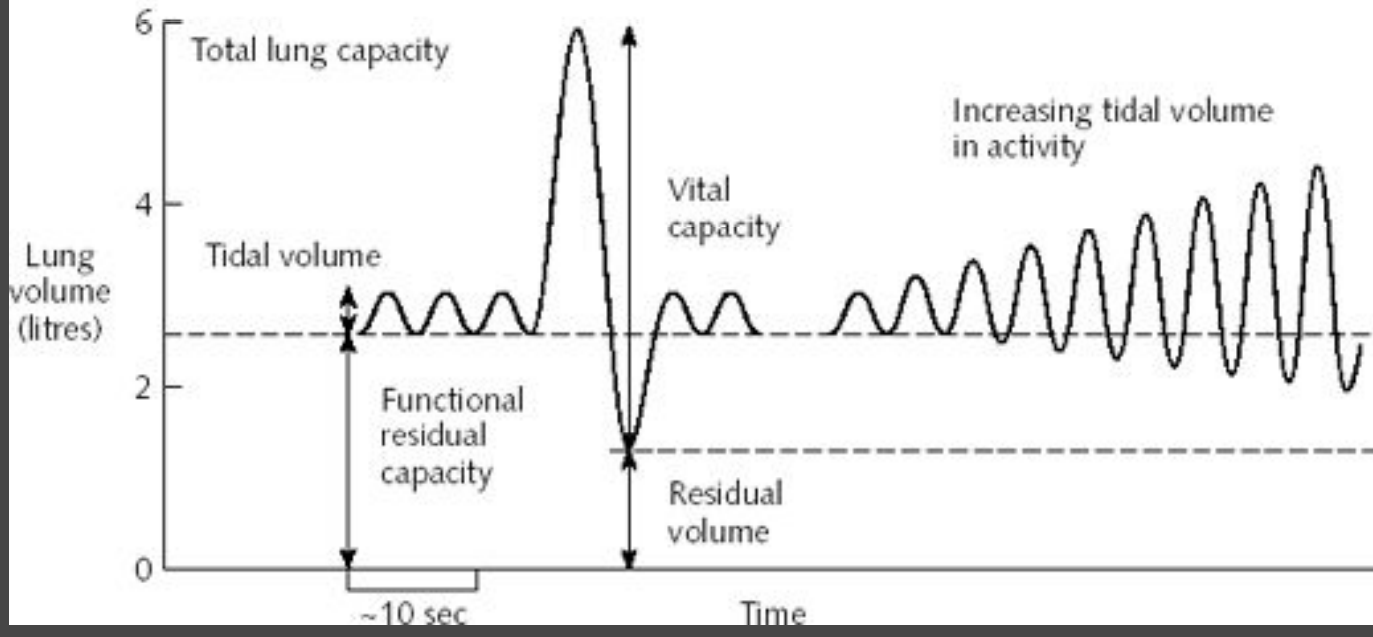
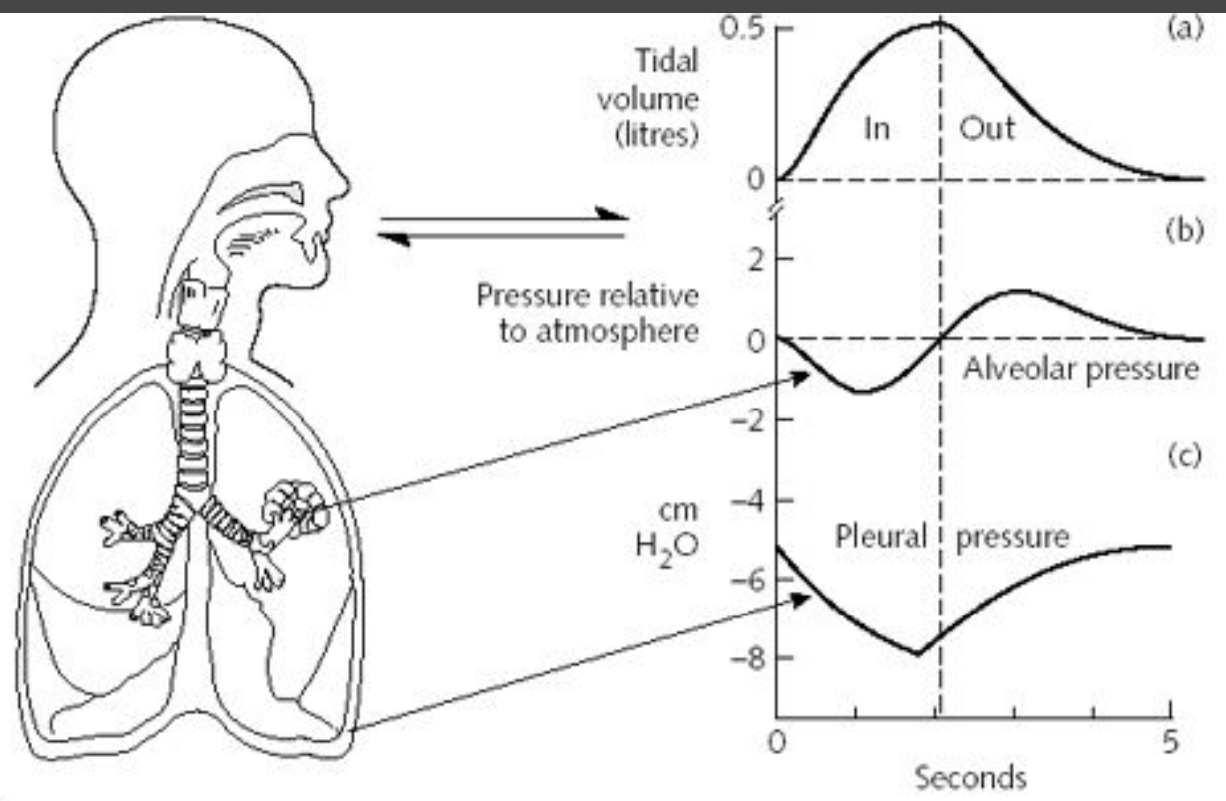


NEUROPULMONOLOGY 101



William M. Coplin, MD, FCCM



Respiratory Patterns in Coma

- ❑ Cheyne – Stokes respiration: bilateral hemispherical dysfunction
 - ▣ Or congestive heart failure
- ❑ Central reflex hyperpnea: midbrain dysfunction causing neurogenic pulmonary edema
 - ▣ Rarely see true central neurogenic hyperventilation with this lesion; central hyperventilation is common with increased ICP

Respiratory Patterns in Coma

- ❑ Apneustic respiration (inspiratory cramp lasting up to 30 sec): pontine lesion
- ❑ Cluster breathing (Biot breathing): pontine lesion
- ❑ Ataxic respiration: pontomedullary junction lesion

Increased Intracranial Pressure

- Hyperventilation ($\text{PaCO}_2 < 35 \text{ mmHg}$) works by decreasing blood flow and should be reserved for emergency treatment and only for brief periods.
 - ▣ Major determinant of arteriolar caliber is the extracellular pH, not actually the PaCO_2 , but this is the parameter we can control.

Classification of Neurogenic Respiratory Failure

- ❑ Oxygenation failure (low PaO₂)
 - ❑ primary difficulty with gas transport
 - ❑ usually reflects pulmonary parenchymal disease, V/Q mismatch, or shunting
- ❑ Primary neurologic cause is neurogenic pulmonary edema.

Neurogenic Pulmonary Edema

- A state of increased lung water (interstitial and sometimes alveolar):
 - ▣ as a consequence of acute nervous system disease
 - ▣ in the absence of
 - - cardiac disorders (CHF),
 - - pulmonary disorders (ARDS), or
 - - hypervolemia

Causes of Neurogenic Pulmonary Edema



Common

- SAH
- head trauma
- intracerebral hemorrhage
- seizures or status epilepticus



Rare

- medullary tumors
- multiple sclerosis
- spinal cord infarction
- Guillain-Barré syndrome
- miscellaneous conditions causing
- intracranial hypertension
- many case reports of other conditions

Classification of Neurogenic Respiratory Failure

- ❑ Ventilatory failure (inadequate minute ventilation [VE] for the volume of CO₂ produced):
 - ❑ In central respiratory failure, the brainstem response to CO₂ is inadequate, and the PaCO₂ begins to rise early.
 - ❑ In neuromuscular ventilatory failure, the tidal volume begins to fall, and the PaCO₂ is initially normal (or low).

Causes of Neurogenic Ventilatory Failure

- Most common causes are:
 - ▣ Myasthenia gravis
 - ▣ Guillain-Barré syndrome
 - ▣ Critical illness polyneuropathy, myopathy
 - ▣ Cervical spine disease

- Many rarer causes

Management of Neurogenic Ventilatory Failure

- Airway protection and mechanical ventilation
 - ▣ Don't wait for the PaCO₂ to rise
- Specific therapies
 - ▣ Myasthenia: IgIV, plasma exchange
 - ▣ Guillain-Barré: plasma exchange, IgIV
 - ▣ Critical illness polyneuropathy, myopathy: time

STROKE AND THE LUNGS



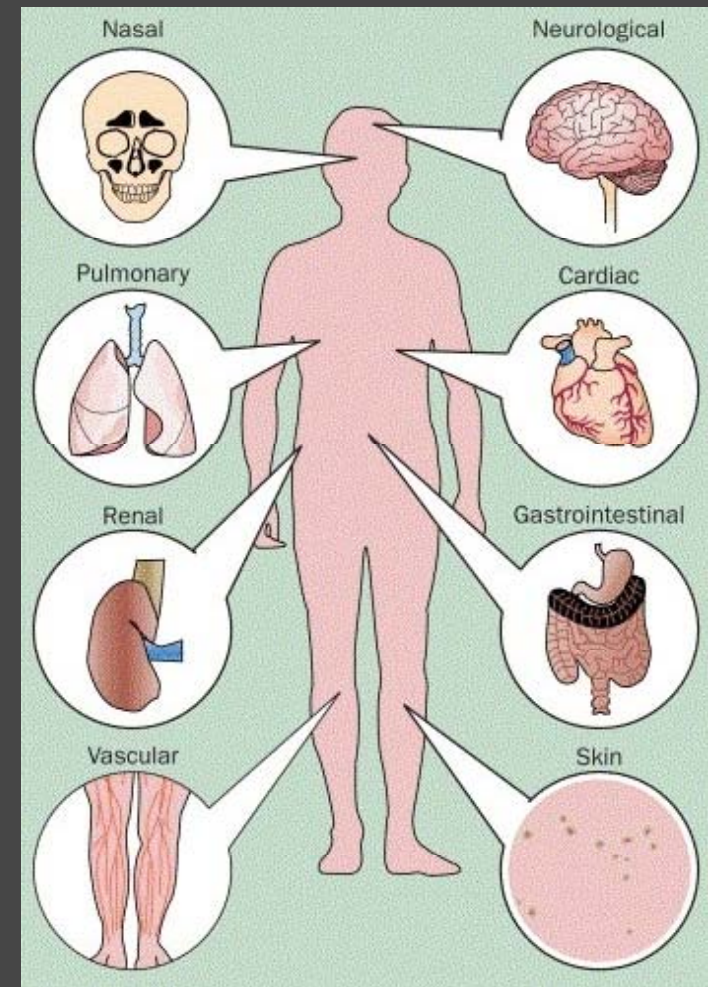
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Stages of Brain Damage after Stroke

- Primary - occurs at moment of injury
 - ▣ Ischemia
 - ▣ Intracranial hemorrhage
- Secondary - initiated at moment of injury
 - ▣ Ischemia
 - ▣ Neuronal injury cascade
 - ▣ Edema (elevated intracranial pressure)
 - ▣ Delayed hematoma formation
 - ▣ Exposure to secondary brain insults

General Management of Neurological Patients

- ❑ Airway protection
- ❑ Blood pressure management
- ❑ Neurological examination
- ❑ Antiplatelet/anticoagulant therapy
- ❑ Volume state
- ❑ Fever control
- ❑ Blood glucose control
- ❑ Neuroimaging
- ❑ Vascular access and monitoring
- ❑ Agitation control
- ❑ Avoidance of harmful medications
- ❑ Dysphagia and nutrition



Common Complications of Neurological Patient Management

- ❑ Infection
- ❑ Dysrhythmia and coronary ischemia
- ❑ Cerebral edema and elevated ICP
- ❑ Hyperosmolar therapy
- ❑ Hyperventilation
- ❑ Other medical interventions
- ❑ Seizures
- ❑ Deep venous thrombosis and pulmonary embolus
- ❑ Alcohol and substance use/abuse
- ❑ Contrast nephropathy
- ❑ Hemorrhagic complications of stroke
- ❑ Anticoagulation after stroke

Secondary Brain Injury

- ❑ Ischemia
- ❑ Edema (leading to elevated ICP)
- ❑ Delayed hematoma formation
- ❑ Apoptotic cell injury cascade
 - ▣ Neuronal apoptosis in gray matter at 48 hours
 - ▣ Oligodendroglial apoptosis in WM at 2 weeks

The primary focus of Emergency and Neurocritical Care for stroke is the prevention, identification, and treatment of secondary brain injury

Secondary Injury In Neurological Patients

- Hypoxia and hypotension are the two major causes of secondary CNS injury following neurovascular disease
- Even in the best intensive care units, these complications occur frequently
- Preventing hypoxia and hypotension could have the greatest effect of any currently available treatment for neurovascular disease

Alphabet Soup

- Airway
- Breathing
- Circulation perfusion pressure
- Disability / DVT prophylaxis
- Edema
- Food
- Glucose / GI prophylaxis
- Hemicraniectomy/Hypothermia
- Invasive monitoring

Stroke: Acute Resuscitation

Overall Goal: Perfuse and Oxygenate the Brain

- Pulmonary edema
 - ▣ FiO₂ & PEEP
 - ▣ Diuresis
 - ▣ Inotropes (dobutamine)
- Hypotension (MAP <70 mm Hg)
 - ▣ Volume
 - ▣ Catecholamines (norepinephrine)
 - ▣ IABP
- Hypertension (MAP >130 mm Hg)
 - ▣ Nicardipine or labetalol
- Normoglycemia
 - ▣ Insulin drip
- Induced normothermia
- Induced hypothermia

Monitoring After Stroke

- ❑ Pulse oximetry
- ❑ Blood pressure: at what interval?
 - ▣ Assessment for arterial catheter
- ❑ ECG
- ❑ Vascular access
- ❑ Urine output determination
- ❑ Body temperature reading
- ❑ Possibly central venous pressure
- ❑ Neurophysiological workup?

Clinically Identified Secondary Brain Insults

- Hypoxia (PaO₂ < 60 torr) or hypotension (SBP < 90 mmHg) prior to or during resuscitation (Traumatic Coma Data Bank)

Chesnut et al. J Trauma 34: 216-222, 1993. (1434 patients)

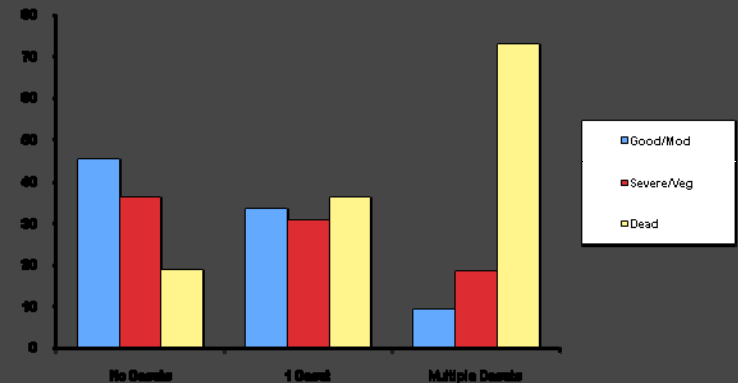
Secondary Insults	% of Total Patients	Good/Mod	Severe/ Vegetative	Dead
Total Cases	100	43.0	20.2	36.8
Neither	43.0	53.9	19.2	26.9
Hypoxia	22.4	50.3	21.7	28.0
Hypotension	11.4	32.9	17.1	50.0
Both	23.2	20.5	22.3	57.2

Secondary Brain Insults

- ❑ Exacerbate injury in vulnerable cells
- ❑ Decreased substrate delivery
 - ▣ Hypotension
 - ▣ Hypoxia
- ❑ Increased metabolism
 - ▣ Fever
 - ▣ Seizures
- ❑ Worsen hemorrhage or edema
 - ▣ Hypertension
- ❑ Cellular toxicity
 - ▣ Hyperglycemia

Secondary Brain Insults and Outcome

- Any occurrence
 - TCDB: hypotension or hypoxia
 - Fever at hospital admission: ICH and AIS
 - Hyperglycemia at hospital admission: AIS
 - ? admission BP (diabetics): ICH expansion
- Number of occurrences
 - S_{ij}vO₂ desaturations
 - < 50% for > 10 min
- Duration/% time above:
 - TCDB: ICP
 - Fever: ICH



What me worry?

- Relative hypoxemia (wide A-a gradient)
 - ▣ As many as 50%?
- Many initially with normal CXR

Neuropulmonology 101

- Pneumonia
 - ▣ Aspiration
 - ▣ Nosocomial
- Hydrostatic pulmonary edema
- Neurogenic pulmonary edema
- Acute lung injury
- ARDS
- Pulmonary embolism

Hydrostatic pulmonary edema

- Congestive heart failure
 - Premorbid
 - Acute onset
 - 12-20% with acute cardiac ischemia
 - Subendocardial vs transmural
 - Dysrhythmias

NPE

- Abrupt onset
- Increased pulmonary vascular permeability
 - ▣ Protein and water extravasation
 - ▣ Post capillary venule constrictors
- Poor lung compliance
- Abnormal gas exchange
- Immune response overdrive
- J/stretch receptors?
 - ▣ Vagal innervation

Causative factors

- ❑ Increased ICP
 - ▣ Blunts reflexive pulmonary vasoconstriction to hypoxia
- ❑ Intracranial hemorrhage
- ❑ Seizures
- ❑ Hypothalamic and medullary lesions
- ❑ Aspiration
- ❑ Drugs
 - ▣ Heterocyclics
 - ▣ Catecholamines
 - ▣ CCBs

ARDS

- Acute onset respiratory failure
 - ▣ $\text{PaO}_2/\text{FiO}_2 \leq 200$ (300)
- Radiographic bilateral infiltrates
- Absence of clinical evidence of left heart failure
- Annual incidence 31-75/100,000
- Mortality 30-50%
- Surfactant

Pneumonia

- ❑ Fever
- ❑ Leukocytosis
- ❑ New/progressive roentgenographic infiltrate
- ❑ Purulent sputum
- ❑ Med-Surg ICU incidence 10-11%
- ❑ Neuro-ICU incidence 26-42%

Aspiration pneumonia

- ❑ ASPIRATION DOES NOT NECESSARILY MEAN PATIENTS WILL DEVELOP PNEUMONIA!!!
- ❑ When/if they do
 - ▣ Oral flora
 - Penicillin/ampicillin
 - Clindamycin

Nosocomial pneumonia

- Oral/GI
 - ▣ Strep./Enterococci (Amp)
 - ▣ Bacteroides
- LF GNR
 - ▣ Coliforms (Amp, TMP/SMZ...)
- NLF Ox+ GNR
 - ▣ Pseudomonads (UreidoPCN or Ceph 3/4 and APAG)
- NLF Ox- GNR
 - ▣ Acinetobacter (AM/SB >> carbapenems)
- S. aureus (Nafcillin >> vancomycin)

Pulmonary embolism

- Incidence of DVT about 11%
 - ▣ Thromboxane A2 release
 - ▣ Inactivity/paresis
- Incidence of PE < 4%
- Relation to instrumentation
- PIOPED study

Intubation: therapy or marker?

- Laryngeal instrumentation
 - ▣ Increased BP and ICP
 - ▣ Risk for secondary ischemia or rebleeding
- Indications
 - ▣ Neurological deterioration
 - Inability to protect airway
 - ▣ Prolonged seizures
 - ▣ Hypercarbia (e.g., PaCO₂ > 60 mm Hg)
 - ▣ Hypoxemia
 - ▣ Increased WOB (e.g., RR > 40/min)
 - ▣ Left heart failure with pulmonary edema

Intubation: therapy or marker?

- 30-day mortality with MV after stroke is 65%
 - ▣ ICH > ischemic stroke
- Predictors
 - ▣ GCS < 10
 - ▣ Clinical deterioration after intubation
 - ▣ Brainstem dysfunction
 - ▣ Cardiac ischemia
 - ▣ Older age
 - ▣ Early in course of stroke
 - ▣ Oxygenation problems (vs. ventilatory)
 - ▣ Fever

VENTILATOR MANAGEMENT IN NEUROLOGICAL DISEASE

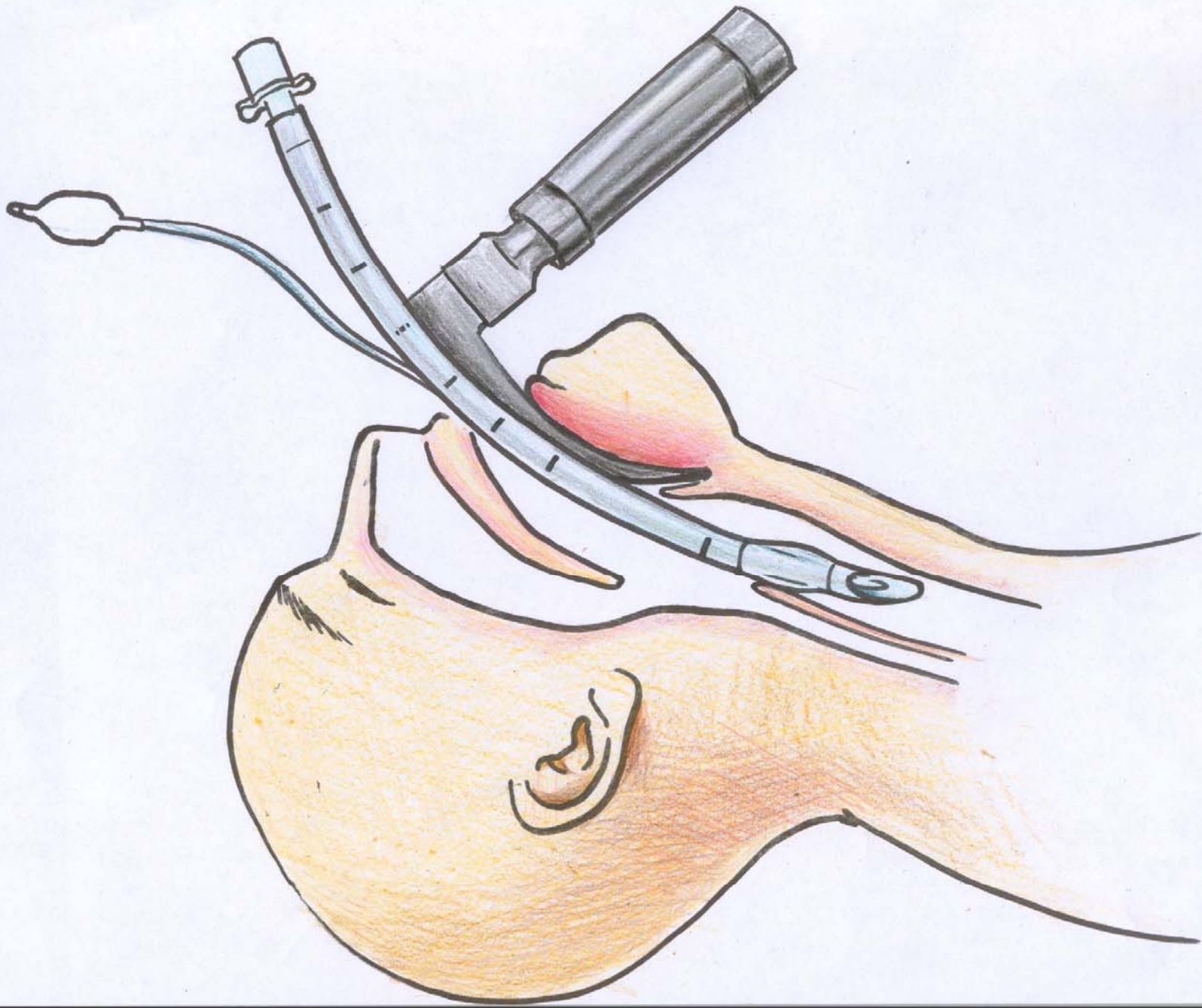


WAYNE STATE
UNIVERSITY

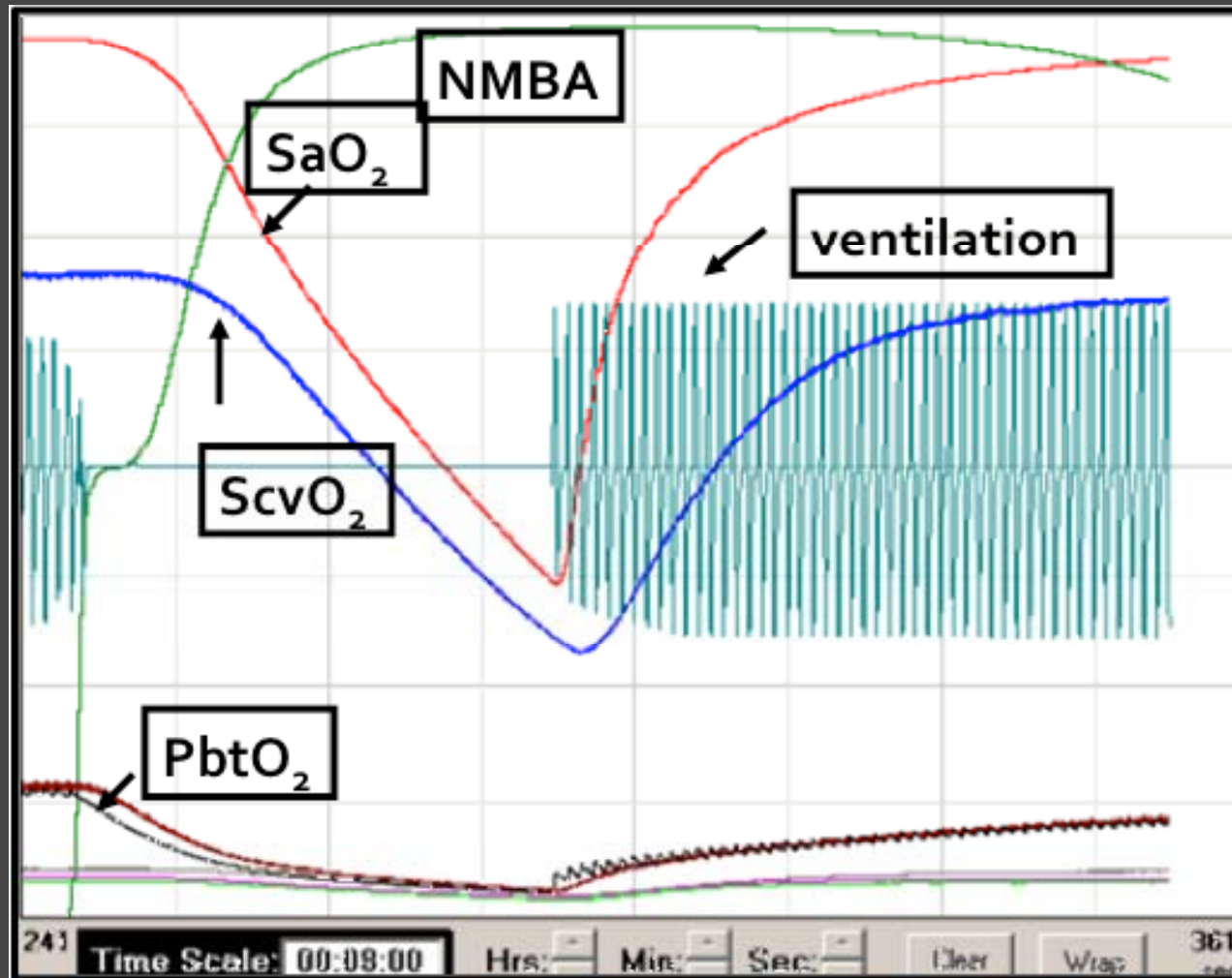
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Endotracheal Intubation and Mechanical Ventilation

- Essential to resuscitating patients who have acute brain injury
- Fulfills multiple goals
 - Ensures airway protection
 - Tissue oxygen delivery
 - Indirectly modulates cerebral vascular reactivity
- Risks
 - Ventilator-associated pneumonia (VAP)
 - Ventilator-induced lung injury (VILI)
 - Delirium
 - Frequent need for sedation
 - Decreases sensitivity of neurological assessment
- Positive pressure ventilation may adversely affect cerebral perfusion pressure (CPP)
 - Importance of effect may be overestimated in most clinical settings
- Patients who have severe brain injury at increased risk for acute lung injury/acute respiratory distress syndrome (ALI/ARDS)
 - May develop VILI
- With concurrent intracranial hypertension and ALI/ARDS, therapies aimed at optimizing brain physiology may conflict with MV strategies aimed at lung protection



Time \downarrow SaO₂ = 3 min 36 sec



Airway Protection

- 8% deteriorate and get intubated
 - ▣ Bulbar dysfunction
 - ▣ Decreased level of consciousness
 - ▣ Aspiration of gastric contents or oral secretions
 - ▣ Concomitant medical process marked by cardiopulmonary failure
- Airway failure
 - ▣ Impaired cerebral oxygen delivery
 - ▣ Aspiration pneumonia
 - ▣ Increased agitation related to ventilatory/respiratory distress with elevated intracranial pressure

Stroke

- 65% 30-day mortality for mechanically ventilated stroke patients
 - ▣ No difference among stroke subtypes
 - ▣ Predictors: GCS < 10, deterioration after intubation, brainstem dysfunction, ischemic cardiomyopathy
 - Mayer, 2000
- Mortality slightly higher in patients with intracranial hemorrhage
 - ▣ Predictors: older age, early intubation
 - Gujjar, 1998

Airway, Breathing, Oxygenation...

- Intubated patient should be constantly monitored and treated for
 - ▣ Agitation
 - ▣ Uncleared secretions
 - ▣ Hypoxemia
- PaO₂ ≥ 80 mm Hg
- SaO₂ ≥ 95%?
 - ▣ Some concern that excessive oxygenation may lead to free radical production and additional secondary cerebral injury

Mechanical Ventilation

- Minimize intrathoracic pressure
 - ▣ May decrease ICP via enhanced drainage
- PEEP
 - ▣ No effect on ICP up to 12-15 cm H₂O?
- Mode
 - ▣ No clear relation to ICP
 - Including IRV
 - Clarke, 1997
- Endotracheal suctioning -- pretreat!

Keystone Initiative: the Ventilator Bundle

- Group of interventions with common purpose
- Ventilator bundle consists of six things, all of which together reduce risk of infection
 - ▣ Elevate head of bed to at least 30 degrees
 - ▣ Appropriate sedation (daily “sedation vacation”)
 - ▣ Daily assessment of readiness to extubate
 - ▣ Venous thromboembolism prophylaxis
 - ▣ Peptic ulcer prophylaxis
 - ▣ Tight glucose control
 - ▣ Chlorhexidine oral swabs

Prophylactic Hyp~~er~~ventilation

