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SPECIAL FEATURE

Management of Nontraumatic Intracranial Emergencies: A Clinical Update

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Dr. Sharma reports no financial relationships relevant to this field of study.

While not an exhaustive review of the literature, this article attempts to provide evidence-based, practical guidelines to the frontline clinician in the nontrauma intensive care setting. Management strategies are reviewed for intracranial bleeding and acute ischemic stroke.

INTRACRANIAL HEMORRHAGE ASSOCIATED WITH NOVEL ORAL ANTICOAGULANTS

Most intensivists will be confronted with reversing the effect of anticoagulation for patients on oral anticoagulants with spontaneous intracranial hemorrhage (ICH). In ICH associated with any anticoagulant, the goal (in addition to immediate discontinuation of the anticoagulant) is rapid reversal of the anticoagulant effect specific to the drug in question and then, more importantly, maintenance of the reversal for at least three to four days.

Novel oral anticoagulants (NOACs) in use include dabigatran (thrombin antagonist/direct thrombin inhibitor, or DTI) and apixaban, rivaroxaban, and edoxaban (Xa antagonists). Any kidney or moderate to severe hepatic impairment is reason to suspect the accumulation of either dabigatran or the Xa antagonists. Dabigatran has a longer half-life (seven to nine hours; up to 14 hours in the elderly) compared to the Xa antagonists. If three to five half-lives have elapsed since the last dose, reversal agents may not be needed. The caveat is renal insufficiency or hepatic impairment. It is generally accepted that normal laboratory testing in the face of life-threatening bleeding in a patient on a NOAC requires treatment. In the case of dabigatran, an elevated partial thromboplastin time (PTT) indicates the presence of (possibly) supratherapeutic levels of dabigatran; however, a normal PTT does not exclude

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its presence. A normal thrombin time essentially excludes any residual effect. Among patients on Xa inhibitors, the international normalized ratio (INR) may be elevated, but the anticoagulation effect is best assessed with anti-Xa activity. Four-factor prothrombin complex concentrate (4F-PCC; Kcentra) is recommended in the setting of bleeding related to both Xa antagonists and DTIs.[1] The usual dose is 50 units/kg (lower doses are not recommended)[2]; 4F-PCC dosing is not typically repeated unless, for example, significant kidney disease raises the suspicion of drug accumulation. In that scenario, dosing can be repeated in 12-24 hours. The effect of dabigatran can be reversed using a specific monoclonal antibody (idarucizumab) dosed at 5 g intravenously. Repeat dosing is usually indicated in overdose scenarios, persistent prolongation of the activated partial thromboplastin time (aPTT), or with ongoing severe bleeding. Dabigatran can be removed using hemodialysis.[3] Andexanet alfa is a specific reversal agent available for bleeding in the setting of Xa antagonists. In contrast to dabigatran, Xa antagonists cannot be dialyzed because of their highly protein-bound state. It may be important to consider specific antagonism for both DTIs and Xa antagonists over 4F-PCC in scenarios where thrombosis may be catastrophic (e.g., prosthetic heart valves).

INTRACRANIAL HEMORRHAGE ASSOCIATED WITH VITAMIN K ANTAGONISTS

An INR of > 1.4 indicates the presence of a vitamin K antagonist (VKA). Reversal strategies include fresh frozen plasma (FFP), 4F-PCC, and vitamin K. 4F-PCC is preferred (typically dosed at 50 U/kg), given the lower risk of infection and more rapid/sustained correction of INR. Intravenous vitamin K dosed at 10 mg is recommended as well and may be repeated if the INR stays elevated.[4] This strategy will correct the INR out to 48 hours, and repeat dosing with FFP or 4F-PCC typically is not needed but may be considered if the INR continues to be elevated or if bleeding is life-threatening. A repeat INR 15-60 minutes after 4F-PCC administration is recommended and at six- to eight-hour intervals thereafter for 24-48 hours.

INTRACRANIAL HEMORRHAGE ASSOCIATED WITH ANTIPLATELET THERAPY

Reversal of the antiplatelet effects of aspirin/clopidogrel and glycoprotein IIb/IIIa inhibitors in the face of catastrophic intracranial bleeding remains controversial. The controversy has been amplified with publication of the PATCH trial.[5] In this trial, researchers randomized patients with supratentorial intracranial bleeding (excluding epidural and subdural bleeds, as well as those patients with planned surgical intervention within 24 hours and those on vitamin K antagonists). The patients had to have been taking antiplatelet therapy (aspirin/clopidogrel/ticagrelor/dipyridamole or a COX-2 inhibitor) within seven days of presentation. Patients receiving dipyridamole received five units of platelets, and those receiving adenosine-diphosphate (ADP) receptor blockers (clopidogrel or ticagrelor family), with or without an additional antiplatelet agent, received 10 units of platelets. No differences in outcomes were found irrespective of single or dual antiplatelet therapy. More patients in the platelet transfusion group died or had residual deficits at three months. No differences in the rate of hematoma growth were evident at 24 hours. Based on this single trial, routine platelet transfusion cannot be recommended in the setting of antiplatelet therapy-related ICH; another trial is ongoing (Clinicaltrials.gov NCT00699621). Desmopressin promotes platelet "stickiness," and there is a suggestion of benefit based on small studies in the setting of ICH[6]; a single 0.4 mcg/kg administered intravenously is reasonable. Desmopressin also is reasonable in patients with uremia. Patients with ICH while being treated with glycoprotein IIb/IIIa inhibitors will not benefit from platelet transfusion given the long half-life of these drugs, but based on very low-quality evidence, cryoprecipitate may be used.

INTRACRANIAL HEMORRHAGE ASSOCIATED WITH FIBRINOLYSIS

In addition to discontinuation of the thrombolytic infusion, administration of antifibrinolytics and fibrinogen typically is initiated. Symptomatic ICH is more likely to occur with fibrinogen levels < 150 mg/dL, and recent guidelines recommend measuring and sustaining fibrinogen levels above this cutoff,[7] with some clinicians

choosing a 200 mg/dL cutoff. Cryoprecipitate enables repletion of fibrinogen and typically will result in an increase in levels by 70 mg/dL in a 70-kg adult per 10 units administered. Fibrinogen levels should be measured at regular intervals (typically every six hours) to assess the need for further therapy. Tranexamic acid or e-aminocaproic acid inhibits fibrin degradation by antagonism of the conversion of plasminogen to plasmin. Case studies describe the use of these agents in the setting of ICH wherein transfusion of blood products was not possible.[8] When indicated, e-aminocaproic acid is dosed at 4 g to 5 g intravenously and tranexamic acid at 10 mg/kg to 15 mg/kg intravenously over 20-30 minutes. Coexistent thrombocytopenia and/or uremia may be indications for platelet transfusion.

MANAGEMENT OF SPONTANEOUS INTRACRANIAL HEMORRHAGE

Malignant processes or an arteriovenous malformation is more likely among younger patients (< 55 years), presentations with lobar hemorrhage, and among those without a history of hypertension. This group of patients may be targeted for early secondary imaging with either contrast computed tomography (CT) angiography or magnetic resonance imaging (MRI).[9] Three trials (STICH, STICH II, and STITCH, which looked at trauma-associated intracranial bleeds) have assessed the utility of early surgery in the setting of lobar intracranial hemorrhage. These trials excluded aneurysmal bleeds or bleeds related to arteriovenous malformations. The STICH and STICH II trials were negative from the perspective of early surgery, but were thought to be due to "mixing" of patients who may benefit from surgery vs. those who were harmed. A recent reanalysis of these trials suggests that large spontaneous ICH and trauma-related ICH with an intermediate Glasgow Coma Scale (GCS) score of 10-13 are more likely to benefit from surgery, which mirrors practice in clinical settings wherein a high GCS implies a net harm from surgery and a low GCS implies no benefit.[10] With respect to posterior fossa bleeds (cerebellar bleeds, for example), a patient-level meta-analysis of recent studies (2005–2016) suggests that functional outcomes may relate to hematoma volume.[11] Brain stem bleeds portend an extremely poor outcome, and surgical evacuation is not recommended. Minimally invasive procedures (clearance of ICH with thrombolytics instilled in the hematoma and stereotactic aspiration of hematomas) have been studied with randomized trials published recently.[12] No differences in death or disability were noted at one year.[13]

Intraventricular bleeds and intraventricular extension of ICH are associated with a high risk for the development of hydrocephalus. Immediate ventriculostomy and external ventricular drainage are indicated if ventricular enlargement is noted on imaging. The presence of edema around the hematoma or large-volume ICH, and suspicions for intracranial hypertension, are usual indications for infusion of saline and maintaining a serum sodium level of 145 mEq/L to 150 mEq/L. This is discussed further later.

SUBDURAL HEMATOMAS

Early surgical intervention is indicated if a significant (> 4 mm) midline shift is noted, the hematoma is > 10 mm in size, or the patient has signs of herniation or is deteriorating neurologically. Smaller hematomas may be managed by observation alone, especially in the elderly and if the hematomas are chronic.[14] Some of these will disappear completely over time, but for those that do not, burr hole drainage followed by placement of a drain into the subdural space was safe and reduced mortality and recurrence at six months in one study.[15]

BLOOD PRESSURE GOALS IN INTRACRANIAL HEMORRHAGE

Two trials, ATACH2[16] and INTERACT2,[17] confirm the safety of rapid reduction of systolic blood pressure (SBP) to < 140 mmHg and diastolic blood pressure (DBP) to < 80 mmHg. Functional outcomes at 90 days were improved with intensive lowering of blood pressure in INTERACT2. However, mortality was not reduced in either ATACH2 or INTERACT2. A secondary analysis of the INTERACT2 data suggested that larger reductions in SBP (≥ 20 mmHg compared to < 10 mmHg) in the first hour after randomization translated into better outcomes.[18] An optimal SBP of 130-139 mmHg is suggested, and intensive blood pressure lowering across a wide range of presenting blood pressures is safe and efficacious, since the blood pressure prior to randomization did not predict outcomes.[19]

MANAGEMENT OF ACUTE ISCHEMIC STROKE

Intravenous thrombolysis is the standard of care for patients presenting with acute ischemic stroke (AIS), and the data support administration up to 4.5 hours after symptom onset.[20] MRI may guide thrombolytic therapy when the symptom onset time is unclear, and mechanical thrombectomy can be used up to 24 hours after symptom onset in carefully selected patients.[21,22] Anterior circulation stroke may benefit specifically from the use of mechanical thrombectomy procedures up front.[23] Although there are no randomized trials to guide blood pressure goals in patients with AIS, guidelines suggest that blood pressure should be reduced to < 180/105 mmHg in patients being considered for intravenous thrombolytics and should be maintained in the same range for at least 24 hours after administration of thrombolytic therapy. Identical goals are recommended for those undergoing mechanical thrombectomy and for 24 hours after therapy. One study provides guidance with respect to blood pressure

goals in patients awaiting mechanical thrombectomy,[24] with an SBP goal of > 150 mmHg recommended while the proximal artery awaits reperfusion. Maintenance of collateral flow is the goal in this setting. Once reperfusion has been achieved, a normal SBP (< 140 mmHg) is recommended.[25]

On occasion, comorbid conditions such as aortic dissection, acute myocardial infarction, and acute pulmonary edema related to congestive heart failure may demand a reduction in blood pressure. In these scenarios, an acute reduction in blood pressure by 15% is recommended, followed by slower reductions to goal. In all of these scenarios of AIS, rapid reduction in blood pressure risks exacerbation of ischemia. For patients not receiving reperfusion therapies, a goal of < 220/120 mmHg is appropriate and a 15% reduction acutely if higher than 220/120 mmHg is reasonable and likely safe.

Nicardipine is the workhorse of hypertension treatment in the setting of AIS. The usual starting dose is 5 mg/kg/hour, titrating to a maximal dose of 15 mg/kg/hour. Alternatively, labetalol may be used at 2-8 mg/minute. In extreme elevations of blood pressure (DBP > 140 mmHg, for example), a nitroprusside drip is recommended if there are no contraindications. Hydralazine and enalaprilat are less effective but may be used as well. It seems important to avoid variation in blood pressure in the first 24 hours after reperfusion; in one study, standard deviations in SBP variation of 15.5 ± 5.4 mmHg were found in those with worse outcomes compared with 13.8 ± 5.1 mmHg in patients with good outcomes.[26]

The treatment of relative hypotension in the setting of AIS is controversial, with expert opinion suggesting augmentation of blood pressure with vasopressors (typically phenylephrine or norepinephrine) is safe.[27] One case report suggested improvement in both cerebral perfusion and symptoms with mean arterial pressure (MAP) augmented from 90 to 110 mmHg with vasopressors.[28] A symptom-driven therapeutic trial in patients with relative hypotension seems reasonable, but no specific recommendations can be made with respect to the type of fluid administered or the type of vasopressor to increase blood pressure; most practitioners would prefer normal saline and avoid albumin.

MANAGEMENT OF SUBARACHNOID HEMORRHAGE

Most patients with subarachnoid hemorrhage (SAH) will receive endovascular delivery of a coil based on the pivotal International Subarachnoid Aneurysm Trial (ISAT) that randomized patients to coiling or neurosurgical clipping. One survey of neurointensivists found a large variation in blood pressure goals in the setting of SAH. In this survey, most targeted an SBP of < 140 mmHg prior to treatment of the aneurysm and allowed permissive hypertension with SBP ranging from 160-240 mmHg after treatment. No randomized trials provide guidance for blood pressure goals in the setting of SAH.[29] Delayed cerebral ischemia (DCI), previously identified as cerebral vasospasm, manifests as new neurological findings in a patient with no new bleeding or infarct on imaging and typically manifests six to 10 days after presentation. Some centers proactively monitor for DCI with transcranial Doppler (TCD) looking for increasing peak velocities in the large vessels that serves as a surrogate for vasospasm. The sensitivity of TCD is worst for the anterior cerebral artery (ACA) at 45%, followed by the middle cerebral artery (MCA) at 64%, and is best for the internal carotid artery (ICA) at 80%.[30] The specificity is worst for the ICA (77%), followed by the MCA (78%), and is best for the ACA (84%).[30] Normal saline infusions and norepinephrine or phenylephrine to allow for a 15% increase in MAP if there are new findings suggesting DCI are typical interventions to try and counteract vasospasm. Refractory vasospasm portends a poor prognosis, and step up to interventional techniques may be necessary. Other manifestations of SAH can include fever, anemia, a prothrombotic state leading to venous thromboembolism, as well as cardiac complications ranging from arrhythmias to cardiogenic shock and neurogenic pulmonary edema. Anemia (defined as hemoglobin < 10 g/dL) is common, with the prevalence increasing over the weeks after presentation. Anemia was found to be an independent factor for poor long-term outcome and death.[31] A multicenter cohort study suggested that transfusion once hemoglobin drops below 8 g/dL is common practice.[32] A randomized trial is planned to assess conservative vs. liberal transfusion goals in this setting.[33] The presentation of neurogenic pulmonary edema closely mimics cardiogenic pulmonary edema. It is hypothesized that a sudden surge of catecholamines that occurs at the time of an acute increase in intracranial pressure is the common pathway for both cardiac abnormalities and neurogenic pulmonary edema.[34] Since cardiac abnormalities may lead to pulmonary edema as well, it is prudent to exclude worsening left ventricular function as a cause of worsening pulmonary opacities, hypoxemia, and frothy blood-tinged secretions. Neurogenic pulmonary edema typically resolves within 72 hours of onset, coincident with a drop in catecholamine levels. Management is supportive, and mechanical ventilation to ARDSNet standards is recommended. Cardiac abnormalities, including Takotsubo cardiomyopathy and a non-ischemic cardiomyopathy leading to cardiogenic shock, may occur. Electrocardiogram (ECG) changes can span the spectrum from sinus tachycardia to ST-T changes mimicking acute myocardial infarction or ischemia. Echocardiographic findings can include regional wall

motion abnormalities in any coronary artery territory.[35] Management in these scenarios is supportive.

SEIZURE PROPHYLAXIS IN INTRACRANIAL EMERGENCIES

Seizures may manifest simply as fluctuations in the level of consciousness in the absence of any other explanation or they may present as full-blown status epilepticus. Focal seizures with secondary generalization are frequent. A continuous EEG study showed that electrographic seizures occur less frequently in the setting of acute ischemic stroke (6%) compared with intracerebral hemorrhage (28%), with most occurring within three days.[36] This study also noted that seizures were most common in lobar hemorrhages but occurred frequently (21%) in subcortical hemorrhages.[36] SAH extension in the setting of lobar hemorrhage was associated with an increased risk of early seizures.[37] While seizure risk increases with increased proximity to the cortex (with the highest risk within 1 mm of the cortex), patients with infratentorial hemorrhages also may be at increased risk.[38] Another study assessed the risk of seizure activity in the setting of ICH with continuous EEG and found seizure activity in one-third of patients, with half of these seizures only evident on EEG.[39]

There currently is no consensus on seizure prophylaxis in the setting of ICH. Most practitioners would choose to treat prophylactically only those with the highest risk for seizures (lobar hemorrhages, expanding hematomas, subcortical hemorrhages — especially those within a millimeter to the cortex — and those with SAH extension and selected subdural hematomas). Access to continuous EEG monitoring may influence the decision to treat prophylactically as well. In the absence of clinical trials, levetiracetam is the preferred drug because of a more favorable pharmacokinetic and safety profile compared to phenytoin. Specifically, in the setting of SAH, extended prophylaxis (longer than three days and up to seven days) with levetiracetam has been recommended.[38,40] It is not clear whether discontinuation of prophylactic antiepileptic drug therapy after definitive aneurysm therapy has been delivered affects outcomes. Most patients with subdural hematomas will be treated prophylactically with antiepileptic drugs for a short duration. The optimal duration is not defined, but most patients receive up to seven days of therapy.

MANAGEMENT OF INTRACRANIAL HYPERTENSION

Intracranial hypertension manifests as the movement of brain structures due to cerebral edema, either across the midline ("midline shift") or herniation of the brain stem. One study published recently found a threshold of 4 mm of midline shift as "the optimal threshold associated with a poor outcome in ICH patients."[41]

Intracranial pressure (ICP) ≥ 20 mmHg is associated with worsening outcomes in intracranial emergencies, including ICH and SAH. Treatment of life-threatening intracranial hypertension relies mostly on neurosurgical intervention, although osmotherapy with hypertonic saline or mannitol is a key intervention. Ventilating to hypocapnia (target $PaCO_2$ to 25-30 mmHg) and raising the head of the bed to decompress the brain also may be tried. There still is no consensus on which agent (mannitol or hypertonic saline) is preferable in the setting of high ICP. A single meta-analysis of multiple randomized trials suggested that hypertonic saline was superior to mannitol in the treatment of episodes of intracranial hypertension.[42] A survey of neurointensivists done in 2011 showed that more (55%) preferred hypertonic saline.[43] Mannitol is dosed intravenously 1 mg/kg bolus followed by 0.25 mg/kg to 0.5 mg/kg every four to six hours to target a serum osmolality of 290-300 mosm/L and the plasma osmolal gap of < 55.

If hypertonic saline is used, 3% saline infusions are used to target a serum sodium of 145-155 mEq/L and supplemented with 30 mL boluses of 23.4% saline as needed for acute increases in ICP. Electrolytes and serum osmolarity are measured every four to six hours. The former is a potent diuretic and may induce hypovolemia due to an osmotic effect; the latter may induce hypertension and worsen coexisting congestive heart failure given the sodium content of the solution. It is recommended that serum osmolarity not exceed 320 mosm/L and that serum sodium not exceed 155-160 mEq/L regardless of infusion type. ■

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ABSTRACT & COMMENTARY

Laryngeal Injury Is Common After 12 Hours of Intubation

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Dr. Nadler reports no financial relationships relevant to this field of study.

SYNOPSIS: After 12 hours of intubation, most patients showed laryngeal injury, including mucosal ulceration, that led to impaired breathing and voicing 10 weeks after extubation.

SOURCE: Shinn JR, Kimura KS, Campbell BR, et al. Incidence and outcomes of acute laryngeal injury after prolonged mechanical ventilation. *Crit Care Med* 2019;47:1699-1706.

Tracheal intubation is a common medical procedure, often for a limited time. However, many patients in the intensive care unit (ICU) are intubated for 12 hours or more. Intubation for this length of time is understood to cause tracheal injuries due to the deformation of the endotracheal tube by the posterior tongue placing pressure on the posterior glottis. The true incidence of these injuries has not been evaluated contemporaneously with extubation, but rather they have been studied once symptoms arise.

The current study prospectively evaluated patients with prolonged intubation for the presence of laryngeal injury. In a single center, patients 18 years of age or older who were intubated for more than 12 hours underwent endoscopic evaluation of the trachea within 36 hours of extubation. Patients who presented with tracheostomies, known laryngeal injuries, or previous radiation exposures were excluded. Acute laryngeal injury (ALgI) was defined by one or more features, such as glottic mucosal ulceration/granulation or subglottic granulation tissue/stenosis. For this study, researchers screened 833 patients to identify 487 who survived to extubation, 100 of whom underwent nasolaryngoscopy. ALgI was documented in 57 of these patients.

In a multivariate logistic regression, a larger endotracheal tube (ETT) size, the presence of diabetes, and elevated body mass index (BMI) were shown to predict ALgI. There was no significant difference between the groups regarding Acute Physiology and Chronic Health Evaluation (APACHE) scores, pressor needs, steroid use, delirium, or acute respiratory distress syndrome (ARDS). Ten weeks after extubation, patients were contacted by phone and validated questionnaires were administered to evaluate for voice and breathing changes. The Voice Handicap Index (VHI)-10 and Clinical Chronic Obstructive Pulmonary Disease Questionnaire (CCQ) were used. Of the 67 patients who completed the questionnaires, a higher proportion of patients with ALgI reported breathing and voice concerns compared to patients without ALgI.

■ COMMENTARY

This prospective study demonstrated a high incidence of laryngeal injury (57%) in patients intubated for more than 12 hours. Both modifiable and unmodifiable factors contributed to the injuries. Clearly, the patient's weight, BMI, and the presence of diabetes cannot be controlled. However, the length of intubation and the size of the ETT can be prospectively changed. Although not statistically significant, for each three-day increase in duration of intubation, the odds ratio (OR) for ALgI increased by 1.49 (95% confidence interval (CI), 0.79-2.82; $P = 0.22$). Compared with 7.5 mm ETTs, 7.0 mm ETTs showed an OR for ALgI of 0.04 (95% CI, 0.004-0.43; $P = 0.007$). When compared with 8.0 mm ETTs, the use of 7.0 ETTs had an OR for ALgI of 0.03 (95% CI, 0.003-0.31; $P = 0.003$). The authors delineated those patients with "appropriately" or "inappropriately" sized ETTs based on previous morphometric studies.[1] Although with a smaller group size, the difference was not statistically significant, patients with "inappropriately" sized ETTs had a higher incidence of ALgI (76.5% vs. 53%, $P = 0.075$). Interestingly, critical care and emergency medicine providers tended to place larger ETTs compared with anesthesiologists and other emergency medical personnel.

This study also examined difficulties patients experienced with vocalization and breathing 10

weeks after extubation. Patients with ALgI reported worse voice outcomes compared to those without ALgI (median VHI 2 vs. 0; $P = 0.005$), although the minimal clinically significant change for this metric is 4. The CCQ is used more commonly to evaluate populations with chronic obstructive pulmonary disease, but with this application, patients with ALgI reported worse breathing compared to those without ALgI (median CCQ 1.05 vs. 0.20; $P < 0.001$) with the minimal clinically significant change being 0.4. Thus, even 10 weeks after extubation, patients with ALgI experienced clinically significant changes in breathing.

Intubation for more the 12 hours is common in the ICU, and laryngeal injury is common in this group. Patients with ALgI had a higher incidence of persistent breathing difficulties compared with patients without ALgI, even 10 weeks after extubation. This risk of ALgI and long-term breathing difficulties may be reduced by using smaller ETTs and extubating patients as soon as is safe. ■

REFERENCE

1. Coordes A, Rademacher G, Knopke S, et al. Selection and placement of oral ventilation tubes based on tracheal morphometry. *Laryngoscope* 2011;121:1225-1230.

CME/CE INSTRUCTIONS

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CME/CE QUESTIONS

1. **In patients in whom seizure prophylaxis therapy is indicated in the setting of intracranial hemorrhage, what is the typical duration of therapy?**
   a. Two days
   b. Five days
   c. 14 days
   d. Seven days

2. **Which of the following is the preferred drug for seizure prophylaxis in intracranial emergencies in the critically ill?**
   a. Valproic acid
   b. Levetiracetam
   c. Phenytoin/fosphenytoin
   d. Carbamazepine

3. **In the study by Shinn, which of the following risk factors increased the likelihood of acute laryngeal injury in patients intubated for more than 12 hours?**
   a. Greater endotracheal tube size (7.0 mm vs. 8.0 mm)
   b. Poor dentition
   c. Decreased duration of intubation
   d. Lower body mass index

CME/CE OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- identify relevant topics in the practice of critical care medicine;
- utilize recommendations from current clinical guidelines; and
- manage common critically ill patient and ICU administration scenarios.