Neurocritical Care & Multimodality Monitoring (MMM)

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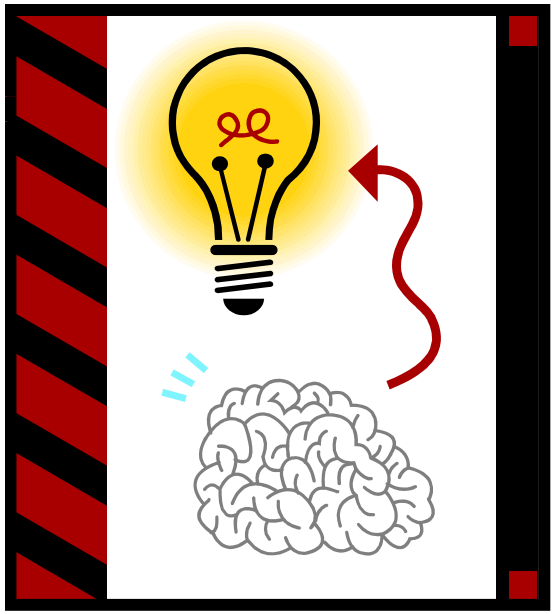
- COI—Masimo

Why neuro is so EASY☺



- Only 2 things can go wrong:
  - Too much stuff in there
  - Not enough blood flow
- *Change is the name of the game!*
- Figure out which problem you have and where & the basic management will follow

What Is Mulitimodality Monitoring

- Combined use of multiple monitors
  - Clinical evaluation correlated with:

Systemic hemodynamics	Glucose and nutrition
ICP & CPP	Hemostasis and hemoglobin
Cerebral autoregulation	Temperature and inflammation
Systemic and brain oxygenation	Cellular damage and degeneration
Cerebral blood flow	ICU processes of care
Electrophysiology	Informatics, data integration, display, and analysis
Cerebral metabolism	Monitoring in emerging technologies

Why Is MMM Important?

- Supplement to the clinical examination of the patient
  - Neuro exam is often confounded by disease progression, sedation/analgesia/neuromuscular blockade or coma
- Some monitors have strong anecdotal evidence of benefit
- Physiologic information from several monitors may translate into outcome differences for some patients
- Impact on prognostication, management and outcome

Why We Monitor Patients With Neurologic Disorders Who Require Critical Care

- Detect early neurological worsening before irreversible brain damage occurs
- Individualize patient care decisions
- Guide patient management
- Monitor the physiologic response to treatment and to avoid adverse effects
- Allow clinicians to better understand the pathophysiology of complex disorders
- Improve neurologic outcome and quality of life of survivors
- Begin to develop new mechanistically oriented therapies where treatments are currently lacking or are empiric in nature

The Consensus Group

- Who they are
- What they did
- How they came up with the consensus statements
- Intention of the consensus statements
  - Acute Brain Injury (ABI)

The Recommendations

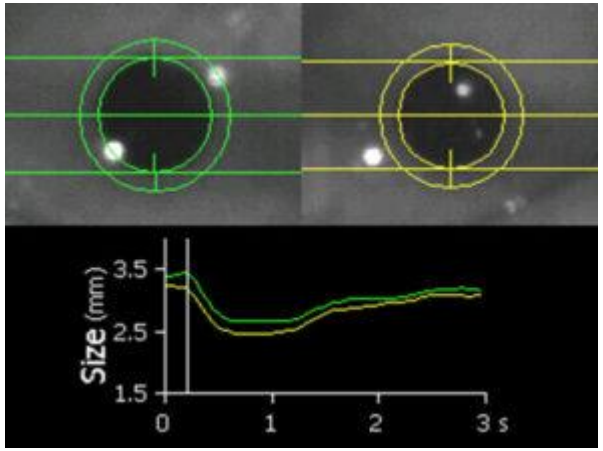
Clinical Exam

- Still remains an essential key

Pupillometer

- Numerical value (index) of pupil size and reactivity
- Trend over time
- Important key assessment in clinical exam
- Removes clinician discrepancies/exam subjectivity

Measured Value*	Assessment
3.0 – 4.9	Normal/“Brisk”
< 3.0	Abnormal/“Sluggish”
0	Non-Reactive or Atypical Response



GCS vs FOUR Score

- Use and accuracy of one over the other

Pain Scales

- NRS vs BPS vs CPOT vs NCS-R

Sedation Scales

- SAS and RASS

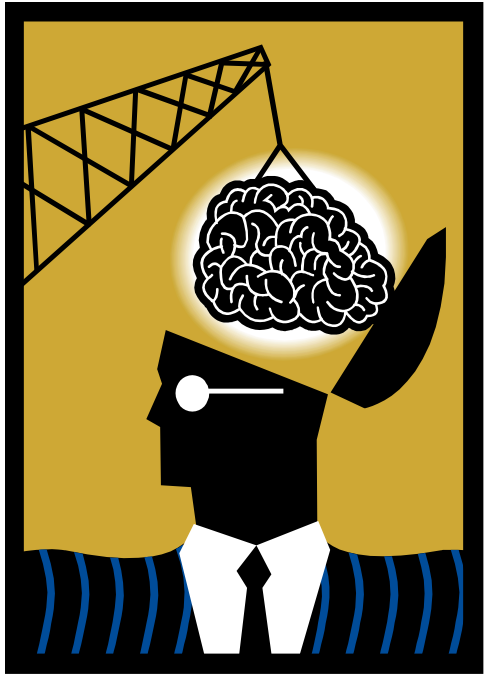
Delirium Prevention

- Wake-up tests
- CAM-ICU vs ICDSC

Systemic Hemodynamics

- BP, HR, pulse ox
- Baseline assessment of cardiac function with echocardiography

Monroe-Kellie Hypothesis



- Skull is a rigid compartment
  - Contains 3 components:
    - Brain tissue (80%)
    - Arterial and venous blood (10%)
    - Cerebrospinal fluid (10%)
- Components are balanced in a state of dynamic equilibrium
  - If an increase occurs in the relative volume of one component the volume of one or more of the other components must decrease or an elevation in ICP will result
  - Small increases in volume result in large increases in ICP
  - Slow vs rapid accumulations

ICP & CPP

- Fundamental to care of patients with ABI
- Threshold of 20 mmHg
- MAP and CPP
- Parenchymal vs EVD

BEST:TRIP

- Summary of study
- Challenges core beliefs in ICP monitoring
- ICP treatment is best guided by ICP monitoring, clinical imaging and clinical evaluation in combination and in the context of a structured protocol

BTF Algorithm For ICP Placement

ALGORITHM FOR THE MANAGEMENT OF INTRACRANIAL HYPERTENSION AND ISCHEMIA IN PATIENTS WITH SEVERE TBI

Place ICP monitor*

ICP > 20

Tier 1

-CSF drainage (continuous or intermittent)
-Low dose mannitol 0.25-1.0/kg bolus

Tier 2

-Therapeutic mild hypothermia T 35-37
-Hypertonic saline by bolus or continuous infusion
-High dose mannitol > 1.0g/kg bolus
-Moderate hyperventilation to PaCO2 30-35 (not recommended in the presence of ischemia)

Tier 3

-High Dose barbiturates
-Neuromuscular blockade
-Decompressive craniectomy in the absence of surgical mass lesions
-Therapeutic Deep hypothermia T 32-34

Ischemia †

Tier 1

-Ventilation manipulation to maintain Pa02 > 90
-CPP manipulation to 50-70 with vasoactive medication

Tier 2

-Therapeutic mild hypothermia T 35-37

Algorithm Options

- Use ICP>20 and/or Ischemia pathways as needed.
- Treatments in a tier box can be used in any order.
- It is not necessary to use all of the modalities in a given tier prior to moving to the next tier.
- There is no rank ordering within a tier.
- When considered advantageous, tiers can be skipped when advancing treatment (e.g. early decompresssive craniectomy.)

Place ICP monitor* for GCS<9 and abnormal CT. Normal CT plus 2 out of 3 of the following parameters is an indicator for ICP Monitoring: GCS motor 1 or 2, Age >40, or SBP < 90 mm Hg at any time. Follow the recommendations in the Guidelines for the Management of Severe TBI-www.braintrauma.org.

Ischemia† in this algorithm is defined in the manuscript text of the Algorithm for Management of Severe TBI-A Systematic Approach for Achieving Consensus.

This algorithm is not intended to be a substitute in any way for care and treatment by a qualified healthcare professional.

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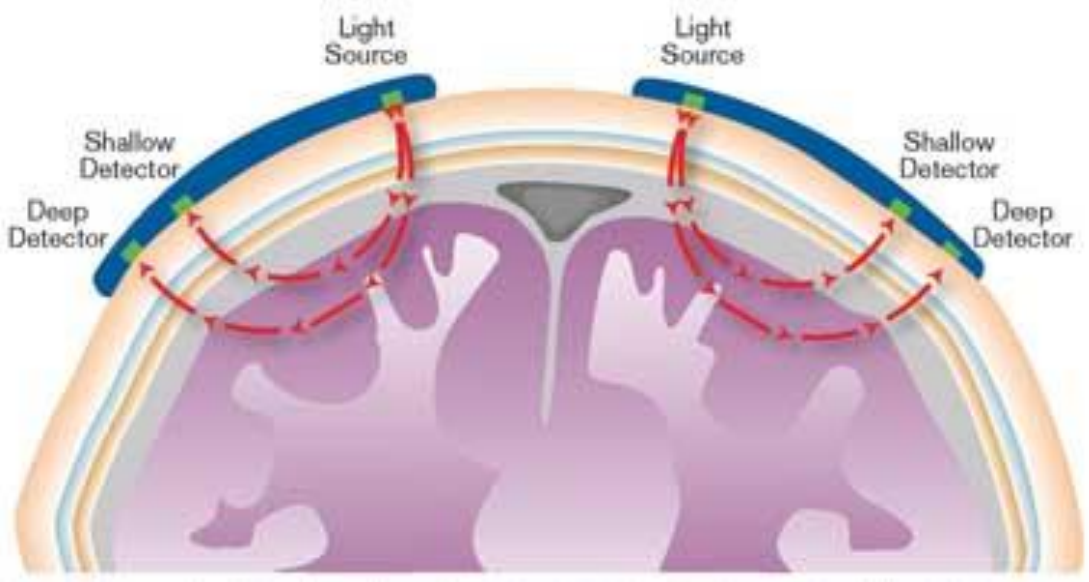
Autoregulation

- PRx and CPPopt
- TCDs, NIRs

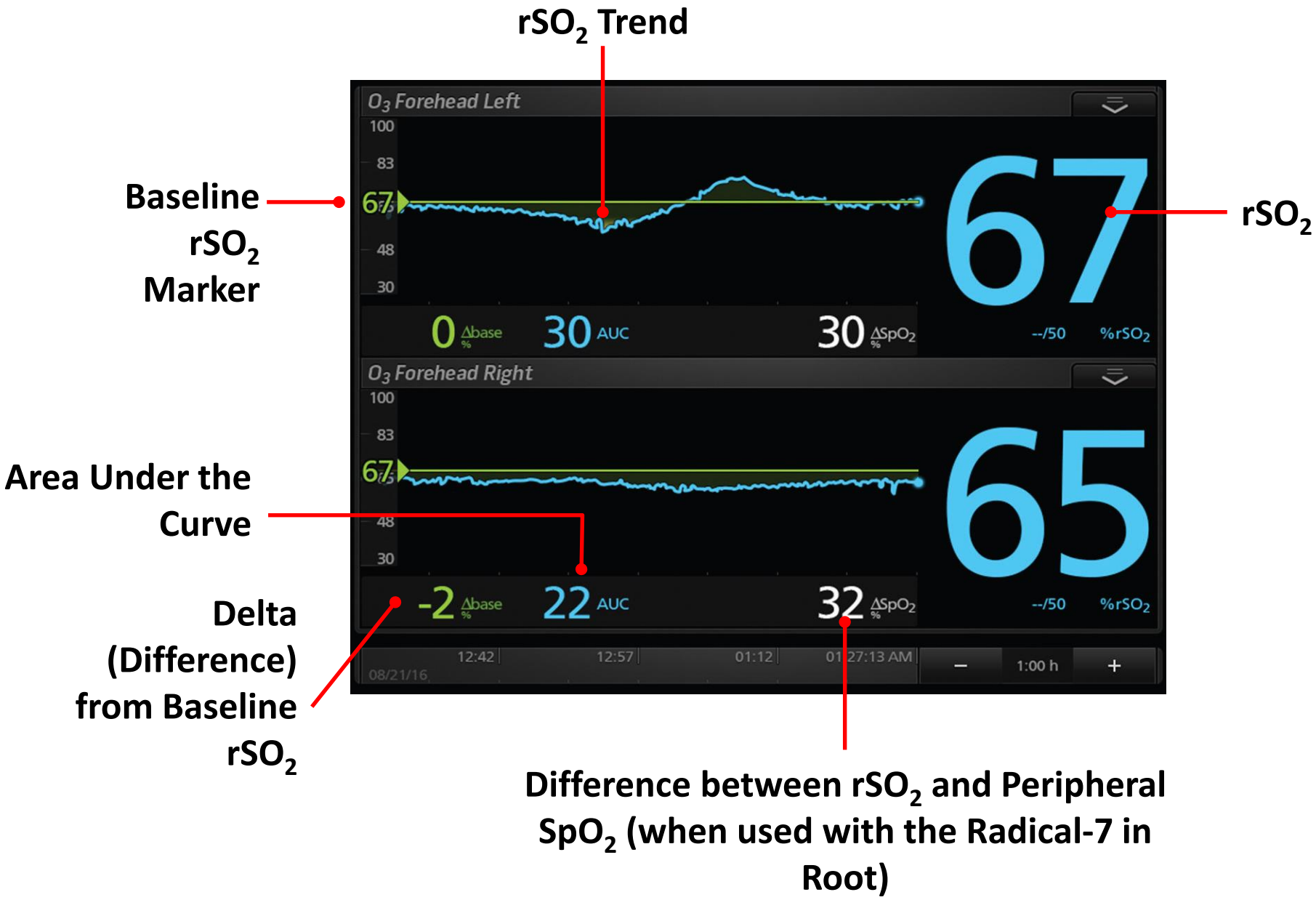
Systemic and Brain Oxygenation

- ABGs
- PaCO2 vs EtCO2
- PbtO2 vs SjvO2

NIRS Technology



- LED source on forehead emits near infrared light.
- Some light is “transflected” to detectors.
- “Shallow” detector is more sensitive to superficial light pathways.
- Light absorbance is converted into Hb oxygen saturation of tissue beneath sensor.
- “Regional O_2 Saturation” -- sensitive to both arterial and venous saturation.
  - **Typically assumes 70% venous blood and 30% arterial blood. This ratio can vary with time.**
- Can use two sensors to assess both hemispheres of brain.


rSO2 Trend
Baseline rSO2 Marker
rSO2
Area Under the Curve
Delta (Difference) from Baseline rSO2
Difference between rSO2 and Peripheral SpO2 (when used with the Radical-7 in Root)
O3 Forehead Left
100
83
67
48
30
0 Δbase %
30 AUC
30 ΔSpO2 %
--/50
%rSO2
67
O3 Forehead Right
100
83
67
48
30
-2 Δbase %
22 AUC
32 ΔSpO2 %
--/50
%rSO2
65
12:42
12:57
01:12
01:27:13 AM
08/21/16
1:00 h

ECMO Study with Brain Oxygenation

Cerebral Blood Flow

- TCD
- Transcranial color-coded duplex sonography
- Thermal dilution flowmetry
- Laser Doppler flowmetry

Electrophysiology

- Continuous EEG
- Evoked potentials

Brain Function Monitoring/Processed EEG

- SCCM Guideline recommendations
- Depth of sedation/anesthesia
  - Delirium prevention from oversedation prevention

- stroke patient on BFM/Sedline monitor (de-identified image)

- patient who is brain dead on BFM/Sedline monitor (de-identified image)

- Video of BFM/Sedline monitor of patient who goes into burst suppression (de-identified video)

Cerebral Microdialysis

- Key parameters
  - Lactate/pyruvate ratio
  - Glycerol
  - Brain glucose
  - Glutamate
- Metabolism assessment
  - Glucose

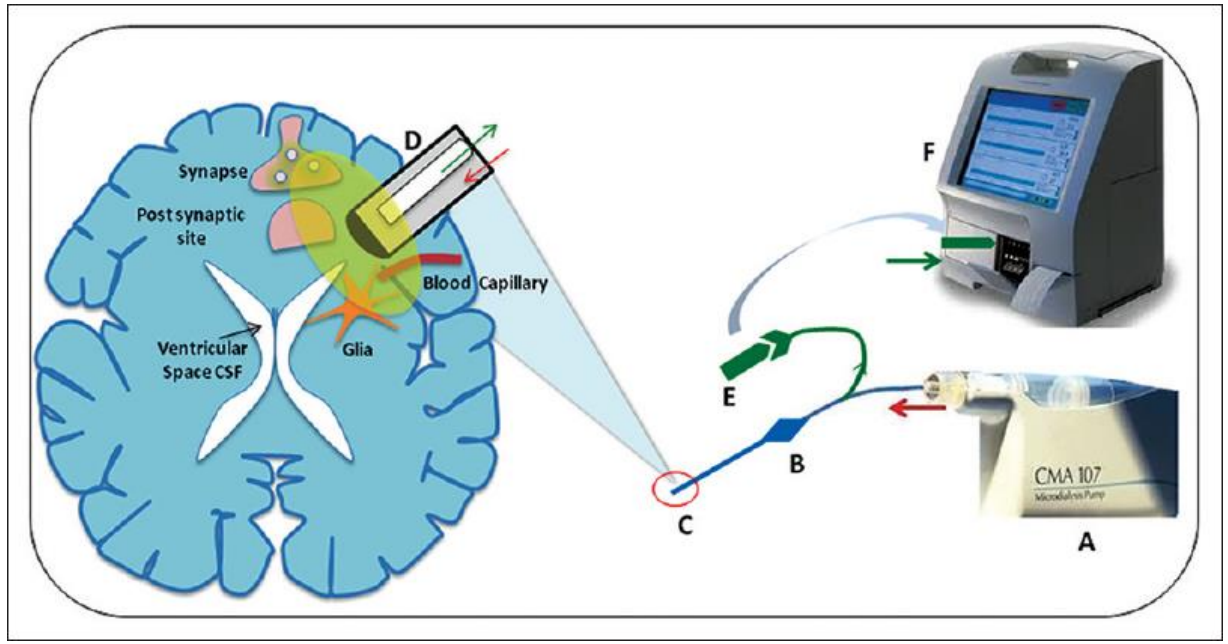


Table 1 Normal and threshold values of metabolic parameters important in neuromonitoring of patients with traumatic brain injury

Parameter	Normal levels	Threshold value
Glucose, mean (SD), mg/dL	30.6 (16.2)	14.4
Pyruvate, mean (SD), mg/dL	1.46 (0.33)	0.27 (fatal)
Lactate mean (SD), mg/dL	26.1 (8.1)	80.2 (fatal)
Lactate to pyruvate ratio	23 (4)	30
Glycerol, mg/dL	184-460	21

SI conversion factors: To convert glucose to mmol/L, multiply by 0.0555; pyruvate to μmol/L, multiply by 113.56; lactate to mmol/L, multiply by 0.111; glycerol to mmol/L, multiply by 0.1086.

Summary

- “Monitors alone cannot save patients, but wise application of the data from monitoring the injured brain can…”