Ischemic Stroke and ICU Care

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ABSTRACT

Acute ischemic stroke is a common disorder with a significant impact on morbidity and mortality in the United States. The number of interventions for acute stroke patients has increased over the past 15 years and patients increasingly require intensive care. There are several issues that are specific to ischemic stroke patients in intensive care unit (ICU) settings, including the care of the postthrombolytic stroke patient, respiratory issues in stroke care, evaluation of worsening or change in neurological status, and attention to factors that affect the ischemic penumbra. The management of the stroke patient in the critical care setting is discussed in this article.

KEYWORDS: Ischemic stroke, hypertension, intracranial pressure, neurocritical care

Ischemic stroke diagnosis, management, and treatment have undergone significant changes in the past 15 years. This period includes the Food and Drug Administration (FDA) approval of an intravenous technique for acute thrombolysis, expansion of interarterial thrombolysis with medications and clot retrieval devices, and clarification of secondary prevention interventions vetted by several prospective treatment trials. Treatment is not the only change; the way health care providers view acute stroke has changed. At least in some ways, the acute thrombolysis trials changed ischemic stroke. Stroke has gone from a diagnosis that was important and life threatening, but without effective treatments, to one where "every second counts" in the impact of intervention to outcome. In many ways, how patients and health care providers think about stroke has changed. The American Stroke Association through the Get with the Guidelines (GWTG) Program organized basic treatments and interventions that stroke patients should receive and a database structure so that individual hospitals and the stroke community at large could keep data. In 2003, the American Board of Psychiatry and Neurology began testing and certifying neurologists as Vascular Neurologists, officially designating a subspecialty in Neurology. About the same time, The Joint Commission on the Accreditation of Healthcare Organizations (JCAHO) began to evaluate and accredit centers as primary stroke centers with an eye to specific parameters required for stroke patients and a structure to monitor and enforce high-quality care for those designated programs. Lastly, the role of the critical care unit in stroke treatment has become more important as the complexity of stroke care increases and the requirements for subspecialty expertise changes. The future of acute stroke care and the role of critical care will be increasingly intertwined. In this article, I will discuss stroke care in the intensive care unit (ICU) setting, ICU care of stroke patients after fibrinolytic therapy has been delivered and before transfer to the floor where secondary stroke prevention management strategies begin, as well as future challenges in the prevention and treatment of ischemic stroke.

STROKE UNITS AND NEURO INTENSIVE CARE UNITS

The concept of the stroke unit has been in the literature for some time. There have been many publications that

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address the need for one place or organizational structure to take care of stroke patients. This is not unlike cardiac or pulmonary care where a team of health care providers sensitive to issues surrounding the diagnosis seems to provide better care than general medical–surgical floors do for specific diagnoses. Stroke units have shorter length of stay, lower rates of deep vein thrombosis (DVT)/pulmonary embolism (PE), lower cost, earlier involvement of rehabilitation specialists, and improved outcomes after discharge.[1–3]

The prevailing concept is that stroke units are an ideological or cultural unit where diagnostic and therapeutic issues are addressed by clinicians and nurses with training and experience in the care of stroke patients. This is probably in the best interest of stroke patients. Stroke is an extremely heterogeneous disorder, more so than many primarily vascular disorders because of the complexity of the end organ affected. In addition, stroke patients can be more ill than other patients with neurological problems, as stroke is not an isolated disorder.[4] Patients with stroke or transient ischemic attack (TIA) are more likely to have peripheral vascular disease, coronary artery disease, atrial fibrillation, and to develop pneumonia and DVT/PE, and therefore often require global care of medical issues.[5] Taken to the extreme, patients with severe strokes, either in size or in the amount of deficits, may need at least a brief period in an ICU.

It is probably not necessary to admit all patients with ischemic stroke to an ICU. One retrospective study by Briggs et al,[6] compared outcome of stroke patients that were admitted to general wards versus an ICU. They found that patients with mild stroke (National Institute of Health [NIH] stroke scale < 8) who were admitted to a ward (non-ICU) service had lower complication rates, more favorable discharge Rankin scales, and lower cost than those admitted to an ICU. They also found that strokes of moderate severity (NIH 8 to 16) did similarly between ward service and ICU, but there was a trend toward better Rankin scores on discharge from an ICU than ward service. This study did not address those patients with severe or complicated strokes.

There is emerging literature showing that neuro ICU (NICU) care may be superior to general ICU care for intracerebral hemorrhage and other issues with primary neurological or neurosurgical disorders. There also appears to be an impact of a NICU or the neuroscience ICU on neurosurgical patient outcomes and cost of care.[7–9] Although there is evidence-based support for an intensivist-directed specialty ICU model of care with the issues of increased intracerebral pressure, neurological worsening, fever, requirements of intubation, and progression of deficits are likely similar, but this is unproven at this time. It is probably the case that neurocritical care is an important adjunct to acute stroke care; centers that feel they are capable of providing comprehensive care of all forms of stroke and intracerebral hemorrhage should probably have access to one, either on the premises or through a closely allied institution.[10]

That being said, there are limited resources for the expansion of the neurocritical care unit, and a majority of primary JCAHO Stroke Centers have access to an ICU, but not necessarily to a neurocritical care unit. Thus, a discussion of the issues that commonly arise in stroke patients cared for in neurocritical care units or otherwise is vital. The issues related to stroke patients in the ICU to be discussed are (1) care of the postthrombolytic stroke patient; (2) specific respiratory issues in stroke care; (3) evaluation of worsening or change in neurological status; and (4) attention to factors that affect the ischemic penumbra.

Management of the Postthrombolytic Stroke Patient

The present recommendations state that a patient postfibrinolytic therapy for acute ischemic stroke should be admitted to a monitored setting, such as a critical care unit, for 24 hours after intervention. Although a majority of postintravenous/intraarterial (IV/IA) tissue plasminogen activator (tPA) patients does not develop ongoing medical problems, a small proportion of patients may develop problems in the course of the admission.

A known post fibrinolytic problem is allergy to the tPA itself. There are numerous examples of not only allergies, rash, and neck edema, but also life-threatening angioedema post-tPA.[11–14] The hypersensitivity usually occurs in minutes and begins with perioral tingling and lip and tongue swelling. This is a serious complication as angioedema can restrict the patient's airway, and require intubation and potentially tracheostomy, as the patient is usually within the 24-hour period where the tPA is still active. This can be treated like any other allergic reaction with H1 and H2 blockers and steroids.[15] If absolutely necessary, small doses of subcutaneous epinephrine 0.3 cc of 1:1000 can be given. It should be remembered that even with subcutaneous administration, epinephrine can be dangerous as it may increase systemic blood pressure, thereby increasing the risk of hemorrhagic conversion with active tPA in the patient's system. A majority of the cases in the literature resolve without epinephrine.

Fibrinolytic therapy induces a profound hypercoagulable state. There is maximal inhibition of the coagulation cascade, including the conversion of plasminogen to plasmin, which initiates local thrombolysis but can lower total levels of fibrinogen.[16,17] This can lead not only to intracerebral hemorrhage (ICH) but to extracerebral hemorrhage as well. The rate of symptomatic hemorrhagic conversion of ischemic stroke in the IV tPA National Institute of Neurological Disorders and

Stroke (NINDS) trial was 6%, and 8 to 10% in the IA tPA PROACT I and II studies and Interventional Management of Stroke (IMS) studies.[18–22] The most common presenting symptoms of hemorrhagic conversion are worsening of deficits, somnolence, new cranial nerve palsies, and/or new neurological deficits. Thus, careful monitoring of the postfibrinolytic patient is vital, and computed tomography (CT) imaging of the brain for even seemingly trivial changes in mental status should be considered in the first 24 hours. If early (< 24 hours after drug) symptomatic hemorrhagic conversion of ischemic stroke occurs, neurosurgery should be consulted and a reversal protocol should be considered.

Significant extracerebral hemorrhages are not very common but do occur. The majority of extracerebral hemorrhages occur from gingiva, gastrointestinal (GI) system, urogenital organs and retroperitoneal areas, but the rates are significantly lower than with streptokinase. These can occur especially from the site of arterial catherization.[17] Cardiac tamponade secondary to cardiac hemorrhage has been described but is less common when tPA is used for acute stroke[23] compared with acute coronary ischemia trials of tPA. The most concerning sign of impending extracerebral hemorrhage is acute hypotension, and can be seen in cardiac tamponade as well as significant GI hemorrhages. The cardiac tamponade in these cases is not associated with elevated cardiac enzymes, but only with hypotension, bradycardia, and electrocardiogram (EKG) changes. Normalization of systolic blood pressure can be seen after tPA infusion with clot thrombolysis. Presumably, this is due to reperfusion of the brain and loss of compensatory autoinduced hypertension to improve perfusion. This can be seen in patients with recovery from deficits after tPA, especially those who required significant antihypertensive medications to control blood pressure within the acceptable range for tPA guidelines. In the case of a precipitous drop in blood pressure after tPA administration, complete blood count (CBC) and EKG should be performed acutely, and especially if there is no improvement of neurological function accompanying arterial reperfusion. An echocardiogram should also be considered.

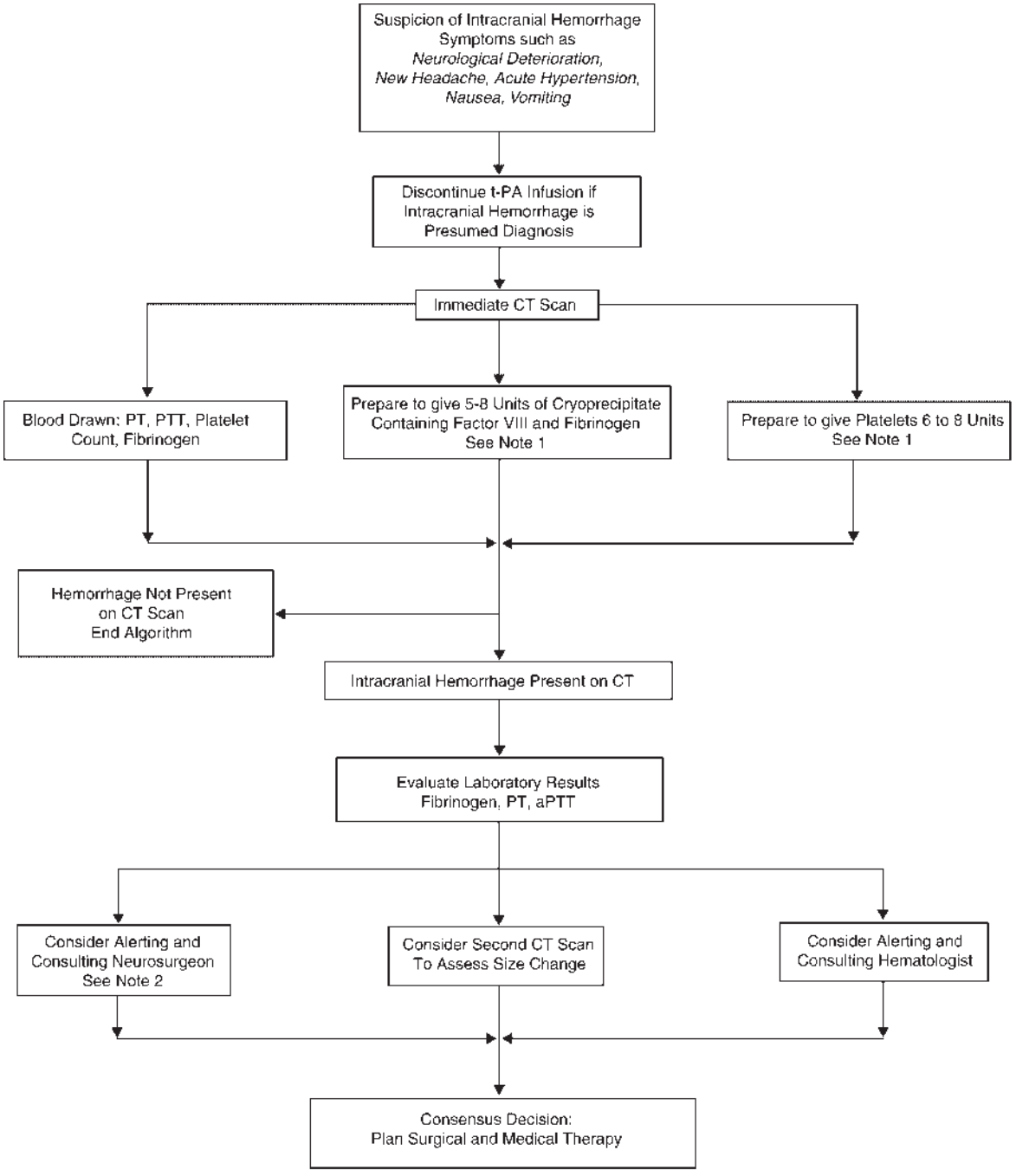
In the case of significant symptomatic extracerebral hemorrhage before the 24-hour window, a tPA reversal protocol should be considered. This should be done only in life-threatening situations, as reversal of tPA will inhibit stroke thrombolysis. The Brain Attack Coalition maintained by the NINDS (www.strokesite.org) describes a reversal protocol in cases of life-threatening intra- and extracerebral hemorrhage, which includes the infusion of cryoprecipitate 5 to 8 units and platelets 6 to 8 units (Fig. 1). Careful monitoring of blood pressure, pulse, and hematocrit during the first 24 hours is vital. Special care should be taken to avoid procedures involving noncompressible sites unless absolutely necessary. Examples of this are central line placement, arterial line placement, arterial blood gas measurement, nasogastric tube or Foley catheter insertion, endotracheal intubation, or any other surgical procedure at a noncompressible site.

Ventilatory Support

Airway evaluation is the first step for management of all critically ill patients, including stroke patients. Stroke patients, especially with multiple cranial nerve involvement, are at high risk for pneumonia and aspiration. A patient should have endotracheal intubation, if necessary to protect the airway, for excessive secretions, loss of protective reflexes, but not necessarily simply in the absence of a gag reflex. Once a stroke patient is intubated, the morbidity and mortality is high and prognosis is guarded.[24–26] Patients who are intubated are often sedated for comfort and better ventilation management. This can compromise stroke care as stroke recurrence, progression, and the development of increased intracranial pressure typically first manifest as a decrease in mental status.

In general, after intubation the head of the bed should be positioned at 30 degrees from the horizontal plane. Obstruction of drainage through the jugular veins from the cranial cavity directly affects intracranial pressure, both by restricting blood flow and reducing cerebrospinal fluid (CSF) resorption.[27–29] In critical care units, attention should be paid to how the endotracheal tube (ETT) is placed and secured. The ETT is usually taped at the maxilla in NICU. Tying the ETT around the neck can impede venous outflow and contribute to ICP issues (Fig. 2). This can be seen when there is significantly decreased compliance due to high ICP and when the patient's head is allowed to flex laterally, leading to the compression of the draining vein.[27–32]

The ventilation settings are best managed with the pulmonary needs of the patient in mind. Care should be taken to avoid, if possible, ventilatory settings that can worsen increased ICP. In those circumstances where patients are intubated for hyperventilation and ICP management is being considered, care should be taken using positive end expiratory pressures (PEEP). PEEP can be directly transmitted to the cranial vault and can exacerbate ICP issues. In general, a PEEP of 10 cm H_20 or less is well tolerated, but higher PEEP can increase ICP.[29,33,34] This point is debated in the literature, and the issue may be that there is no effect of PEEP on ICP unless cerebral autoregulation is lost due to intracranial hypertension. PEEP's effect may also mainly be on mean airway pressures, which when elevated, can increase ICP.[35–37] The use of high PEEP may be necessary in certain cases to maintain adequate



Note 1: Preparations for giving platelets and Cryoprecipitate can be initiated at the first suspicion of hemorrhage so that they would be ready if needed.
Note 2: It is highly recommended to have a plan for obtaining neurosurgical advice.

Figure 1 Intracerebral or extracerebral hemorrhage evaluation and action flow chart (www.stroke-site.org). CT, computed tomography; PT, prothrombin time; PTT, partial thromboplastin time; tPA, tissue plasminogen activator.

oxygenation. Hypoxemia itself leads to cerebral vasodilatation, and if significant, leads to ICP elevation. A careful balance of PEEP and oxygen delivery must be tailored to the individual patient. Mean airway pressures should be kept as low as possible and monitoring of these pressures in developing ICP issues is vital. If patients develop neurogenic pulmonary edema or acute respiratory distress syndrome (ARDS), careful monitoring of ICP, positioning of the head and neck, head of the bed at 30 degrees, mean airway pressure monitoring, and careful oxygenation are required. The effect of elevated carbon dioxide on intracranial vasodilatation and ICP elevation must also be carefully considered in the ventilator strategies in these patients.

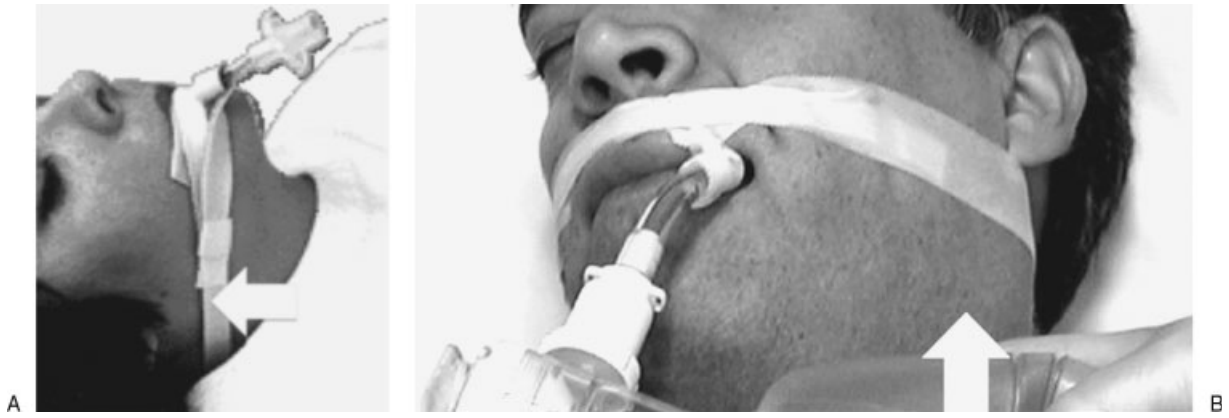


Figure 2 Two positions are shown for securing the endotracheal tube (ETT). (A) Shows the ETT secured below the mandible, which can occlude the bilateral jugular veins and slow venous outfluow from the cranium. However, the head is in a good straight position. (B) Shows correct mandibular ETT securing, but the head is rolled to the right, which can reduce flow through the right jugular vein. (Image A modified from http://www.bpmedicalsupplies.com/images/1200281712412-1968075932.gif. Image B modified from http://www.accessce.com/online_modules/legacy_images/images/var/57478d39.jpg).

Evaluation of Worsening or Change in Neurological Status

Ischemic stroke is not a singular event with a predictable outcome. Just like any other acute or devastating disorder, the goal is to address acute decompensation after the initial event. Similarities can be drawn to the development of malignant arrhythmias in acute myocardial infarction or critical illness polyneuropathy/myopathy or ARDS in patients with acute pulmonary processes. So too patients with acute ischemic stroke can develop complexities associated with the initial event that are primarily neurological in origin. The rate of neurological worsening in stroke patients is probably on the order of 13 to 25%, with most common causes being progressive or recurrent stroke and increasing ICP.[2,38]

The nature of stroke is that while objective measures such as serial CT scans or acute magnetic resonance imaging (MRI) testing can show progression, the best and most dependable way to monitor stroke progression is through serial neurological examinations. This includes the NIH stroke scale or basic neurological examination. The neurological examination looks not only at the degree of deficits, but also more importantly at the nearby cortical and subcortical function. A right anterior division middle cerebral artery (MCA) stroke affecting frontal lobe may have deficits of aphasia and hemiparesis. Critical worsening may not be worsening weakness, but the development of a new neglect or field cut. This suggests a new area of cortical involvement. To determine that new imaging is required and that there is a new deficit, two things must be present. First, there needs to be the basic skill set of the intensivist to perform a neurological evaluation, not simply testing and retesting of the baseline deficit, but the ability to explore neurological function of adjacent brain tissue. Second, there needs to be a culture in the ICU of avoidance of long-acting sedative/hypnotic medications in patients with stroke, so that new or expanded deficits can be found on examination.

SEDATIVES IN STROKE PATIENTS

Sedative medications are useful for patient comfort and in ICP management, but overuse or use of long-acting medications that cannot quickly be reversed should probably be avoided.[39] Oversedation of stroke patients was one of the difficulties of the CLASS study and other neuroprotective trials. Concerns were raised about the ability to care for sedated stroke patients and detection of progression and increased intracranial pressure.[40,41] Effective critical care units that treat stroke avoid long-acting sedative medications and understand that agitation can be the preceding symptom of worsening attention and arousal, and not necessarily the indication for higher doses of stronger sedatives. A more comprehensive discussion on the sedation of the neurological patient in the ICU is provided elsewhere in this issue.

RECURRENT STROKE AND HEMORRHAGIC CONVERSION

New alteration of mental status in stroke patients may represent recurrent embolic stroke. New nondominant infarcts can present with a confusional state that resembles toxic/metabolic processes (Fig. 3).[42] Consideration should be given to either embolization or progression of stroke in the patient with an alteration in mental status. This is especially the case before a stroke etiology has been ascertained or at any time a patient is receiving unfractionated heparin for stroke treatment. Hemorrhagic transformation of ischemic stroke is another important cause of progression of neurological deficits. The frequency of hemorrhagic transformation of stroke outside the setting of tPA

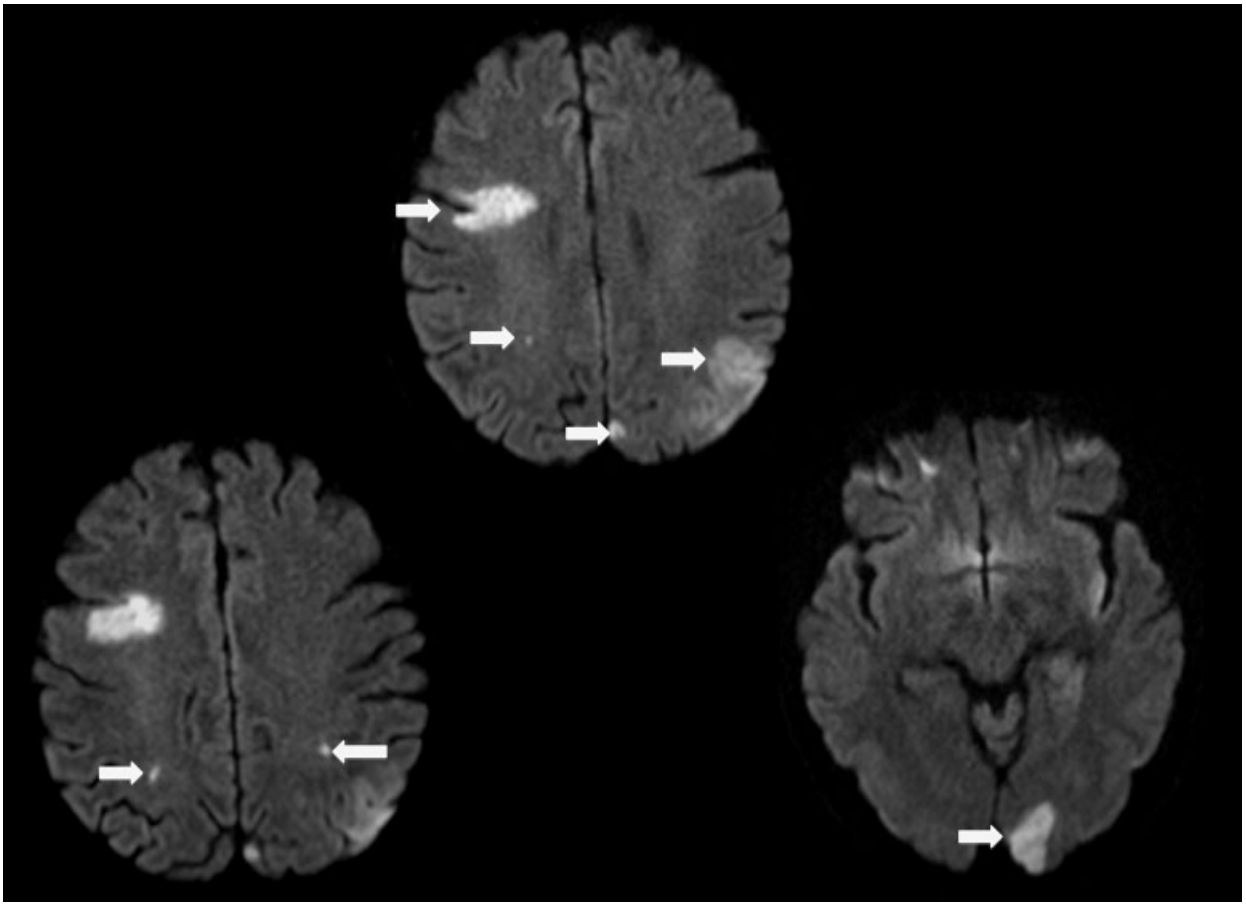


Figure 3 This is patient in the intensive care unit with acute change in mental status without clear focal motor neurological defects. The person was found to have a field cut, neglect, and mild aphasia. In this figure, three slices of a diffusion weighted magnetic resonance image (MRI) scan are shown. The bright lesions (white arrows) represent acute strokes. The diffuse distribution of the strokes suggests an embolic shower as the cause of the alteration of mental status.

use was noted in the International Stroke Trial (IST) as ~2.8% of stroke from atrial fibrillation treated with anticoagulation.[43]

SEIZURES

Seizures are another cause of either progression of deficits or alteration of mental status. First seizure associated with acute stroke is not very common, but the substrate for seizures electrographically may be.[44–46] It is more of an important issue with the acute evaluation of stroke patients and the consideration of IV/IA thrombolytics. Seizures may occur as often as in 2% of strokes at onset.[44] Seizures are more common in intracerebral hemorrhage than in acute ischemic stroke; most of the risk occurs months to years after the event, presumably when the old ischemic lesion leads to scar formation and an epileptic focus. Status epilepticus is quite uncommon, and typically the seizures are single focal motor or generalized tonic-clonic seizures.[47] New seizures should be evaluated with an acute imaging scan to rule out hemorrhagic conversion, and sedating medications to control seizures should probably be avoided. A single seizure should not prompt intubation and general anesthesia before imaging. Medications such as intravenous lorazepam or phenytoin should be reserved for recurrent seizures or ongoing clinical or electrographic status epilepticus. General anesthesia, phenytoin, and lorazepam can make clinical monitoring of the stroke patient in the ICU difficult due to sedation and the not infrequent blood pressure falls that can accompany lorazepam and more often IV phenytoin.[48] There is some concern that medications that are first-generation antiepileptic medications, such as phenytoin, phenobarbital, and benzodiazepines, may impede stroke recovery as well as contribute to sedation while in the ICU.[49] Other first-generation antiepileptic drugs may also contribute to further complicating the patient's medical issues by interacting with antiplatelet or anticoagulant therapy, especially in the case of phenytoin and valproic acid. Medications such as lamotrigine, gabapentin, oxcarbazepine, and levetiracetam do not demonstrate interaction with anticoagulant or antiplatelet therapy and tend not to cause significant sedation.[49] Therefore, consideration of medications for

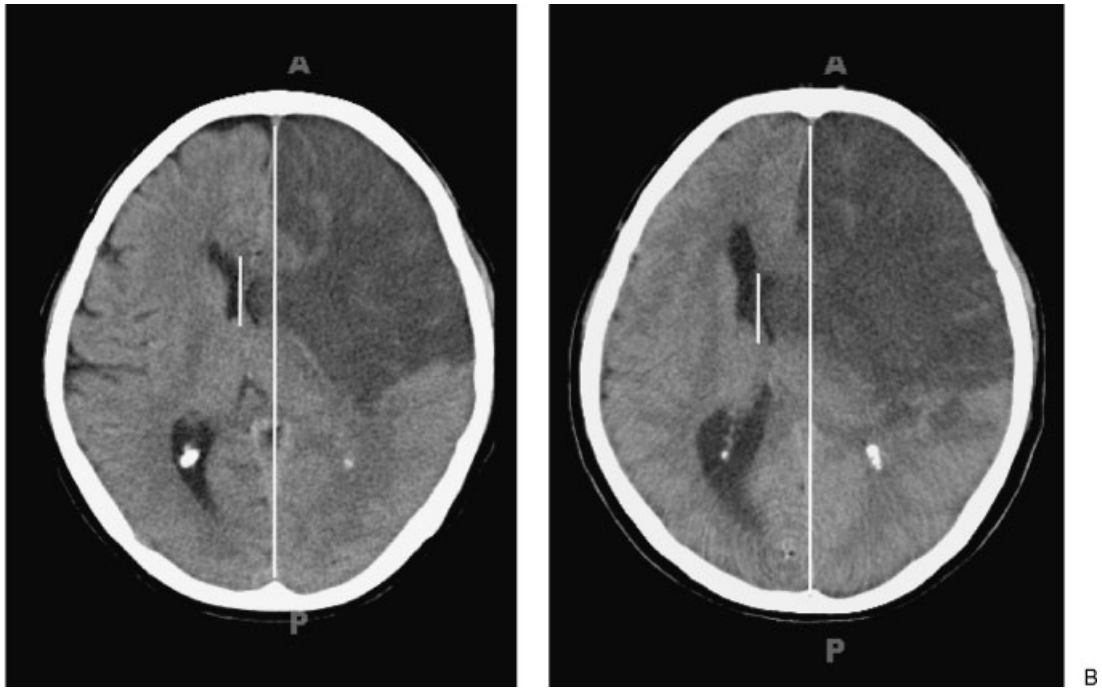


Figure 4 Computed tomography (CT) scans of a patient with a large carotid/ middle cerebral artery (MCA) distribution stroke. (A) Shows midline shift and a large area of cytotoxic edema. (B) Shows the same patient 2 days later despite maximal intracranial perfusion management. The patient had worsening mental status over the 2 days despite no new stroke events.

seizure control, especially if there is a single seizure without status epilepticus, should be done with attention to the patient's neurological status, medical issues, and potential neurological recovery.

INCREASED INTRACRANIAL PRESSURE

Intracranial pressure can increase 3 to 5 days after the initial insult (Fig. 4). The basic management of increased ICP acutely involves positioning of the patient's head at 30 degrees, hyperventilation, osmotic or hypertonic solutions, pressure monitoring, and sometimes surgical decompression (i.e., hemicraniectomy with duraplasty). A comprehensive approach to ICP management is provided elsewhere in this issue.

Care of the Ischemic Penumbra

In addition to the above issues of worsening progression or symptomatic worsening secondary to the stroke itself is the issue of the ischemic penumbra. The ischemic penumbra can be defined as the area of brain that is dysfunctional secondary to ischemia, but is not yet infarcted (Fig. 5). This area of tissue that may infarct or not infarct is a focus of drug research. But, at this point, there have been no clear pharmacological interventions that slow expansion of infarcted tissue into ischemic tissue. Several basic critical care interventions can help, including induced hypertension and hypothermia, fever, and hyperglycemic control.

BLOOD PRESSURE MANAGEMENT

Management of arterial blood pressure is an important and recurrent issue. Several studies have shown that both significant hypertension or hypotension predict poor outcomes. In general, there is an automatic response to central nervous system (CNS) ischemia in the form of hypertension, and the duration of this hypertension can be a little as 24 hours.[50] There is evidence that precipitously lowering blood pressure in patients suffering from an acute ischemic stroke can be detrimental due to impairment of the compensatory elevation of perfusion.[51] Permissive hypertension, defined as allowing the blood pressure to rise without intervention or gentle use of antihypertensive medications and occasionally giving crystalloid IV fluids can be useful if done carefully. The practice of completely withdrawing antihypertensive medications in patients when the details of the cardiovascular and hypertensive history are not known is probably not advisable. Complete withdrawal of angiotensin-converting enzyme (ACE) inhibitors can cause an acute increase in afterload, which may precipitate cardiac ischemia and worsen hypotension in patients with low left ventricular ejection fraction (LVEF) or aortic stenosis. Withdrawal of β-blockers and calcium channel blockers used for rate control of atrial fibrillation can precipitate rapid ventricular rate. Withdrawal of clonidine, ACE inhibitors, methyldopa, calcium channel blockers, and some β blockers can result in significant rebound hypertension.[52–56] Although this has not been carefully studied, antihypertensive medications should

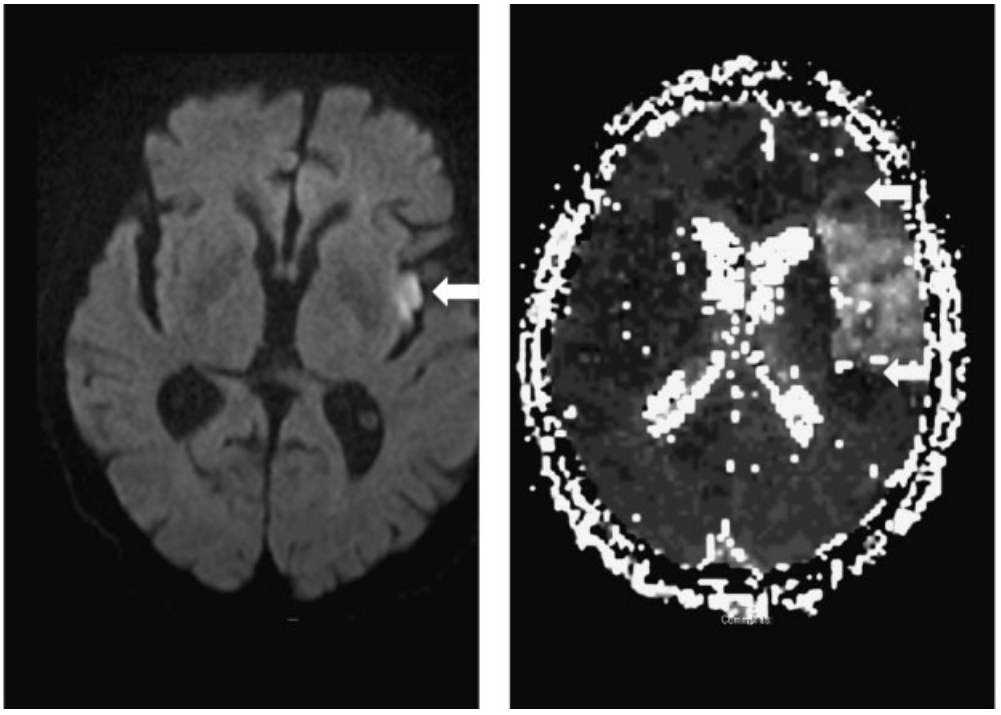


Figure 5 (A) A diffusion-weighted image MRI (DWI) scan shows a small stroke in the left insula (arrow). (B) A perfusion weighted MRI (PWI) scan reveals that the stroke involves a much larger area as shown by arrows. This patient presented with right-sided weakness and aphasia better described by the lesion on PWI seen here than on DWI. This likely represents the ischemic penumbra or brain area at risk.

probably be either continued or reduced by 50% at maximum. Blood pressure can be supported, if necessary, with careful use of crystalloid IV fluids with special attention to fluid overload states.

Induced Hypertension Induced hypertension is a technique of using vasopressors, such as phenylephrine, or a larger volume crystalloid infusion to increase systolic pressure and mean arterial pressure (MAP). Induced hypertension has been studied in several small trials, and when used in conjunction with diffusion weighted/perfusion weighted imaging (DWI/PWI), appears to be useful in the short term.[57–60] The difficulty is that not all acute stroke patients will have a favorable DWI/PWI mismatch or measurable ischemic penumbra. It is likely that patients who have "open artery" physiology with no mismatch may not benefit from induced hypertension. Many institutions do not have access to methods for measuring a perfusion mismatch in a critically ill patient. However, induced hypertension can be a very helpful technique in the ICU for dealing with some patients with fluctuations caused by local hypo-perfusion states. Improvement in objective measures can be seen when the MAP is increased to a point where underperfused brain begins to receive normal perfusion (Fig. 6).[59] It is important to be able to document a mismatch of ischemic/infarcted brain tissue with DWI/PWI or other techniques that show cerebral hypoperfusion without infarct as there are potential adverse effects from induced hypertension, such as bradycardia and myocardial ischemia. The long-term benefit has not been proven yet and the impact on morbidity and mortality is as yet unclear.

TEMPERATURE MANAGEMENT

Hyperpyrexia has been shown to worsen the outcome of stroke patients.[61–63] This effect is independent of the underlying cause of hyperpyrexia;for example, pneumonia, DVT, urinary tract infections, or related increases in serum glucose. Experimentally there is a faster rate of consumption of substrates with increasing temperature and theoretically increased rate of ischemic brain converting to infarcted brain in the area of the ischemic penumbra.[64] Normopyrexia is recommended at least for the first 24 hours to reduce the changes in increased metabolic states. Conversely, it seems reasonable that cooling of the acute stroke patient with an ischemic penumbra may be beneficial. Unfortunately, pharmacological neuroprotectants have not shown benefit.

There are small trials with mixed results showing that cooling improves outcomes and other trials which do not.[65–67] In general, no recommendation can be made about whether outcome improves with cooling. It may be said of cooling that, like all other neuroprotectant interventions, if cerebral blood flow to the ischemic area is not reinstated, neuroprotection may not be helpful. In this way cooling for ischemic stroke is different than

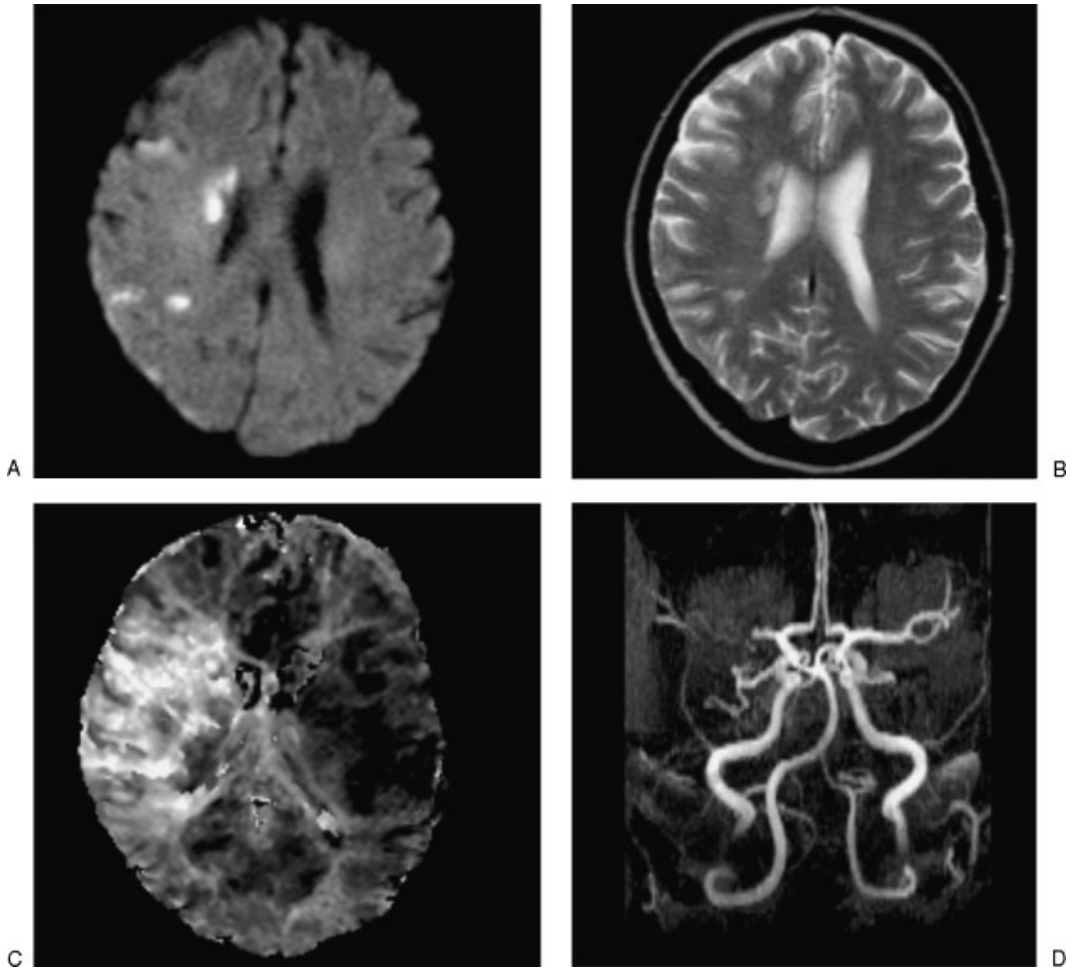


Figure 6 Magnetic resonance imaging (MRI) scans of a patient who recurrently developed left hemiplegia and neglect during dialysis. The blood pressure would drop below his baseline during the procedure and, when his blood pressure was supported, the deficits would resolve. (A) This diffusion-weighted MRI (DWI) shows scattered strokes in the middle cerebral artery (MCA) distribution. (B) This T2-weighted MRI scan reveals no significant findings besides old strokes. (C) This MRI scan shows a larger area of hypoperfusion in the right MCA distribution. (D) An MCA occlusion can be seen.

cooling in cardiac arrest where the patient is cooled around the time that perfusion is reinstated.

GLUCOSE CONTROL

There are several studies suggesting that elevated serum glucose in acute stroke patients on admission is associated with higher rates of deep venous thrombolysis, pneumonia, and poor outcome.[60,68–70] There has been debate as to whether this is simply a marker of poorly controlled diabetes, which is a risk for the above medical problems, or whether the high glucose itself has a detrimental impact on the ischemic penumbra. Experimentally there seems to be an effect of hyperglycemia on ischemic neurons,[71,72] but it has not been shown clinically that there are larger strokes or that the ischemic penumbra is directly affected. Outcomes do appear to be affected with tight glucose control within the first 24 hours after admission. It is probably beneficial to maintain tight glucose control in stroke patients, but how tight and through what technique is unclear at this time.

CONCLUSION

The acute stroke model of care should mirror the care that acute myocardial infarction patients receive. Care in the field by emergency medical services and in the Emergency Department should focus on diagnosis and clot lysis by the designated stroke team. The focus of the ICU team should be preserving the ischemic penumbra and identifying stroke worsening, recurrent hemorrhage, or increased ICP. The management of the acute stroke patient with a large infarct or after fibrinolytic therapy can be quite complex. It requires a basic understanding of the neurological examination and the more sophisticated the knowledge the better. It also includes measures to limit obscuration of the examination as well as avoidance of interventions that can worsen the stroke

itself. Finally, maintenance of the ischemic penumbra to ensure there is as little expansion of the stroke as possible is of the utmost importance. Ideally, a multidisciplinary approach with intensivists, neurologists, neurosurgeons, physiatrists, nursing, and rehabilitation therapists is the best way to appropriately care for the stroke patient.

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