Table 81. Causes of Ataxia

Form of Ataxia	Major Causes	Other Causes
Acute ataxia	Ingestion	Migraine
	Postinfectious cerebellitis	Neuroblastoma
Acute recurrent ataxia	Migraine	. . .
	Metabolic disease	
Chronic ataxia	Congenital disorders with mental deficiency	. . .
Chronic progressive ataxia	Brain tumors	Ataxia-telangiectasia
	Neuroectodermal tumors	Friedreich ataxia

Table 82. Glasgow Coma Scale

Eyes open		Best motor response	
Spontaneously	4	Obey commands	6
To speech	3	Localize pain	5
To pain	2	Withdrawal	4
None	1	Flexion to pain	3
Best verbal response		Extension to pain	2
Oriented	5	None	1
Confused	4		
Inappropriate	3		
Incomprehensible	2		
None	1		

Adapted from Fleisher G, Ludwig S, eds. *Textbook of pediatric emergency medicine*, 3rd ed. Baltimore: Williams & Wilkins, 1993:272.

Table 83. Glasgow Coma Scale (GCS) for Adults and Children and Modified Score for Infants

	Glasgow Coma Score (Adults/Older Children)		Modified Glasgow Coma Score (Infants)
Eye Opening	Spontaneous	4	Spontaneous
	To verbal stimuli	3	To speech
	To pain	2	To pain
	None	1	None
Best Verbal Response	Oriented	5	Coos and babbles
	Confused speech	4	Irritable, cries
	Inappropriate words	3	Cries to pain
	Nonspecific sounds	2	Moans to pain
	None	1	None
Best Motor Response	Follows commands	6	Normal spontaneous movements
	Localizes pain	5	Withdraws to touch
	Withdraws to pain	4	Withdraws to pain
	Flexes to pain	3	Abnormal flexion
	Extends to pain	2	Abnormal extension
	None	1	None

Table 84. Drugs that Can Cause Delirium or Coma

Drug	Physical Findings
Barbiturates	Small, reactive pupils; hypothermia; flaccidity; doll's eye reflex may be absent
Opiates	Pinpoint, reactive pupils; hypothermia; hypotension; hypoventilation; bradycardia
Psychedelics	Small, reactive pupils; hypertension; hyperventilation; dystonic posturing
Amphetamines	Dilated pupils, hyperthermia, hypertension, tachycardia, arrhythmia
Cocaine	Dilated pupils, hyperthermia, tachycardia
Atropine-scopolamine	Dilated pupils; hyperthermia; flushing; hot, dry skin; supraventricular tachycardia
Glutethimide	Midposition, irregular fixed pupils; hypothermia; flaccidity
Tricyclic antidepressants	Hyperthermia, hypotension, supraventricular tachycardia
Phenothiazines	Hypotension, arrhythmia, dystonia
Methaqualone	Same as with barbiturates; if severe tachycardia, dystonia

From Packer RJ, Berman PH. Coma. In: Fleisher GR, Ludwig S, eds. *Textbook of pediatric emergency medicine*, 3rd ed. Baltimore: Williams & Wilkins, 1993:126, with permission.

Table 85. Prognostic Indicators of Poor Neurologic Outcome in Near-Drowning Victims[a]

At the scene
Submersion time >4–10 minutes
Delay in beginning CPR
Resuscitation >25 minutes
In the emergency department
Necessity for CPR
Fixed, dilated pupils
pH <7.0
GCS score <5
After initial resuscitation
Persistent GCS score <5
Persistent apnea

CPR, cardiopulmonary resuscitation; GCS, Glasgow Coma Scale.
[a] Applies to victims of warm water near-drownings only. Hypothermic victims of cold water near-drownings may have a better prognosis.

Table 86. Relationship of the Lesion to the Physical Findings

Lesion	Findings
Upper motor neuron involving corticospinal tract, thalamus, centrum, semiovale, motor cortex	Altered, normal or increased reflexes, bulk normal; strength normal or decreased
Cerebellum	Uncoordinated
Spinal (upper and lower motor)	Local pain, bowel and bladder dysfunction, if anterior horn cells involved, weakness and bulk, decreased absent reflexes, fasciculations
Peripheral	Loss of distal muscles, fasciculations less than spinal lesions; sensation is affected
Muscle	Weakness, muscle atrophy, decreased reflexes, pain, cramping, stiffness
Corticospinal tract	Increased tone, clasp knife character in flexion of arms and extension of legs
Extrapyramidal (basal ganglia)	Rigidity, normal reflexes, absent Babinski, voluntary movement is preserved, may have tremor, chorea, athetosis or dystonia

Table 87. Poisons Causing Coma

Coma with miosis
- Barbiturates and other sedative/hypnotics
- Bromide
- Chloral hydrate
- Clonidine
- Ethanol
- Narcotics
- Organophosphates
- PCP
- Phenothiazines
- Tetrahydrozoline

Coma with mydriasis
- Atropine/diphenoxylate
- Carbon monoxide
- Cyanide
- Cyclic antidepressants
- Glutethimide
- LSD

LSD, lysergic acid diethylamide; PCP, phencyclidine hydrochloride.

Table 88. Poisons Causing Seizures

- Amoxapine
- Amphetamines
- Anticonvulsants
  - Phenytoin
  - Carbamazepine
- Antihistamines and anticholinergic drugs or plants
- Camphor
- Carbon monoxide
- Chlorinated hydrocarbons
- Cocaine
- Cyanide
- Cyclic antidepressants
- Isoniazid
- Lead
- Lidocaine
- Meperidine
- PCP
- Phenothiazines
- Phenylpropanolamine
- Propoxyphene
- Propranolol
- Theophylline

PCP, phencyclidine hydrochloride.

Table 89. Differential Diagnosis of Metabolic Neurologic Dysfunction

Prominent Symptom	Diagnoses to Consider	Diagnostic Test	Metabolic Therapy
Myoclonic seizures	Ceroid	DNA, tissue EM	
	Lafora body disease	Muscle biopsy	
	Prion (GSS)	DNA	
	Mitochondrial	DNA, muscle biopsy	CoQ, other vitamins
	Aminoacidopathies	Blood biochemistry	Dietary
	Biotinidase deficiency	Blood biochemistry	Biotin supplement
	Organic acidurias	Blood biochemistry	Dietary, vitamins
Stroke	Homocystinuria	Blood/Urine test	B vitamins, betaine
	Mitochondrial	DNA, muscle biopsy	
Coma	Organic aciduria	Blood/Urine test	
	MSUD	Blood/Urine test	
	Hyperammonemias	Blood test	Dietary
Spasticity	Leukodystrophy	MRI, fibroblast analysis	Dietary
Visual loss	Leukodystrophy	MRI	
	Mitochondrial	DNA, muscle biopsy	
Psychosis	Leukodystrophy	MRI, blood biochemistry, DNA, fibroblast analysis	
	Porphyria	Blood/Urine	Avoid precipitants
		Biochemistry	
	Wilson disease	Copper excretion, DNA	Penicillamine
	Homocystinuria	Blood biochemistry	(See above)
	Ceroid	DNA, tissue electron microscopy	
	Huntington disease	DNA	
Microcephaly	Ceroid	DNA, tissue EM	
	Rett syndrome	(Clinical features)	
	Krabbe disease	Blood biochemistry	
Macrocephaly	Storage disorders	DNA, blood biochemistry	
	Canavan disease	Urine biochemistry, DNA	
Neuropathy	Krabbe disease	MRI and blood biochemistry	
	Metachromatic leukodystrophy		
	Porphyria	Blood/Urine biochemistry	
	Mitochondrial	DNA, muscle biopsy	
	Friedreich ataxia	(Clinical features)	
	Abetalipoproteinemia		
	Disorders/deficiency of vitamin E	Blood biochemistry	Vitamin E
	Mitochondrial	Vitamin E level	Vitamin E
	Neuroaxonal dystrophy	DNA, muscle biopsy	
		MRI, nerve biopsy	
Myopathy	Fukuyama disease	MRI	
	Mitochondrial	DNA, muscle biopsy	
	Lactic acidoses	Blood biochemistry	
Ataxia	Ataxia telangiectasia	DNA	
	Leukodystrophies	MRI, blood biochemistry	
	Friedreich		
	Mitochondrial	(Clinical features)	
	Hartnup	DNA, muscle biopsy	
	Hyperammonemias	Blood biochemistry	
	Abetalipoproteinemia	Blood biochemistry	
	Sphingolipidoses	Blood biochemistry	
	Machado-Joseph, SCA-1 (hereditary ataxias)	Blood biochemistry, fibroblast analysis, DNA	

Table 90. Acquired Disorders Associated with Progressive Neurologic Dysfunction

Structural	Hormonal	Infectious	Environmental	Toxic	Immunologic
Hydrocephalus Brain tumor Vascular anomalies	Hypothyroidism Congenital adrenal hyperplasia (visuospatial deficits)	SSPE HIV Spirochetes	Malnutrition/Malabsorption syndromes Vitamin/Trace element deficiency (niacin, thiamine, folic acid, vitamin E, B_{12}, essential fatty acids) Physical abuse/neglect	Lead Organic chemicals Carbon monoxide Cocaine, hallucinogens, hypnotics Phenytoin (cerebellar degeneration)	Demyelination/Multiple sclerosis Opsoclonus/Myoclonus or cerebellar ataxia (neuroblastoma) Sydenham chorea Rasmussen encephalitis

Table 91. Epidural versus Acute Subdural Hematoma

	Epidural Hematoma	Subdural Hematoma
Common mechanism	Blunt direct trauma, frequently to parietal region	Acceleration-deceleration injury
Etiology	Arterial or venous	Venous (bridging veins below dura)
Incidence	Uncommon	Common
Peak age	Usually >2 years	Usually <1 year Peak at 6 months
Location	Unilateral Commonly parietal	75% bilateral Diffuse, over cerebral hemispheres
Skull fracture	Common	Uncommon
Associated seizures	Uncommon	Common
Retinal hemorrhages	Rare	Common
Decreased level of consciousness	Common	Almost always
Mortality	Rare	Uncommon
Morbidity in survivors	Low	High
Clinical findings	Dilated ipsilateral pupil, contralateral hemiparesis	Decreased level of consciousness
	Period of lucidity prior to acute decompensation and rapid progression to herniation	Irritability, lethargy
Onset	Acute	Acute (within 24 hours), subacute (within 1 day–2 weeks), or chronic (after 2 weeks)
Findings on CT	Convex "lens-shaped" cerebral hemisphere	Concave, diffusely surrounding cerebral hemisphere

CT, computed tomography.