocardial disease – ischaemic. Cardiomyopathy 5. Hypertension 6. Bacterial embolism 7. Feline ischemic Encephalopathy 8. Anoxia 9. Pulmonary disease 10. Coagulopathies 11. Nutritional deficiency (thiamine) 12. Anemia, blood loss 13. Carbon monoxide poisoning 14. Hypoadrenocorticism 15. Hypothyroidism 16. Post-ictal depression 17. Toxicity – ethylene glycol, lead, barbituates, cannabinoids, alcohol	**Hyper-osmolar states** 1. Hyperglycemia 2. Diabetes mellitus 3. Hypernatremia 4. Diarrhoea 5. Diabetes insipidus 6. Severe water loss **Hypo-osmolar states** 1. Water intoxication

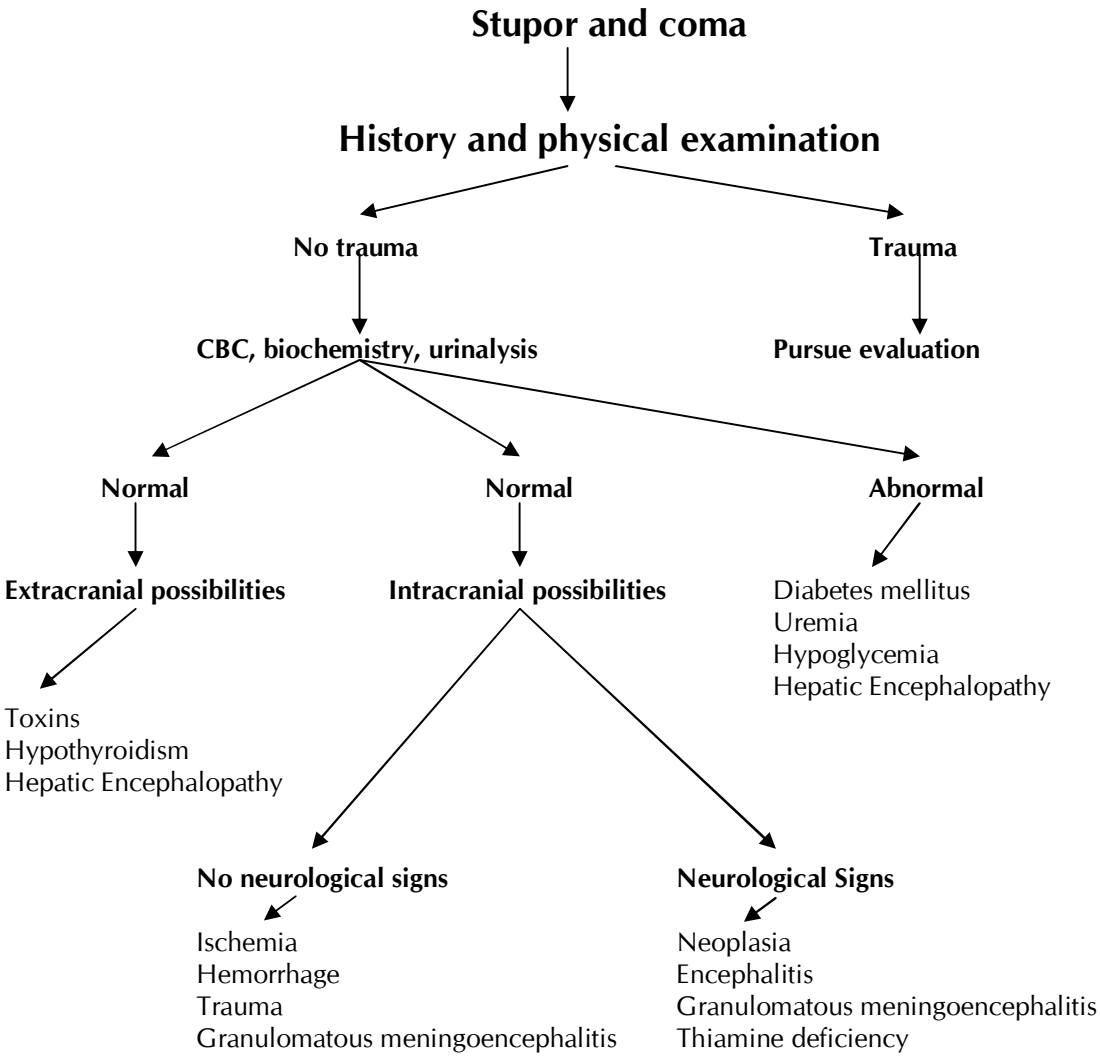
Clinical Signs in Coma

Location of Lesion	Motor Function	Pupillary Light Reflex	Eye Movements
Diffuse Cerebral Disease	tetraparesis, may have locomotor movements but postural reactions are abnormal	normal	normal but no visual following
Metabolic/Toxic Encephalopathy	tetraparesis, reflexes may be depressed	may be normal or abnormal depending on etiology	normal or abnormal depending on etiology
Bilateral Tentorial Herniation	tetraparesis, increased extensor tone (decerebrate rigidity)	dilated or mid-position unresponsive	bilateral ventrolateral strabismus poor vestibular eye movements
Unilateral Tentorial Herniation	hemiparesis or tetraparesis, increased extensor tone on affected side	dilated ipsilateral	ipsilateral ventrolateral strabismus poor vestibular eye movements
Brainstem Hemorrhage	tetraparesis with decerebrate rigidity	bilateral midposition	no vestibular eye movements may have bilateral ventrolateral strabismus

Location of Lesions Causing Stupor and Coma

Location of lesion	Possible Clinical Signs
Cerebrum	• Seizures • Normal or constricted pupils that respond to light • Roving eye movements • Cheyne-Stokes respirations
Midbrain	• Hyperventilation • Loss of oculocephalic response • Negative caloric test • Pinpoint or dilated pupils that do not respond to light
Medulla	• Irregular respiration pattern • Cardiac arrhythmias, or irregular heart rate and rhythm

Diagnostic Approach to the Patient with Stupor and Coma



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