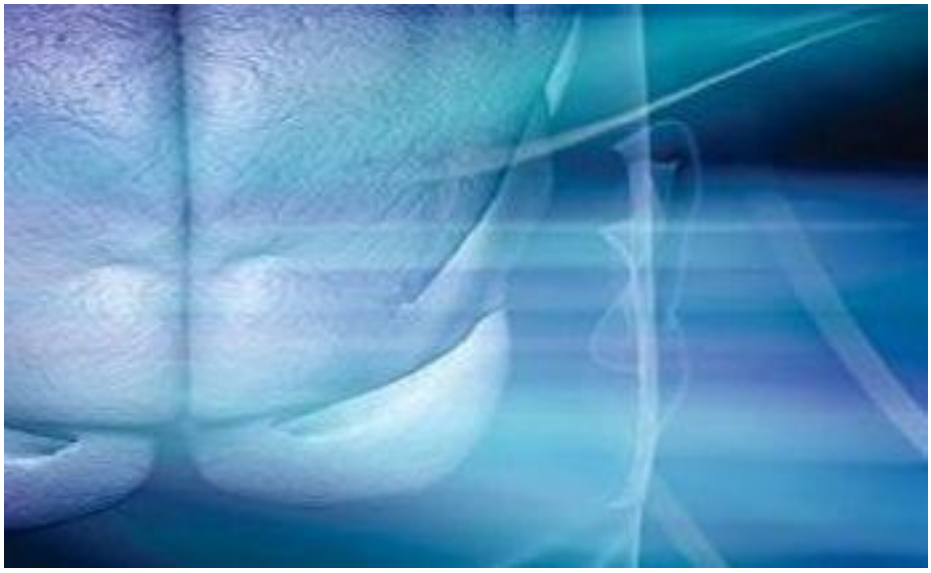

OFFICIAL JOURNAL OF THE AMERICAN ORGANIZATION OF
NEUROLOGICAL SURGEONS AND ACOS NEUROSURGICAL SECTION

VOLUME 13, 2013

# INSTRUCTIONS FOR AUTHORS

Papers submitted should be original documentation, including photographs. The papers should be single column, double-spaced. The title should be in title case and bold, followed by Authors, degree, organization and city, state.

The papers should contain an abstract and be separated into sections with bold typing of the section title. The page set-up should be 0-6.5 inches. Paragraphs should be indented 0.5 inches. All tables should be submitted separate from the paper. If possible make the tables up to 3 inches wide so that they could fit into a column. This will allow quicker scanning and preparation.

References should be numbered, tab, name of authors. Title of paper. Journal. Year volume:pages.

All papers, correspondence and DUES can be submitted to the:

American Organization of Neurological Surgeons

The JAONS is produced and published jointly by the AONS & ACOS-NSS.

# EDITOR'S PAGE

Physicians in training, learn and practice research "To formulate, ingrain, and measure, a method of thought, investigation, and evaluation necessary for physicians to have multi-lateral information exchange and communication with experts in areas of scientific and medical discovery, knowledge, and analysis, in order to continuously and efficiently improve human health and patient care." Understanding and performing quality research provides students and residents the tools to propel quality medical care into the community and into the future.

Welcome to the Journal of the American Organization of Neurological Surgeons and the American College of Osteopathic Surgeons Neurosurgical Section. This volume is composed of the Residents' annual papers that were submitted but not published elsewhere. It is therefore dedicated to the future Neurosurgeons and their education. All papers were reviewed by the peer review committee and selected for awards. The papers submitted are excellent, representing some of our talented colleagues. Issues will be published annually. I hope that this issue will spread the knowledge of our residents and our section. We will continue to solicit annual papers and all papers submitted at the annual meeting. This is your Journal paid for by your annual dues. This issue is available on our website AOANeurosurgery.org. This is your organization; please support it as you can.

Thank you,

Dan Miulli, D.O, F.A.C.O.S
Editor

2014 Annual Resident Paper Winners

1st Place Joseph F. Georges, DO, PhD Columbus- Aquaporin 1 Predicts Survival of Malignant Glioma Patients and Increases Glioma Cell Invasiveness. Award $1500

2nd Place Derek Kroll, D.O., M.S. Bromenn –Rate of Procedural and Periprocedural Morbidity and Mortality in Patients Treated with Intracranial Angioplasty and Stenting for Intracranial Atherosclerotic Disease. Award $1000

3rd Marietta Walsh, DO, Bromenn - Profound hypoglycemia presents with reversible symmetrical ADC signal changes isolated to eloquent cortex of the parietal lobe. Award $500

All Residents will be expected to be at the ACA in Boston September 18-21 for the 1st Day of Presentation to give a 10 minute talk about their research.

**Profound hypoglycemia presents with reversible symmetrical ADC signal changes isolated to eloquent cortex of the parietal lobe.**

**Marietta Walsh, DO**

**Key words:** Hypoglycemia; Diffusion signal; Apparent diffusion coefficient

## ABSTRACT

MRI diffusion/ADC signal change with reversal, symmetrically isolated to the cortex of the precentral gyri in profound hypoglycemia has not been previously described. We present a case in which minimal cortical signal change without deep grey matter involvement and subsequent reversal occurred without significant clinical improvement. Correlation of the reversal of diffusion/ADC signal to findings by EEG evaluation has been described in animal studies and not in humans before.

## INTRODUCTION

This to our knowledge is the first case report describing MRI diffusion signal change with apparent diffusion coefficient (ADC) reversal, symmetrically isolated to the cortex of the precentral gyri in profound hypoglycemia. Extensive cortical diffusion signal change with or without deep gray matter involvement is identified in hypoglycemic coma. Reversibility of ADC signal change in hypoglycemia has been previously described and has been correlated with varied clinical outcomes. We describe a case in which minimal bihemispheric parietal lobe eloquent cortical diffusion signal change without deep grey matter involvement and with reversal of ADC signal occurred in the context of profound hypoglycemic coma without significant clinical improvement.

## CASE STUDY

A 71 year old diabetic female was brought to the emergency room after she was found unresponsive at her nursing home. The patient was on monotherapy for her diabetes (glyburide). An accucheck ® assessment showed a glucose of 32 mg/dl. The patient was given glucagon and D50 with subsequent serum glucose level improvement from 45 mg/dl to 92 mg/dl. This resulted in no change in her level of consciousness.

The neurological exam revealed an obtunded patient with no posturing, no response to tactile stimulation and no spontaneous movement. Her Glasgow coma scale (GCS) was 3 (1= no eye opening, 1= no verbal response, 1= no motor response). She was intubated to secure and maintain an airway. She was normotensive with stable vitals. Her pupils were equal and reactive to light. She had no nystagmus or facial twitching. A day prior to presentation, she was alert, able to feed herself, and was conversing appropriately with no focal neurologic deficit. Her past medical history was significant for multiple medical comorbidities. This included schizophrenia/bipolar disease, non insulin dependent diabetes mellitus and chronic obstructive

airway disease. Laboratory evaluation and a head CT scan without contrast was otherwise unremarkable.

Within one hour of arrival at the emergency department, the patient underwent an MRI of the brain without contrast. This demonstrated bilateral symmetric increased signal within the cortex of the precentral gyri on the diffusion weighted imaging (DWI) sequences. Corresponding signal dropout was identified on ADC mapped sequences. No associated change in signal on FLAIR/T2 or T1 images was identified (Fig. 1 A, B, C and D). The initial MRI study was otherwise unremarkable. A follow-up MRI of the brain was performed approximately 43 hours later. The comparison study showed a diminution in high intensity diffusion signal with a resolution on ADC mapped sequences. No delayed FLAIR /T2 or T1 signal abnormality was noted (Fig. 2, A, B, C and D)

An electroencephalogram (EEG) performed approximately 15 hours after admission showed diffuse slow wave activity of moderate to severe degree consistent with bilateral cerebral hemispheric dysfunction. This was characterized by slow wave activity of 1.5 to 2 Hertz in the frontal central areas. Posterior and central rhythms of 7 Hertz were identified. No epileptiform activity was seen. Five hours prior to the patient's follow-up MRI of the brain, a repeat EEG revealed improvement in the diffuse slow wave activity with higher amplitudes relative to the prior study. In addition some temporal sharp wave activity was noted bilaterally. These findings were consistent with improving encephalopathy associated with hypoglycemia.

Despite rapid correction of the glucose level, she initially remained comatose, intubated and unresponsive. Her clinical status over a total of 19 days post-admission, showed slow and gradual improvements. The patient was weaned of the ventilator after 6 days. After extubation, she began to grimace to painful stimuli and had occasional minimal spontaneous movements of the lower extremities. She had no movement of the upper extremities, even with painful stimuli. At discharge, she was awake and more alert. She was not able to follow commands and made some vocalizations without meaningful conversation. She spontaneously grimaced and opened her eyes. Upon discharge her GCS was 7 (4 = eye opening spontaneously, 2 = incomprehensible sounds, 1 = no motor response). A decision was made by the family to admit the patient to hospice.

**DISCUSSION**

The clinical presentation of hypoglycemia is non specific and requires early and accurate diagnosis and management for improved outcomes. The varied signs that include anxiety, memory loss, headaches, focal deficits, generalized weakness, seizure and coma are common to other acute neurologic events such as infarction. Status epilepticus and generalized tonic clonic seizures from other etiologies present with a pattern of cortical diffusion signal change and ADC

reversal similar to the case presented and are important diagnoses to exclude in the emergent management setting (1, 2).

DWI and ADC signal changes in severe hypoglycemia have been previously reported in patients with varied and selective involvement of grey and white matter areas (3, 4, 5). Correlation of the location of signal change on DWI/ADC sequences in hypoglycemic patients with prognosis, as well as correlating the reversibility of such lesions with patient outcomes has been suggested by prior case reports and a study by Kang et al (1, 3, 4, 6). Extensive bi-hemispheric cortical lesions with or without basal ganglia involvement is observed in severely affected patients with hypoglycemia (3, 4, 5). In a study of MR imaging features of hypoglycemia, single lobe involvement resulted in complete recovery. This study also identified complete recovery with isolated white matter involvement (1). DWI/ADC signal change with post therapeutic reversal isolated only to the precentral gyri of the parietal lobes in a comatose hypoglycemic patient has not been previously reported. Despite isolated limited cortical involvement of the eloquent brain with ADC signal reversal, the clinical prognosis of such a pattern in our patient was similar to more extensive cortical and or deep grey matter involvement.

The changes in diffusion signal with hypoglycemia occurs initially in the cortex with progressive involvement of the deep grey matter structures and hippocampi (3, 6, 7), where selective vulnerability to disruption of protein synthesis has been identified (5). The time over which diffusion signal change occurs and the level of hypoglycemia that induces these changes in humans is not known. The resultant signal change and neurologic deficit also appears to be specific to the individual. In animal models permanent neurologic deficit was identified in only 73% of cases after a period of 6 hours (8). A recent publication demonstrated no diffusion signal change in non comatose patients subjected to short term hypoglycemia (9). Reported studies have shown reversal of ADC signal change with and without clinical improvement. Clinical recovery with signal reversal was noted in one case report at 10 days and was noted in another case at 22 days post ictus with a poor outcome (3, 4, 5, 6). Our patient's hypoglycemia was corrected within 4 hours of its discovery. The reversibility of the reduction in ADC was demonstrated within 48 hours of the initial MRI.

The mechanism of diffusion signal change in hypoglycemia occurs as a result of cellular membrane sodium potassium pump failure and cellular swelling with extracellular volume depletion, as it does in infarction (3). In infarction however, intracellular acidosis occurs due to hypoxia. Consumption of intracellular metabolic acids with resultant alkalosis is present in hypoglycemia (5). The reversal of ADC signal is an important consideration in excluding infarction. White matter structures affected by hypoglycemia are the internal capsules, corona radiata and the splenium of the corpus callosum. Associated signal change is thought to occur by a different mechanism, with extracelluar excitatory peptide secretion (glutamate) resulting in glial cell and myelinic sheath edema (1, 3, 5, 7). Involvement of the corona radiata and centrum

semiovale occurred in 64% of patients in a study evaluating the imaging findings of hypoglycemia (1). In neonates poor outcomes are associated with involvement of the occipital and parietal lobes (10). Symmetric early phase involvement of the occipital lobes with gradual recovery in adults has been noted (11). Early phase parietal pattern of involvement in adults may also portend a poor outcome as was identified in our case. Occipitoparietal involvement is thought to occur when hypoperfusion complicates hypoglycemia, as these regions are more vulnerable to diminished blood flow (12, 13). Our patient was not hypotensive on presentation to the emergency room.

The duration of EEG isoelectricity correlates with the severity of hypoglycemia. In an animal study looking at the MRI findings after temporary severe hypoglycemia, ADC signal change within the cortex and periventricular regions was identified before EEG isoelectricity. Global ADC signal change was noted with the onset of cerebral isolectricity (7). This corroborates the greater earlier vulnerability to the cortical regions as in our patient, prior to overall brain neuronal dysfunction. Improvement in EEG activity and ADC signal occurs with glucose infusion. In our study, the patient had diffuse slowing of a moderate to severe degree on EEG at 15 hours after admission. Prior to the patient's follow-up MRI and after the patient's glucose had normalized, the EEG showed improvement in slow wave activity with bilateral temporal sharp activity. This appears to correlate with aspects of the animal study. In our study reversal in ADC signal was seen within 5 hours after the EEG showed improvement.

## CONCLUSION

DWI hyperintense lesions in hypoglycemic patients not involving the deep grey matter structures, and involving solely the cortex have a better prognosis. Increased diffusion signal with reversal of ADC signal in a hypoglycemic comatose patient with limited eloquent cortical parietal lobe involvement and without significant neurologic improvement has not been previously described. The reversal of ADC signal within 48 hours of the insult also corresponded to commensurate improvement on EEG evaluation as has been seen in animal studies and not in humans before.

## REFERENCES

1. Kang EG, Jeon SJ, Choi SS, et al. Diffusion MR Imaging of Hypoglycemic Encephalopathy. AJNR Am. J. Neuroradiol 2010;31(3):559 - 64.

2. Kim JA, Chung JI, Yoon PH, et al. Transient MR Signal Changes in Patients with Generalized Tonicoclonic Seizure or Status Epilepticus: Periictal Diffusion-weighted Imaging. American Journal of Neuroradiology 2001;22(6):1149-60.

3. Lo L, Tan AC, Umapathi T, et al. Diffusion-weighted MR imaging in early diagnosis and prognosis of hypoglycemia. AJNR Am J Neuroradiol 2006;27:1222–4.

4. Lee BW, Jin ES, Hwang HS, et al. A case of hypoglycemic brain injuries with cortical laminar necrosis. J Korean Med Sci 2010;25(6):961-5.

5. Albayram S, Ozer H, Gokdemir S, et al. Reversible reduction of apparent diffusion coefficient values in bilateral internal capsules in transient hypoglycemia-induced hemiparesis. AJNR Am J Neuroradiol 2006;27:1760–2.

6. Aoki T, Sato T, Hasegawa K, et al. Reversible hyperintensity lesion on diffusion-weighted MRI in hypoglycemic coma. Neurology 2004;63:392–3.

7. Hasegawa Y, Formato JE, Latour LL, et al. Severe transient hypoglycemia causes reversible change in the apparent diffusion coefficient of water. Stroke 1996;27:1648–56.

8. Myers RE, Khan KJ. Insulin-induced hypoglycemia in the nonhuman primate. II: Long-term neuropathological consequences. In: Bradley JB, Meldrum BS, editors. Brain hypoxia, 1971:195–206. Reprinted in Clin Devel Med.

9. Schmidta P, Böttchera J, Ragoschke-Schumma A, et al. Diffusion-Weighted Imaging of Hyperacute Cerebral HypoglycemiaAmerican Journal of Neuroradiology 2011;32:1321-7.

10. Cakmakci H, Usal C, Karabay N, Kovanlikaya A. Transient neonatal hypoglycemia: cranial US and MRI findings. Eur Radiol 2001;11:2585–8.

11. Maekawa S, Aibiki M, Kikuchi K, et al. Time-related changes in reversible MRI findings after prolonged hypoglycemia. Clin Neurol Neurosurg 2006;108:511–13

12. Tallroth G, Ryding E, Agardh CD. Regional cerebral blood flow in normal man during insulin-induced hypoglycemia and in the recovery period following glucose infusion. Metabolism 1992;41:717–21

13. Sontineni SP, Lee JM, Porter J. Hypoglycemia-induced pontine infarction in a diabetic male with basilar artery stenosis: insight into the mechanisms of hypoglycemic stroke. Cerebrovasc Dis 2008;25:281–2

**FIGURES**

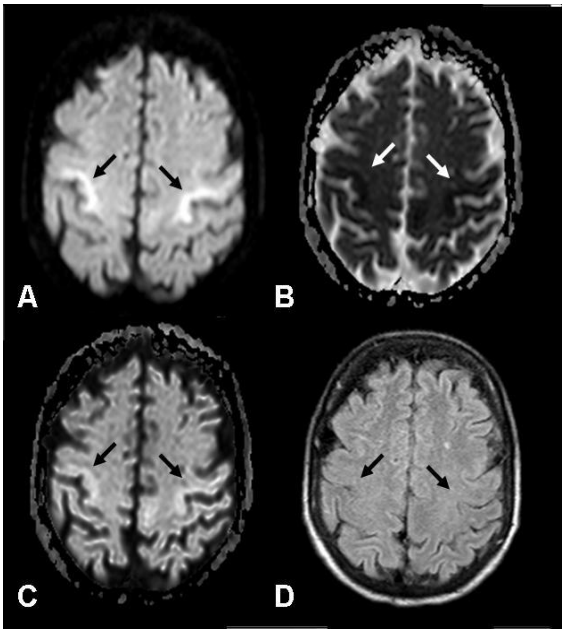


**Figure 1**

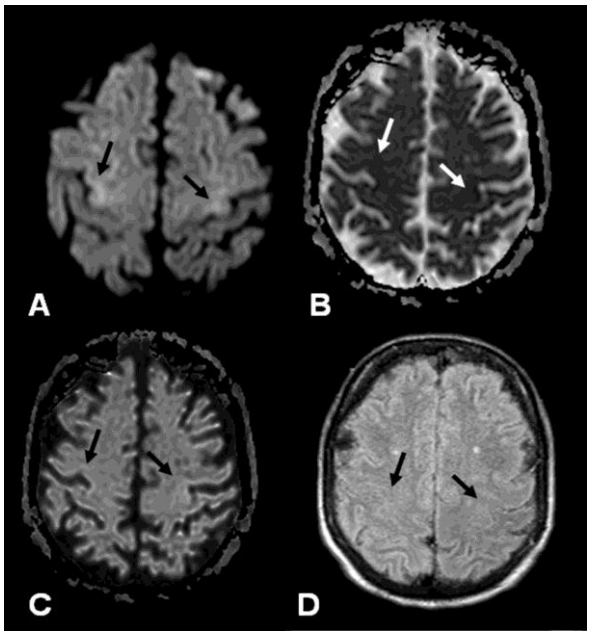


**Figure 2**

**Figure 1:** Select axial MR image sequences acquired 4 hours after onset of ictus demonstrating symmetric increased diffusion signal isolated to the precental gyri (A) with corresponding change in ADC (B) and eADC signal (C). No associated FLAIR signal change (D).

**Figure 2:** Select axial MR image sequences acquired at 43 hours post ictus demonstrating reversal of diffusion (A) ADC (B) and eADC (C) signal in the precentral gyri bilaterally. No delayed FLAIR signal change (D).

# An Innovative, Multidisciplinary, Process-Driven Approach to Acute Stroke in a Community Health System Network

Richard D Fessler, M.D.1, Chiu Yuen To, D.O.2, Vickie Gordon, NP-C, Ph.D.3, Carrie Stover, MSN, NP-C 4, Robert Dunne, M.D. 5

1 Chairman, Department of Surgery, Providence Hospital and Medical Center, St. John Hospital and Medical Center 2 Department of Surgery, Division of Neurosurgery 3 Nurse Practitioner, Neuroscience St. John Providence Health System 4 Vice President, Van Elslander Neuroscience Center of Excellence St. John Providence Health System 5 Vice Chief of Emergency Medicine St. John Hospital and Medical Center

Corresponding Author:
Richard D. Fessler, M.D. Chairman, Department of Surgery, 22151 Moross Road, Ste. 212
Detroit, Michigan 48236, Email: Richard.fessler@stjohn.org

## Abstract

Stroke is one of the major causes of death and disability in the United States, yet it is under treated by many major medical centers across the country. Timely recognition and treatment of acute ischemic stroke remains a challenge due to confusing clinical presentations, hospital logistics, communication barriers among providers, and lack of standardized treatment algorithms. By creating a system-wide code stroke protocol, St. John Providence Health System (SJPHS) improved documentation, increased intravenous tissue plasminogen activator (tPA) delivery, reduced specialist call back times, improved door-to-computer tomography (CT) scan and door-to-needle time, and identified appropriate patients for endovascular therapy.

*Key Words: Code Stroke, Algorithm, Stroke Triage, Stroke Alert*

## Introduction

Stroke is a leading cause of mortality among developed countries, accounting for 15 million deaths worldwide each year.(1) In the United States, it is the fourth leading cause of death and the number one cause of adult disability. An estimated 6.8 million Americans over the age of 20 have experienced a stroke, comprising 2.8% of the population.(2) Approximately 795,000 people have a new or recurrent stroke yearly in the United States, out of which 134,000 (17%) are likely to die.(1–3) Stroke accounted for approximately 134,000 deaths in 2008.(4) On average, someone suffers a stroke every 40 seconds and someone dies of a stroke every 3.1 minutes (1,2) and among all patients with a transient ischemic attack (TIA), 12% will die within one year due to stroke or related causes.(6) Meta-analyses of cohorts of patients have shown the short-term risk of stroke after TIA to be between 3-10% at 2 days and 9-17% at 90 days. (2,7,8) Immediate care of stroke patients accounted for $28.3 billion in direct medical expenses (9) and $53.9 billion in indirect medical expenses in 2008. (3) More than half of the total cost of caring for a stroke patient accrues 3 years after the initial stroke occurs. (3)

In 2003, the Joint Commission established criteria for the designation of Primary Stroke Centers. These sites meet specific requirements for the delivery of treatment to this vulnerable population.

(10) St John Providence Health System (SJPHS) has established three Primary Stroke Centers with 5 campuses in Southeast Michigan that are accredited by the Joint Commission. Members of the stroke team at each hospital campus met on a weekly basis to review process issues and developed a system wide code stroke protocol. All stroke team members maintain NIHSS certification and participate in yearly mock "Code Stroke" drills to assess and refresh their skill sets. The three Primary Stroke Centers offer a comprehensive, multi-specialty team approach that has improved stroke treatment. Quantified patient outcomes clearly demonstrate these improvements and indicate that utilizing a system-wide code stroke protocol can result in highly effective and efficient care.

The Code Stroke Process

At SJPHS, the stroke response process is known as "code stroke." When a patient presents with stroke symptoms to an SJPHS emergency department (ED), a streamlined fully accountable process is initiated (Figure 1). This protocol is set in motion if the patient presents within 8 hours of stroke symptom onset or if the time of onset is unknown, The initial assessment is completed within five minutes when emergency medical services (EMS) or triage in the ED identifies a patient as having stroke-like symptoms. Those who meet the criteria for an acute stroke are placed in a high acuity ED bed. Within 10 minutes, the ED physician evaluates the patient, confirms stroke symptoms, generates a National Institute of Health Stroke Scale (NIHSS) score, and readies the patient for a CT scan. The Stroke Alert protocol is activated by the 15 minute mark.

Once the Stroke Alert is activated, an automated alert is sent to the on-call stroke neurologist, neuroendovascular surgeon, CT tech, radiologist, ED operational manager, and bed coordinator. During minutes 16-45, CT brain and CT perfusion (CTP) studies are performed and a final radiologist read is called in to the ED physician. The Code Stroke process includes CTP studies on all acute stroke patients in order to identify those who are potential candidates for neurointervention. CTP also assists with clinical decision making for patients with unclear signs and symptoms despite a relatively low NIHSS. When performed at the same time as an initial CT, a CTP study adds minimal scanning time and does not have a significant impact on tPA delivery time. Once the studies are completed, the ED physician, neurologist, and neuroendovascular surgeon confer and reach a consensus on a treatment recommendation which may include intravenous tissue plasminogen activator (IV-tPA/alteplase), neuroendovascular treatment versus conservative treatment, or potential randomization into a clinical trial if indicated.

The key component to SJPHS's Code Stroke process is the Stroke Alert packet which is placed on all charts in ED triage when a possible stroke patient has been identified. The Stroke Alert packet contains the Recommended Time Line (Figure 1.), (which is also replicated within the electronic health record), an informational handout for patients and families, and a Reference Sheet for Health Care Providers (Figure 2.) providing talking points relative to the risk and

benefit of IV-tPA delivery. The packet also includes an IV-tPA inclusion/exclusion criteria checklist (Figure 3), a worksheet for the NIH Stroke Scale, an IV-tPA dosing chart, an algorithm for management of intracranial hemorrhage following thrombolytic therapy (Figure 4), and an order set for intracranial hemorrhage following initiation of thrombolytic therapy (Figure 5).

Quality Assurance

Depending on volume, multi-disciplinary quality assurance meetings were held on a weekly, biweekly, or monthly basis at each SJPHS hospital to evaluate all code stroke activations in terms of timeliness of assessment and treatment, and to conduct a review of any complications. These evaluations allow for the identification of trends and provide continuous opportunities for improvement. Physician representatives from the four service areas (neurology, emergency medicine, radiology, and endovascular neurosurgery) meet to address care and logistical issues internally. Multiple ancillary specialties are represented, including X-Ray technicians, CT technicians, stroke team members, quality assurance personnel, etc. ED physicians, rapid response team members, neurologists, neuroendovascular surgeons, and radiologists are provided with benchmarks for response times, treatment rates, and outcomes.

The Code Stroke program at SJPHS was initiated in mid 2009. Figure 6a shows intravenous tissue plasminogen activator (IV-tPA) administration rates from 2005 to 2011. Figure 6b shows all patients discharged with a diagnosis of stroke over the same time period. The graph shows an improvement in IV-tPA administration at one of the hospital sites from less than 2% to greater than 10% during the three years following initiation of the program.

Quality Metrics for Improving Code Stroke

During the multi-disciplinary quality assurance meetings, physician response times, NIHSS documentation, tpa treatment or reason for non-treatment, endovascular candidates, CT times, and radiology read times were reviewed. The SJPHS team made several improvements to improve these metrics, including:

*Monthly call-back schedules and Perfectserve electronic paging system*: by creating a monthly stroke call-back schedule for neurology and endovascular neurosurgery, both services are called simultaneously and provide back up for one another. For any call to a specialist not answered within fourteen minutes, a Perfectserve notification is automatically generated to the chief of the neurology telemedicine network program or the endovascular neurosurgery program director. Response times are then shared amongst specialists on a monthly basis.

*National Institutes for Health Stroke Scale (NIHSS) documentation*: prior to initiation of the Code Stroke Program, nearly one third of Emergency Department evaluations lacked NIHSS documentation which, along with response time, were designated as primary quality metrics. All patient charts are now reviewed each week at a multidisciplinary stroke quality assurance

meeting. Individuals failing to appropriately document stroke evaluations are contacted and encouraged to improve their clinical documentation. The rate of NIHSS documentation improved from 70% to 100% after initiation of benchmarking and education, and the average neurologist call back time to the Emergency Department decreased from more than 22 min to less than 2 minutes without further intervention.

Results of the Code Stroke Process:

ED Triage and Time to CT

As the Code Stroke program was expanded to other facilities within SJPHS, the data consistently indicated that in those institutions where the Code Stroke protocol is followed, the times to treatment and overall IV-tPA delivery rates are several times faster than the national average. (11–13) Those facilities that do not participate wholeheartedly in the process tend to deliver at higher rates than the national average, but are far lower than SJPHS's internal best standard. Figure 7 provides a 6-month snapshot of data from late 2010, collected from 4 hospital campuses within SJPHS.

Sites 1, 2, and 3 have neuroendovascular coverage 24/7 and tele-radiology is available for immediate viewing of imaging studies at all facilities. Each facility had access to an InTouch RP-Lite robot. (Figure 8.) Two sites routinely activated code stroke alerts upon patient triage in the EDs; alerting CT, neurology, and the neuroendovascular team. This activation occurred uniformly for well over 90% of all patients at Sites 1 and 2.

Two campuses had differential protocol adherence: Site 3 failed to routinely activate the code stroke until after the ED physician evaluation and image acquisition. Site 4 altered the code stroke activation to an evaluation by resident staff and mid-level providers rather than initiating immediate alerts to neurology and neuroendovascular specialists. Sites 1 and 2 delivered IV-tPA at an 11% rate to all patients triaged as stroke victims during 2011. Patients presenting at Sites 3 and 4 received treatment with IV-tPA at a rate of 4%. These rates are percentages of the total number of IV-tPA doses given relative to the total number of stroke discharges, not the percentage of eligible patients. In addition, Sites 3 and 4 acquired a greater number of imaging studies per stroke patient than Sites 1 and 2. Data evaluation has resulted in an ongoing program of process improvement at each hospital in the health system.

Time to CT is a primary determinant of door-to-needle time, and is highly process dependent. A Code Stroke alert notifies the CT team and the room is readied for an emergent study. An intravenous line is started in the ED and a non-enhanced CT Head is done, followed by CT Perfusion and/or CT Angiogram of Head and Neck. A radiologist interprets these studies, and the final read is expected to occur within the 45-minutes from arrival to allow for treatment within an hour (or 15-minutes for non-enhanced CT Head as well as CT Perfusion). A history of contrast reaction is not considered an absolute contraindication to intravenous contrast and these

patients are not delayed in their evaluation.(14) The interpretation times and accuracy of interpretation between different radiologists and sites are compared and reviewed on a weekly or biweekly basis.

The initial interpretation time averaged 26 minutes from CT completion. Several changes were made to the process to shorten this time frame. Upon downloading images into the pictorial archiving system (PACS), studies are now designated as STAT to alert the radiologist at his/her reading station. Radiologists also receive a notification page with Code Stroke alerts. On time reads were difficult to obtain initially; however, through continued education, benchmarking, and peer review, average interpretation times dropped from 26 minutes to 8 minutes after completion of the study.

Intravenous tPA Administration

Once the patient returns from CT, the decision as to whether the patient is an intravenous tPA (IV-tPA) candidate has already been made based on colleague-to-colleague discussions, care algorithms, and potential CT results. To decrease the delivery and mixing time for IV-tPA, the medication is now stored within the ED for immediate availability. Patients are then transferred to the neuroscience intensive care unit for observation and ongoing evaluation.

Determination of Appropriate Candidates for Intervention

Identifying patients as potential interventional candidates is accomplished with physiologic imaging as combined with guidelines published by the American Heart Association and Society of Neurointerventional Surgery. (15-17) For patients with major vessel occlusion, CT perfusion is relied upon to identify areas of reversible ischemia. (18-20) Every patient who is part of the Code Stroke process receives a CT perfusion scan that is reviewed by a neuroendovascular specialist at the time of completion. This not only identifies potential candidates for interventional therapy, but also identifies those patients who have completed infarcts not demonstrated by a plain CT. By vigilantly adhering to the code stroke protocol, complications related to recanalization of ischemic dead tissue have been minimized.

Discussion

Despite the high prevalence of acute ischemic stroke, it remains a major challenge for many hospital systems to accurately diagnose these patients in a timely manner in order to provide life-saving treatments that are time dependent. Many hospital systems across the world are working on ways to address this concern by creating triage systems, emergency medical system (EMS) algorithms, new scoring systems, new protocols in the Emergency Department, or by developing a hub-spoke relationship with tertiary stroke centers and incorporating telemedicine in this process. (21,22,24–27)

Rapp et al were among the first to introduce the concept of "code stroke" in 1997 when they described the concept of developing a pathway in order to facilitate identification and subsequently provide treatments of these patients with rt-PA in the National Institute of Neurological Disorders and Stroke (NINDS) study. (28) They detailed an EMS stroke alert activation process preferentially triaging these patients and obtaining non-contrast CT scan as a priority, followed by beginning intravenous thrombolytics. Insufficient details, however, were provided in terms of how to successfully achieve these benchmarks in a timely manner with the exception of creating a pathway and placing these patients on a high acuity level. The main purpose of the paper was to educate the medical community in recognizing stroke as a medical emergency and that reperfusion/thrombolytic therapy should be the main goal of treatment in the initial phase of stroke.

The Europeans have extensive experience in rapidly identifying and treating patients with acute ischemic strokes, particularly in Spain. In 2004, Alvarez-Sabin et al. described the clinical benefits following the implementation of a specialized urgent stroke care system by going through a three step process: 1) developing a stroke team and code stroke protocol; 2) creating a stroke unit; and 3) incorporating an on-call stroke neurologist. Noted improvements include a decreased length of stay, hospital mortality, and institutionalization of these patients. (29) De Leciñana et al. described the relevance of a code stroke protocol, stroke unit and stroke network in the organization of acute stroke care which emphasized the need for coordinated care lead by the stroke neurologist, available consultants in a multitude of specialties, and the creation of a dedicated stroke unit. While these elements helped to improve implementation of the treatment protocols, details of the protocol itself were not provided. (30) Clua-Espuny et al. described the implementation of stroke code model in Terres de l'Ebre, Spain. Among 380 patients that were treated, a 13.9% thrombolytic usage rate was achieved (31), however the article was published in non-English literature and the differences between the health care systems of the United States and Spain have to be taken into account. Nevertheless, achieving a 13.9% intravenous thrombolytic rate is a noteworthy accomplishment.

Prabhakaran et al. recently published a study stating that IV-tPA infusion rate increases in primary stroke centers over time are significantly higher than nonstroke certified centers. (32) By having a specific stroke protocol, more patients may be qualified for treatment with IV-tPA. Finally, Theiss et al. evaluated the effect of the Telestroke network over a four-year period and found that the rate of thrombolytic use increased and in-hospital mortality decreased. (33)

Gonzalez et al. from Harvard University recently published their acute stroke imaging algorithm that incorporated non-enhanced CT Head, CT angiogram and diffusion MRI (Magnetic Resonance Imaging) in the identification of penumbra and quantifying the ischemic core. (34) They defer to CT Perfusion only if a patient cannot receive diffusion MRI. Although their

algorithm is very reasonable and has achieved positive results, it is often difficult to reproduce due to the limited availability of MRI scanners.

In order to develop a code stroke process, a few key components must be addressed: 1) recognizing potential stroke patients from presentation (prehospital, in the emergency department, or on the floor); 2) obtaining fast and appropriate diagnostic studies; 3) interpreting complex clinical presentations together with the diagnostic results; and 4) providing timely treatment. Though each of these components appears simple, many obstacles may lie ahead, hence developing a code stroke protocol at an institutional level remains the most feasible way to tackle this problem. While developing the protocol at SJPHS, logistical issues were encountered such as physician call back time, imaging availability, bed availability, time for radiology interpretation, delay from pharmacy for mixing and delivering of IV-tPA, and availability of neuroendovascular services. Full support from hospital administration, physicians, clinical staffs, and ancillary personnel are absolutely vital to the success of such a process. Through a step-wise approach, each and every one of these obstacles was identified, and attempts were made to address them in an effective manner. This resulted in the creation of a code stroke model that has ultimately translated into significant improvements in objective benchmark measures that are successfully reproducible on a system-wide basis. The model is also being adapted by many other health systems across the nation. (23,35) A particularly valuable component of the SJPHS approach is the built-in mechanism that continues to evaluate the protocol by means of weekly reviews of all stroke patients. These reviews identify the deficits, missed benchmarks, and reasons for not providing certain interventions that may be otherwise overlooked. The resulting recommendations are then translated directly into everyday clinical practice across the system. No model is perfect of course, which is why the SJPHS protocol remains dynamic in nature so that it can adapt to new advances and seamlessly incorporate new evidence-based practices, guidelines and techniques in treating this challenging disease.

Summary

The outcome following an acute ischemic stroke depends on a timely diagnosis and reperfusion of the ischemic brain. By creating a code stroke protocol, the process of identifying and subsequently providing appropriate treatments for patients with ischemic stroke in a timely and effective manner has been streamlined. A code stroke protocol delivers specialty care to the patients' points of access to the health system and eliminates traditional obstacles with objective and measurable outcome improvements. The process was successfully implemented at one hospital site, then subsequently scaled to the health system, and, finally, to a network level. The SJPHS protocol may be used as a model for other centers to enhance their stroke programs and achieve similar positive results.

References

1. Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, De Simone G, et al. Heart disease and stroke statistics--2010 update: a report from the American Heart Association. Circulation. 2010 Feb 23;121(7):e46–e215.
2. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Borden WB, et al. Heart disease and stroke statistics--2013 update: a report from the American Heart Association. Circulation. 2013 Jan 1;127(1):e6–e245.
3. CDC - Chronic Disease - Heart Disease and Stroke Prevention - At A Glance [Internet]. [cited 2012 Dec 25]. Available from: http://www.cdc.gov/chronicdisease/resources/publications/AAG/dhdsp.htm
4. Towfighi A, Saver JL. Stroke declines from third to fourth leading cause of death in the United States: historical perspective and challenges ahead. Stroke. 2011 Aug;42(8):2351–5.
5. Foraker RE, Rose KM, McGinn AP, Suchindran CM, Goff DC Jr, Whitsel EA, et al. Neighborhood income, health insurance, and prehospital delay for myocardial infarction: the atherosclerosis risk in communities study. Arch. Intern. Med. 2008 Sep 22;168(17):1874–9.
6. Weintraub WS, Spertus JA, Kolm P, Maron DJ, Zhang Z, Jurkovitz C, et al. Effect of PCI on quality of life in patients with stable coronary disease. N. Engl. J. Med. 2008 Aug 14;359(7):677–87.
7. Parkin L, Sweetland S, Balkwill A, Green J, Reeves G, Beral V. Body mass index, surgery, and risk of venous thromboembolism in middle-aged women: a cohort study. Circulation. 2012 Apr 17;125(15):1897–904.
8. Heit JA, Melton LJ 3rd, Lohse CM, Petterson TM, Silverstein MD, Mohr DN, et al. Incidence of venous thromboembolism in hospitalized patients vs community residents. Mayo Clin. Proc. 2001 Nov;76(11):1102–10.
9. Heidenreich PA, Trogdon JG, Khavjou OA, Butler J, Dracup K, Ezekowitz MD, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. Circulation. 2011 Mar 1;123(8):933–44.
10. Advanced Certification Comprehensive Stroke Centers | Joint Commission [Internet]. [cited 2013 Apr 3]. Available from: http://www.jointcommission.org/certification/advanced_certification_comprehensive_stroke_centers.aspx
11. Reeves MJ, Arora S, Broderick JP, Frankel M, Heinrich JP, Hickenbottom S, et al. Acute stroke care in the US: results from 4 pilot prototypes of the Paul Coverdell National Acute Stroke Registry. Stroke. 2005 Jun;36(6):1232–40.
12. Kleindorfer D, Lindsell CJ, Brass L, Koroshetz W, Broderick JP. National US estimates of recombinant tissue plasminogen activator use: ICD-9 codes substantially underestimate. Stroke. 2008 Mar;39(3):924–8.
13. Cronin CA. Intravenous tissue plasminogen activator for stroke: a review of the ECASS III results in relation to prior clinical trials. J Emerg Med. 2010 Jan;38(1):99–105.

14. Brockow K, Christiansen C, Kanny G, Clément O, Barbaud A, Bircher A, et al. Management of hypersensitivity reactions to iodinated contrast media. Allergy. 2005 Feb;60(2):150–8.
15. Latchaw RE, Alberts MJ, Lev MH, Connors JJ, Harbaugh RE, Higashida RT, et al. Recommendations for imaging of acute ischemic stroke: a scientific statement from the American Heart Association. Stroke. 2009 Nov;40(11):3646–78.
16. Meyers PM, Schumacher HC, Higashida RT, Barnwell SL, Creager MA, Gupta R, et al. Indications for the performance of intracranial endovascular neurointerventional procedures. A scientific statement from the American Heart Association Council on Cardiovascular Radiology and Intervention, Stroke Council, Council on Cardiovascular Surgery and Anesthesia, Interdisciplinary Council on Peripheral Vascular Disease, and Interdisciplinary Council on Quality of Care and Outcomes Research. J Neurointerv Surg. 2010 Jun;2(2):177–88.
17. Blackham KA, Meyers PM, Abruzzo TA, Albuquerque FC, Alberquerque FC, Fiorella D, et al. Endovascular therapy of acute ischemic stroke: report of the Standards of Practice Committee of the Society of NeuroInterventional Surgery. J Neurointerv Surg. 2012 Mar;4(2):87–93.
18. Silvennoinen HM, Hamberg LM, Lindsberg PJ, Valanne L, Hunter GJ. CT perfusion identifies increased salvage of tissue in patients receiving intravenous recombinant tissue plasminogen activator within 3 hours of stroke onset. AJNR Am J Neuroradiol. 2008 Jun;29(6):1118–23.
19. Turk AS, Magarick JA, Frei D, Fargen KM, Chaudry I, Holmstedt CA, et al. CT perfusion-guided patient selection for endovascular recanalization in acute ischemic stroke: a multicenter study. J Neurointerv Surg. 2012 Nov 26;
20. Kleinman JT, Mlynash M, Zaharchuk G, Ogdie AA, Straka M, Lansberg MG, et al. Yield of CT perfusion for the evaluation of transient ischaemic attack. Int J Stroke. 2012 Dec 11;
21. Schwamm LH, Holloway RG, Amarenco P, Audebert HJ, Bakas T, Chumbler NR, et al. A review of the evidence for the use of telemedicine within stroke systems of care: a scientific statement from the American Heart Association/American Stroke Association. Stroke. 2009 Jul;40(7):2616–34.
22. Schwamm LH, Audebert HJ, Amarenco P, Chumbler NR, Frankel MR, George MG, et al. Recommendations for the implementation of telemedicine within stroke systems of care: a policy statement from the American Heart Association. Stroke. 2009 Jul;40(7):2635–60.
23. Brown EV. Robotic assistance remedy. The Michigan Stroke Network utilizes remote presence robots to bring needed specialists to stroke patients at remote hospitals. Health Manag Technol. 2008 Jul;29(7):18–21.
24. Sattin JA, Olson SE, Liu L, Raman R, Lyden PD. An expedited code stroke protocol is feasible and safe. Stroke. 2006 Dec;37(12):2935–9.
25. Tai YJ, Weir L, Hand P, Davis S, Yan B. Does a “code stroke” rapid access protocol decrease door-to-needle time for thrombolysis? Intern Med J. 2012 Dec;42(12):1316–24.

26. Asimos AW, Norton HJ, Price MF, Cheek WM. Therapeutic yield and outcomes of a community teaching hospital code stroke protocol. Acad Emerg Med. 2004 Apr;11(4):361–70.
27. Dalloz MA, Bottin L, Muresan IP, Favrole P, Foulon S, Levy P, et al. Thrombolysis rate and impact of a stroke code: a French hospital experience and a systematic review. J. Neurol. Sci. 2012 Mar 15;314(1-2):120–5.
28. Rapp K, Bratina P, Barch C, Braimah J, Daley S, Donnarumma R, et al. Code Stroke: rapid transport, triage and treatment using rt-PA therapy. The NINDS rt-PA Stroke Study Group. J Neurosci Nurs. 1997 Dec;29(6):361–6.
29. Alvarez-Sabín J, Molina C, Montaner J, Arenillas J, Pujadas F, Huertas R, et al. [Clinical benefit following the implementation of a specialized urgent stroke care system]. Med Clin (Barc). 2004 Apr 17;122(14):528–31.
30. De Leciñana-Cases MA, Gil-Núñez A, Díez-Tejedor E. Relevance of stroke code, stroke unit and stroke networks in organization of acute stroke care--the Madrid acute stroke care program. Cerebrovasc. Dis. 2009;27 Suppl 1:140–7.
31. Clua-Espuny JL, Piñol-Moreso JL, Gil-Guillén FV, Orozco-Beltran D, Panisello-Tafalla A, Lucas-Noll J. [The stroke care system in Terres de l'Ebre, Spain, after the implementation of the Stroke Code model: Ebrictus Study]. Med Clin (Barc). 2012 May 19;138(14):609–11.
32. Prabhakaran S, McNulty M, O'Neill K, Ouyang B. Intravenous thrombolysis for stroke increases over time at primary stroke centers. Stroke. 2012 Mar;43(3):875–7.
33. Theiss S, Günzel F, Storm A, Hausn P, Isenmann S, Klisch J, et al. Using Routine Data for Quality Assessment in NeuroNet Telestroke Care. J Stroke Cerebrovasc Dis. 2012 Feb 22;
34. González RG, Copen WA, Schaefer PW, Lev MH, Pomerantz SR, Rapalino O, et al. The Massachusetts General Hospital acute stroke imaging algorithm: an experience and evidence based approach. J NeuroIntervent Surg [Internet]. 2013 Mar 14 [cited 2013 Jun 4]; Available from: http://jnis.bmj.com/content/early/2013/03/13/neurintsurg-2013-010715
35. Fessler R. Michigan Stroke Network | About Us | Meet Clinical Team | Richard Fessler, MD [Internet]. [cited 2012 Dec 26]. Available from: http://www.michiganstrokenetwork.com/about_us/clinical_team/v/Richard_Fessler_MD/about.aspx

## Figures

Figure 1. Recommended Time Line for Code Stroke Alert

## EMERGENT CARE OF THE ACUTE STROKE PATIENT
## RECOMMENDED TIME LINE

**Time Line**

**0 min** — **Patient arrives to ED with Stroke Symptoms.**

**5 min** — **RN Assessment Completed**

**10 min** — **Physician Assessment Completed**

| ED PHYSICIAN / RAPID RESPONSE TEAM | ED NURSE / STAFF NURSE |
|---|---|
| • Determine last time known normal<br>• Obtain a brief history and physical examination | • O2 2L/Nasal Cannula<br>• IV access 2 (large gauge catheters) |
| • Neurological Examination with NIHSS<br>• Identify any **ABSOLUTE** exclusions for IV tPA | • Telemetry<br>• Pulse oximetry<br>• Vital signs and Neurological examination |
| • STAT CT | • Prepare for CT Scan<br>• Glucose (POC) |
| • STAT labs | • Blood drawn and sent STAT |

**15 min** — **Activate Stroke Alert:**

**25 min** — **Brain Imaging Completed**

**45 min** — **Brain Imaging Results Reported/Lab Results Reported**

**60 min** — **Initiate Treatment Option**

Figure 2. Reference Sheet for Healthcare Providers

**REFERENCE SHEET FOR HEALTH CARE PROVIDERS**

| **Benefit of tPA** | **Benefit of NO tPA** |
|---|---|
| **Less than 3 hours TPA (alteplase)**<br>17% - Normal at 24 hours<br>31% - Normal at 3 months<br>20% - Less chance of moderate to severe disability or death | **Less than 3 hours No tPA (alteplase)**<br>3% - Normal at 24 hours<br>20% - Normal at 3 months |
| **3 to 4.5 hours* TPA (alteplase)**<br>52% - Normal at 3 months (mRS 0-1)<br><br>28% - chances or returning to an independent lifestyle compared to no tPA | **3 to 4.5 hours No tPA (alteplase)**<br>45% - Normal at 3 months (mRS 0-1) |
| **Risk of tPA** | **Risk of NO tPA** |
| **Less than 3 hours TPA (alteplase)**<br>17% - Mortality<br>40% - Severe Disability or Death<br>17% - Deterioration within 36 hours<br>6.4% - Symptomatic ICH | **Less than 3 hours NO tPA (alteplase)**<br>21% - Mortality<br>48% - Severe Disability or Death<br>18% - Deterioration within 36 hours<br>0.6% - Symptomatic ICH |
| **3 to 4.5 hours* TPA (alteplase)**<br>7.7%- Mortality<br>7.9% - Symptomatic ICH | **3 to 4.5 hours NO tPA (alteplase)**<br>8.4% - Mortality<br>3.5% - Symptomatic ICH |
| ***This does not extend the treatment window for acute stroke. Early treatment remains essential. Treatment with alteplase (tPA) within the first 1.5 hours after onset of stroke doubles the efficacy compared to 1.5-3 hours after onset. For 1 patient to have a favorable outcome (mRS 0-1) after 3 hours, 14 patients will require treatment.** | |
| **Frequency of ICH by Baseline NIHSS Score with tPA treatment:** | |
| NIHSS 0- 5 2% - ICH<br>NIHSS 6-10 3% - ICH<br>NIHSS 11-15 5% - ICH<br>NIHSS 16-20 4% - ICH<br>NIHSS >20 17% - ICH | Note: Patients with NIHSS greater than 20 return to normal more frequently with tPA; the rate of death or severe disability is still high (65-70%) either way. |
| **Intracerebral Hemorrhage** | |
| Symptoms ICH | — NIHSS decrease in LOC (1a) greater than or equal to 1<br>— Increase in NIHSS greater than or equal to 4<br>— Acute hypertension<br>— Nausea<br>— Vomiting<br>— New headache |
| If you suspect ICH | — Discontinue tPA (alteplase)<br>— Obtain stat CT<br>— Draw blood for PT, PTT, Fibrinogen, Type and Screen<br>— Prepare Cryoprecipitate 5-8 Units<br>— Prepare Platelets 6-8 Units<br>— Fresh Frozen Plasma 5-8 Units |
| ICH verified on CT | — Obtain lab results<br>— Stat Neurosurgery Consult<br>— Consider Cryoprecipitate and Platelets<br>— Consider Hematology consult |

Figure 3. IV-tPA inclusion/exclusion criteria checklist

| | |
|---|---|
| | — Consider second CT to assess ICH progress |

**CONSIDERATION FOR INTRAVENOUS THROMBOLYTICS**
**IV t-PA INCLUSION/EXCLUSION CHECKLIST**

| YES | NO | INCLUSION CRITERIA |
|---|---|---|
| | | Onset of symptoms less than or equal to 4.5 hrs (270 min.) prior to treatment |
| | | Age greater than or equal to 18 years |
| | | Clinical diagnosis of stroke with measurable deficit |

| YES | NO | ABSOLUTE EXCLUSION CRITERIA |
|---|---|---|
| | | Evidence of Intracerebral hemorrhage on pretreatment CT head |
| | | CT findings of infarct greater than 1/3 territory |
| | | Clinical presentation suggestive of subarachnoid hemorrhage |
| | | Active internal bleeding |
| | | Known bleeding diathesis including:<br>— Platelet count less than 100,000<br>— On heparin (w/in 48 hrs) and elevated partial thromboplastin time (PTT)<br>— International normalized ratio (INR) greater than or equal to 1.7<br>— Direct thrombin inhibitor (e.g. dabigatran) **OR** direct factor Xa inhibitor (e.g. rivaroxaban) within 72 hours |
| | | Intracranial surgery, serious head trauma or previous stroke within 3 months |
| | | Any history of intracerebral hemorrhage, arteriovenous malformation, cerebral aneurysm |
| | | Blood pressure: systolic greater than 185 or diastolic greater than 110 on repeated measurement at time of treatment |
| | | Glucose less than or equal to 50 per BGM at time of treatment |
| | | Patient and family declined and/or refused treatment |

| YES | NO | ADDITIONAL EXCLUSION CRITERIA 3-4.5 Hours following symptom onset |
|---|---|---|
| | | Age greater than 80 |
| | | Major neurological deficits NIHSS >25 (stroke severity) |
| | | History of stroke **AND** diabetes |
| | | Receiving anticoagulant therapy regardless of INR |

| YES | NO | CAUTIONS/WARNINGS |
|---|---|---|
| | | Low molecular weight heparin (LMW) administered in the past 24 hours at therapeutic doses (Excludes prophylactic doses of 40mg or less) |
| | | Minor symptoms or rapid improvements |
| | | Major neurological deficits (multi-lobar) CT greater than 1/3 cerebral hemisphere |
| | | Major surgery or serious trauma (excluding head trauma) within 14 days |
| | | History of frank GI or GU hemorrhage within 21 days |
| | | Arterial puncture at non-compressible site 7-days |
| | | Acute MI in previous 3 months (not concurrent) |
| | | Seizure at onset of stroke symptoms suggestive of postictal neurological impairment |
| | | Lumbar puncture within 7 days |
| | | Pregnancy |
| | | |

Figure 4 Algorithm for management of intracranial hemorrhage following thrombolytic therapy

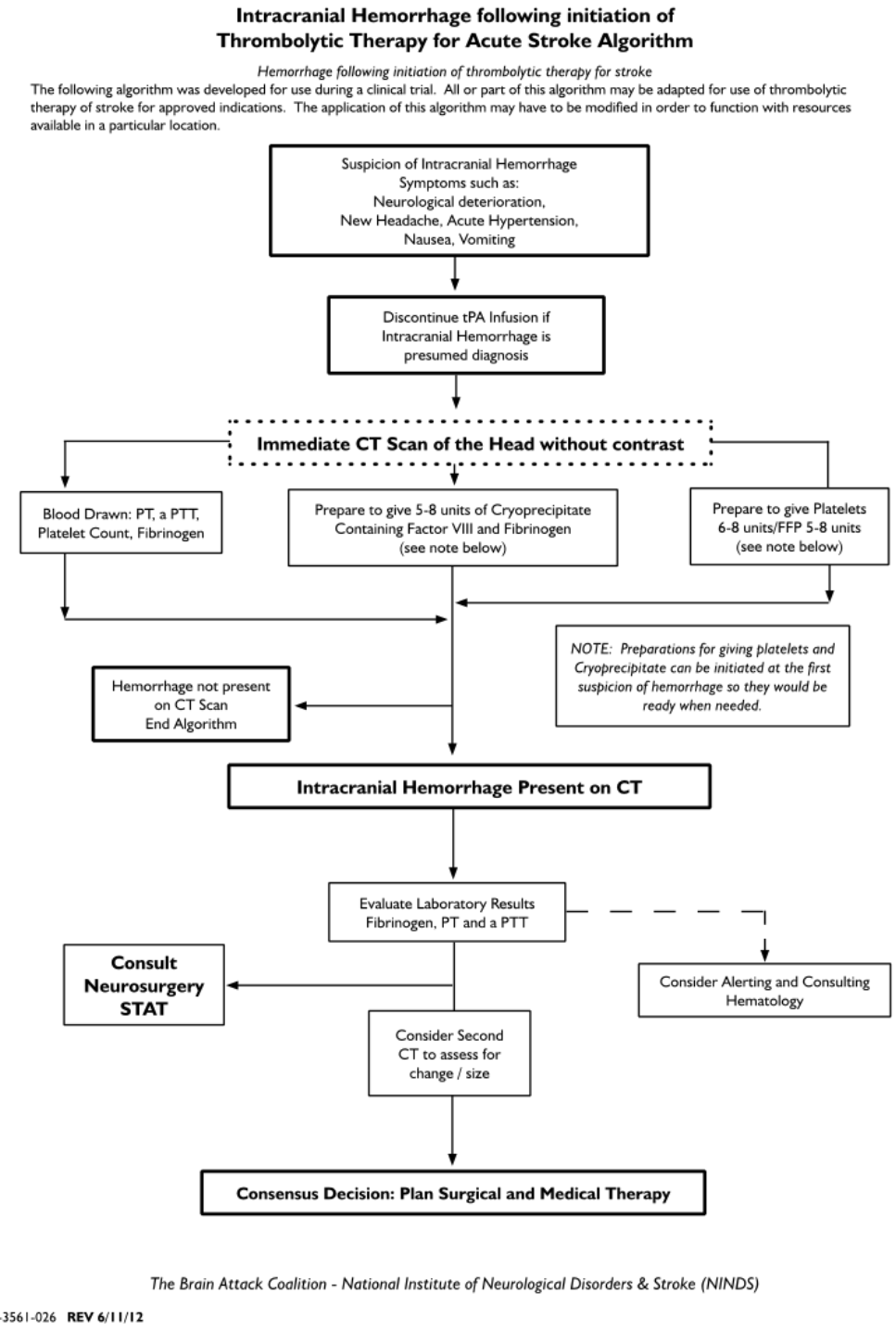


Figure 5. Order set for intracranial hemorrhage following initiation of thrombolytic therapy

**Intracranial Hemorrhage following Initiation of Thrombolytic Therapy for Acute Stroke Orders**

| Allergies/Reactions | Height: Inches Cm | Weight: Actual Estimated | Kg Lb |
|---|---|---|---|
| | | | |

**For use with patients who experience neurological deterioration, sudden severe headache, acute uncontrolled hypertension, nausea and vomiting and intraparenchymal hemorrhage is considered likely:**

☒ STOP tPA Infusion

☒ STAT CT of the Head without contrast RE: Intracranial Bleed

☒ STAT Labs: CBC, a PTT, PT/INR, Fibrinogen, Type and Cross

☒ Neuro checks every 15 minutes

☒ Vital signs every 15 minutes

Cryoprecipitate _____ units (5-8 units recommended)

Fresh Frozen Plasma _____ units (5-8 units recommended)

Platelets _____ units (6-8 units recommended)

☒ STAT Consult Neurosurgery if ICH comfirmed

| Emergency Verbal Order or Telephone Order / Read back by: | | Date: | Time: |
|---|---|---|---|
| Transcriber's Signature: | | Date: | Time: |
| **Prescriber's Printed Name:** | Noting Nurse's Signature: | Date: | Time: |
| **Prescriber's Signature:** | **Complete Call Back Number:** ( ) ___ ___ ___ - ___ ___ ___ ___ | **Date:** | **Time:** |

Form transmitted to pharmacy: Date/Time: _____ By: _____

80-561-027 **REV 6/11/12** Original - Chart Copy - Transmit or send to Pharmacy

**Intracranial Hemorrhage following Initiation of Thrombolytic Therapy for Acute Stroke Orders**

Figure 6a. Number of patients received IV-tPA at one of the participating hospitals from 2005 to 2011. 6b. Number of patients with acute ischemic stroke as a discharge diagnosis from 2005 to 2011.

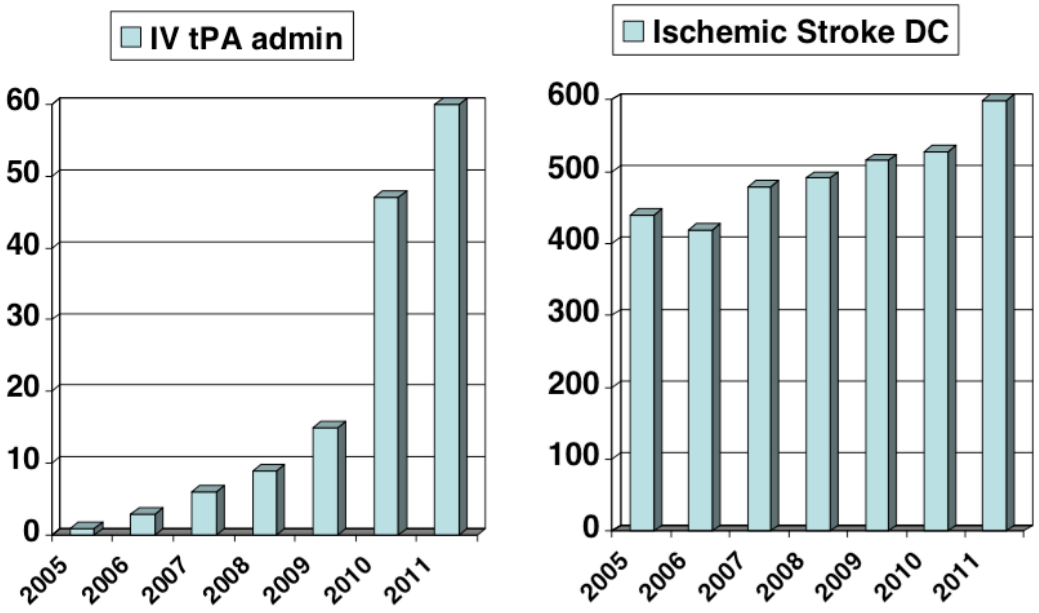


Figure 7. Percentage of Code Stroke Activations of patients with acute ischemic stroke at four primary stroke center designated emergency departments system wide as well as IV-tPA administration rate in each ED in 2010.

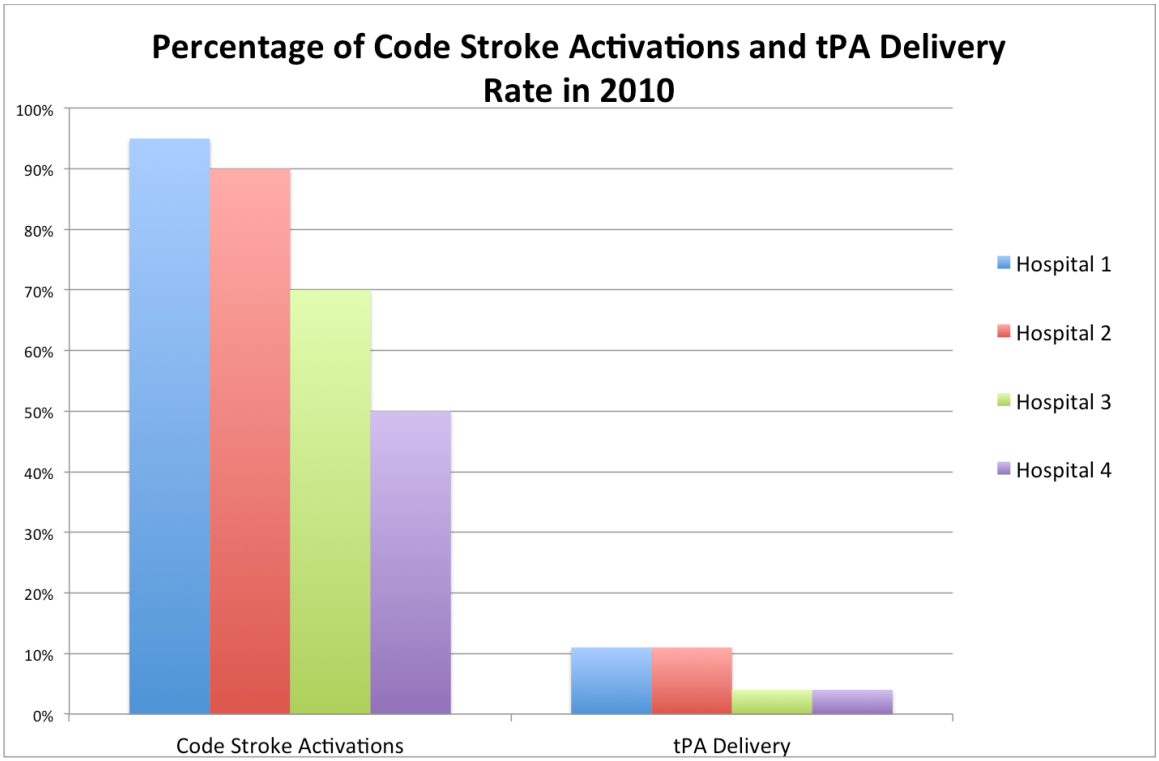

Figure 8. Intouch RP-Lite Robot allows stroke neurologist to remotely assess the patient via the use of telemedicine technology in the form of high-quality videoconferencing (HQ-VTC).