EANS/UEMS European examination in neurosurgery

Variants of questions with answers (compilation - Vyacheslav S. Botev, Department of Neurosurgery, M.Gorky Donetsk National Medical University)

Neurocritical Care

Cerebral Metabolism

1. What is cerebral perfusion pressure (CPP)?
2. What range of cerebral perfusion pressures are accommodated by cerebral autoregulation?
3. CPP should be maintained above what number after a severe head injury?
4. How does one calculate MAP?
5. At what blood flow rate does electrical activity of the cerebral cortex fail?
6. What is cerebral autoregulation?
7. How does pressure autoregulation work?

CPP and ICP

8. What conditions can alter the cerebral metabolic rate of oxygen $CMRO_2$?
9. How does the cerebral metabolic rate of glucose (CMRG) vary in head injury?
10. What does this hyperglycolysis state indicate?
11. What is the normal cerebral blood flow (CBF)?
12. What are the three different groups of patients with severe TBI based on CBF?

ICP Monitoring

13. What is a normal ICP waveform?
14. What are pathological ICP waveforms?
15. Is it ever appropriate to place an ICP monitor in a patient with a normal CT?
16. What is considered an abnormal CT?
17. Is the Camino bolt the “gold standard” for ICP monitoring?
18. What are some of the complications of ventriculostomy catheter placement?
19. What is the only factor that will significantly reduce the risk of infection?
20. What are the factors that are not associated with infection?
21. Does an antibiotic-coated catheter decrease the risk of infection?

22. Is it good practice to routinely exchange EVD catheters to reduce infection risk?
23. What first-tier interventions can be done to decrease ICP?

ICP Management

24. Will ICP always be low after a large intracranial hematoma evacuation?
25. What are some of the factors that contribute to increase ICP?
26. Is a CPP of 70 mm Hg an appropriate goal?
27. Is it OK to have SBP <90 mm Hg, as long as PaO_2 is >90?
28. What is the antihypertensive agent of choice to treat hypertension in TBI?

29. Why are sympathomimetic-blocking antihypertensives the agents of choice to treat hypertension in TBI?

30. Are there selected TBI patients in whom steroids are indicated?

31. What are the measures used to treat medically refractory intracranial hypertension (second tier)?

32. Has the use of propofol supplanted pentobarbital in the management of the TBI patient?
33. Describe the propofol infusion syndrome.
34. What is the dose in which the propofol infusion syndrome can occur?

35. What other alternatives can be used for sedation?

36. Which anesthetic agent decreases CBF and $CRMO_2$ and suppresses adrenocortical response?

37. Which anesthetic agent decreases CBF and $CRMO_2$ and produces cardiovascular depression?

38. Which anesthetic agent induces seizures discharges?
39. What are the major complications of neuromuscular blockade?
40. Is hypertonic saline (HTS) equivalent to mannitol for treatment of elevated ICP?
41. What are the mechanisms by which mannitol exerts its beneficial effect?
42. What are the rheological effects of mannitol?
43. What is important to know about the osmotic effects of mannitol?
44. Why is it better to administer mannitol in boluses that as an infusion?

45. What is the dose of mannitol typically given for reduction of ICP?
46. What are the mechanisms by which hypertonic saline exerts its effect?
47. In which scenario does hypertonic saline have an advantage over mannitol?
48. Is hyperventilation ever indicated in TBI?

49. Explain the rebound effect of hyperventilation.

50. What is the protocol for barbiturate use in the setting of increased ICP? (Is the Eisenberg et al [1988] randomized controlled trial, currently still in use?)

51. What is the mechanism of action of barbiturates to lower ICP?
52. What complications have been reported that can occur during barbiturate coma?
53. Is hypothermia (target 32–33ºC) effective in reducing mortality in TBI patients?
54. What is the risk of vasospasm in TBI?
55. Why is it important to maintain normoglycemia in patients with TBI?
56. What are the measures to treat medically refractory intracranial hypertension?

Advanced Neuromonitoring

57. List the different types of whole-brain monitoring.
58. List the different types of regional/focal brain monitoring.
59. What is the fundamental goal of $SjvO_2$ monitoring?
60. Where should one place the $SjvO_2$ catheter?
61. What are the normal $SjvO_2$ parameters?
62. What $SjvO_2$ level is consistent with irreversible ischemic injury?
63. What abnormal values can we see in TBI?
64. What are the causes of low $SjvO_2$ (50% or less)?
65. What does the $PbtO_2$ measure?
66. What are the normal $PbtO_2$ values?
67. What are the therapeutic measures to treat a low $PbtO_2$?

68. Is the use of a brain oxygen monitoring system (e.g., Licox™ monitoring) associated with improvement outcomes?

69. What is cerebral microdialysis?
70. What are the uses of microdialysis?
71. What are the indications for transcranial Doppler?

72. What does an increase in the flow velocity mean?
73. How can one differentiate between vasospasm and hyperemia?
74. What does xenon-enhanced CT measure?
75. What are the two methods for continuously measuring local CBF?
76. Why is the use of near-infrared spectroscopy limited?
77. What is the goal of critical care management in patients with severe TBI?
78. What are the most common causes of cerebral ischemia?

Seizures

79. What are the six general types of seizures that you should be able to recognize?
80. What is the definition of an “early” posttraumatic seizure?
81. Describe a simple partial seizure. How is it treated?
82. Describe complex partial seizures. How are they treated?
83. Give the classic description of an absence seizure.
84. How do you recognize a tonic-clonic seizure?
85. Define febrile seizure.
86. What are the common causes of secondary seizures? How are they treated?
87. What are the significant risk factors for late posttraumatic seizures?

88. Which antiemetic medication lowers seizure threshold and should be avoided in neurosurgery patients?

89. Do anticonvulsants prevent long-term seizures?
90. Plasma levels of phenytoin are increased by which medications?

91. What are the four major neurobiological rationales for the use of EEG in the ICU?

92. What different pathologies can be detected with the EEG in the ICU?
93. Define status epilepticus.
94. Which drug is best in the immediate control of seizures in status epilepticus?
95. What is the treatment algorithm for status epilepticus?
96. True or false: Hypertension can cause seizures.
97. What do you need to remember when giving anticonvulsants to women?

Fluid and Electrolytes

98. What is the incidence of electrolyte abnormalities in TBI patients?

99. What is the optimal rate for administration of maintenance fluids after resuscitation in TBI?
100. What three most common electrolyte abnormalities occur in TBI?
101. Describe the two most common Hyponatremia syndromes.
102. Define diabetes insipidus.
103. What is the treatment for diabetes insipidus?
104. Why does hyperglycemia need to be treated?

Pulmonary Complications

105. What is the ventilation pattern of choice in nonparalyzed patients with CNS dysfunction?

106. Why is a small amount of pressure support used in SIMV to prevent respiratory fatigue?

107. What ventilation mode should be used in neurosurgical patients who are chemically paralyzed?

108. What is ACV?
109. Why are SIMV and ACV the ventilation modes most used in neurosurgical patients?

110. What are the potential problems with ACV?
111. What is the main characteristic of pressure-controlled ventilation (PCV)?
112. Why is the use of PCV limited in patients with intrinsic pulmonary dysfunction?
113. What is IRV?
114. What is the advantage of IRV?
115. What is the disadvantage of IRV?
116. Which patients most likely benefit from IRV?
117. What is the pathophysiology of ARDS?
118. Define neurogenic pulmonary edema.

Coagulation and Deep Venous Thrombosis

119. Prolongation of bleeding time occurs in which conditions and situations?
120. Name some common drugs that alter the effects of warfarin.
121. Which of the coagulation factors has the shortest half-life?
122. Which of the coagulation factors are vitamin K–dependent factors?

123. Which laboratory finding in disseminated intravascular coagulation (DIC) correlates with bleeding?

124. Which group of patients has an increased risk for pulmonary embolism?
125. What are the prophylactic measures for deep venous thrombosis (DVT)?

126. When can heparin be started in a patient who underwent a cranial neurosurgical procedure without a risk of hemorrhage?

127. What do the Guidelines for the Management of Severe TBI (2007) recommend regarding infection prophylaxis?

128. Is it safe to give DVT prophylaxis 3 days postinjury?

Hemodynamics

129. What are the most common arrhythmias in the critical care setting?
130. What is the most common cause of AF in the immediate postoperative period?
131. What are the treatment options for AF?
132. What is the most appropriate drug for a stable patient with a narrow complex supraventricular tachycardia?
133. What it the most common wide-complex tachycardia in the ICU?
134. What is the treatment for VT?
135. What are the data parameters that a Swan-Ganz catheter (SGC) can provide?
136. What are the normal ranges for the SGC measurements?
137. What is another use of the SGC?
138. What are the complications of SGC placement?
139. What are the characteristics of hypovolemic shock?
140. What are the characteristics of cardiogenic shock?
141. What are the characteristics of septic shock?
142. Define neurogenic stunned myocardium.
143. What is the treatment of neurogenic stunned myocardium?

Preoperative Assessment

144. What is the most common side effect of mannitol?
145. When do the majority of perioperative myocardial infarctions occur?

146. What is the best method in current use to assess cerebral metabolism quantitatively?

147. Why are inhalational anesthetics referred to as “uncoupling” agents with respect to cerebral hemodynamics and metabolism?

148. When should the use of nimodipine in vasospasm be reconsidered?

149. What should be considered in the evaluation of a patient who is scheduled for an elective craniotomy for meningioma who is hyponatremic and hypotensive, but otherwise healthy?

150. What types of coagulopathies are not detected by prothrombin time/partial thromboplastin time/international normalized ratio (PT/PTT/INR) and platelet counts?

151. What disorders can lead to platelet sequestration?

152. In what patient population does steroid use increase the risk of gastrointestinal hemorrhage?

153. Oxygen transport is maximal when hematocrit is in what range?

154. What finding on a CT scan of the head would be predictive of the success of a third ventriculostomy for hydrocephalus?

Trauma and Emergencies

Trauma

155. What is the most common cause of cerebrospinal fluid leakage?
156. How can one differentiate if nasal drainage is CSF or nasal secretion?
157. What is the best initial treatment for a CSF leak?
158. What is the major cause of spontaneous intracranial hypotension?
159. What is the microscopic hallmark of diffuse axonal injury?

160. What radiologic view is necessary to fully appreciate an occipital bone fracture on plain x-ray films?

161. Which allele predisposes one to greater risk of Alzheimer disease after a head injury?

162. What area of the intracranial facial nerve is most commonly damaged by blunt trauma?

163. What is the Schirmer test?

164. What type of temporal bone fractures more frequently result in external manifestations such as otorrhea of CSF and tympanic membrane perforation?

165. Why is an EEG sometimes ordered in cases of lowered level of consciousness posttrauma?

166. What brainstem reflexes are mandatory to test in performing brain death evaluation?

167. In using auditory evoked potentials in the evaluation of brain death, what wave is necessary for the test to be valid?

168. What are some medications that are neuroprotective?

169. Why is a bifrontal exposure often needed in trauma cases for persistent rhinorrhea?

170. What does the literature state about prophylactic antibiotics for CSF leaks after traumatic head injury?

171. What does the literature state about hyperventilation in the setting of traumatic brain injury?

172. How can an acute subdural hematoma appear isointense to brain in a multitrauma patient?

173. Barbiturates are the most common class of drugs used to suppress cerebral metabolism in the setting of major cerebral trauma. What is the typical dosage of pentobarbital and what tests can be used to make sure the right amount is given?

174. What are some shortcomings of the Glasgow Coma Scale (GCS)?

175. Finding a linear skull fracture on radiologic exams in a conscious patient increases the risk of intracranial hematoma by how much?

176. What are the prerequisites for a growing skull fracture?

Emergencies

177. A fall in end tidal CO_2 could be the only clue to what emergency?
178. How is air embolism treated?
179. What are some cases where hyperemia of the brain can occur?

180. What are the signs and symptoms of myxedema coma and how is it properly treated?

181. What are the three places that a shunt may be occluded?
182. How should life-threatening cerebellar swelling from infarction be managed?
183. What are the drugs used in neuroleptic malignant syndrome?
184. What is the most common cerebral vascular complication encountered during pregnancy?

185. What is the most common site of hypertensive cerebral hemorrhage?

186. What are the signs and symptoms of Addisonian crisis and how is it properly treated?

187. What is neurogenic pulmonary edema?
188. How is an acute attack of migraine headache best treated?

189. What potential emergency can occur intracranially if nitrous oxide anesthesia is not discontinued prior to closure of the dura during surgery?

190. What are the most common complications of the transoral operative route?

Neurocritical Care Answers

Cerebral Metabolism

1. Cerebral perfusion pressure equals the mean arterial pressure minus the intracranial pressure (CPP =MAP—ICP).
2. 60–160 mm Hg.
3.70 mm Hg.
4. MAP is twice the diastolic pressure, which is added to the systolic pressure; then all are divided by three. MAP = ((2D) + S)/ 3). It is two times the diastolic because the majority of the cardiac cycle is in diastole.

5. About 20 mL/100 g/min.

6. It is the capacity to maintain blood flow at a relatively constant level during changes in blood pressure (BP) and is usually observed between a MAP of approximately 50 and 150 mm Hg.

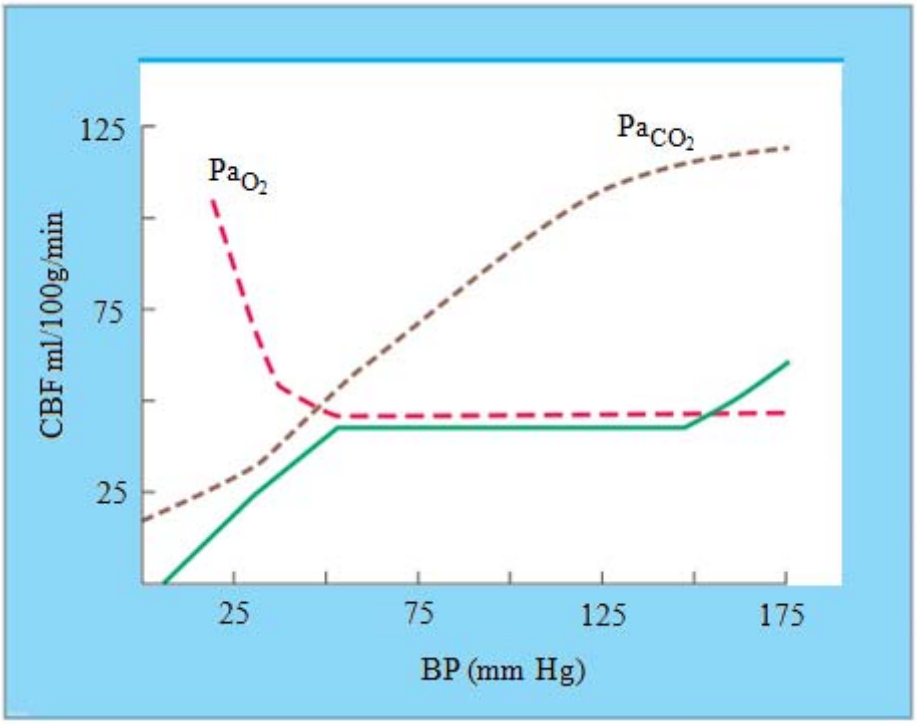


Figure 1. Autoregulation of cerebral blood flow (*solid line*). Cerebral perfusion is constant over a wide range of systemic blood pressure. Perfusion is increased in the setting of hypoxia or hypercarbia. *BP*, blood pressure; *CBF*, cerebral blood flow; $PaCO_2$, arterial carbon dioxide tension; PaO_2, arterial oxygen tension.

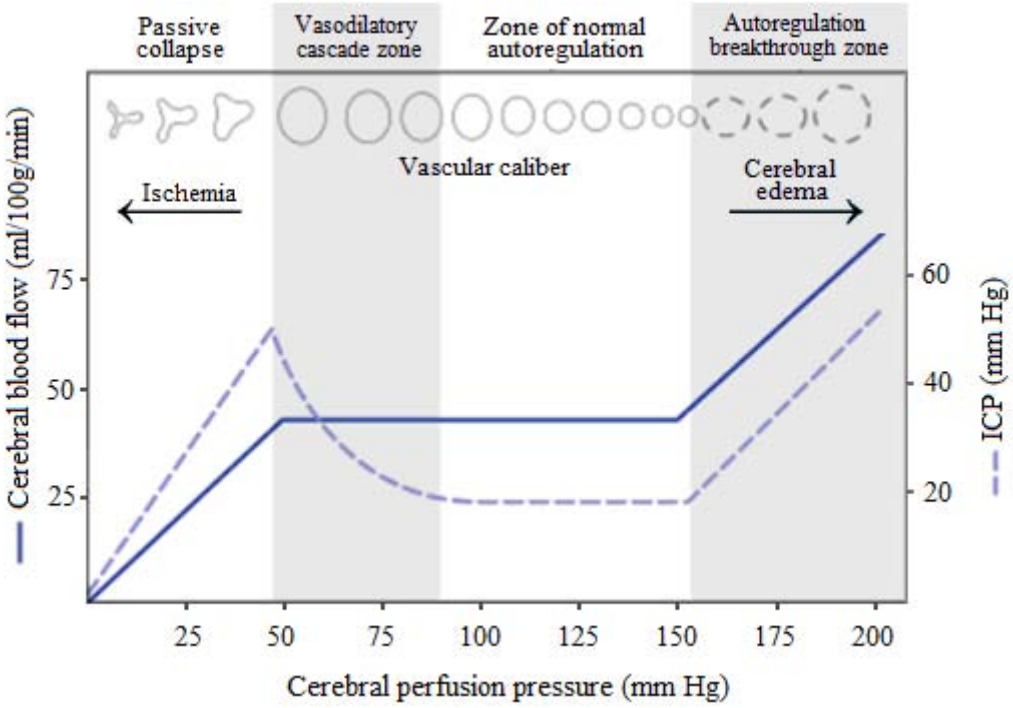


Figure 2. Relationship of intracranial pressure (ICP) to cerebral perfusion pressure (CPP) in states of reduced intracranial compliance. In both the vasodilatory cascade zone and the perfusion pressure breakthrough zone, increased cerebral blood volume leads to an increase in ICP.

7. A decrease in CPP results in vasodilation and allows CBF to remain unchanged. This vasodilation can result in increased ICP, which further perpetuates the decrease in CPP (vasodilatory cascade); likewise, an increase in CPP results in vasoconstriction and may reduce ICP.

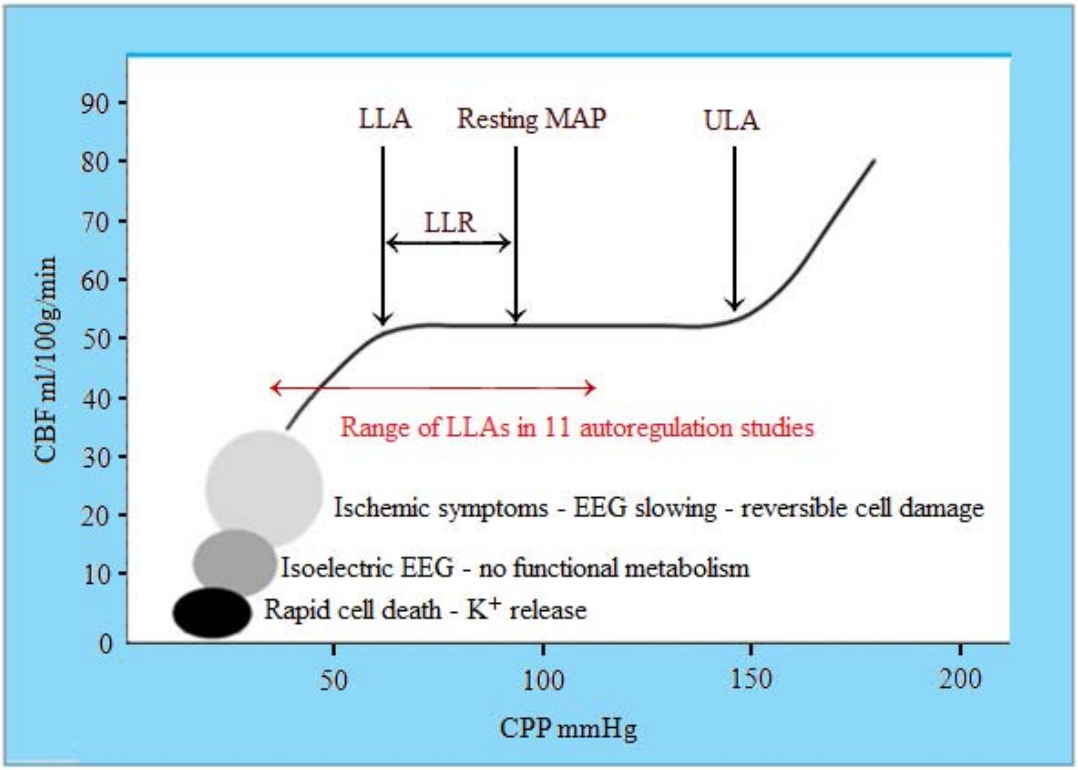


Figure 3. Lassen's classic autoregulation curve.

The cerebral vasculature maintains a stable CBF over a wide range of CPP (typically CPP 60–140 or MAP 70–150 mm Hg). The lower end of the autoregulatory range is the LLA, where there is maximal cerebral vasodilation. The ULA is when there is maximal cerebral vasoconstriction. The area between the baseline MAP and the LLA is known as the lower limit reserve (LLR). Symptoms and signs (EEG slowing) of cerebral ischemia occur at approximately 24 to 38 mL/100 g/min CBF and neuronal damage is still reversible. Functional metabolism ceases between 15 and 6 mL/100 g/min with an isoelectric EEG, and neurons will eventually die. Destruction of cell integrity occurs at approximately CBF <6 mL/100 g/min and rapid cell death ensues. The lower the CBF at ischemic levels, the shorter the duration allowable before irreversible neuronal damage. Red line represents the range of LLAs from 11 autoregulation studies in awake humans.

CPP and ICP

8. Head injury, anesthesia, and hypothermia decrease $CMRO_2$. Fever and seizures increase $CMRO_2$.
9. CMRG may be elevated regionally and even globally when $CMRO_2$ is reduced.

10. Mitochondrial damage, inability to metabolize oxygen normally, and glucose depletion. This alteration can be another cause of secondary ischemic injury.

11. Normal CBF in humans averages 50 mL/100 g brain tissue per minute.
12.
• Low CBF (<33 mL/100 g/min); these patients are more prone to develop cerebral ischemia.
• Relative hyperemia (33–55 mL/100 g/min)
• Absolute hyperemia (>55 mL/100 g/min).

ICP Monitoring

13. Normal ICP waves usually consist of three arterial components superimposed on the respiratory rhythm (Figure 4).

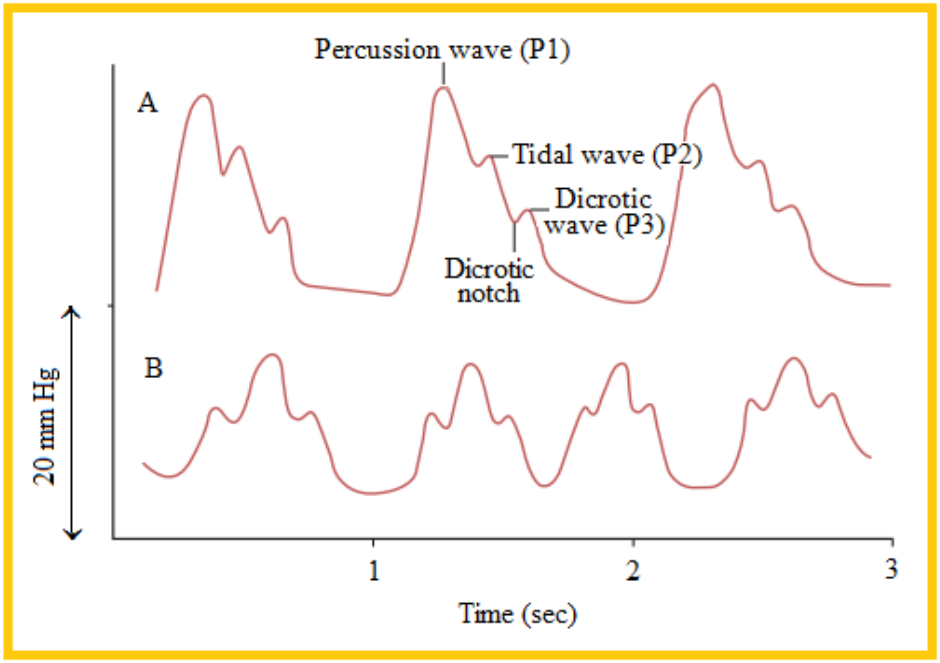


Figure 4. **A,** Intracranial pressure (ICP) waveform under physiological conditions. Three peaks are generally recognizable. The fist, and usually tallest, is the percussion wave, followed by the tidal wave, the dicrotic notch, and the dicrotic wave. Note that the tidal and dicrotic waves have progressively lesser amplitudes than the percussion wave (P1 > P2 > P3). **B,** Abnormal intracranial waveform with high ICP. The amplitude of the tidal wave exceeds the amplitude of the percussion wave.

Percussion wave: arterial pressure transmitted from the choroid plexus.

Tidal wave: ventricular relaxation.

Dicrotic wave: closure of aortic valve.

Compliance: dV/dP (Figure 5).

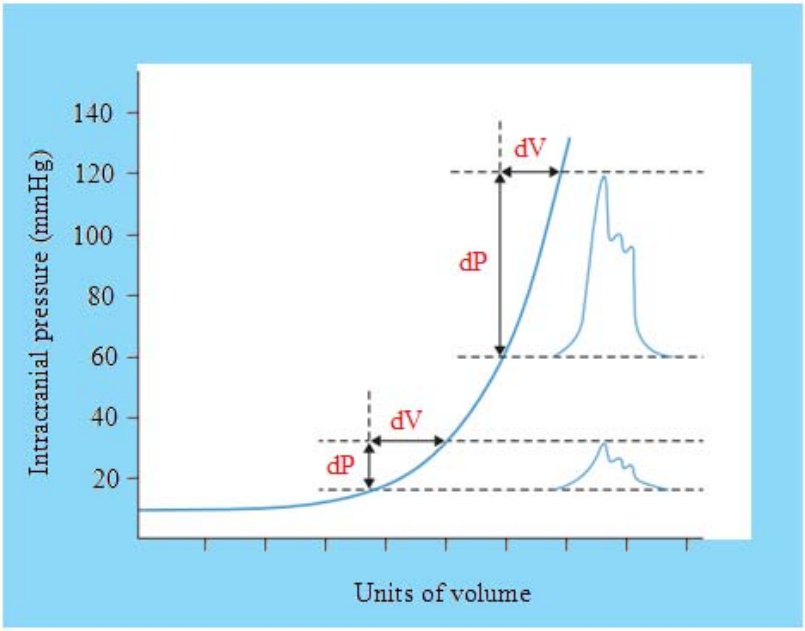


Figure 5. Compliance.

14.

1. Lundberg A waves (plateau waves).
These indicate an abrupt increase in ICP ≥50 mm Hg for 5–20 minutes followed by rapid fall in ICP. The plateau wave represents a transient increase CBV, possibly secondary to CO_2 retention.

2. Lundberg B waves (pressure pulses).
Amplitude of 10–20 mmHg with rhythmic variation 0.5–2 min.

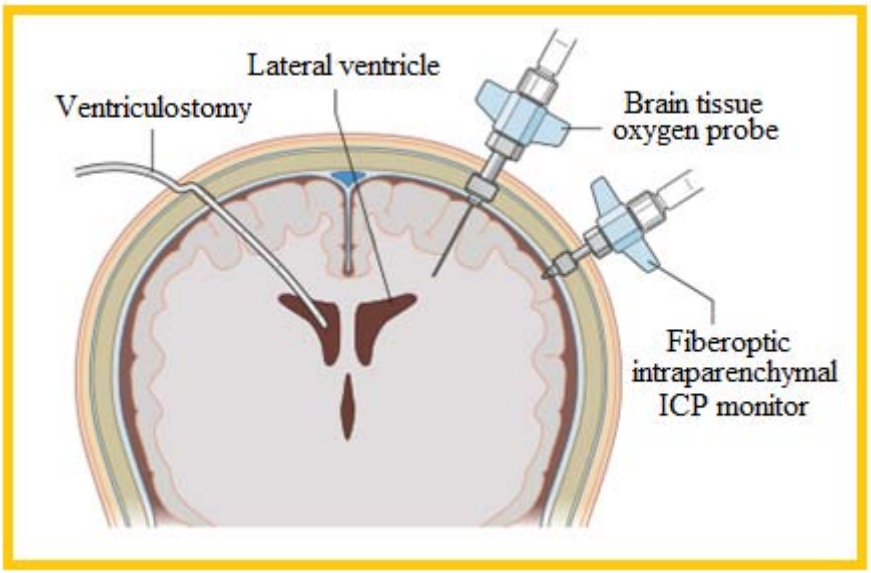
3. Lundberg C waves.
Frequency of 4–8/min. Low amplitude C waves may be seen in a normal ICP waveform. High amplitude C wave may be pre-terminal, and may sometimes be seen on top of plateau waves.

15. Yes, based on the Guidelines for the Management of Severe TBI 2007.
Class II evidence: ICP should be monitored in all salvageable patients with a severe TBI (GCS score of 3 to 8 after resuscitation) and an abnormal CT (see below).

Class III evidence: ICP should be monitored in patients with severe TBI with a normal CT if two or more of the following factors are noted at admission: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure <90 mm Hg.

16. An abnormal CT is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns.

17.



No, based on the Guidelines for the Management of Severe TBI 2007. A ventricular catheter connected to an external strain gauge is the most accurate, cost-effective, and reliable method of monitoring ICP. It can be recalibrated in situ. Parenchymal ICP monitors cannot be recalibrated during monitoring. Parenchymal ICP monitors, using micro strain pressure transducers, have negligible drift. Subarachnoid, subdural, and epidural monitors are less accurate.

18. Infection incidence of 5 to 14% (a nonlinear increase of risk in the first 10 to 12 days, after which the rate diminished).
Other complications (overall incidence of 1.4%) include hemorrhage as well as catheter malfunction, obstruction, and malposition.

19. Appropriate sterile technique.
20.
• Insertion of catheter in NICU vs. OR
• Previous catheter
• Drainage of CSF
• Use of steroids

21. Yes, antibiotic-coated catheters reduce the risk of infection (ranges of infection rates are from 9.4 to 17% for nonimpregnated catheters to 1.3 to 2.4% for impregnated catheters).

22. No, based on the Guidelines for the Management of Severe TBI 2007 regarding infection prophylaxis.
Class III evidence: Routine ventricular catheter exchange or prophylactic antibiotic use for ventricular catheter placement is NOT recommended to reduce infection.

23.
• Head elevation to 30 degrees and in neutral position
• Airway and ventilation control
• Sedation and analgesia
• Treatment of anemia
• Control of fever
• Control of hypertension
• Prevention of seizures

ICP Management

24. No, this is a misconception. Intracranial hypertension (IC-HTN) will occur in 50 to 70% of patients with evacuated intracranial hematoma.

25.
• Traumatically induced masses
• Cerebral edema (after evacuation of mass lesions, this is the primary cause)
• Hyperemia owing to vasomotor paralysis
• Hypoventilation that leads to hypercarbia
• Hydrocephalus
• Increased intrathoracic and intraabdominal pressure (from mechanical ventilation, posturing, agitation, Valsalva maneuvers).

26. Based on the Guidelines for the Management of Severe TBI 2007:
Class II evidence: Aggressive attempts to maintain CPP above 70 mm Hg with fluids and pressors should be avoided because of the risk of adult respiratory distress syndrome (ARDS).
Class III evidence: CPP <50 mm Hg should be avoided.
The CPP target value lies within the range of 50 to 70 mm Hg. Patients with intact pressure autoregulation tolerate higher CPP values.

27. This should usually be avoided, based on the Guidelines for the Management of Severe TBI 2007.
Class II evidence: Blood pressure should be monitored and hypotension (SBP <90 mm Hg) avoided.
Class III evidence: Oxygenation should be monitored and hypoxia (PaO_2 <60 mm Hg or O_2 saturation <90%) avoided.

28.
Sympathomimetic-blocking
• beta-blocking drugs (labetalol, esmolol)
• central-acting α-receptor agonists (clonidine).

29. Because they reduce blood pressure without affecting the ICP.

30. Class I evidence: Steroids are not recommended for improving outcome or reducing ICP. In patients with moderate or severe TBI, high-dose methylprednisolone is associated with increased mortality and is contraindicated (CRASH [Corticosteroid randomisation after significant head injury] trial).

31.
In this order:
• Heavy sedation and paralytics
• Hyperosmolar therapy
• Hyperventilation
• Barbituric coma
• Hypothermia (this last one is controversial).

32. Based on the Guidelines for the Management of Severe TBI 2007:
Class II evidence: Prophylactic administration of agents to induce burst suppression EEG is not recommended.
High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.
Propofol is recommended for the control of ICP, but not for improvement in mortality or 6-month outcome. High-dose propofol can produce significant morbidity.

33. Common clinical features include hyperkalemia, hepatomegaly, lipemia, metabolic acidosis, myocardial failure, rhabdomyolysis, and renal failure resulting in death.

34. Extreme caution when using doses >5 mg/kg/h or >8 µg/kg/min or when usage exceeds 48 hours.

35.
• Morphine sulfate, although tachyphylaxis is extremely common
• Fentanyl and sufentanil have become increasingly popular because of their brief duration of action. However, these agents have been shown to cause a mild but definite elevation in ICP.

36. Etomidate.
37. Thiopental.
38. Enflurane.
39.
• Myopathy (increased when the neuromuscular blocking agent is combined with B_2 -agonists, corticosteroids or antibiotics [aminoglycosides])
• Polyneuropathy
• Prolonged neuromuscular blockage.

40. For the most part, yes, based on the Guidelines for the Management of Severe TBI 2007.
Class II evidence: Mannitol is effective for control of raised ICP at doses of 0.25 g/kg. Arterial hypo tension (SBP <90 mm Hg) should be avoided.
Class III evidence: Restrict mannitol use prior to ICP monitoring in patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes.
In a recent study on 47 TBI patients, mannitol was shown to be as effective as HTS in decreasing ICP, although both failed to improved cerebral metabolism. HTS also demonstrated a stronger effect on cerebral perfusion in the presence of cerebral ischemia.

41. Rheological and osmotic effects.
42.
• Immediately plasma-expanding
• Reduction of hematocrit
• Increases deformability of erythrocytes
• Reduction of blood viscosity
• Increases CBF and oxygenation

43. Mannitol takes from 15 to 30 minute to be effective and will increase serum tonicity. Stop mannitol when osmolarity reaches 320 mOsm to prevent hypovolemia, hyperosmolarity, and renal failure.

44. To prevent rebound edema.
45.
• A bolus of 0.25 to 1 g/kg
• 1 g/kg should be given when urgent reduction of ICP is needed.
• Higher doses (1.4 g/kg) may give significantly better results in extreme critical situations.

46.
• Osmotic mobilization of water across the blood–brain barrier (BBB).
• Dehydration of endothelial cells and erythrocytes, which increases the diameter of the vessels and deformability of erythrocyte leading to an increase in CBF.
• Reduces leukocyte adhesion in the traumatized brain.

47. In hypotensive and hypovolemic patients (a very common scenario in TBI).

48. Based on the Guidelines for the Management of Severe TBI 2007:

Class II evidence: Prophylactic hyperventilation ($PaCO_2$ of 25 mm Hg or less) is not recommended.
Class III evidence: Hyperventilation is recommended as a temporizing measure for the reduction of elevated ICP.
Hyperventilation should be avoided during the first 24 hours after injury when CSF is often critically reduced.

If hyperventilation is used, jugular venous oxygen saturation ($SjvO_2$) or brain oxygen tension ($PbrO_2$) measurements are recommended to monitor oxygen delivery.

49. The vasoconstrictive effect on cerebral arterioles lasts 11 to 20 hours because the pH of the CSF equilibrates to the new $PaCO_2$, and then cerebral arterioles re-dilate (possibly to a larger caliber). When hypocarbia is induced and maintained for hours, it should be reversed slowly (over days) to minimize this rebound hyperemia.

50.
• Loading dose 10 mg/kg over 30 minute; 5 mg/kg every hour × 3 doses
• Maintenance 1 mg/kg/h
• Titrate to serum levels of 30 to 50 μg/mL or burst suppression pattern on EEG.

51. Unclear, but likely reduces CBF and $CMRO_2$; its action is closely tied to the retention of CO_2 reactivity by the brain.

52.
• Hypotension
• Hypokalemia
• Respiratory complications
• Infections
• Hepatic dysfunction
• Renal dysfunction.

53. Based on the Guidelines for the Management of Severe TBI 2007:
Class III evidence: Prophylactic hypothermia is not significantly associated with decreased mortality when compared with normothermic controls. However, preliminary findings suggest that a greater decrease in mortality risk is observed when target temperatures are maintained for more than 48 hours. Prophylactic hypothermia is associated with significantly higher Glasgow Outcome Scale (GOS) scores when compared with scores for normothermic controls.

54.

• 20 to 35% demonstrated by transcranial Doppler ultrasound studies for severe TBI.
• Use of calcium channel blockers may improve outcome (Cochrane database)

55. Hyperglycemia increases neuronal metabolism, inducing rapid conversion to anaerobic metabolism and accelerating the oxidative stress on the cell. Maintaining the patient in the normoglycemic range has been shown to prevent further ischemic insults and seizures and to reduce mortality.

56.

• Evacuation of mass lesions
• CSF drainage
• Decompressive craniectomy

Advanced Neuromonitoring

Parameter	Normal values
ICP	5 - 15 mm Hg
CPP	60 - 160 mm Hg
CBF (global)	50 - 100 ml/100g/min (global brain CBF) 80 ml/100g/min (gray matter) 20 ml/100g/min (white matter)
$CMRO_2$	3.2 ml/100g/min (global brain $CMRO_2$) 6 ml/100g/min (gray matter) 2 ml/100g/min (white matter)
$PbtO_2$	25 - 30 mmHg
$SjvO_2$	55% - 70%
$AjvDO_2$	4 – 8 ml/dL

$AjvDO_2$, Arteriojugular venous oxygen difference, calculated value; *CBF*, cerebral blood flow, calculated as: CBF = CPP ÷ cerebrovascular resistance; *$CMRO_2$*, cerebral metabolic rate of oxygen; *CPP*, cerebral perfusion pressure; *ICP*, intracranial pressure; *$PbtO_2$*, brain tissue oxygenation; *$SjvO_2$*, jugular venous oxygen saturation.

57.

• ICP measuring devices
• Jugular bulb catheter ($SjvO_2$)
• Electroencephalography (EEG)
• Evoked potentials

58.
• Tissue probes (pO_2, pCO, pH)
• Microdialysis catheter
• Transcranial Doppler
• Xenon CT
• Laser Doppler and thermal diffusion flowmetry
• Near-infrared spectroscopy

59. Continuous index of the changing balance between cerebral oxygen delivery and consumption. $SjvO_2$ and SaO_2 from an A-line allow one to calculate arteriovenous oxygen difference ($AVDO_2$).

60. The tip of the catheter at the level of the skull base (jugular bulb). In cases of diffuse brain injury, the catheter should be placed on the dominant side, or on the side of the major focal lesion.

61. 55 to 71%.
62. <20% is consistent with irreversible ischemic injury.

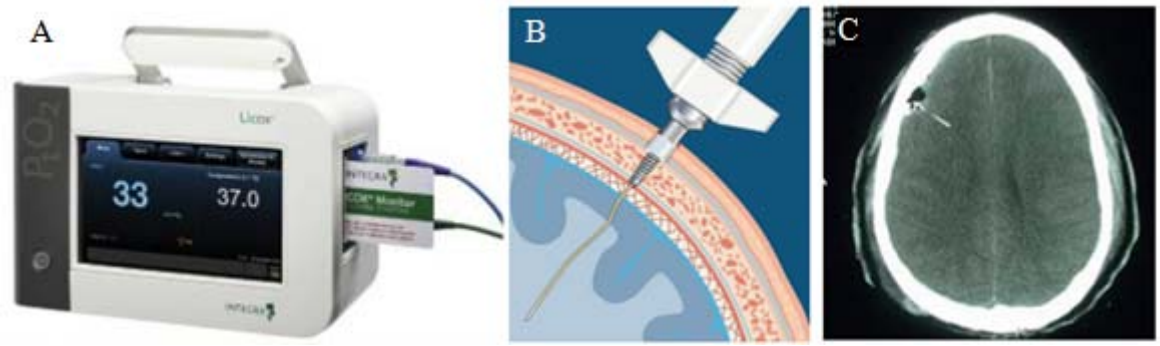
63. Greater than 75% or lower than 55%.
<20% is consistent with irreversible ischemic injury.

64.
• Catheter malposition
• Improper calibration
• Arterial hypoxia
• Anemia
• Inadequate CBF

65. Regional cerebral oxygenation (partial pressure of oxygen in brain tissue).
66.
• 20 to 40 mm Hg
• Critical values are 8 to 10 mm Hg

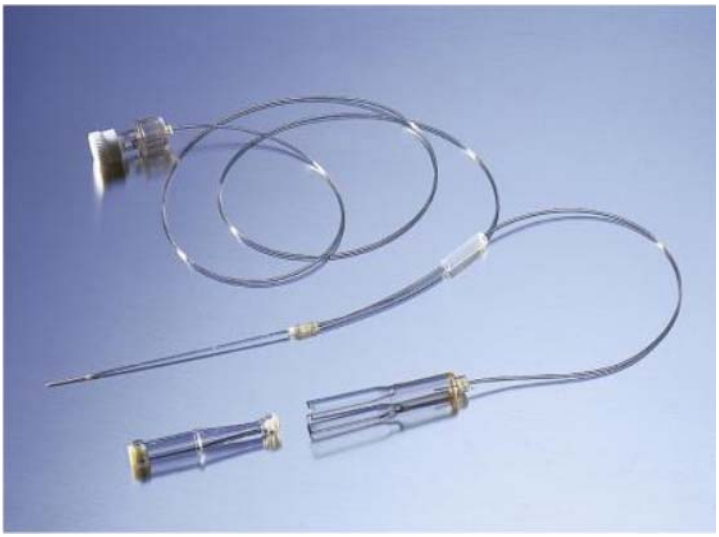
67.
• Elevation of arterial blood pressure
• Increase of FiO_2
• Transfusion of red blood cells
• Reduction of ICP

68. This remains undetermined at this time, based on the Guidelines for the Management of Severe TBI 2007.
Class III evidence: $SjvO_2$ <50% or $PbtO_2$ <10–15 mm Hg are treatment thresholds, in addition to ICP monitoring.
There is insufficient evidence to determine whether this information is useful for patient management or prognosis.



A. Licox brain tissue oxygen ($PbtO_2$) and brain temperature monitor and calibration “smart card”. B. Schematic diagram of the Licox probe that illustrates placement through a cranial bolt into the cerebral tissues. Placement is similar to an intracranial pressure (ICP) monitor and is frequently used through the same bolt. C. A computed tomography (CT) head scan that demonstrates the position of a Licox probe in the white matter of the right frontal cortex.

69. It is a technique of sampling the extracellular space of the brain tissue. It allows for continuous and on-line monitoring of changes in brain tissue chemistry.



Components of clinical microdialysis catheter.

70.
• TBI
• Subarachnoid hemorrhage
• Epilepsy
• Brain tumors

• Ischemic stroke

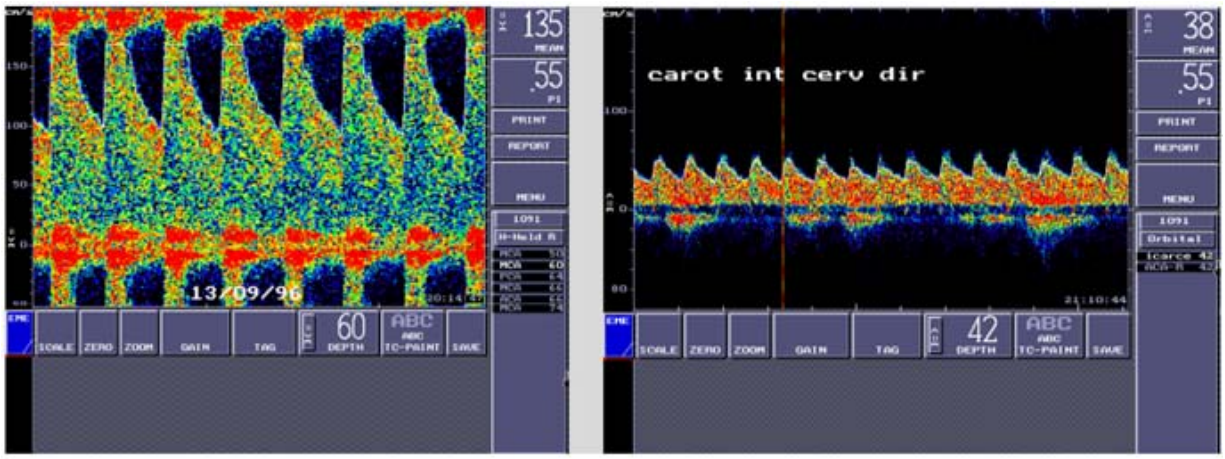
71.

Assess regional cerebral blood flow indirectly from cerebral artery flow velocity (e.g., TBI, vasospasm). In normal individuals, the pulsatility reflects the distal cerebrovascular resistance.

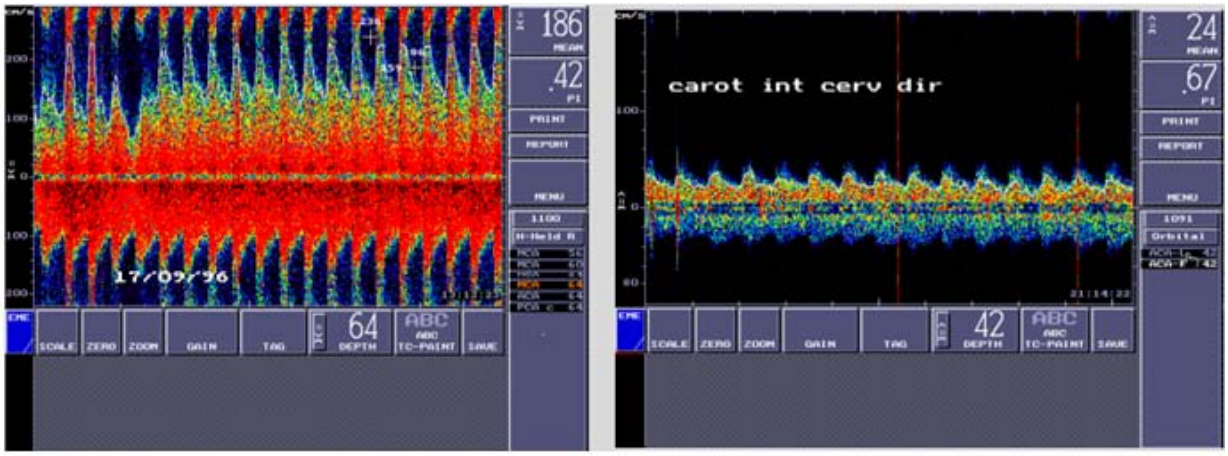
72.

• Arterial spasm will show velocities in the range of 120 to 200 cm/sec 2 (severe spasm >200 cm/sec^2)
• Hyperemia

73.



Moderate vasospasm:
MCA / ICA ratio (Lindegaard) < 6 (3,55)



Severe vasospasm:
MCA / ICA ratio (Lindegaard) > 6 (7,75)

Using the Lindegaard or hemispheric index, which is the middle cerebral artery/extracranial internal carotid artery flow velocity ratio (normal: 1.7)
• Hyperemia: raised flow velocity on both
• Vasospasm: high-flow velocity in intracranial vessels (flow velocity ratio of 3).

74. Global and regional quantitative CBF.

75. Thermal diffusion and laser Doppler flowmetry (design for measuring flow in capillaries). The probe needs to be on the surface of the brain.

76. This is due to its inability to differentiate between intracranial and extracranial change in blood flow oxygenation.

77. Enhance cerebral perfusion and avoid therapy that may cause regional cerebral ischemia.

78. Hypotension, hypoxia, and intracranial hypertension.

Seizures

79.

- Simple partial
- Complex partial
- Absence (petit mal)
- Tonic-clonic
- Febrile
- Secondary

80.
An early posttraumatic seizure is one that occurs within the first 7 days of an injury; those that come after 7 days are called late posttraumatic seizures. About 10% of adults with early seizures will develop status epilepticus. Prophylactic phenytoin therapy may be stopped in ~1 to 2 weeks to prevent early posttraumatic seizures; however, there is no proven advantage that phenytoin or other anticonvulsants prevent late posttraumatic seizures.

81. Simple partial (local or focal) seizures may be motor (e.g., Jacksonian march), sensory (e.g., hallucinations), or psychic (cognitive or affective symptoms). The key point is that consciousness is not impaired. The first-line

agents for treatment are carbamazepine, lamotrigine, oxcarbazepine, and levetiracetam.

82. Complex partial (psychomotor) seizures are any simple partial seizure followed by impairment of consciousness. Patients perform purposeless movements and may become aggressive if restraint is attempted (however, people who get in fights or kill other people are not having a seizure). The firstline agents for treatment are valproate, lamotrigine, and levetiracetam.

83. Absence (petit mal) seizures do not begin after the age of 20 years. They are brief (10 to 30 seconds in duration), generalized seizures in which the main manifestation is loss of consciousness, often with eye or muscle fluttering. The classic description is a child in a classroom who stares into space in the middle of a sentence, then 20 seconds later resumes the sentence where he left off. The child is not daydreaming; he or she is having a seizure. There is no postictal state (an important differential point). The first-line treatment agents are ethosuximide and valproate.

84. Tonic-clonic (grand mal) seizures are the classic seizures that we knew about before we went to medical school. They may be associated with an aura. Tonic muscle contraction is followed by clonic contractions, usually lasting 2 to 5 minutes. Associated symptoms may include incontinence and tongue lacerations. The postictal state is characterized by drowsiness, confusion, headache, and muscle soreness. The first-line agents for treatment are valproate, lamotrigine, or levetiracetam.

85. Children between the ages of 6 months and 5 years may have a seizure caused by fever. Always assume another cause outside this age range. The seizure is usually of the tonic-clonic, generalized type. No specific seizure treatment is required, but you should treat the underlying cause of the fever, if possible, and give acetaminophen to reduce fever. Such children do not have epilepsy, and the chances of their developing it are just barely higher than in the general population. Make sure that the child does not have meningitis, tumor, or another serious cause of the seizure. The Step 2 question will give clues in the case description if you should pursue work-up for a serious condition.

86.

- Mass effect (tumor, hemorrhage)
- Metabolic disorder (hypoglycemia, hypoxia, phenylketonuria, hyponatremia)

- Toxins (lead, cocaine, carbon monoxide poisoning)
- Drug withdrawal (alcohol, barbiturates, benzodiazepines, withdrawing anticonvulsants too rapidly)
- Cerebral edema (severe or malignant hypertension; also watch for pheochromocytoma and eclampsia)
- Central nervous system (CNS) infections (meningitis, encephalitis, toxoplasmosis, cysticercosis)
- Trauma
- Stroke

Treat the underlying disorder and use a benzodiazepine (lorazepam or diazepam) and/or phenytoin or fosphenytoin acutely to control seizures. For all seizures (primary or secondary), secure the airway, and, if possible, roll the patient onto his or her side to prevent aspiration.

87.
• Brain contusions
• Subdural hematoma
• Skull fracture
• Loss of consciousness
• Amnesia for more than 1 day
• Age 65 years or older

88. Promethazine (Phenergan^{TM}).

89. No, based on the Guidelines for the Management of Severe TBI 2007. Class II evidence: Prophylactic use of phenytoin or valproate is not recommended for preventing late posttraumatic seizures (PTSs). Anticonvulsants are indicated to decrease the incidence of early PTS (within 7 days of injury). However, early PTSs are not associated with worse outcome.

90. Sulfonamides, Coumadin, isoniazid, and cimetidine.
91.
• Its close relationship to cerebral metabolic rate
• Its sensitivity in detecting hypoxic-ischemic neuronal dysfunction at an early stage
• Monitoring seizure activity
• Cerebral localization

92.
• Nonconvulsive seizures and status epilepticus

• Clinical impression of metabolic encephalopathy
• Psychogenic unresponsiveness
• Locked-in state
• Focal mass lesions
• Cerebral ischemia (including vasospasm)

93. Status epilepticus is defined as a seizure that lasts for a sufficient length of time (usually 30 minutes or longer) or is repeated frequently enough that the individual does not regain consciousness between seizures. Status epilepticus may occur spontaneously or result from withdrawing anticonvulsants too rapidly.

94. Lorazepam is better than diazepam or phenytoin.

95. Lorazepam 4 mg (or 0.1 mg/kg) intravenously (IV) over 2 minutes, may repeat after 5 minutes. Simultaneously load with phenytoin 1200 mg (or 20 mg/kg), or 500 mg if already on phenytoin. Phenobarbital may be given up to 1400 mg at a rate of less than 100 mg/min. If seizures continue, consider general anesthesia. It is also important in the initial stages of status to send laboratories for electrolyte levels and antiepileptic drug levels if the patient is already on an agent. A normal saline IV drip may be started and 50 mL of 50% glucose given, as well as 100 mg of thiamine. Other agents that may be used if the above measures are not effective include pentobarbital (while watching for circulatory depression and being prepared to use a pressor), midazolam, or propofol.

96. True. Remember hypertension as a cause of seizures or convulsions, headache, confusion, stupor, and mental status changes.

97. All anticonvulsants are teratogenic, and women of reproductive age need counseling about the risks of pregnancy. Do a pregnancy test before starting an anticonvulsant and offer birth control. Valproic acid is a major contributor to the risk. Polypharmacy increases the risk. There is limited human information of the risks to the fetus with the newer antiepileptic medications.

Fluid and Electrolytes

98. 59%.
99. 35 mL/kg/d (0.9% normal saline).

100.

• Hyponatremia
• Hypernatremia
• Hyperglycemia

101.

	SIADH	CSW
Volume status	Normovolemic or slightly expanded	Hypovolemic
Pathophysiology	Enhanced secretion of ADH	Elevated levels of circulating natriuretic factors
Serum Na	< 135 mEq/L	< 135 mEq/L
Serum Osm	< 280 mOsm/L	< 280 mOsm/L
Urine Na	> 40 mEq/L	> 40 mEq/L
Urine Osm	> serum Osm	> serum Osm
Treatment	Fluid restriction 800-1000 mL/d, Demeclocycline, Hypertonic (3%) saline	Normal saline or hypertonic saline, Salt tablets

102. Inadequate circulating quantities of ADH that result in an inability to concentrate urine, causing hypovolemic hypernatremia. In TBI, diabetes insipidus often indicates a grave prognosis.

103.

• Mild to moderate cases may be treated with water replacement.
• In TBI, water replacement may exacerbate IC-HTN!
• DDAVP (desmopressin acetate) 2–4 µg IV is an option.
• Correction of hypernatremia can exacerbate IC-HTN; it should be done slowly over 48 hours.

104.

Hyperglycemia has been associated with a poor neurological outcome in TBI:
• Reflects the occurrence of a more severe injury
• Exacerbates the secondary injury processes
Glucose levels >200 mg/dl should always be treated.
Maintenance fluids should always be glucose-free.

Pulmonary Complications

105. Synchronized intermittent mandatory ventilation (SIMV), which delivers volume-cycled breaths that coincide with spontaneous lung inflation.
106. SIMV requires spontaneous breathing through a ventilatory circuit of high resistance, which can result in increased work of breathing and respiratory muscle fatigue.

107. ACV (assist-control ventilation).

108. A form of volume-cycled ventilation that provides breaths at a preselected rate, and assists each spontaneous breath regardless of respiratory rate.

109. They are volume-cycled forms that deliver larger lung inflation volumes. The expiration phase is prolonged, allowing adequate lung deflation in the presence of larger tidal volumes.

110.
• Hyperinflation (auto–positive end-expiratory pressure [PEEP])
• Respiratory alkalosis
• Increased work for patient with tachypnea

111. It delivers pressure-cycled mechanical breaths, preventing overinflation and lung injury.

112. In patients with increased airway resistance or decreased lung compliance, the inflation volumes are decreased with PCV.

113. Inverse ratio ventilation, which is the prolongation of the inflation time; this can be done with PCV.

114. Facilitates alveolar recruitment.

115. Hyperinflation → increase in transthoracic pressure→ decrease in cardiac output → increase in ICP.

116. Patients with acute respiratory distress syndrome (ARDS).

117. Results from a diffuse systemic inflammatory response, leading to damage to the pulmonary capillary endothelium and accumulation of exudates within the

lung parenchyma. Causes include pulmonary contusion, sepsis, long bone fracture, and blood product transfusion, among others.

118. Increased transmural pulmonary vascular pressure by activation of α- and β-adrenoreceptors secondary to massive sympathetic activation; named for its association with neurological disease, but can also be a sequela of heart failure.

Coagulation and Deep Venous Thrombosis

119.
• Uremia (check BUN and Cr before you operate on a patient with renal insufficiency!)
• von Willebrand's disease
• Use of nonsteroidal antiinflammatory drugs.

120.
• Bactrim (decreases clearance of warfarin)
• Barbiturates and rifampin (accelerate clearance of warfarin)
• Cimetidine (inhibits the metabolism of warfarin)

121. Factor VII.
122. Factors II, VII, IX, and X.
123. Decrease fibrinogen.
124. Patients with severe trauma, malignancy, or lower extremity paralysis, or those undergoing major surgery.

125.
• Graded compression stockings
• Low-dose heparin
• Low-molecular-weight heparin
• Pneumatic compression boots.

126. The optimal time is unknown, but 1 week after surgery is recommended.

127. Class II evidence: Periprocedural antibiotics for intubation should be administered to reduce the incidence of pneumonia. However, periprocedural antibiotics do not change the length of stay or mortality.

128. Based on the Guidelines for the Management of Severe TBI 2007:

Class III evidence: Graduated compression stockings or intermittent pneumatic compression (IPC) stockings are recommended, unless lower extremity injuries prevent their use. Use should be continued until patients are ambulatory.
Low molecular weight heparin or low-dose unfractionated heparin should be used in
combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial hemorrhage. There is insufficient evidence to support recommendations regarding the preferred agent, dose, or timing of pharmacological prophylaxis for DVT.

Hemodynamics

129. Atrial flutter and atrial fibrillation (AF).
130. Electrolyte imbalances (Mg, K).
131.
• Calcium channel blockers
• Cardioselective beta-blockers (esmolol is recommended for its short half-life).

132. Adenosine (initial dose of 6 mg, followed by a repeat dose of 12 mg PRN).
133. Ventricular tachycardia (VT).
134. If hemodynamically stable: lidocaine or amiodarone.
If hemodynamically unstable: direct current cardioversion.

135.
• CVP (central venous pressure)
• PCWP (pulmonary capillary wedge pressure)
• CO (cardiac output)
• CI (cardiac index)
• SVR (systemic vascular resistance)

136.
• CVP: 1–6 mm Hg
• PCWP: 6–12 mm Hg
• CI: 2.4-4.0 L/min/m^2
• SVR: 1–6 mm Hg 900–1200 dynes/cm^2

137. Facilitates the diagnosis and differentiation of various shock states.

138. Pneumothorax, arterial injury, cardiac arrhythmias, line sepsis, and pulmonary hemorrhage and infarction.

139. Decreased venous and intracardiac pressure, increased SVR, hypotension, and tachycardia.

140. Low CI, elevated PCWP and CVP, and decreased systolic blood pressure.
141. Low filling pressures, decreased SVR, and normal or increased CO.
142. Cardiac hypokinesis leading to reduced ejection fraction and elevated cardiac enzymes caused by a catecholamine surge that is triggered at the level of the hypothalamus. It is seen in TBI and subarachnoid hemorrhage.

143.
• Milrinone if SBP >90 mm Hg and SVR is increased or when patient is on chronic beta-blockers
• Dobutamine if SBP <90 mm Hg.

Preoperative Assessment

144. Renal failure.
145. Between postoperative days 3 and 5.
146. Positron emission tomography (PET).

147. They decrease cerebral metabolism, but increase cerebral blood flow through vasodilatation. If an inhalational agent is to be given to a patient with poor intracranial compliance, hyperventilation should be initiated prior to induction. Isoflurane has less of an effect on cerebral blood flow than other agents.

148. In the face of diminished cardiac contractility, use of nimodipine (which is a negative inotrope) may exacerbate the cardiac complications.

149. Adrenal insufficiency.
150. Dysfibrinogenemia, von Willebrand disease, factor XIII deficiency, aspirin/Plavix use.

151. Hypersplenism associated with cirrhosis, Gaucher disease, sarcoidosis.
152. Patients with preexisting ulcer disease.
153. 30–32%.
154. Triventricular hydrocephalus (or obstructive hydrocephalus) from aqueductal stenosis or blockage of third ventricular outflow.

Trauma and Emergencies

Trauma

155. Head trauma; having a skull fracture doubles the patient's risk of a cerebrospinal fluid leak. Cerebrospinal fluid leaks may occur from the nose (rhinorrhea), ear (otorrhea), or orbit (mimicking tears).
156. The primary distinction between CSF and nasal drainage is the glucose level. Glucose is present in CSF (at 50% of the serum level) and not present in nasal drainage. A protein level of less than 1 g per liter is suggestive of CSF. The double-ring sign ("halo sign") seen on the bed sheets or clothing of patients with nasal drainage is only suggestive of a CSF leak; the β2-transferrin test can confirm the presence of CSF.

157. Bed rest and head elevation. Most leaks stop within 3 days. If after 3 days the leak persists, lumbar drainage may be used. Rarely is surgery needed to repair the source of the leak. The use of prophylactic antibiotics is controversial and may select for more virulent bacteria should infection occur.

158. Spontaneous CSF leaks. Diffuse pachymeningeal enhancement on magnetic resonance imaging (MRI) is the most common imaging finding. Patients often complain of headache that is alleviated by lying flat. A CT myelogram or radionucleotide cisternogram may be used to find the leak site.

159. Corpus callosum and superior cerebellar peduncle.
160. The Towne view.
161. Apolipoprotein E4 (APOE4).
162. The area of the facial nerve around the geniculate ganglion.

163. This test distinguishes facial nerve injuries proximal and distal to the geniculate ganglion. The test involves placing a narrow strip of thin paper on the conjunctiva to assess for lacrimation. Injuries proximal to the geniculate ganglion tend to produce a dry eye, whereas injuries distal to the ganglion do not interfere with lacrimation. Whether the location of the facial nerve injury is proximal or distal to the geniculate ganglion is important because the choice of surgical approach differs with different sites of injury.

164. Longitudinal fractures more frequently result in external signs of injury, whereas transverse fractures generally spare the middle ear, tympanic membrane, and external auditory canal. For this reason, transverse fractures manifest fewer external signs of injury. Transverse fractures most commonly

pass through the otic capsule; longitudinal fractures typically spare the otic capsule.

165. To rule out subclinical status epilepticus.

166. Pupil reflex, corneal reflex, oculovestibular reflex, oculocephalic reflex, gag reflex. Additional tests that should be performed are checking for a response to deep central pain and the apnea test. The patient should be checked for normothermia and normal blood pressure, and show no evidence of drug or metabolic intoxication.

167. Wave I, at least on one side.
168. Corticosteroids, free-radical scavengers, calcium channel blockers, glutamate antagonists, mannitol, and barbiturates.

169. Fractures of the anterior fossa often extend across the midline.

170. An article in Lancet (1994) states that the use of prophylactic antibiotics only encourage the resistance and late attacks of meningitis; therefore, they are not recommended.

171. Hyperventilation of head-injured patients may do more harm than good by decreasing cerebral perfusion pressure and delivery of O_2 and glucose. There are no good prospective, randomized studies to date to support the use of hyperventilation in head injury.

172. When the hematocrit is less than ~23, this may cause an acute subdural to appear isointense to brain. Another possibility is in the setting of coagulopathy.

173. A loading dose of 10 mg/kg is administered over 30 minutes then 5 mg/kg per hour is administered over 3 hours. If systolic blood pressure drops by more than 10 mm Hg or the perfusion pressure falls below 60 mm Hg, the loading dose infusion should be slowed. A maintenance infusion of 1 to 3 mg/kg per hour is begun after loading is completed. The infusion is titrated to burst suppression on the EEG and a serum level of 3 to 4 mg/dL. When checking for brain death, remember that the level of pentobarbital must be less than 10 µg/mL.

174. It does not allow the assessment of eye opening in periorbital trauma, verbal response in intubated patients, and brainstem function or reflexes. The GCS also works poorly for patients in the first 2 years of life. The GCS,

however, remains the standard for defining the level of consciousness after head injury and is a reliable and independent predictor of long-term outcome. The GCS is also used for patients who have not sustained trauma, such as postoperative patients.

175. 400-fold according to a study by Mendelow et al.

176.
1. The skull fracture occurs in infancy or early childhood.
2. There is a dural tear at the time of the fracture.
3. There is brain injury at the time of the fracture with displacement of leptomeninges and possibly brain through the dural defect.
4. There is subsequent enlargement of the fracture to form a cranial defect.

Emergencies

177. Air embolus.
178. Packing the wound with wet sponges, lowering the patient's head, using jugular venous compression, rotating the patient's left side downward, aspirating from the venous line that is in the right atrium, and ventilating the patient while maintaining adequate blood pressure and heart rate.

179. Head trauma, after carotid endarterectomy or stenting, and after excision of an arteriovenous malformation (AVM).

180. Myxedema coma is an emergency of hypothyroidism. The signs are hypotension, bradycardia, hyponatremia, hypoglycemia, hypothermia, and hypoventilation. Treatment consists of IV fluids, intubation if necessary, IV glucose, 400 mg hydrocortisone IV over 24 hours, and 0.5 mg levothyroxine IV followed by 0.05 mg levothyroxine per day.

181. The entry point (proximal occlusion), the valve system (valve obstruction), and the distal end (distal catheter occlusion). A CT scan of the head, a shunt series, and palpation of the valve are important in determining the site of the occlusion.

182. Resection of cerebellar infarction has been advocated in patients in whom life-threatening deterioration is occurring from focal cerebellar swelling, herniation, and brainstem compression or secondary fourth ventricular obstruction and hydrocephalus. A ventriculostomy may be needed as a

temporizing measure in anticipation of surgery; however, one must be cognizant of the risk of upward herniation.

183. Bromocriptine (dopamine receptor agonist) and dantrolene (muscle relaxant). Neuroleptic malignant syndrome is a rare condition seen with dopamine antagonist and long-acting depot neuroleptic preparations. Drowsiness, fever, tremor, and rigidity occur suddenly.

184. Subarachnoid hemorrhage is the most common cerebral vascular complication encountered during pregnancy. The risk of rupture parallels the hemodynamic changes, reaching an apex in the third trimester, in concert with blood volume changes. Aneurysms are most prone to rupture during the seventh and eighth months of pregnancy and at delivery.

185. The putamen.

186. Addisonian crisis is an adrenal insufficiency emergency with symptoms of mental status changes and muscle weakness. Signs of postural hypotension, shock, hyponatremia, hyperkalemia, hypoglycemia, and hyperthermia may be seen. For a glucocorticoid emergency, administer 100 mg IV hydrocortisone (Solu-Cortef) immediately (STAT) and then 50 mg IV every 6 hours. Concurrently, one should also give cortisone acetate 75 to 100 mg intramuscularly (IM) STAT, and then 50 mg IM every 6 hours. For a mineralocorticoid emergency, it is best to give desoxycorticosterone acetate 5 mg IM twice daily.

187. Neurogenic pulmonary edema is associated with SAH, head trauma, and seizure disorder. It is caused by an increased capillary permeability in the lungs associated with an increased in sympathetic discharge. Treatment is aimed at reducing ICP, maintaining positive pressure ventilation, and supportive care.

188. Prochlorperazine (Compazine) 10 mg IV.
189. Tension pneumocephalus.
190. CSF leakage and infection.