The Stroke Center Handbook Second Edition Organizing Care for Better Outcomes



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To the stroke teams at Saint Luke's Neuroscience Institute and The University of Cincinnati

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Preface

Stroke is the second leading cause of death worldwide and the leading cause of adult disability. Since the first edition of this book in 2007, new treatments have become available for acute intervention, and there is new evidence regarding the best approaches to the prevention of complications, secondary prevention, and neurorehabilitation. It is important to design processes and infrastructure that will ensure every stroke victim has the best possible outcome.

The purpose of this book is to provide insight and information to physicians, nurses, therapists, and administrators who are planning and evolving the programs necessary to do this work, regardless of the size of the hospital or available workforce. The recommendations for the management of stroke that are included are based on current evidence and future trends, but they will change as new data become available. The hope, however, is that the infrastructure and processes described in this book are foundations that will stand the test of time and ensure standardization of care in stroke centers while providing the flexibility to incorporate new therapies as evidence evolves. In addition to an update of all the content in the previous edition, there are new chapters on transient ischemic attacks and neurocritical care.

Providing good outcomes to stroke victims is time dependent, labor intensive, and complicated. Nevertheless, it is the responsibility of every healthcare organization to have a plan in place to meet the challenge when the patient arrives at the door.

chapter one

Setting the goal for the stroke center

Introduction

The goal of the stroke center is to provide the best possible outcome for every patient. An organized approach to care can be achieved in every hospital, and the first issue to address in planning is to determine what level of care can be realistically provided.

The continuum of care for stroke can be divided into four distinct phases, each requiring different resources, personnel, and infrastructure.

Phase I: Acute stroke treatment

It is very likely that acute stroke treatment will always be time dependent, with the best outcomes resulting from the earliest intervention. Options for acute stroke treatment fall into two categories. The following examples have not all been proven effective but are meant to be illustrative.

Noninvasive treatment requires no surgical or interventional access.

- Intravenous (IV) thrombolytics
- Neuroprotective agents
- Ultrasound-enhanced thrombolysis

Invasive treatment requires interventional or surgical access.

- Mechanical embolectomy
- Intra-arterial (IA) thrombolysis
- Aneurysm coiling or clipping
- Surgical clot removal
- Injection of thrombolytic into intracerebral or intraventricular hemorrhage
- Hemicraniectomy
- Ventriculostomy

Important questions to be addressed if acute stroke intervention is one of the goals:



Figure 1.1 Algorithm for acute stroke treatment.

- 1. Is time-dependent acute stroke intervention going to be offered 24/7?
- 2. Is noninvasive acute treatment going to be provided? (Checklist 1)
- 3. Is invasive acute treatment going to be provided? (Checklist 2)

If the answer to question 1 is no and acute stroke treatment is not going to be available or only available during the day, then it is critical to have a plan for rapid transfer or bypass for patients who present within the treatment time window (Figure 1.1 and Chapter 3).

Checklist 1: Resources required for noninvasive acute treatment

- Emergency department (ED) personnel trained to triage and diagnose acute stroke
- Physician trained in acute stroke treatment available 24/7
- Neuroimaging available to be done in 25 minutes and interpreted in 45 minutes
- Stat lab
- Pharmacy to rapidly mix drug or, preferably, drug available in ED
- Intensive care unit (ICU) or step-down unit for patient admission
- Stroke care path/flowchart
- Order set for stroke and for acute treatment selected
- Nurses trained in neuro assessment and stroke management

Checklist 2: Additional resources required for invasive stroke treatment

- Neurointerventional and neurosurgical teams ready in 30 minutes
- Neurointerventionalist and neurosurgeon available 24/7
- Properly equipped neurosurgical and neurointerventional suite(s)

Phase II: Medical/surgical management

There is conclusive evidence that stroke victims, even those ineligible for acute intervention, who are managed in an organized stroke center have better outcomes and decreased mortality.¹ Care paths, standing order sets, telemetry, and highly trained nurses make up the basic infrastructure that guarantees standardized care in five areas.

- Tight control of physiologic parameters (Chapter 8)
- Prevention of complications (Chapter 9)
- Diagnosis of the cause of the stroke (Chapter 10)
- Plan for secondary prevention (Chapter 10)
- Early rehabilitation (Chapter 11).

Should structural disease such as significant carotid stenosis be diagnosed, then surgical or interventional treatment should be considered (Chapter 10).

Checklist 3: Resources to achieve best practice medical/surgical management of stroke

- Physicians knowledgeable in the diagnosis and management of stroke
- Stroke center nurses trained in neurological assessment and competent to use specialized monitoring equipment
- Staffing level high enough to guarantee frequent neurological assessments and blood pressure (BP) monitoring by nurses
- Neuroimaging resources, including emergent computed tomographic (CT) scan at minimum and possibly computerized tomographic angiography (CTA), computerized tomographic perfusion (CTP) sequences, magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), magnetic resonance diffusion-weighted imaging and perfusion-weighted imaging (MRI DWI and PWI, respectively), digital subtraction biplane cerebral angiography
- · Carotid artery imaging: ultrasound, CTA, MRA
- Echocardiography: two-dimensional (2D) echo, transesophageal echo with protocol for diagnosis of atrial septal defect or patent foramen ovale (PFO)

- Care paths for ischemic and hemorrhagic stroke and transient ischemic attack (TIA) to address acute and subacute management, prevention of complications, secondary prevention and early rehabilitation (Appendix)
- Standing order sets for ischemic and hemorrhagic stroke, stroke interventions, and TIA (Appendix)
- Designated neuro ICU and step-down stroke unit beds

Phase III: Rehabilitation

It is clear that early access to rehabilitation specialists improves outcomes and decreases length of stay on the acute hospital unit. The goal would be to have evaluations by physical, occupational, and speech therapy on day 1 and initiation of plans for the next phase of rehabilitation should it be needed.

Checklist 4: Rehabilitation services available

- Physical therapy
- Occupational therapy
- Speech therapy
- Neuropsychology
- Social services
- Inpatient rehabilitation unit.

If the patient is transferred to the inpatient rehabilitation unit, the care path from the stroke center can coordinate with the care path on the rehabilitation unit. In addition to the focus on physical rehabilitation, it is important to assess and treat cognitive impairment and depression. The work of the stroke center does not end at discharge from the rehabilitation unit.

Phase IV: Postdischarge monitoring

Discharge with a diagnosis of stroke is associated with a fivefold increased risk of mortality and more than two times increased risk of readmission for ischemic stroke, heart failure, cardiac events, any vascular event, pneumonia, and hip fractures in the first year as compared with a matched nonstroke cohort. The increased risk of mortality and rehospitalization persisted beyond the first 30 days postdischarge and beyond the first year.² This may, in part, be due to poor compliance with secondary prevention measures instituted during the hospital stay. In one report of 3,467 patients followed poststroke discharge, compliance in achieving goals was 56% for blood pressure, 36% for low-density lipoproteins, 41%

for international normalized ratio (INR), 40% for glycosylated hemoglobin target, and 39% for depression management.³

Communication of treatment goals and coordination of care with primary care providers from the inpatient to the outpatient setting is a significant challenge as much of this work is not reimbursed but is essential to better outcomes. Organized outpatient follow-up care in a stroke prevention clinic has been reported to decrease 1-year mortality rates by 25%.⁴ This kind of clinic could do double duty as the place for evaluating TIA cases in a timely manner (Chapter 5). In addition to follow-up assessment for secondary prevention, the widely accepted outcome measure for stroke is the modified Rankin Scale (mRS) score at 90 days. This assessment can be done by phone but requires personnel at the stroke center to see the patient or make the phone call at 90 days. Patient and family satisfaction with the care provided is also important in evaluating the effectiveness of the stroke program.

Checklist 5: Postdischarge monitoring

- Trained personnel to make 90-day mRS follow-up calls
- Plan for coordination and monitoring of secondary prevention goals
- Consider organizing a stroke prevention clinic

Once the analysis of the level of care that can be realistically provided for the four phases of care is complete, then the pertinent sections of this handbook can be used to guide the stroke center's development. Ideally, hospitals capable of delivering primary care will establish relationships with tertiary care institutions to carry out the more sophisticated and resource-intense interventions, evaluations, and follow-up.

Stroke centers: Levels of care

The Brain Attack Coalition (BAC) has published recommendations for the components of a primary stroke center (PSC) ⁵ and of a comprehensive stroke center (CSC):⁶

- A PSC would provide acute care to most patients with stroke, be able to use some acute therapies such as IV tPA (tissue plasminogen activator) and admit the patient if it had a stroke unit.
- A CSC would provide care to those patients with large or complex strokes or hemorrhagic strokes, patients who required specialized treatments (endovascular, surgery), or those with multisystem involvement.
- A third type of facility, acute stroke–ready hospitals, includes those that have made an institutional commitment to effectively and efficiently evaluate, diagnose, and treat most ED stroke patients but do

not have fully organized inpatient stroke systems of care. These hospitals would have relationships with regional PSCs and CSCs for additional support and transfer agreements.⁷

There are over 800 certified PSCs in the United States. By submitting data on agreed-on measures, these centers are able to benchmark performance with other PSCs.

In the United States, DNV (Det Norsk Veritas) Healthcare and the Joint Commission (JC; formerly the Joint Commission on Accreditation of Healthcare Organizations or JCAHO) offer certifications for primary and CSCs. In addition, many states are certifying hospitals for levels of stroke care.

Major components of a primary stroke center as recommended in 2000

Patient care areas:

- Acute stroke teams
- Written care protocols
- Emergency medical services (EMS)
- Emergency department
- Stroke units in hospitals planning to admit stroke patients
- Neurosurgical services

Support services:

- Commitment and support of medical organization; a stroke center director
- Neuroimaging services
- Laboratory services
- Outcome and quality improvement activities
- Continuing medical education

Based on the evolution of stroke care since the original recommendations, in 2011 the BAC published revised and updated recommendations for PSCs in 2011.⁷

Revised and updated recommendations for primary stroke centers (2011)

- Emphasis on the importance of acute stroke teams
- Emphasis on the importance of stroke units with telemetry monitoring

- Capability to perform brain imaging with MRI and diffusionweighted sequences
- Assessment of cerebral vasculature with MRA or CTA
- Cardiac imaging
- Early initiation of rehabilitation
- Certification by an independent body, including a site visit and disease performance measures

Performance measures for a primary stroke center

Details regarding how to collect these measures (i.e., the numerators and denominators) for each of these are available at the Centers for Medicare and Medicaid Services (CMS) website (http://cms.gov).

- Venous thromboembolism (VTE) prophylaxis
- Discharge on antithrombotics
- Patients with atrial fibrillation receiving anticoagulation therapy
- Thrombolytic treatment in eligible patients
- Antithrombotic medication within 48 hours of hospitalization
- Stroke education
- Assessment for rehabilitation

The core measures reported to JC are the same as those submitted to CMS as of 2013. There is likely to be increasing linkage between performance and reimbursement from payers and more emphasis on the entire episode of care, including postdischarge compliance with secondary prevention measures and reduction in readmission rates.

Major components of a comprehensive stroke center (2005)

A CSC should have the following major components: Personnel with expertise in the following areas:

- Vascular neurology
- Vascular neurosurgery
- Advanced practice nursing
- Vascular surgery
- Diagnostic radiology/neuroradiology
- Interventional/endovascular procedures
- Critical care medicine
- Physical medicine and rehabilitation
- Rehabilitation therapy (physical, occupational, speech therapy)

- Stroke nursing
- Swallowing assessment

Diagnostic techniques:

- MRI, including DWI
- MRA/magnetic resonance venography (MRV)
- CTA
- Digital cerebral angiography
- Transcranial Doppler (TCD)
- Carotid duplex ultrasound
- Transesophageal echocardiography (TEE)

Surgical and interventional therapies:

- Carotid endarterectomy
- Clipping and coiling of intracranial aneurysm
- Placement of ventriculostomy
- Hematoma removal/draining
- Placement of intracranial pressure transducer
- Endovascular ablation of aneurysms/arteriovenous malformations (AVMs)
- IA reperfusion therapy
- Endovascular treatment of vasospasm

Infrastructure:

- Stroke unit
- ICU
- Operating room staffed 24/7
- Interventional services coverage 24/7
- Stroke registry

Educational/research programs:

- Community education, including prevention
- Professional education
- Patient education

Leadership is critical to the successful integration of these components into a finely tuned stroke treatment system. The BAC recommends there be a physician and nurse director for the program and strong relationships with the ED and EMS providers.

The basic requirements for certification of CSCs by the JC that began in September 2012 did not substantially change from the original BAC list of recommendations,⁶ but the specifics around each requirement are described in detail at the JC website.

Candidate performance measures for CSCs are being tested in a pilot project. The list of metrics is summarized in a paper published January 2011 in *Stroke*.⁸ The core metrics are likely to evolve over time as centers test the validity of these measures. A sample of possible measures is listed.

Candidate performance measures for comprehensive stroke centers

- National Institutes of Health Stroke Scale (NIHSS) Score on Arrival
- mRS at 90 days
- Severity measurement on arrival for subarachnoid hemorrhage (SAH) (Hunt and Hess Scale) and intracerebral hemorrhage (ICH) (ICH score)
- Median time to treatment with a procoagulant reversal agent for ICH cases
- Median time to INR reversal for hemorrhagic stroke
- Hemorrhagic complication for patients treated with IV tPA without catheter-based reperfusion
- Hemorrhagic complication for patients treated with IA thrombolytic therapy or mechanical endovascular reperfusion procedure with or without IV tPA
- Nimodipine treatment initiated in SAH cases
- Median time to recanalization therapy (time from arrival to femoral puncture) in ischemic stroke cases treated with IA thrombolytic or mechanical recanalization therapy
- Thrombolysis in cerebral infarction (TICI) post-treatment reperfusion grade in ischemic stroke cases treated with IA therapy

Components of an acute stroke-ready hospital

- Written emergency stroke care protocols
- Written transfer agreement with a hospital with neurosurgical expertise
- Director of stroke care to oversee hospital stroke policies and procedures
- Ability to administer intravenous recombinant tissue plasminogen activator (rtPA)
- Ability to perform emergency brain imaging (e.g., CT scan) at all times
- Ability to conduct emergency laboratory testing at all times
- Maintenance of a stroke patient log

Setting annual goals

Once the stroke center planning team has determined the level of service to be provided, then setting yearly goals for the stroke center helps focus energy and provides a basis for measuring success. The following are examples of categories of goals:

- Performance measures
 - Clinical: case volume, severity adjusted (baseline NIHSS score) mortality rate, disposition, length of stay, acute treatment rate, complication rate
 - Financial: product line contribution margin, cost per case
 - Patient and family satisfaction
- Educational programs and stroke screenings: public, professional
- Research
- Infrastructure and personnel
- Marketing

Questions to consider while setting the goals for the stroke center

- 1. What kind of stroke services will be provided?
- 2. What is the feasibility of success and acceptance of the stroke program?
- 3. What are the opportunities and barriers?
- 4. Who will be the core team members? Who do we already have, and who do we need to recruit?
- 5. What will it take to get buy-in from key constituents: neurologists, neurosurgeons, radiologists, administrators, primary care physicians, board members?
- 6. Who should be on the planning team?
- 7. What will the budget be?
- 8. How will we communicate what we are doing?

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Stroke center organization

Introduction

By the very nature of the disease, stroke care requires coordination of multiple clinical services. This chapter addresses the components of a comprehensive stroke program that would include research and education as well as the core clinical work. Primary stroke centers and acute stroke–ready hospitals can adapt this structure to their resources, personnel, and goals.

For the stroke center to be successful, it must have strong leadership and be backed up by organizational will. The strongest stroke centers are led by an administrator committed to the success of the program and a physician and nurse champion. Taking excellent care of patients with strokes requires cooperation from multiple hospital departments and dedicated clinicians. An effective partner in administration is important to the success of the clinicians. It is also important to develop a written vision, mission, and strategic/business plan for the stroke center early in the process of organization and to gain support for that vision and plan from the highest levels of responsibility in the organization, the board of trustees, and senior hospital or health system administration. These written documents will vary in complexity with the size of the program, but even a basic plan serves as a guide for the work of the stroke center. Yearly review of the documents provides an opportunity to evaluate progress and set goals for the future (Box 2.1). A board member or committee of the board that serves as a liaison from the stroke center leaders to the board of the hospital or health system and to the community can be important to the successful launch and maintenance of the stroke program.

People

Core leadership team

Designating a medical director, nurse coordinator, and administrator for the stroke program sets up lines of responsibility for program development and clinical quality. In the most basic programs, these responsibilities may be as simple as developing the policy and mechanism for transfer of acute stroke patients to a center capable of acute intervention and standards of care for those patients not eligible for transfer. The job

BOX 2.1 STROKE PROGRAM

Stroke program vision: The stroke program will provide the best possible outcome for every patient.

Stroke program mission: The mission of the stroke program is to decrease the incidence and morbidity from stroke in the region through clinical excellence, research, and education.

Stroke program plan:

- 1. Stroke team leadership and personnel
- 2. Stroke center location
- 3. Clinical tools
- 4. Budget
- 5. Policies

descriptions that follow are typical for tertiary care community hospital stroke centers but can be easily adapted to less-complex programs or to academic programs with neurology residents or stroke fellows.

Medical director

The medical director is often a neurologist. As more fellowship-trained vascular neurologists become available, it would be ideal to have a person with that kind of experience leading the program, but this is not a requirement. A physician with interest and expertise in the care of stroke patients is all that is needed. In larger programs, this position is generally financially compensated by the hospital based on the time commitment. The role of the medical director is to provide leadership in strategic planning, clinical quality, communication, marketing, research, and fund-raising. The job description may include the following tasks:

- Develop vision, mission, and strategic plan for the stroke program
- Set and review yearly goals (Chapter 1)
- Communicate information regarding the stroke program to the hospital/health system board, administration, medical staff, laboratory, pharmacy, radiology and emergency departments (EDs), marketing, emergency medical service (EMS) providers, lay community supporters, and outlying community medical personnel
- Ensure clinical quality by developing and updating standardized protocols and order sets based on current evidence as well as conducting regular case and performance measure reviews
- Continually review current evidence regarding stroke prevention, diagnosis, treatment, and rehabilitation

- Lead development and maintenance of stroke database and stroke research program
- Review clinical and cost outcome data on a regular basis
- Assist in development of educational programs for professional staff and the community
- Develop philanthropic support for the stroke program
- Publish in peer-reviewed journals and present data at local and national meetings
- Write an annual report for the stroke program

Nurse coordinator

The role of the nurse coordinator is essential regardless of the size or complexity of the stroke program. In some programs, this may be an advanced practice nurse (APN) who can do independent patient assessment and billing, but in most settings, the nurse coordinator is a hospital employee with expertise in neuroscience. Depending on the size of the program, the role of stroke team nurse coordinator may be combined with other related responsibilities. The nurse coordinator works closely with the medical director and shares responsibility for stroke center operations and clinical quality. The job description may include these tasks

- Set and review yearly goals for the stroke program
- Lead development of care paths, protocols, and standing order sets for ischemic and hemorrhagic stroke, transient ischemic attack (TIA), and the like and update yearly based on current evidence
- Provide education for nurses in all locations of the hospital who are taking care of patients with strokes, including instruction aimed at National Institutes of Health Stroke Scale (NIHSS) certification (Appendix)
- Review clinical and service quality using case reviews for stroke center nurses
- Support entry of information into the stroke database
- Support stroke clinical research
- Provide education to EMS providers, emergency room staff in referring hospitals, and members of the community
- If trained to an APN level, provide first response to acute stroke calls

Stroke program administrator

In larger programs, the job of administrator for the stroke center may be a full-time position. However, in many hospitals this assignment may be combined with other related responsibilities. The job description may include these tasks:

- Strategically plan with the stroke team clinicians to anticipate equipment and personnel needs
- Set and review yearly goals for the stroke program
- Represent the stroke center at administrative and budget meetings
- Build hospital processes that support the clinical and research activities of the stroke team
- Provide space and supplies for the stroke team
- Supervise the members of the stroke team who are hospital employees

The three roles described bridge the activities of the stroke teams and the rest of the stroke program operations (Figure 2.1).



Figure 2.1 Stroke teams: composition and communication.

Clinical team

The composition of the clinical team will vary depending on what level of service is to be provided. If all phases of clinical services for stroke are to be offered, then it is ideal to have neurologists, neurosurgeons, neurointerventionalists, neuroradiologists, neurointensivists, ED physicians, acute stroke team nurses who respond to stroke cases in the ED 24/7, rehabilitation physicians, nurses and therapists, stroke center nurses who care for the patients at the bedside in the intensive care unit (ICU) and in the stroke center, and technical support staff in radiology, lab, and pharmacy represented on the team. Some members of this team will need to be available 24/7. Programs may choose to use hospitalists, intensivists, or emergency medicine or primary care physicians with stroke expertise in the role of the stroke neurologist. The ED, radiology, lab, and pharmacy may find it beneficial to designate one person who is the principal communication link to the stroke team. The responsibilities of the clinical team are to take excellent care of the patient, to ensure the best possible outcome, and to communicate with the family, referring physicians, and EMS providers. The members of the team will change depending on where the patient is and what care is being provided.

For each of the following phases of care, a list of team members should be developed:

- Acute intervention in the ED, neuroradiology suite or operating room
- · Postintervention management in the ICU or step-down unit
- Prevention of complications, evaluation of cause of the stroke, and plan for secondary prevention in the acute care stroke center
- Rehabilitation in the rehabilitation department or acute care units
- Postdischarge monitoring

Expanded role for specialty-trained neuro nurses

The expertise of the nurses involved in the care of stroke patients is one of the critical success factors for an outstanding stroke program. Nurses in the ED, ICU, and acute stroke center should have organized stroke education and be certified to use the NIHSS (Appendix). Many will choose to become certified by the American Association of Neuroscience Nurses. Offering group study sessions led by stroke center physicians and nurses in preparation for the examination encourages nurses to attain certification.

Some comprehensive stroke centers have expanded the role of neuro APNs or neurocritical care nurses as 24/7 first responders to stroke cases arriving in the ED. The role is called the *code neuro nurse* at Saint Luke's Neuroscience Institute in Kansas City, Missouri, and duties include the following:

- facilitating transfers from referring hospitals
- meeting the patient and family on arrival
- assisting with ED assessment
- coordinating communication between the ED staff, neurologists, neurointerventionalists, computed tomography (CT) technicians, and the interventional suite staff
- documenting inclusion/exclusion criteria for intravenous tissue plasminogen activator (IV tPA)
- providing frequent updates to the family while the patient is in the interventional suite
- ensuring neurocritical care-level nursing for the patient from the ED until the patient is admitted to the ICU
- responding to in-hospital stroke alerts (Stroke Watch Action Team [SWAT] calls)
- communicating outcomes back to the referring ED and EMS crew

This is an effective strategy to improve quality of care and outcomes as well as patient and family satisfaction.

Stroke program team

Additional team members can enrich and support the activities of the stroke program. Some of these people work primarily to support the stroke program, and some function in a liaison role to connect the stroke team with other resources and departments of the hospital.

Data manager

Tracking clinical and financial data is critically important in operating an efficient and high-quality stroke program. Administrative data may be used for an overview of mortality, disposition, complications, and costs. Using a stroke-specific database gives much more detailed information and is useful for clinical analyses, trending, benchmarking, and research. Most primary stroke centers submit data to Get With the Guidelines, a database developed by the American Heart Association/American Stroke Association. Performance measures required for stroke center certification can be uploaded to the Joint Commission (Joint Commission on Accreditation of Healthcare Organizations). Once the performance metrics for comprehensive stroke centers are determined, these will be added to Get With the Guidelines. The nurse coordinator may be able to manage the data, but most larger programs have a dedicated position for this work. The job description may include the following:

- Choose the most appropriate database or develop one
- Enter data for all stroke admissions

- Determine what outcome measures will be tracked (e.g., mortality, complications, disposition, 90-day function [modified Rankin Scale score])
- Develop process for obtaining 90-day outcome data
- Coordinate with administrative and financial datasets to avoid duplication of effort
- Produce regular reports tracking volume, outcomes, and performance measures
- Provide data to support clinical and research operations.

Research coordinator

The opportunities for stroke centers to participate in clinical research continue to grow. Clinical trials can be successfully run in community hospitals as well as academic centers and are often a source of excitement and pride for the team. The research coordinator is generally a nurse experienced in the care of stroke patients. A good clinical nurse can be trained to understand the regulatory requirements and other aspects of directing a research trial. Formal courses are available for in-depth training, and some coordinators may choose to become formally certified (Box 2.2). After the research program is up and running, the salary for the coordinator can be paid from income from the studies. In some programs, the nurse coordinator for the stroke team may also be the research coordinator. If the clinical trial involves acute stroke management or intervention, then the question of rotating research coordinator coverage during nonbusiness hours has to be addressed. The job description may include

- Obtain training in all aspects of clinical research
- Submit research protocols to the institutional review board (IRB) and comply with all IRB rules
- Develop the research budget
- Submit the contract for legal review
- Coordinate and train all participating departments and personnel
- Develop standing order sets for trials
- Develop checklists of inclusion/exclusion criteria to aid clinicians in enrolling appropriate subjects
- Obtain consent from and enroll subjects
- Submit timely case report forms
- Meet with study monitors
- Ensure research accounts are in order
- Attend investigator meetings

Administrative assistant

Once the program grows, it is essential to budget for a position to support the activities of the stroke team, including scheduling of meetings and

BOX 2.2 CERTIFICATION PROGRAMS FOR RESEARCH STAFF

There are two options to obtain certification for the staff working on stroke center research. The Association of Clinical Research Professionals (ACRP) offers global certification programs as a formal recognition of clinical research professionals for clinical research coordinators (CRCs), clinical research associates (CRAs), and clinical trial investigators (CTIs) (http://www.acrpnet.org/certification/ fda/crc/index.html). The Society of Clinical Research Associates Incorporated (SoCRA) is a nonprofit professional organization dedicated to the continuing education and development of clinical research professionals (http://www.socra.org).

These certification examinations document the clinical research professional's knowledge and standards for professional practice. In addition, they grant industry recognition for the clinical research professional.

The following are benefits of certification:

- Certification is increasingly recognized by today's global clinical research industry.
- Study sites use certification for documentation to sponsors and contract research organizations (CROs) that the site is profession-ally managed.
- The largest investigator online databases include a request for the study coordinator's certificate number.
- Certification assists the public, health-care professionals, and the industry itself by identifying standards for professional practice.

taking minutes. The person in this position might also take responsibility for managing the research accounts.

Liaison team members

- *Neuroscience unit nurse manager.* In most centers, the stroke nurse coordinator does not function as the nurse manager for the neuroscience unit. The stroke coordinator and the nurse manager for the unit need to work together closely to ensure clinical quality and maintenance of the expertise of the nurses staffing the stroke center.
- *Rehabilitation services.* It is productive to identify one person from the rehabilitation department to be a member of the stroke program

team for the purposes of communication back to the therapists and nurses in the rehabilitation department(s).

- *Marketing.* Internal and external marketing are important to the success of the stroke program. Having a dedicated person from the marketing department attached to the stroke program team is a big asset. Telling the stories of patients who have had good outcomes is a powerful marketing and educational technique. Permission to tell the story can be obtained while the patient is still in the hospital.
- *Human resources (HR).* There will be personnel issues that have an impact on the stroke program, and a liaison from HR familiar with the stroke center is key to successful resolution of these issues.
- *Foundation*. A liaison to the hospital foundation, if there is one, can be instrumental in helping raise funds for research and education projects.

In larger programs, many people from multiple departments are involved in the operations of the stroke program, and most of these people are managed by and report through their own departments. However, there are some positions in the program that logically report to the stroke program administrator, as illustrated in the sample organizational chart (Figure 2.2).



Figure **2.2** Sample stroke program organizational chart. CEO, chief executive officer; COO, chief operating officer.
Meetings

Planning

In the planning phase of the stroke center described in Chapter 1, the medical director, nurse coordinator, and administrator will constitute the core leadership team and will generally meet weekly. It is important to have one meeting where everyone potentially involved in the stroke program comes together to dream about what the best possible stroke program looks like. Once the scope of the program is determined, the planning team will need to communicate with representatives from the departments involved in the care of the stroke patients. Achieving a balance between obtaining input from key clinicians and technicians with minimal disruption of their clinical activities is the challenge. One way to approach this is to have the core team concentrate on one department at a time. For example, when the planning involves ED operations, the core team can meet with the representatives from the ED to discuss the pertinent issues. This same process can occur with key physicians and personnel in neurology, neurosurgery, neurointervention, neurocritical care, pharmacy, lab, radiology, rehabilitation services, marketing, and so on.

When there are overlapping issues (e.g., stocking tPA in the ED), then representatives from the pharmacy and ED can both meet with the core team. This process will protect the time of the people in the various departments. Only the core team will participate in all of the meetings. It is important to circle back and make sure that the results of the planning are communicated to the entire department (e.g., ED) for comments and suggestions. The core leadership team functions as the communication hub, obtaining input from all the departments and clinicians and communicating back. This same communication plan can facilitate discussion of quality and service issues once the program is up and running.

Clinical operations

Meetings to discuss clinical operations are generally already established in most hospitals. The neuroscience department/institute meetings and the morbidity and mortality conferences, for example, can serve as a forum for discussion of stroke center clinical issues, difficult or interesting cases, and review of data regarding complications, outcomes, and volumes. These meetings will be attended by members of the clinical team and core leadership team (Figure 2.1) as well as the data manager. Highvolume acute stroke intervention centers find it useful to review all the interventional cases on a weekly basis. A peer review process is a requirement for comprehensive stroke center certification.

Stroke program operations

After the program is up and running, it is useful to have a monthly meeting of the key people involved in the stroke program. The core leadership team plus the data manager, research coordinator(s), and representatives from the nursing staff of the stroke center, marketing, the hospital foundation, and rehabilitation department might constitute the stroke program operations team. Discussions regarding the clinical, research, and educational activities and reports from the data manager, marketing, and foundation representative could be regular agenda items. This team could be responsible for setting yearly goals for the stroke program and evaluating whether these are achieved. Stroke program clinicians could look to this operations team to solve problems they identify. The SWAT (see Appendix to this chapter), organized to identify and manage in-hospital strokes, is an example of the operations team devising a process to address an issue raised by a concerned clinician.

Board committee

If the stroke program has a committee of the board of the hospital attached to it, then meeting with that committee once or twice a year to report activities is important.

Location

The complete spectrum of care for the stroke patient is going to occur in multiple locations in the medical center: ED; operating room; neurointerventional suite; ICU; step-down unit; medical/surgical hospital unit (stroke unit, rehabilitation unit, stroke prevention/TIA clinic); and outpatient offices. The excellent outcomes for stroke patients in dedicated units do not depend on the physical space but on the expertise of the staff and the standardized protocols for care. Nurses in all of the units need stroke education and, ideally, NIHSS certification (Appendix).

Neuro ICU

Many comprehensive stroke centers have a neuro ICU staffed by physicians and nurses trained in neurocritical care. This would be the logical place to admit hemorrhagic stroke cases and individuals with ischemic strokes that are more complicated. An NIHSS score should be done on admission, in 24 hours, and at discharge from the unit.

Neuro step-down unit

A step-down unit where staffing levels would be 3:1 (patients/nurse) is a useful concept for the care of stroke victims. Patients who have had uncomplicated acute stroke intervention with IV tPA or intra-arterial (IA) therapy, carotid stenting, or uncomplicated clipping or coiling of an aneurysm may not need ICU-level care, but need closer observation than in the stroke center, where nurses are caring for five patients. The care in the step-down unit could include frequent neurological assessment, careful blood pressure monitoring, and intravenous drip therapy for blood pressure, glucose control, and NIHSS score recording as in the ICU.

Stroke unit

Once the patient is ready to be cared for on a medical/surgical unit, it is worthwhile to have a specified place where stroke patients go. This could be accomplished by simply designating some number of beds on a given unit as the "stroke center beds." Even in large-volume centers, 8–10 beds are generally enough. This way, nursing expertise can be achieved by training a core number of stroke center nurses, and protocols and standing orders can be used routinely. The patient-to-nurse ratio on this unit should be no more than 4 or 5:1. If stroke patients are scattered throughout the general units of the hospital, it is almost impossible to achieve the nursing expertise and routine use of standardized tools. Stroke center beds should always have cardiac monitoring available, as unsuspected arrhythmias are common in stroke victims.

Stroke center work

- Prevent complications
 - Aspiration pneumonia
 - Deep-vein thrombosis (DVT) and pulmonary embolism (PE)
 - Urinary tract infection (UTI)
 - Falls
 - Skin breakdown
- Determine the cause of the stroke with diagnostic studies
- Institute secondary prevention
 - Provide education about stroke and stroke prevention to patients and families
- Institute early rehabilitation
 - Admission NIHSS score
 - Plan for postdischarge monitoring

By concentrating the patients in one location, the nurses become familiar with neurological evaluation and the diagnostic workup for stroke. This knowledge is helpful in communicating with families and referring physicians and can potentially decrease length of stay. For example, if the nurse is aware that the physician would like to order an echocardiogram if the carotid artery evaluation is unremarkable, this could be accomplished without the physician having to return to the unit. Both studies might be accomplished in one day.

It is important to have a sign identifying this part of the unit as the stroke center. A poster-size version of the stroke clinical path can be hung on the wall. It helps the staff as well as the patients and their families know that something unique for the care of stroke is going on in this place.

Tools

The purpose of all of the tools is to standardize care as much as possible: prevent complications, institute early rehabilitation, perform appropriate diagnostic studies, identify issues in secondary prevention, ensure that appropriate treatment is given, and communicate effectively with patients, families, and referring physicians.

Care paths

Stroke paths can be organized in days or phases (Appendix). Ideally, the path is initiated in the ED and carries through the ICU, step-down unit, and stroke unit and interfaces with a stroke rehabilitation care path. Usually, the path is a tool used by nurses rather than physicians and can be used as a nursing charting tool to avoid double documentation. The path can also be the source for documenting specific data elements, such as verification that stroke education and information on smoking cessation were provided to the patient.

Paths for ischemic and hemorrhagic stroke may differ from paths for subarachnoid hemorrhage and TIA. Many centers develop "patient paths" that track the same information but in lay terminology for patients and families. The paths should be reviewed annually by the stroke team nurse coordinator to be sure that they are based on current evidence.

Standing order sets

If the care paths are for nurses, the correlating tools for physicians are the standing order sets. Ideally, each path has an associated order set, and as electronic medical records evolve, these tools become completely integrated. The first order on stroke standing orders should be to initiate the stroke care path. This automatically sets into motion the measures to prevent complications and initiate early rehabilitation. There is sometimes resistance on the part of physicians to use standing order sets. However, no diagnostic test is done or medication given without specific orders from the attending physician. Once physicians become familiar with use of the order set, they find it saves time. It is important when developing these orders to write a draft version and ask the physicians who will be admitting patients with strokes to make suggestions and changes. The physicians should also be given the opportunity to review the yearly updates to the order sets.

One technique to encourage the use of order sets by physicians is to require any patient who is admitted to a stroke unit bed to be on the care path. Empower the stroke unit nurses and the unit secretary to place the standing orders on the chart or initiate the electronic version. When it becomes obvious that the standardization of care for the stroke patient ensures shorter length of stay and improved outcomes, most physicians will agree to use the tools. They must feel, however, that they have input into the content. The medical director and nurse coordinator can facilitate communication between the attending physicians and the stroke center staff.

In addition to having specific order sets addressing various types of strokes and TIA, it is helpful to have standing orders for blood pressure management; pre- and postinterventional procedures such as stenting, clipping, and thrombolysis; and pre- and postsurgical procedures.

Protocols/flowcharts

Whenever the process of care is complex, developing a protocol or flowchart can be helpful in making sure everything is done properly and that all the providers of care are communicating accurate information. One obvious example is the administration of tPA for acute stroke. The example of an ED flowchart shown in the Appendix puts all the pertinent information onto one page. Another example is the tracking of the neurological status of the patient over time. Most stroke centers do an NIHSS evaluation on admission, at 24 hours, and at discharge. However, more frequent evaluation of the neurological status of the patient is often critical. The Neuro Frequent Assessment Tool (included in the Appendix) developed by Saint Luke's Hospital stroke team was so successfully implemented in the stroke center that it is now the tool used by all units in the hospital that need to track the neurological status of the patient, such as the cardiovascular ICU and cardiac catheterization labs. Additional clinical tools from the University of Cincinnati stroke team are included in the Appendix.

Stroke and risk factor education

Education about stroke, stroke warning signs, and stroke prevention is important and challenging to accomplish. Educational materials are available from the American Stroke Association and the National Stroke Association. Many stroke centers develop their own stroke education booklet. As risk factors are identified, it is important for the stroke center nurses and physicians to discuss those with the patient throughout the hospital stay and at discharge (Appendix).

Questions to consider

- 1. Will there be a dedicated stroke unit? Location?
- 2. How many beds are required for the ICU and step-down unit?
- 3. Who can admit patients to each unit?
- 4. What policies for use of care paths and order sets will be in place on the unit?
- 5. What equipment is needed?
- 6. What will the staffing ratio be for each unit?
- 7. Who will train the nurses?
- 8. What care paths and order sets are needed?
- 9. Who will write the standardized tools?
- 10. What kind of neurological assessment will be done on the unit, and how often?
- 11. Will all the nurses be certified to use the NIHSS?
- 12. Who should be on the stroke program team?
- 13. How often should the team meet?
- 14. What outcome and performance measures will be tracked?
- 15. What dataset will be used?

Appendix to Chapter 2: Stroke Watch Action Team (SWAT)

Hospitalized patients who suddenly develop symptoms of stroke need rapid assessment to determine if any intervention should be considered. The usual chain of events whereby the nurse taking care of a patient calls the house staff or attending physician can delay immediate neurological evaluation, resulting in missed opportunities for acute stroke treatment. The Stroke Watch Action Team (SWAT) developed by Saint Luke's Stroke Center in 1996 is a rapid response team specifically aimed at evaluating people with sudden change in neurological status. It takes advantage of the expertise of the stroke center nurses and code neuro nurses, who are all trained to use the NIHSS as a tool for assessment.

Code SWAT

See Figure A2.1 for details of code SWAT in action.

- Patient develops sudden change in neurological status.
- Floor nurse caring for the patient needs assistance in sorting out what is going on.
- Floor nurse calls hospital operator to page a code SWAT to the code neuro nurse pager.
- Floor nurse checks patient's glucose level.
- Code neuro nurse picks up a SWAT flowsheet (Figure A2.1) and goes to the room of the patient in question.
- A CT scan is done on a STAT basis, and the neurologist can make a recommendation regarding how to treat the patient.

Since its inception in 1996, there have been 40–70 SWAT calls per year. Initially, the stroke center nurses answered these calls, but currently the code neuro nurses respond. The floor nurses are satisfied that they now have a method to obtain an immediate evaluation of their patients.



Total time < 30 minutes

Figure A2.1 Stroke Watch Action Team (SWAT) algorithm for rapid identification and treatment of in-hospital strokes.

			Neurolog	gical E	vent				
	Time of No vious Assessm		iset: n No Apparent Ne	eurolo	 aical Deficit	s:			
PATIENT ASSESSMENT		MODIFIED NIH *		*See 1	*See the NIH Key on back of form		oke otoms	Recommendations	
CODE SWAT	TIME:					[]Mo	tor	[] Neuro consult	
BP		LOC 0-3				[]Se		[]CT Scan	
PULSE		LOC 0-2 QUESTIONS				[]Language []Visual		[] Observation [] Heparin Drip [] ASA [] tPA [] Carotid Doppler [] Arteriogram [] MRI/MRA [] Other [] None	
RHYTHM (Check one)	[] Irregular [] Regular	LOC	COMMANDS 0-2		[]Dysarthria [[]None [
RESP.		MOTOR ARMS 0-4 MOTOR LEGS 0-4		L	Α.	[] Other (Check ALL that apply)			
				F	RA				
O2 Sat				L	.L				
					RL				
Blood Gluc	ose	FACIA	L PALSY 0-3]		(Neurologist	
General Patient Information		Total		tal				Recommendations for Treatment)	
[] Recent Date:	Surgery	[] Recent Invasive Procedure Date:]				
[] CV Sur	inal surgery gery ar surg/fem pop	/ AAA	[] PTCA/PA [] Car [] A-gram [] Oth AAA						
Rea	son for Admissio	on/Diagnosis			Additional Per		Pertinent Health History		
[] Arrhythmia [] CVA/TIA [] HTN [] CEA [] CEA [] CHF [] CAD [] ChF [] CAD [] Chr [] Chal [] Chr [] Chr			I/PE ephalopathy umonia er		[] CVA/TIA [] CAD [] AFIB [] HTN [] DM [] Carotid Disease [] Hyperlipidemiaa		[] DVT/PE [] CHF/Cardiomyopathy [] COPD [] Renal Disease [] Abdominal Surgery [] Other		
[] Dr. Arkin [] Dr. Ryme Time Neuro	- 989-2203	[]Dr. []Dr.	Bettinger – 989-2 Schwartzman -	2198	[7819 [] Dr. Bo] Dr. We	utwell – s einstein -		
Patient Labe	l		RN	I Activ	ating Code	SWAT _ AT RN _		Signature	
Patient assess	ment and form to	be com	pleted by SWAT RN	I	Original:			by: SWAT Satchel	

Figure A2.2 Frequent neurological assessment key (back of form).

Concerned families are satisfied with the quick response and evaluation for intervention. Clearly, not all the calls are actual strokes, and not all of the strokes can be treated, but the process has elevated the level of care; all patients who are eligible receive treatment. Nurses, physicians, and house staff new to the hospital are educated about the services of SWAT. In some centers, the rapid response team fills this role.

FREQUENT NEUROLOGICAL ASSESSMENT KEY

LOC (Leve	l of Consciousness)
-----------	---------------------

- 0 = Fully alert, immediately responsive to verbal stimuli; is able to cooperate completely.
- 1 = Drowsy; consciousness is slightly impaired; arouses when stimulated verbally or after shaking; responds appropriately.

If the patient scores either 2 or 3 in this section of the neuro check, proceed to the Glasgow Coma Scale

- 2 = Stuporous; aroused with difficulty, often painful stimuli must be applied; arousal usually incomplete; responds inadequately; reverts to original state when not stimulated.
- 3 = Comatose; unresponsive to all stimuli or responds with reflex motor or autonomic effects.

LOC QUESTIONS

- 0 = Patient knows his age and the month (only initial answer is scored).
- 1 = Patient answers one question correctly.
- 2 = Patient unable to speak, to understand, or answers incorrectly to both questions.

LOC COMMANDS

- 0 = Patient grips hand and closes/opens eyes to command.
- 1 = Patient does one correctly.
- 2 = Patient does neither correctly.

MOTOR: ARM (Right & Left)

The patient is examined with arms outstretched at 90° if sitting, or at 45° if lying down. Request full effort for <u>10 seconds</u>. If consciousness or comprehension is abnormal, <u>cue</u> patient by actively lifting arms into position as the request for effort is verbally given.

- 0 = No drift (Limb holds at 90° if sitting, at 45° if lying down for full 10 seconds).
- 1 = Drift (Limb holds position, but drifts before 10 seconds; does not touch the bed).
- 2 = Some effort against gravity (Limb falls to the bed before the full 10 seconds)
- 3 = No effort against gravity (Limb falls, no effort against gravity, some voluntary movement observed).
- 4 = No movement.
- U = Untestable due to amputation.

MOTOR: LEG (Right & Left)

While supine, the patient is asked to maintain the leg at 30° for <u>five seconds</u>. If consciousness or comprehension are abnormal, <u>cue</u> patient by actively lifting leg into position while the request for effort is verbally given.

- 0 = No drift (Leg holds 30° for five seconds).
- 1 = Drift (Leg falls to intermediate position by the end of five seconds).
- 2 = Some effort against gravity (Leg falls to bed by five seconds).
- 3 = No effort against gravity (Leg falls to bed immediately, with no resistance to gravity, some voluntary movement observed).
- 4 = No movement.
- U = Untestable due to amputation.

FACIAL PALSY

Ask the patient to show teeth, raise eyebrows, squeeze and shut eyes.

- 0 = Normal
- 1 = Minor
- 2 = Partial
- 3 = Complete

Figure A2.2 (continued) Frequent neurological assessment key (back of form).

chapter three

Regional stroke networks

Introduction

Organizing regional networks linking primary care hospitals and physicians to comprehensive stroke centers (CSCs) capable of providing the entire spectrum of acute stroke intervention remains essential to increasing the number of stroke victims who receive acute interventions. Intravenous (IV) recombinant tissue plasminogen activator (rtPA) use has increased very gradually from 1.4% in 2001 to 4.5% in 2009 in the United States. This increase is demonstrated in two different national administrative databases, and the initiation of stroke center certification seemed to be a key turning point.¹ Continuation on this trajectory is needed.



National estimates of recombinant tissue-type plasminogen activator (rtPA) use in the United States [1].

BOX 3.1 GUIDELINE PRINCIPLES FOR STROKE NETWORK ORGANIZATION

- Successful stroke intervention is time dependent.
- Stroke victims will most often go to or be taken to the closest hospital.
- Hospitals will have varying capabilities to render acute stroke intervention.

Determine the type of stroke center you will be

One of the first steps in organizing a stroke center is to determine the level of care that can be provided, as discussed in Chapter 1. Once that decision is made, then a logical next step is to find out the capabilities of other hospitals in the region and determine where your center fits in the spectrum of services available in these hospitals. The most likely pattern is that there will be one or more CSCs in the large urban center, surrounded by hospitals capable of primary stroke care, surrounded by hospitals that will be "stroke ready" (i.e., able to administer IV rtPA), and then surrounded by hospitals that will ideally be bypassed.

The most sophisticated treatments for stroke, such as mechanical clot retrieval or aneurysm clipping and coiling, require a neurointerventional and neurosurgical team. This would mean that both the hospitals capable of administering intravenous thrombolytics and the hospitals without that capability should be linked to a CSC to provide the best possible outcome for each patient.

See Table 3.1 for added components of CSCs as compared to primary stroke centers (PSCs) based on Joint Commission standards. (http://www.jointcommission.org/certification/advanced_certification_ comprehensive_stroke_centers.aspx) Similar components are required for certification by Det Norske Veritas (DNV) (http://dnvaccreditation.com/ pr/dnv/primary-stroke-center-certification.aspx).

Primary components	Additional comprehensive components
24/7 acute stroke team available within 15 minutes Written protocols EMS to identify and route strokes and prenotify ED ED that is stroke/tPA ready Stroke unit Neurosurgery Diagnostics: lab results within 45 minutes Rehabilitation and recovery: PT, OT, speech, rehabilitation placement Imaging services: noncontrast CT and vascular imaging	Imaging (24/7 digital substraction angiography, MRI and MRA, CTA, ultrasound, echocardiogram) Posthospital care coordination for patients Neurocritical care Peer review process to review/monitor care Participation in stroke research Monitoring of periprocedural complications Minimum volumes of SAH, coiling/ clipping, and IV rtPA patients Professional educational activities within and outside stroke center Public educational activities

Table 3.1 Key Components of Primary versus Comprehensive Stroke Centers

Note: CT, computed tomography; CTA, computerized tomographic angiography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; OT, occupational therapy; PT, physical therapy; SAH, subarachnoid hemorrhage.



Figure 3.1 Examples of regional networks in the United States (clockwise: Cincinnati, OH; Kansas City, MO; San Diego, CA; Peoria, IL; Augusta, GA; Houston, TX).

Examples of successful regional networks in the United States are shown in Figure 3.1. They vary in their approaches toward organizing stroke care. For example, there are medium-size cities where one stroke team based at the CSC drives to several hospitals in the area and provides in-person evaluation and treatment and might provide care to more distant hospitals via telemedicine. Examples of this approach include that of the University of Cincinnati, University of California-San Diego, and University of Texas-Houston. Others provide acute stroke care primarily via telemedicine, such as Our Lady of St. Francis Stroke Network (Peoria, IL) and Medical College of Georgia REACH Network (Augusta, GA) as spoke hospitals are in primarily rural areas. Still others, such as Saint Luke's Neuroscience Institute (Kansas City, MO), treat a large proportion of their patients in outlying areas by phone and then "drip and ship" them to the CSC. Drip and ship refers to starting IV tPA at the referring hospital and then continuing the IV infusion while the patient is being transported by ambulance or helicopter to the CSC. In addition, some hospitals may not participate in a network based on region, but rather achieve their stroke readiness or their PSC status by staffing through commercial telemedicine organizations, such as Specialists on Call, Incorporated. These companies consist of a national rotation of stroke physicians who staff multiple hospitals across the country to provide acute stroke care. (See case studies for descriptions of the stroke networks in Cincinnati, OH, and Kansas City, KS.)

BOX 3.2 CASE STUDY 3.1: UNIVERSITY OF CINCINNATI STROKE TEAM

A well-established model exists in the greater Cincinnati/northern Kentucky region, where the acute stroke team has been available on call 24 hours per day and 7 days per week since 1988. Emergency department (ED) physicians call the stroke pager (immediately on patient arrival and ideally prior to receiving computed tomographic [CT] scan results) and speak directly to a stroke physician on call.

If the hospital is one of the 16 in the greater Cincinnati/northern Kentucky region and the patient sounds as though he or she may be eligible for acute treatment, then The University of Cincinnati (U.C.) stroke team physician on call immediately travels to that hospital. The patient is evaluated and administered IV rtPA if appropriate. The patient is typically admitted to that local hospital with the stroke team physician on consultation for 24 hours. Reasons for transfer (by ambulance or helicopter depending on the urgency and weather) to University of Cincinnati Medical Center (UCMC) include severe stroke that may benefit from acute endovascular therapy or neurointensive care monitoring for either malignant cerebral edema and future decompressive hemicraniectomy or fluctuating deficits that warrant careful blood pressure management. If the patient remains at the local hospital, then the stroke physician signs off at 24 hours and hands off the patient's stroke care, including secondary prevention and recovery issues, to the local neurologist.

The U.S. Stroke Team also receives calls from regional hospitals. Currently, seven regional hospitals are linked by telemedicine, and several others are guided toward IV rtPA treatment by phone only. Hospitals in the local network and the region receive education, protocol templates, and recommended order sets from the stroke team. (See Appendix for examples.)

Stroke team activation and treatment rates have increased significantly over the last several years (Figure 3.2), and they have increased at many of the individual hospitals (Figure 3.3). In 2011, there were 1,943 calls made to the stroke team, 398 individuals were seen in person, and 230 were treated with IV rtPA or endovascular therapies.

Links in the chain of successful stroke intervention (focusing on ischemic stroke)

Public awareness

Many stroke victims do not experience pain and, because the brain is the affected organ, they cannot process what is happening. It is usually



Figure 3.2 U.C. Stroke Team volume (calls, in-person evaluations, and acute reperfusion treatments).

someone else on the scene who recognizes the signs of stroke. It is important for everyone, not just the "at-risk population," to know the warning signs of stroke and to know that calling 911 is the best course of action.

Stroke center personnel are often the source of this education within a community.² A useful public awareness message uses the acronym FAST in evaluating a possible victim of stroke: F for face, A for arms, S for speech, and T for time. This message was developed by the U.C. Stroke Team based on the Cincinnati Prehospital Stroke Scale (CPSS) and has been developed into illustrations and campaigns nationally and internationally (Figure 3.4). Another useful message that may be more sensitive and comprehensive, but possibly less easy to remember, is the SUDDENS message created by the Brain Attack Coalition (Figure 3.5).

Emergency medical services

Emergency medical services (EMS) providers play a key role in the success of acute stroke intervention. While on the scene, they can determine the time of stroke onset (i.e., the time that the patient was last known to be well) and perform a quick neurological assessment. The most commonly used assessments are the CPSS³ or the Los Angeles Prehospital Stroke Screen (LAPSS)⁴ (Appendix). With the aid of these tools, the EMS provider can rapidly identify a stroke with a high degree of accuracy and then prenotify the hospital of impending arrival. This allows the ED to be "stroke ready," and the ED may also prenotify the stroke physician on call.



Figure 3.3 Treatments by hospital in the U.C. Stroke Team Network.

EMS transport systems are locally organized. In most communities, stroke victims are taken, either by ambulance or helicopter, to only those EDs that are stroke ready.⁵ It is important for stroke centers to become familiar with the local EMS providers, offer education about stroke if it is needed, and understand the policies that determine where stroke victims are routed in their specific region.

BOX 3.3 BLUEPRINT FOR EMS

The following list is a blueprint for EMS^{6,7}:

- Dispatch stroke patients at highest level of care available in the shortest time possible.
 - Time between receipt of call and dispatch of response team is less than 90 seconds.
- EMS response from call to arrival at scene is less than 8 minutes.
 - Dispatch time less than 1 minute
 - Turnout time to being en route less than 1 minute
- On scene in less than 15 minutes
- Travel time should be like trauma and acute myocardial infarction (AMI) calls.
 - Consider air transport if ground transport will be longer than 60 minutes.





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(b)

Figure 3.4 (a) English public awareness campaign using the acronym FAST and (b) Spanish public awareness campaign. (Illustration produced by the Massachusetts Department of Public Health through funding by the U.S. Centers for Disease Control and Prevention [CDC].)



Figure 3.5 Public awareness campaign using SUDDENS Message. (Illustration developed by the America Heart Association/American Stroke Association.)

Primary hospital evaluation

The stroke–ready primary hospital will be able to administer intravenous tissue plasminogen activator (tPA) to the eligible stroke patient within 1 hour after arrival. Administering tPA intravenously requires that the CT scan is done and interpreted, the necessary laboratory studies are complete, and the drug is mixed rapidly. Detailed discussion of this process is provided in Chapter 5.

A tPA checklist is helpful to make sure that the patient is an appropriate candidate. A flowsheet can be helpful as well. The example in the Appendix from the Saint Luke's Hospital Stroke Center has two possible paths. If the patient presents within 3.5 hours of symptom onset, then the primary hospital capable of administering IV tPA can follow that pathway. If the patient is IV tPA ineligible and has a severe stroke, then there is still an option for endovascular treatment, and the transfer protocol can be instituted.

The ED physician typically administers IV tPA with consultation from a neurologist in person, by telemedicine,⁸ or by phone. Quick access to a neurologist at the stroke center hospital is mandatory to ensure treatment of patients as early as possible within the 4.5-hour time window.

There are many aspects of acute stroke care in addition to whether thrombolysis should be administered. These are discussed in Chapter 6 and are also important for the primary hospital to consider.

Rapid communication and transport to a comprehensive stroke center

The primary hospital will benefit greatly by having a relationship with a CSC. It is critical that the CSC make communication and transport efficient. After administration of IV tPA, some patients may be transferred

BOX 3.4 CASE 3.2: THE SAINT LUKE'S HOSPITAL STROKE CENTER REGIONAL NETWORK

Since opening in 1993, Saint Luke's Stroke Center (SLSC) has developed a relationship with many hospitals in the region. Stroke center physicians and nurses provide on-site stroke education to the staff of these hospitals.⁹

Regarding rapid transfer, all referring physicians/hospitals can use one phone number to transfer a patient or to speak to a stroke neurologist. The phone number accesses a trained triage nurse, who asks a series of questions regarding time of onset, availability of CT scan, and so on. The transfer nurse then pages the neurologist on call with a 911 page. The neurologist receives the information from the nurse and calls the referring ED to discuss options, including making a decision regarding whether to give IV tPA in the ED at the primary hospital. If tPA is going to be administered, a set of orders and a transport protocol are faxed to the primary hospital (Appendix). That ensures the primary hospital has up-to-date order sets and the transporting EMS crew has guidelines to follow.

If the patient is to be transferred, the stroke neurologist speaks to the patient or family by phone to discuss what might happen after transport and gets a cell phone number so that further discussion can occur after the patient arrives by ambulance or helicopter. The family always arrives later by car, and that time interval is critical. The neurologist then notifies the transfer team nurse to alert the code neuro nurses, who notify the neurointerventional team and coordinate care on arrival. Code neuro nurses are neuro critical care nurses in house 24/7 to respond to every neuro case in the ED. Everyone is ready to go once the patient arrives. After the patient is treated at SLSC, every effort is made to send information on the outcome back to the referring ED physicians and EMS crews. This regional organization of care has resulted in a 29–39% stroke intervention rate (2008–2011).

Regional stroke transfer algorithm Stroke victim presents to rural ED Call to Saint Luke's Hospital (SLH) transfer team (TT) TT nurse questions

- Time of onset
- CT results
- tPA checklist

911 page to neurologist on call with information and ED phone number

Neurologist talks to ED

- Discusses options with family and gets cell phone number
- Decision to give IV tPA with ED doctor
 - If tPA is to be given prior to transfer, tPA order set, dosing schedule, and transport protocol are faxed to the sending hospital.
 - Transfer plans are coordinated by code neuro nurse.

TT notifies

- Interventional radiology
- Admissions
- Neurologist of estimated time of arrival (ETA)

In a series of 1,626 ischemic strokes seen at Saint Luke's Hospital from 2008 to 2010, there were 717 (44%) transferred from 63 referring hospitals. Of these, 20% (145) were drip-and-ship cases for which the IV tPA was administered in the referring hospital in consultation with the stroke team at Saint Luke's. Twenty-nine of the hospitals were critical access hospitals with 25 beds or less. On arrival, 14/145 (9.6%) had blood pressures greater than 180/105. Two of those cases resulted in mortality, one related to symptomatic intracerebral hemorrhage and one not associated with hemorrhage. Overall, there was a 13.7% mortality rate (20/145). Twenty-four percent (35/145) went on to intra-arterial interventions. At 90 days, 63% of the drip-and-ship cases had a good outcome, with modified Rankin Scale (mRS) scores of 0–2. Based on these data, drip and ship appears to be safe, blood pressure is generally well controlled en route, and even very small hospitals can successfully administer IV tPA.

for adjunctive endovascular therapy. Further, some hospitals will transfer patients out of the ED for their post-tPA monitoring, particularly when they do not have an intensive care unit (ICU) or access to a neurologist. Patients with intracranial hemorrhage, including intracerebral hemorrhages and subarachnoid hemorrhages, often require the resources at a CSC as well.

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chapter four

Imaging for diagnosis and selection of therapy

Imaging for acute ischemic stroke

Imaging plays a pivotal role in the evaluation of a patient with acute stroke. The critical information provided by imaging can help triage stroke patients for appropriate treatment. Rapid advances in computed tomographic (CT) and magnetic resonance (MR) technology have enabled comprehensive evaluation of brain parenchyma, vessel status, and perfusion. The role of multimodal imaging and advantages and challenges of CTand MR-based modalities are discussed.

The goals of imaging in acute stroke are to answer the following questions:

- Is the stroke hemorrhagic or ischemic?
- Is there a stroke mimic explaining the neurologic condition of the patient?
- Is there a large-vessel occlusion?
- Is there a "core" or critically ischemic infarcted tissue, and how large is this core?
- Is there salvageable brain tissue?

Computed tomography

The biggest advantage of CT-based technology is that it is easily accessible, relatively rapid, and cost-effective. In addition to standard noncontrast head CT, multimodal CT imaging includes CT angiography (CTA) and CT perfusion (CTP) studies.

Noncontrast CT head

Noncontrast CT (NCCT) remains the workhorse of acute stroke imaging and is considered the gold standard for hemorrhage. An emergent NCCT is essential to detect intracranial hemorrhage and to exclude stroke mimics. It should be available 24 hours per day and 7 days per week. The CT scan should be performed within 25 minutes of arrival; therefore, a technician must be available promptly. In addition, interpretation must be





available within 45 minutes. In many instances, the physician making the decision regarding the use of thrombolytic therapy (i.e., the neurologist or emergency department [ED] physician) will do the initial interpretation of the NCCT; hence, it is important to be familiar with the early CT signs of ischemia.

- Early ischemic changes on CT include loss of gray-white differentiation, sulcal effacement, hypodensity (Figure 4.1) and swelling of the insular cortex, and basal ganglia obscuration. A narrow window width and center-level settings are recommended for better detection of these subtle signs (approximately 25/35 HU).
- The CT ischemic signs have been quantified through a 10-point scoring system called the Alberta Stroke Programme Early CT Score (ASPECTS). In the National Institute of Neurological Disorders and Stroke (NINDS) trial, there was a trend toward reduced mortality and increased benefit to tPA (tissue plasminogen activator) with a greater than 7 ASPECTS score.¹ While potentially prognostically significant, these changes have not been shown to predict hemorrhagic transformation after thrombolytic therapy within 3 hours.



Figure 4.2 Hyperdense left middle cerebral artery.

- Frank hypointensity on CT, involving more than one-third of a middle cerebral artery (MCA) territory, is a strong contraindication to treatment and typically leads to a revised history of the patient being outside the intravenous recombinant tissue plasminogen activator (IV rtPA) time window.
- Other findings on NCCT include the observation of a hyperdense artery (Figure 4.2), indicative of an intraluminal thrombus, a sensitive but nonspecific sign for a large-artery occlusion.

BOX 4.1 EARLY NCCT FINDINGS IN ACUTE STROKE

- Hypodensity in the lentiform nucleus
- Insular ribbon sign
- Loss of gray-white matter differentiation
- Sulcal effacement
- Hyperdense MCA sign

CT angiogram

The main goal of CTA is to evaluate the status of intracranial and extracranial vasculature, including detection of large-vessel occlusion. The site of occlusion can help determine stroke mechanism, assess prognosis, and potentially identify patients who should receive intra-arterial (IA) therapeutic modalities in addition to, or instead of, intravenous therapy if IV tPA ineligible.

CTA source images (CTSIs) with a narrow window are more sensitive than NCCT for the detection of early brain infarction.² The CTSI is reflective of blood volume and can detect areas of hypoperfusion.

CT perfusion

- The proposed advantage of perfusion imaging is that perfusion/ diffusion "mismatch" identifies salvageable brain tissue for delayed reperfusion treatment. Using the concept of penumbral imaging, it may be possible to extend the critical treatment window of a stroke patient.
- CTP studies can provide quantitative maps of cerebral perfusion parameters of mean transit time (MTT), cerebral blood flow (CBF), and cerebral blood volume (CBV).
- Low CBV may estimate irreversible ischemic injury or core (similar to DWI-MRI [diffusion-weighted imaging magnetic resonance imaging], discussed further in the chapter) and predict poor outcome despite recanalization.³ Newer studies have indicated absolute and relative (normalized) thresholded CBF maps may be more accurate for infarct core than CBV maps.⁴
- Identification of reversible ischemic tissue or penumbra may be determined based on a decreased CBF, elevated MTT, and normal or elevated CBV. Untreated or unsuccessfully treated patients with large CBF or MTT/CBV mismatch exhibit significant lesion growth.

Magnetic resonance imaging

Emergent MR is less accessible than CT scanners at most hospitals but remains a promising avenue for diagnosis and therapy selection. Comprehensive stroke centers should have advanced imaging consisting of MRI and MRA available 24 hours a day, 7 days a week. Qualified MRI technologists are required to be available 24 hours a day, 7 days a week (although not necessarily in house).

Stringent MR acquisition guidelines to obtain maximum imaging data in the shortest time should be a priority in the setting of acute stroke. MRI protocols for acute stroke generally include conventional MRI sequences (DWI, fluid-attenuated inversion recovery [FLAIR], gradient echo [GRE]), MR angiogram (MRA), and MR perfusion (MRP) and can be completed in 10–15 minutes.

MRI brain

- 1. Diffusion-weighted imaging
 - DWI can confirm an acute infarct within minutes of onset and has high sensitivity and specificity for acute ischemia that surpasses Non Contrast Computed Tomography (NCCT).^{5,6}
 - The American Academy of Neurology 2010 clinical guidelines recommend that DWI be performed for the most accurate diagnosis of acute ischemic stroke.⁷
 - A positive DWI lesion is analogous to the core of an infarct and may also yield important prognostic information. In nonstandard IV rtPA time windows, a "malignant MRI profile" characterized by a DWI lesion larger than 100 mL or a perfusion–weighted imaging (PWI) lesion larger than 100 mL with longer than 8 seconds of $T_{\rm max}$ delay may have a high risk of symptomatic hemorrhagic transformation and death after rtPA therapy.⁸ Patients with a DWI lesion (core) larger than 70 mL before IA thrombolysis had a poor prognosis and high mortality rate despite a 50% recanalization rate.⁹
- 2. Gradient echo
 - MR-GRE is as accurate as CT for detection of acute hemorrhage and more sensitive for detection of chronic hemorrhages.¹⁰
 - A thrombus in an intracranial vessel appears as an intracranial hypointensity on GRE (susceptibility vessel sign).¹¹
- 3. Fluid-attenuated inversion recovery
 - In the first 6 hours of acute stroke, the FLAIR sequence will generally be normal. A mismatch between positive DWI and negative FLAIR may be indicative of a hyperacute stroke (less than 3 hours).¹² This is under investigation as a potentially useful sign to determine time of stroke onset and reperfusion therapy eligibility, particularly in wake-up stroke.
 - A hyperintense vessel sign on FLAIR may be indicative of slow flow versus collateral flow in the region of acute ischemia, distal to the occluded large vessel.

MR angiography

As with CTA, MRA is useful for diagnosing vascular occlusion and can identify subsets of patients who might be considered for IA therapy in

BOX 4.2 EARLY MR FINDINGS IN ACUTE STROKE

- Hyperintensity on DWI and corresponding hypointensity on apparent diffusion coefficient (ADC) maps corresponding to the core of infarct
- Minimally abnormal or normal FLAIR
- Hyperintense vessel sign on FLAIR indicative of slow flow or possibly collateral flow
- Susceptibility vessel sign on GRE sequence indicative of thrombus

addition to intravenous therapy or instead of intravenous therapy if IV tPA ineligible. Two MRA techniques can be used in clinical practice: contrast-enhanced MRA and time-of-flight (TOF) MRA.

MR perfusion-weighted imaging

- Perfusion imaging of the entire brain is possible with MRI and is a distinct advantage over CTP. Similar to CTP, MRMRP can estimate the ischemic penumbra using MTT, Time to Peak (TTP), and $T_{\rm max}$ parameters
- Regions of brain tissue with normal DWI and increased MTT or T_{max} may represent hypoperfused areas of reversible ischemia. The ischemic penumbra is the DWI-PWI mismatch, that is, the volumetric difference between the DWI lesion (representing ischemic core) and the perfusion defect.

Challenges of CT and MR perfusion imaging

Whether perfusion imaging may allow the selection of patients likely to be responsive to thrombolytic therapy beyond the established time limits remains to be determined. Further, both CT and MR perfusion studies have several pathophysiological and technical challenges.

- The interpretation of perfusion imaging is challenging because of lack of well-defined thresholds for infarct core and ischemic penumbra. There is lack of standardization of perfusion parameter values across different vendor platforms for acquisition and postprocessing, and there is no consensus regarding optimal perfusion algorithm and mismatch thresholds.¹³
- CTP, though promising, is yet to be validated by clinical trials.

BOX 4.3 PRACTICAL ASPECTS OF IMAGING IN ACUTE STROKE

- NCCT remains the initial study of choice in acute stroke imaging.
- Although DWI has excellent diagnostic accuracy, MR evaluation is not practical for most stroke centers due to limited availability.
- Both CTA and MRA are excellent tools for evaluating vascular occlusion.
- Vascular imaging may be considered during the initial imaging workup of acute stroke for patients if endovascular therapy is contemplated for management.
- Perfusion studies may identify and differentiate the infarct core and ischemic penumbra. Accuracy and usefulness of MRP and CTP have not been well established and are the focus of intense research. Better standardization and validation are needed.
- The acquisition of multimodal CT or MR studies should not delay the administration of IV tPA within the 4.5-hour time window.
- Another limitation of CTP is the limited brain coverage. Newer 256and 320-detector scanners are capable of whole-brain perfusion, although at the cost of increased radiation dose.
- Although clinical trials using MRP have provided biological support for the mismatch hypothesis, clinical benefit using penumbral selection was not demonstrated in the recently published MR RESCUE trial with both penumbral and nonpenumbral cases and both treated and untreated cases.^{14–16}
- There is insufficient evidence to support perfusion imaging to aid clinical decision making in acute stroke.^{7,16,17}

Safety considerations

- CTP radiation doses are traditionally higher than NCCT. Moreover, using CT-based multimodal imaging including NCCT, CTA, and CTP adds to the total radiation dose received by the patient. Hence, it is pertinent to set up correct parameters to minimize radiation dose (using 80 kvp and < 200 mA for CTP).¹³
- All patients need to be screened for MRI contraindications, including the presence of cardiac pacemakers and MRI-incompatible and MR-conditional hardware.

Imaging for secondary stroke prevention

Internal carotid artery evaluation

Assessing internal carotid artery stenosis

- Carotid ultrasound is an excellent screening tool. It is reasonably sensitive and specific (80–95% and 95–99% for internal carotid artery [ICA] stenosis > 70%), 100% sensitive and specific for occlusion, and cost-effective.¹⁸
- Three-dimensional (3D) TOF MRA has a comparable profile, but less specificity (90%), for ICA stenosis. It is dependent on flow-related signal changes and subject to velocity and turbulence alterations rather than anatomic depictions of luminal contrast. In other words, stenoses may be overestimated.
- Contrast-enhanced MRA has greater sensitivity and specificity than Doppler ultrasound for extra- and intracranial vessel evaluation.¹⁹ When an MRI is already being performed for other reasons, addition of MRA is a logical screening method. MR contrast agents may be contraindicated in patients with renal failure due to risk of nephrogenic systemic fibrosis.
- The accuracy of CTA is equal to or superior to that of MRA in most circumstances, and in some cases, its overall accuracy approaches that of digital subtraction angiography (DSA).¹⁷ Also, very-high-grade stenosis (string sign) is well depicted by CTA.
- DSA, although a gold standard for vasculature, is not used for screening, due to the risk of complications in less than 1% of patients, unless there is another reason for performing angiography. It is primarily used when there are discrepancies between other noninvasive methods. DSA also provides important information on collateral flow and occult vascular lesions.
- High-resolution plaque imaging is a promising area of research for secondary stroke prevention.

Identifying extracranial artery dissection

An MRI and MRA of the neck are warranted for identifying extracranial artery dissection. A dissection protocol, consisting of T1 axial, fatsaturated MR images of the neck, should be performed. This specific sequence allows an intramural thrombus to be more easily visualized.

Intracranial stenosis evaluation

• CTA is highly sensitive and specific for intracranial stenoses. The application of CTA may be limited with patients with renal dysfunction and contrast allergies.

- TOF MRA also has high accuracy for evaluating intracranial stenosis.
- Transcranial Doppler (TCD) can be used to assess only the proximal vessels in the anterior circulation and has variable accuracy in the posterior circulation, limiting its utility in this setting.²⁰

Imaging of hemorrhagic stroke

The two main types of hemorrhagic stroke are intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH).

Intracerebral hemorrhage

- Although CT is usually the first line of imaging for ICH, MR is equally sensitive for detection of acute hemorrhage and is better for chronic hemorrhages. MR is superior to CT for aging a hematoma and is also useful for determining the cause of the ICH (including amyloid, vascular, and neoplastic etiologies).
- ICH score: The admission ICH score is a simple outcome stratification scale and includes Glasgow Coma Scale (GCS) score, age, ICH volume, presence or absence of intraventricular hemorrhage, and supratentorial versus infratentorial location of the hemorrhage.²¹ The ICH volume is calculated by the ABC/2 method, where the largest cross-sectional diameter of the hematoma is A, second diameter drawn at right angles to the first is B, and C is the height of the hematoma estimated from the number and thickness of CT/MR slices in which the hemorrhage is visible.
- Another imaging variable associated with poor outcome is hematoma expansion as seen on a 24-hour CT.²² CTA is a favored initial diagnostic test in an ICH. CTA "spot sign" or presence of small, enhancing foci in the ICH is associated with progression of the hematoma.²³
- CT/MR angiogram or venogram studies have high sensitivity and specificity at identifying secondary causes of hemorrhage, including arteriovenous malformations, moyamoya, and cerebral vein thrombosis.
- DSA may be considered if clinical suspicion is high or noninvasive CTA/MRA studies are suggestive of an underlying vascular cause.²⁴

Subarachnoid hemorrhage

• If the NCCT demonstrates presence of nontraumatic SAH, it is common practice to obtain an emergent CTA at the same time. As compared to MRA, CTA offers additional advantages, including accurate

measurement of dome versus neck, demonstration of calcification and incorporation of branch vessels, and evaluation of adjacent bony and dural landmarks. All this information can help decide the treatment options, including coiling versus clipping of the aneurysm.²⁵

- The sensitivity of CTA to detect aneurysms approaches that of a conventional angiogram.²⁶ In a recent systematic review, multi-detector CTA reliably depicted ruptured intracranial aneurysms, with a pooled sensitivity of 98% and a pooled specificity of 100%.²⁷ The chance of missing a ruptured aneurysm at CTA is no more than 2%.²⁷ Missed aneurysms at CTA are often small (less than 3 mm) and located near the skull base.
- DSA with 3D rotational angiography is indicated when the CTA is inconclusive or if the CTA is negative in the diffuse aneurysmal pattern of subarachnoid hemorrhage.²⁸

Conclusion

Neuroimaging is crucial in the diagnosis and management of acute stroke and secondary stroke prevention. Since the mid-2000s, stroke imaging has undergone significant advances, giving us further insight into stroke pathophysiology and management options. However, it is important to remember that time is of the essence, and rapid acquisition and interpretation of imaging in acute stroke is critical.

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chapter five

Transient ischemic attack TIA

Introduction

The concept of a transient episode of neurological dysfunction caused by temporary ischemia in a focal area of the brain, spinal cord, or retina has been around since the 1950s. When the symptoms are recognized as warnings of an impending stroke, in many cases the stroke can be prevented. Therein is the importance of this topic. The pathophysiologic mechanisms, risk factors, and secondary prevention strategies for transient ischemic attacks (TIAs) are the same as for ischemic stroke, and these are discussed in the chapter on secondary stroke prevention.

Incidence and diagnosis

The true incidence of TIAs is difficult to discern. Public knowledge of the symptoms is quite low. In one study involving over 10,000 subjects, only 8.6% of those surveyed could identify a symptom.¹ This would lead to the conclusion that TIAs are substantially underrecognized and underreported by patients. The percentage of strokes that are preceded by TIAs, generally reported to be 15–25%,² might be much higher due to unrecognized symptoms. Having a TIA affects long-term survival. A TIA reduces survival by 4% in the first year and by 20% within 9 years in people older than 65.³

For patients presenting to emergency departments with transient neurological symptoms, the diagnosis of TIA is often incorrect. In the Oxfordshire Community Stroke Project, of the 512 cases diagnosed as TIAs, 38% were confirmed as the correct diagnosis.⁴ In another series of 100 cases of TIAs, 40% were confirmed.⁵ Common mimics of TIAs are migraine, syncope, vertigo, epilepsy, transient global amnesia, hypoglycemia, medication side effect, and conversion disorders. As there is no definitive radiologic or laboratory test to confirm the diagnosis, it is often sorted out by the neurologist taking a more in-depth history, assessing the risk profile, reviewing diagnostic tests such as vascular imaging or magnetic resonance imaging (MRI), and following the clinical course.

Evolution of definition

The original definition of a TIA, focal neurological symptoms or signs lasting less than 24 hours, remained intact for decades. More recently, it has become clear that the vast majority of TIAs last less than an hour, and sometimes symptoms resolve in a matter of minutes. Most clinicians feel confident in treating patients with continuing symptoms of stroke as quickly as possible after arrival given the better outcomes with earlier treatment. As more sophisticated imaging modalities have become available, the concept of what constitutes a TIA has evolved even further. About one-third of cases of TIA defined by 24-hour time criteria have an abnormality on diffusion-weighted MRI sequences, indicating actual infarction.⁶ Infarction can be seen even in cases with symptoms of 30- to 60-minutes duration. When symptoms last more than 6 hours before clearing, 50% have evidence of infarction on MRI studies.⁷ This has led to the recommendation by the American Stroke Association that the definition of TIA change from one based on time to one based on tissue: a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction. 8 Many cases that used to be classified as TIAs are now described as strokes with rapid and complete recovery or episodes of transient symptoms with infarction (TSI). While this distinction may be important for purposes of coding and reimbursement, both groups are at risk of a second ischemic event and require the same clinical approach of an appropriate and timely evaluation.

Clinical evaluation

A significant number of patients who have TIAs will go on to have a completed stroke. Using a timed-based definition of TIA (without imaging), stroke risk after a TIA has been estimated at 5% in the first 24–48 hours and up to 12.8% in the first week.^{9,10} Ninety-day stroke risk can be as high as 24%.¹¹ The obvious challenge for the stroke center is to determine who is at higher risk for stroke and what sort of evaluation needs to be done in a given timeframe.

Imaging

MRI diffusion-weighted imaging (DWI) is sensitive for early ischemia. When the MR scan is positive in cases presenting with transient neurological symptoms, this is considered a stroke based on the tissue diagnosis discussed. In this circumstance, the risk of subsequent stroke is 7.1% in the first 7 days as compared to 0.4% if the scan is negative, an 18-fold difference. As noted, these cases are now classified as strokes and should be admitted to the hospital for rapid evaluation, optimization of cerebral blood flow, and treatment with antithrombotics.⁷ The obvious challenge is that most hospitals do not provide 24/7 MRI services, and some rural hospitals have no MRI availability. In addition, some patients are excluded from having an MRI either because of an implant or fear of the enclosed space. Computed tomographic (CT) scans are much more readily available, but a CT scan is insensitive to early ischemia. The newer techniques of CT angiography (CTA) and CT perfusion may be quite useful in this regard but require further study regarding their sensitivity. A CT/CTA-positive metric defined as acute ischemic change seen on plain CT or intracranial or extracranial vessel occlusion or stenosis greater than 50% ipsilateral to the clinically relevant ischemic brain tissue has been found to be as sensitive as MRI/DWI in prediciting early recurrent stroke.¹² However, a negative CT workup by itself does not eliminate the possibility of high-risk TIA and should lead to an expedited stroke workup.

ABCD2

There are certain clinical variables that affect the risk of very early stroke after a TIA. These have been carefully studied, and a risk assessment tool called the ABCD2 score has been used in several studies.¹³

ABCD2 Scoring					
$\overline{A = Age > 60}$	1 point				
B = Blood pressure					
Systolic > 140 or diastolic > 90	1 point				
C = Clinical features					
Unilateral weakness with or without speech	2 points				
Speech impairment without unilateral weakness	1 point				
D = Duration					
>60 minutes	2 points				
<10–59 minutes	1 point				
D2 = Diabetes	1 point				

Scores of 0–3 are considered low risk, 4–5 intermediate risk, and 6–7 high risk for stroke at 2, 7, 30, and 90 days. In TIA cases with a positive DWI scan, the ABCD2 score correlates with 7-day stroke risk: 1.8% for scores of 0–3, 7.5% for scores of 4–5, and 12.5% for scores of 6–7. Cases with a positive scan and low ABCD2 score and cases with a negative scan and high ABCD2 score had similar risks for subsequent strokes.¹⁴

Critics of the ABCD2 score cite low interrater reliability and poor correlation with imaging findings and contend that it may be useful in predicting the risk of severe strokes but is insensitive to the risk of minor strokes that carry a risk of significant disability.¹⁵ In one series, 20% of
cases with a low ABCD2 score were found to have a lesion such as a critical carotid stenosis that warranted immediate attention, and these patients had the same 90-day stroke risk as those with high ABCD2 scores.¹⁶

Timely evaluation and TIA clinics

Early initiation of evaluation for the cause of the ischemic event and appropriate treatment have been shown to decrease the risk of early recurrent stroke by 80%.¹⁷ Statistically, cardioembolism is the cause of TIA symptoms in 10–20% of cases, and large-artery stenosis is the cause in 15–20% of cases. There is widespread agreement that vascular imaging and a cardiac evaluation should be carried out as quickly as possible. Carotid ultrasound, CT angiography, and MR angiography are all viable options. A normal electrocardiogram (EKG) does not rule out paroxysmal atrial fibrillation. Telemetry monitoring and echocardiography should be considered for all patients with TIAs.

Traditionally, all cases of TIA were admitted to the hospital, but an alternative to hospitalization for TIA cases is a TIA clinic that is based in either the outpatient setting or the emergency department. Ideally, patients should be seen within the first 24 hours after the event in these settings, where an expedited diagnostic evaluation could be completed and secondary prevention measures instituted. When that kind of "triage" is available with 24-hour access such as reported in the SOS-TIA trial, only 25% of patients needed hospitalization for management of atrial fibrillation, intracranial hemodynamic compromise, and symptomatic carotid or intracranial stenosis.¹⁸ The evaluation in a TIA clinic is protocol driven and can potentially be managed by an advanced practice nurse in consultation with a neurologist.

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chapter six

Acute stroke interventions

Introduction

The availability of acute reperfusion treatment for acute stroke as of 1996, in addition to supportive care and rehabilitation, has driven efforts to organize and standardize stroke care across the world. Organized stroke centers provide the critical infrastructure to implement and disseminate established and newer reperfusion therapies as they become available. This chapter discusses some of the acute treatment options that are currently being used or tested.

Ischemic stroke

The basic challenge in ischemic stroke is to restore blood flow to the brain before there is irreversible tissue damage and loss of neurological function. The most effective therapies will likely be a combination of pharmacologic and mechanical means of restoring flow while protecting brain tissue.

Intravenous tPA

Thrombolytic revascularization with intravenous (IV) tissue plasminogen activator (tPA) is a proven effective way to reverse neurological deficit in acute ischemic stroke.¹ For every eight people treated with IV tPA within 3 hours, one additional patient will have minimal or no disability. Moreover, for every three treated within 3 hours, it is estimated that one additional patient will have less disability.² The overall benefit of tPA is seen in all subtypes of ischemic strokes (cardioembolic, large-artery atherosclerotic, small vessel, and other less-common subtypes) despite an increased risk of symptomatic intracranial hemorrhage (ICH) of 6.6%, compared with 0.6% in placebo controls, within 36 hours of IV tPA administration. This benefit has been consistently reproduced by additional study, including community experience.^{3,4} More recently, based on the European Cooperative Acute Stroke Study III (ECASS III) trials, we have learned that the benefit of IV tPA can be extended to 4.5 hours from onset, although the treatment benefit is reduced; for every 14 patients treated with IV rtPA, 1 additional patient will have minimal or no disability.5

Emergency departments (EDs) are sometimes faced with the challenge of administering IV tPA without adequate neurological support. Ideally, a hospital faced with an acute stroke treatment candidate would have a relationship with a stroke specialist by phone, video telemedicine, or in person to provide guidance (Chapter 3). Without this support, the ED physician is left with the option of developing skills to treat with tPA independently or triaging acute stroke candidates to other hospitals, preferably before arrival to their ED so that time is not lost.

Timing: The earlier the better

Intravenous thrombolysis should be administered to all eligible ischemic stroke patients within 4.5 hours of stroke onset. Within this 4.5-hour time window, earlier treatment leads to better outcomes. Figure 6.1 demonstrates progressively lower benefit of IV tPA as time goes on in a pooled analysis of all major IV tPA trials to date.⁶

Rapid assessment goals

To expedite treatment, administration of IV tPA within 1 hour or less of presentation at the ED (i.e., door-to-needle time) is strongly recommended. Further delineation of time goals regarding ED assessments within that 1-hour timeframe include the following⁷:

- 1. A physician should evaluate a stroke patient **within 10 minutes** of arrival at the ED doors.
- 2. A physician with expertise in the management of stroke should be available or notified **within 15 minutes** of patient arrival. Depending on the protocol established, this may be accomplished by activating a stroke team.

	Modified	Rankin	Score	0 - 1
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Figure 6.1 Odds ratio for favorable outcome (modified Rankin scale 0–1 at 3 months after IV tPA) according to time from onset to IV tPA administration.

- 3. A computerized tomographic (CT) scan of the head should begin **within 25 minutes** of arrival. In many centers, the patient is taken directly from entry to the ED to the CT scanner.
- 4. The CT interpretation should be obtained **within 45 minutes** of arrival. In many centers, this interpretation is performed by the stroke specialist.
- 5. IV tPA treatment should be initiated **within 60 minutes**. Keep in mind that the earlier the treatment, the better the outcome will be.

Pre-tPA evaluation

Good outcomes with IV tPA are dependent on following the inclusion and exclusion criteria established in the National Institute of Neurological Disorders and Stroke (NINDS) tPA and ECASS III trials.^{1,5}

The most important eligibility criteria are the following:

- Ischemic stroke causing disabling neurologic deficit
- Time the patient was last known well is less than 4.5 hours
 - IV tPA must be administered within 4.5 hours of onset of symptoms.
 - The time of onset is the last time the patient was known to be normal.
 - Example: A patient who went to sleep at 11 p.m. and awakened with stroke symptoms at 6 a.m. is assumed to have onset at 7 hours earlier and therefore is *not* eligible for IV tPA.
- CT head scan negative for blood
- Blood pressure (BP) 185/110 at the time of tPA administration without aggressive antihypertensive management.
 - Initial antihypertensive medication choices include
 - Labetalol bolus of 10 mg over 1–2 minutes; if inadequate response within 10–20 minutes, may repeat with additional bolus, using double the prior dose
 - Nicardipine drip starting at 5 mg/hour, titrated up by 2.5 mg/hour at 5- to 15-minute intervals, up to a maximum dose of 15 mg/hour
 - See Chapter 8 for more extensive discussion of BP management.

Additional exclusions include

- CT scan with area of large (e.g., > 1/3 middle cerebral artery [MCA] territory) and clear (darker than white matter and lighter than cerebrospinal fluid [CSF]) hypodensity
 - If an area of clear hypodensity is seen, it is important to confirm that the time of onset is less than 4.5 hours.
- Intracranial or intraspinal surgery, serious head trauma, or previous stroke within the last 3 months
- History of ICH

- Major surgery within the last 14 days
- Suspicion of subarachnoid hemorrhage (SAH) on pretreatment evaluation
- Arterial puncture at noncompressible site
- Active internal bleeding
- Intracranial neoplasm (except meningioma) or arteriovenous malformation (AVM)
- Known bleeding disorder: platelets less than 100,000; international normalized ratio (INR) greater than 1.7; elevated partial thromboplastin time (PTT)

The following are additional exclusion criteria only within the 3- to 4.5-hour time window based on the ECASS III trial design (and considered to be less stringent by some experts):

- History of stroke and diabetes
- National Institutes of Health Stroke Scale (NIHSS) score greater than 25
- Age above 80 years
- On warfarin (regardless of INR value)

Relative exclusions are the following:

- Rapidly resolving neurologic deficit
 - This exclusion has caused some confusion and requires careful consideration. Unless the rapid resolution is a minor deficit (i.e., not clearly disabling), treatment should still be considered.
- Minor neurological deficit
 - Minor deficits should be determined based on deficits that do not appear clearly disabling and not NIHSS score thresholds alone. Disabling deficits can occur with NIHSS scores of 5 or less.
- Acute myocardial infarction in past 3 months
 - This exclusion is based on risk for hemopericardium and is most relevant for transmural or subacute infarcts or patients with symptoms/signs of pericarditis. Many experts would consider tPA treatment of an acute STEMI (ST elevation myocardial infarction) (or a NSTEMI, non-ST elevation myocardial infarction) concurrent with stroke using the lower stroke dosing (0.9 mg/kg) of tPA (as compared to the cardiac dosing of ~ 1.1 mg/kg).
- Seizure at onset
 - When seizure occurs concurrently to acute stroke as verified or supported by neuroimaging, then treatment should be considered. However, the severity of the stroke deficits may be confounded by deficits from the postictal seizure state.

- Glucose less than 50 or greater than 400
 - If deficit persists after glucose correction or infarct is supported by neuroimaging, then treatment should be considered.

It is important to note that there is no upper age limit for IV tPA eligibility within the 3-hour timeframe.

ED evaluation process involves the following:

- Confirm diagnosis and last-known-well time and perform NIHSS.
- Secure two intravenous lines and administer 0.9% normal saline (NS) at 75–100 mL/hour (unless contraindicated); avoid glucose solutions.
- Nasal O₂ if oxygen saturation less than 93%
- Brain CT scan
- Stroke team activation (preferably prior to or while obtaining CT scan)
- Electrocardiogram
- Weigh patient or estimate weight
- Fingerstick blood sugar
- Stat laboratory tests
 - Serum electrolytes
 - Glucose
 - Creatinine
 - Complete blood count with platelets
 - INR
 - Activated PTT
 - Pregnancy test in selected patients
- Treat BP to required level
- Discuss treatment options with family, including risks and benefits.
 - No written consent is needed for standard IV tPA therapy
- If Foley catheter is needed, insert prior to treatment if possible.

Of the labs ordered, only glucose is necessary for most patients prior to tPA administration. If there is no clinical history suggesting coagulopathy (abnormal INR or PTT) or thrombocytopenia (low platelets), then tPA administration should not be delayed to wait for those lab results.^{8,9}

This process can be greatly aided by an ischemic stroke/tPA flowsheet or checklist in the ED (Appendix).

IV tPA administration and dosing

While eligibility is being determined, tPA should be prepared. If the patient is not ultimately eligible for tPA, the cost of the tPA will be reimbursed by Genentech as per the package insert. tPA should be diluted 1:1 in sterile water or normal saline, and the mixture should be gently swirled, but not agitated. The approved dose is 0.9 mg/kg with 10% administered as a

BOX 6.1 RECOMMENDED STRATEGIES FOR DECREASING DOOR-TO-NEEDLE TIMES FOR IV TPA

- Activate stroke team prior to CT scan
- Glucose is only required lab if no clinical suspicion of thrombocytopenia (platelets), occult bleeding (hemoglobin), or coagulation abnormalities (INR, PTT)
- Store tPA in ED pixus for rapid access
- Mix tPA early in decision-making process
- Standardized communication tools for discussion of risks and benefits with patient and family

bolus over 1–2 minutes, followed by a 60-minute infusion of the remainder. Valuable time can be saved if tPA can be kept in the ED.

Post-tPA management

- Admit to an intensive care unit or a stroke unit
- Perform neurological assessments and BP checks every 15 minutes for the first 2 hours, every 30 minutes for the next 6 hours, every hour for the next 16 hours, and every 4 hours thereafter.
- If the systolic BP (SBP) is greater than 180 or diastolic BP (DBP) is greater than 105, recheck within 5 minutes. If it remains elevated, emergently treat with antihypertensive medications (Chapter 8). The target BP is 180/105 after tPA administration.
- Evaluate for angioedema every 20 minutes starting with infusion.
- Provide maintenance rate of normal saline intravenous fluid (with *no dextrose*).
- Keep the patient's head of bed flat if tolerated.
- No anticoagulants or antiplatelet agents for 24 hours.
- Maintain nothing by mouth (NPO) until swallowing has been adequately assessed.
- If the patient develops severe headache, acute hypertension, nausea, vomiting, drowsiness, or worsening of the neurological exam, discontinue the infusion (if agent is still being administered) and obtain a CT scan of brain on an emergent basis.
- Maintain glucose less than 180.
- Provide mechanical deep venous thrombosis (DVT) prophylaxis.
- Order a head CT for 24 ± 6 hours after the tPA was administered to rule out ICH.

Management of complications

Symptomatic intracranial hemorrhage after IV tPA management. The overall incidence of symptomatic ICH after IV tPA is 6% based on the pivotal NINDS trial, and subsequent trials have replicated this rate.^{1,3–5} Key risk factors include older age, increased stroke severity, and higher baseline glucose, although the benefits of treatment outweigh risks in all these subgroups of patients. Approximately half of all patients who have this complication will develop severe disability or die.

ICH management

- Discontinue tPA infusion if still running.
- Stat CT head scan.
- Stat lab for type and cross, prothrombin time, PTT, platelet count, and fibrinogen level.
- If ICH on CT scan, give 6 units of platelets and either 6–8 units of fresh frozen plasma or 6–8 of cryoprecipitate containing factor VIII.
- Consult neurosurgery for consideration of hematoma evacuation.

Angioedema¹⁰:

- Incidence: estimated 1–2% of all tPA-treated stroke⁸
- Common in patients taking angiotensin-converting enzyme (ACE) inhibitors
- Usually starts near end of tPA infusion
- No standard guidelines available for management

UC stroke team angioedema protocol

- a. Begin examining tongue 20 minutes before IV tPA infusion is completed and repeat several times until 20 minutes after tPA infusion. Look for any signs of unilateral or bilateral tongue enlargement.
- b. If angioedema is suspected, immediately
 - 1. Consider early discontinuation of tPA infusion
 - 2. Benadryl 50 mg IV
 - 3. Ranitidine 50 mg IV or famotidine 20 mg IV
- c. If tongue continues to enlarge after items a and b, give solumedrol 80–100 mg IV.
- d. If any further increase in angioedema,
 - 1. Give epinephrine 0.1% 0.3 mL SC (subcutaneously) or by nebulizer 0.5 mL;
 - 2. Call ear-nose-throat (ENT) specialist/anesthesiology/or appropriate in-house service stat for possible emergent cricotomy/ tracheostomy or fiber-optic nasotracheal intubation if oral intubation is unsuccessful (Figure 6.2).

Endovascular therapy

Endovascular therapies offer the possibility of higher rates of restoring blood flow in an occluded artery with reasonable safety. Currently



Intubation strategies in the setting of angioedema

Figure 6.2 Intubation strategies in the setting of angioedema.

available modalities include IA tPA and mechanical devices. The Stryker MERCI retriever in 2004 was the first device cleared by the Food and Drug Administration (FDA), followed by the Penumbra aspiration system in 2006 and most recently the stent retrievers Covidien Solitaire and Stryker TREVO2 in 2012.¹¹⁻¹⁴ The stent retrievers were both shown to be superior for clot removal in randomized trials against the MERCI device. Still newer devices such as Johnson & Johnson REVIVE device and Penumbra 3D separator are currently in development. Endovascular therapy is discussed by indication in the following material.

IV tPA ineligible strokes

Patients who are not IV tPA candidates should be considered for endovascular therapy, particularly when arriving within 4.5 hours of onset, based on several sources of indirect evidence.

The randomized PROACT II trial has shown that patients may benefit from IA thrombolytic administration, using recombinant pro-urokinase (r-pro-UK), up to 6 hours from stroke.¹⁵ This lytic, r-pro-UK, was not FDA approved due to concerns regarding baseline group imbalances; consequently, r-pro-UK is not available commercially. Subsequently, a Japanese trial called MELT has also provided supportive evidence for this approach using a related lytic, urokinase (UK).¹⁶ Most recently, the Italian SYNTHESIS trial, which randomized patients to IV tPA alone versus endovascular therapy, showed comparable safety and efficacy with both treatment arms.¹⁷ Based on these data, it is reasonable to assume that a patient who arrives early to an ED and is not IV tPA eligible will benefit from endovascular therapy at experienced centers.

Eligibility criteria for endovascular therapy (based on current evidence) should typically include

- Age 85 years old or less
- NIHSS score 8 or more

Severe strokes treated with IV tPA

Given the high morbidity (>60% poor outcome) among moderate and severe ischemic strokes, adjunctive endovascular therapy (after initiation of treatment with IV tPA) became increasingly used, and a randomized trial comparing the combination of IV tPA with endovascular therapy to IV tPA alone was recently performed. This trial, the Interventional Management of Stroke (IMS) III, suggested safety but no superiority of the endovascular approach in unselected moderate and severe strokes (NIHSS score 8 or more).¹⁸ Studies are now under way to consider whether endovascular therapy may be more beneficial using the newer and more technically effective clot retrieval devices (stent retrievers or aspiration) and whether specific subgroups are more likely to benefit from adjunctive endovascular therapies. Subgroups under investigation include those with large-vessel occlusions (ICAT*, M1, basilar artery), longer arterial occlusions (occlusions ≥ 8 mm long), small core infarcts on CT perfusion or MR perfusion (see Chapter 4), earlier times to ED presentation and reperfusion, or the most severe strokes (NIHSS \geq 20).

As newer trials are awaited, the use of adjunctive endovascular therapy must be considered using clinical judgment and limited data; standard guidelines are lacking. Enrollment of these patients in ongoing clinical trials is strongly encouraged.

ICH after endovascular treatment

In addition to the steps taken to reverse tPA if administered (see previous discussion), it should also be noted that unfractionated heparin should be reversed if administered within the preceding 4 hours by giving 1 mg of protamine for every 100 U of heparin.

Future strategies under investigation

In addition to the studies of endovascular therapies mentioned, transcranial Doppler ultrasound to enhance the lytic activity of IV tPA is another promising avenue of investigation. Newer lytic agents such as tenecteplase (TNK), the combination of lytic agents with glycoprotein (GP) IIb/IIIa inhibitors such as eptibatide and argatroban, are also under study.

* Internal Carotid Artery Terminal occlusion.

Identifying patients who may have reversible ischemia beyond the 4.5-hour time window of tPA remains an area of active interest. It is hoped that multimodal imaging modalities, such as CT- and MR-based perfusion, may identify patients eligible for reperfusion therapies based on physiological (rather than clock) time in the future.

Further, despite many disappointing trials, neuroprotection remains an area of interest. If brain tissue could be made more resistant to injury, then there might be a longer window of opportunity for revascularization and decreased functional deficit. Investigations of treatments such as hypothermia and prehospital administration of magnesium are ongoing.

It is likely that new pharmacologic agents and devices for revascularization as well as effective neuroprotective strategies will only be successful in making an impact on stroke morbidity and mortality if an infrastructure for very rapid transport and treatment is in place.

Intracerebral hemorrhage

Except for removal of rapidly expanding life-threatening hematomas such as in the cerebellum, there is currently no effective acute treatment for spontaneous ICH. It is well documented that ICH expands in the first 24 hours.¹⁹ If that expansion could be limited or eliminated, the morbidity from ICH could be substantially reduced, and studies are under way to attempt this. This will also be time-dependent therapy, with administration of the drug within 3–4 hours of symptom onset. Success will be dependent on the same organization of stroke care required for acute ischemic stroke treatment.

For ICH in the setting of warfarin, it is imperative to begin reversal of anticoagulation quickly. Considerations include fresh frozen plasma (FFP) (determine dosing based on INR using an online calculator; note limited by large volume of 2 L typically), four-factor prothrombin complex concentrate (PCCs), or three-factor PCCs with FFP. PCC use offers the possibility of more rapid use but is less well established than PCCs. In addition, intravenous vitamin K (10–20 mg) should be administered. While it takes 12–24 hours to take effect, it will maintain the INR correction after PCCs or FFP effects are gone.²⁰

ICH in the setting of thrombocytopenia warrants rapid platelet transfusion.

ICH in the setting of the novel anticoagulants (such as dabigatran, apixiaban, and rivaroxaban) has no established therapy. Consider PCCs, activated charcoal, or hemodialysis, depending on the agent and rapidly changing evidence.

BOX 6.2 RECOMMENDED READING

The following clinical guidelines for intracerebral hemorrhage, aneurysmal subarachnoid hemorrhage, and acute ischemic stroke are useful references:

- "Guidelines for the Management of Spontaneous Intracerebral Hemorrhage: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association"²⁰
- "Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Statement for Healthcare Professionals from a Special Writing Group of the Stroke Council, American Heart Association"²¹
- "Guidelines for the Early Management of Patients with Acute Ischemic Stroke: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association"²²

Chapter 7 addresses general management of ICH.

Subarachnoid hemorrhage

Definitive treatment of the aneurysm underlying SAH should generally be performed early.¹⁸

Depending on your stroke center's capabilities, this may require emergent transfer to a referral center.

Neurosurgical clipping and endovascular coiling are both options for securing a ruptured aneurysm.²¹ In many cases, the two methods are complementary, with one being better than the other for an individual circumstance. Physicians with a multidisciplinary perspective should carefully consider both treatments. Considerations include

- The patient's age and medical status
- Surgical accessibility of the aneurysm
- Vascular anatomy
- Aneurysmal and parent vessel morphology
- Characteristics of the hemorrhage

When patients are equally good candidates for either intervention, endovascular coiling is often the preferred approach.

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Neurocritical care management of acute stroke

Introduction

The primary goal of clinical management in neurocritical care is to prevent/minimize secondary brain injury after an acute neurological event. In broad terms, this secondary injury may result from the natural history or progression of the primary event, hypoxia or hypotension resulting in brain ischemia, and cerebral edema, hydrocephalus, or intracranial hypertension. Given the topics that have been covered in the chapters on issues in acute management and preventions of complications with regard to airway and blood pressure management and treatment of infections and other hospital complications, we focus this chapter on an overview of critical care management of cerebral edema, hydrocephalus, and intracranial hypertension in acute ischemic stroke (AIS), intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH). Further, we provide a brief review of the different roles and positions required for neurocritical care for comprehensive stroke center (CSC) certification by the Joint Commission (JC) and Det Norske Veritas (DNV).

Acute ischemic stroke

Cerebral edema after ischemic stroke progresses slowly in the first few days and reaches its peak in approximately 3–5 days.¹ Medical management of cerebral edema may include hyperosmolar agents such as hypertonic saline or mannitol. However, these therapies remain unproven. The term *malignant infarction* is used to describe severe cerebral edema that results after proximal occlusion of the middle cerebral artery (MCA).² In the context of malignant MCA infarction, decompressive hemicraniectomy within 48 hours of symptom onset has been shown to improve outcomes and reduce mortality in carefully selected patients.³ Thus, early neurosurgical involvement is warranted for patients who may be candidates for hemicraniectomy. Patients with cerebellar strokes may develop life-threatening edema, resulting in acute hydrocephalus and intracranial hypertension. Placement of a ventricular drain or emergent posterior fossa decompression is recommended in this setting.¹



Figure 7.1 An approach to management of elevated intracranial pressure (ICP). cerebral perfusion pressure (CPP), cerebral perfusion pressure; and cerebrospinal fluid (CSF).

Intracerebral hemorrhage

Depending on the location, size, and intraventricular involvement after ICH, acute hydrocephalus, intracranial hypertension, and cerebral edema may all contribute to rapid and catastrophic neurological deterioration. Patients with small hemorrhages and minimal intraventricular hemorrhage (IVH) often do not require placement of an intraventricular drain or monitoring of intracranial pressure (ICP). However, ICP monitoring and ventricular drainage are recommended for patients with decreased level of consciousness (Glasgow Coma Scale ≤ 8) and acute hydrocephalus, with a goal of cerebral perfusion pressures of 50–70 mmHg.⁴ A possible algorithm for evaluation and management of elevated ICP is presented in Figure 7.1.

Subarachnoid hemorrhage

Prior to securing the causative aneurysm, medical management of SAH is aimed at preventing rebleeding. To this end, blood pressure control is strongly recommended, and administration of antifibrinolytic agents may be considered.⁵ Acute hydrocephalus may occur in up to 30% of patients with SAH,⁶ and ventriculostomy may be beneficial in patients with decreased level of consciousness or ventriculomegaly secondary to

acute SAH with hydrocephalus.⁵ Management of elevated ICP after SAH may also follow the algorithm in Figure 7.1. After securing the aneurysm, prevention of secondary injury after SAH also entails close monitoring for and aggressive management of delayed cerebral ischemia. Oral nimodipine is a class I recommendation for improving outcomes after SAH. Induction of hypertension may also be considered, and in the case of evidence of vasospasm, angioplasty or vasodilators may be considered, although these have not been shown to improve outcomes.⁵

Neurocritical care and comprehensive stroke center certification by Joint Commission

The eligibility criteria for CSC certification by the JC (www.jointcommission. org) issued in September 2012 specify a need for "dedicated neurointensive care unit (ICU) beds for complex stroke patients that include staff and licensed independent practitioners with the expertise and experience to provide neuro-critical care 24 hours a day, 7 days a week." At the individual hospital level, many questions arise regarding what is considered adequate neuro ICU coverage:

- 1. Does there need to be dedicated space for a neuro ICU?
- 2. Do nurses in the neuro ICU require specialty training?
- 3. Does there need to be 24/7 attending critical care physician availability?
- 4. Do the critical care physicians require special certification?

We address each of these questions in turn.

Dedicated space for a neuro ICU

As discussed, the language in the CSC handbook specifies the need for "dedicated neuro-ICU beds." We believe this entails a defined space with contiguous beds dedicated to the care of complex stroke cases. No mention is made in the handbook of other complex neurological/neurosurgical diseases that would typically be cared for alongside stroke patients as is done in most neuro ICUs. Thus, it is conceivable that the requisite neuro-ICU beds may in fact reside within a larger mixed-patient ICU facility. While we believe that complex stroke cases are best cared for in a dedicated neuro ICU space covered by a full-time neurointensivist, we acknowledge that access to dedicated neuro ICUs is limited in the United States.⁷ Further, creating adequate space to accommodate a large neuro ICU with a variety of complex neurological/neurosurgical diseases may not be feasible for all potential CSCs. Hospital systems that otherwise provide comprehensive stroke care but without a large nonstroke neuro ICU may well qualify for CSC designation based on our interpretation of the 2012 CSC handbook.

Specialty training for nurses

No specific certification is mentioned regarding nurses in the neuro ICU at a CSC. However, the skill set outlined for neuro ICU nurses almost certainly requires certified neuroscience registered nurse (CNRN) training. According to the CSC handbook:

> RNs that staff the intensive care unit (ICU) that contains dedicated neuro-ICU beds for complex stroke patients demonstrate expertise in:

- Neurologic and cardiovascular assessment
- Nursing assessment and management of ventriculostomy devices (external ventricular pressure monitoring and drainage)
- Treatment of intracranial pressure
- Nursing care of hemorrhagic stroke patients (intracerebral hemorrhage and subarachnoid hemorrhage)
- Nursing care of patients receiving intravenous thrombolytic therapy and intra-arterial thrombolytic therapy
- Management of malignant ischemic stroke with craniectomy
- Use of therapeutic hypothermia protocols
- Use of intravenous vasopressor, antihypertensive, and positive inotropic agents
- Methods for systemic and intracranial hemodynamic monitoring
- Methods for invasive and noninvasive ventilatory management

These advanced nursing skills are no doubt required for the optimal care of complex stroke patients at a CSC. Nurses with CNRN certification who work in a dedicated neuro ICU on a full-time basis and maintain the necessary continuous education would best fulfill these requirements.

Physician staff

It has been recognized for over 10 years that "high-intensity" intensivist staffing or mandatory involvement of an intensivist in the care of ICU patients is associated with lower hospital mortality and reduced hospital length of stay.⁸ Specifically, neurointensivist-led team models have been

associated with lower mortality, reduced length of stay, and increased likelihood of discharge to home in neuro ICU patients.^{9,10} Thus, physicians with critical care and cerebrovascular experience are required to staff the dedicated neuro ICU in a CSC.

It is important to note that 24/7 *in-house* attending physician staffing of the neuro ICU is not required either by the JC or by safety and quality groups such as the Leapfrog Group. The ICU Physician Staffing (IPS) Safety Standard issued by the Leapfrog Group states:

Hospitals fulfilling the IPS Standard will operate adult or pediatric general medical and/or surgical ICUs and neuro ICUs that are managed or comanaged by intensivists who:

- 1. Are present during daytime hours and provide clinical care exclusively in the ICU and,
- 2. When not present on site or via telemedicine, returns pages at least 95% of the time, (i) within five minutes and (ii) arranges for a FCCScertified physician or physician extender to reach ICU patients within five minutes.

A recent study of 65,752 patients admitted to 49 ICUs in 25 hospitals found that nighttime intensivist staffing did not confer any mortality benefit in hospitals with high-intensity daytime staffing, although it was associated with a reduction in mortality in hospitals with low-intensity daytime staffing.¹¹ Overall, for CSC certification, neurointensivists should be available 24/7 either in person or via adequate daytime staffing plus nighttime coverage by attending physician extenders. These may include acute care nurse practitioners, physician assistants, residents, or fellows.

Critical care physician certification

A CSC is required to have "physicians with critical care and cerebrovascular experience staff the intensive care unit (ICU) that contains the dedicated neuro-ICU beds for complex stroke patients." No mention is made of specific certification, although examples of physicians who have completed neurocritical care and vascular neurology fellowships are used to describe physicians who may be qualified to fill these roles. As such, while the United Council for Neurologic Subspecialties (UCNS) certifies intensivists with neurocritical care expertise, a UCNS-certified intensivist is not currently required for a CSC, although extensive experience caring for complex stroke patients is required.

Neurocritical care and comprehensive stroke center certification by DNV

Eligibility criteria for CSC certification by DNV were issued in September 2012 as well.

DNV requires that the stroke team include physicians with critical care expertise. These physicians managing neurocritical care patients should be

- 1. Accredited critical care fellowship or neurocritical care fellowship trained;
- 2. Care for a minimum of 20 acute stroke patients annually; and
- 3. Attend greater than or equal to 4 hours per year of continuing medical education (CME) activities (or similar educational programs) related to or focused on cerebrovascular disease.

As with JC CSCs, ICUs at DNV-accredited CSCs need not be dedicated neuroscience ICUs, but they need to be able to support ventilated patients, peripheral and pulmonary artery catheters, ventriculostomies, and pressors.

DNV also specifies that the director of the ICU should undertake at least 8 hours CME or equivalent education related to cerebrovascular disease each year, and that attending or resident coverage be available 24/7 with a written schedule.

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Issues in acute management

Introduction

All of the physiologic parameters discussed in this chapter are important in the management of acute stroke. Evidence regarding optimal management will continue to evolve. A standardized approach using a care path and standing orders is the best way to make sure that all patients receive the best care. Each year, the most current evidence should be reviewed and clinical guidelines and protocols updated.

ICU care

Comprehensive stroke centers must have a neurocritical care unit staffed by nurses who are highly trained to care for patients with neurological and neurosurgical conditions. Patients who have received intravenous (IV) thrombolysis or endovascular recanalization and those with aneurysmal subarachnoid hemorrhage (aSAH) or intracerebral hemorrhage (ICH) should be admitted to the neurocritical care intensive care unit (ICU). Other patients who should be admitted to the ICU include patients with large cortical infarcts who are at risk for cerebral edema, patients with significant comorbidities, and those patients who need blood pressure (BP) augmentation to achieve optimal cerebral perfusion pressure. Patients that are less critical and can be monitored less frequently may be admitted to a progressive step-down unit. Examples of these patients include those with nonruptured aneurysms, mild strokes after intravenous thrombolysis, and those requiring insulin infusion monitoring. The ratio of patients to nurse on those units should be no more than 3:1.

Vital signs

In patients treated with intravenous thrombolysis, BP monitoring and neurological assessment should be performed every 15 minutes for 2 hours after the start of the tissue plasminogen activator (tPA) infusion, followed by every 30 minutes for 6 hours, and then hourly up to 24 hours following the administration of IV tPA.¹ The stroke center should

use the National Institutes of Health Stroke Scale (NIHSS) for the initial neurological assessment. An abbreviated assessment tool can be used for the subsequent examinations (see Appendix), but a full NIHSS assessment should be done if there is any neurological deterioration.

Blood pressure management

Blood pressure management is perhaps the most important of all the parameters. Target BP will be different depending on the type of stroke and as evidence continues to evolve. This is an area where the stroke team needs to review the literature regularly so that targets can be revised based on new information. The goal is to maintain maximum cerebral perfusion while minimizing the risk of hemorrhage or extension of hemorrhage. In complicated cases where there is hypertensive encephalopathy, acute myocardial infarction (MI), aortic dissection, acute renal failure, or hemorrhagic transformation of ischemic stroke, target BPs will have to be lower than the following recommendations.

For management in ischemic stroke patients treated with thrombolysis and catheter-based reperfusion therapy,

- BP must be less than 185/110 prior to treatment with IV tPA and be maintained at less than 180/105 for 24 hours after IV tPA therapy. Protocols and agents for managing hypertension are summarized in Box 8.1.
- BP management after catheter-based treatment will vary depending on whether recanalization was achieved but typically follows the same parameters as for those individuals treated with IV recombinant tissue plasminogen activator (rtPA). These guidelines are evolving, and at present, the target BP may be influenced on a caseby-case basis by the degree of recanalization, assessment of collateral circulation, and the patient's clinical status. The goal is to optimize reperfusion and avoid hemorrhagic transformation.

Management in ischemic stroke patients not treated with thrombolysis or reperfusion therapy is as follows:

• In the first 24 hours after acute stroke, hypertension should not be treated unless SBP is greater than 220, DBP is greater than 120 on two consecutive readings at least 5 minutes apart. Treatment options can be found in Box 8.1. It is reasonable to lower BP 15% during the first 24 hours after stroke.¹

BOX 8.1 OPTIONS FOR MANAGEMENT OF HYPERTENSION

The following are options for the management of hypertension¹:

- Labetalol 10–20 mg IV over 1–2 minutes; may repeat once. Cautions: asthma, chronic obstructive pulmonary disease (COPD), left ventricular (LV) failure, second- or third-degree heart block, heart rate less than 50.
- Nicardipine drip 5 mg/hour IV infusion. Increase by 2.5 mg/ hour every 5 minutes to a maximum of 15 mg/hour to achieve target BP.
- Other agents (hydralazine, enalaprilat, etc.) may be considered when appropriate
 - Hydralazine 10–20 mg IV every 4–6 hours. Cautions: ischemic heart disease, aortic dissection, mitral valve disease.
 - Enalaprilat 0.625–1.2 mg IV every 6 hours. Cautions: acute MI, history of angioedema, renal insufficiency.
- If systolic BP (SBP) greater than 180–230 mm Hg or diastolic BP (DBP) greater than 105–120 mm Hg:
 - Labetalol 10 mg IV followed by continuous intravenous infusion 2–8 mg/min, or
 - Nicardipine IV 5 mg/hour, titrate up to desired effect by 2.5 mg/hour every 5–15 minutes, maximum 15 mg/hour
- Intravenous sodium nitroprusside should only be considered if DBP is greater than 140 mm Hg.

For management of hypotension after acute ischemic stroke (AIS):

 It is just as important to treat low BP to maintain perfusion pressure. When clinical instability appears to be due to hypotension, a target mean arterial pressure (MAP) of 120–130 can be achieved with fluid boluses first. If not effective, consider titrating a neosynephrine drip at 0.5–3 µg/kg/min. This should be used with caution in patients with congestive heart failure, coronary artery disease, or renal insufficiency. Pressors should not be initiated if the patient is neurologically stable despite relative hypotension as evidence is lacking for the benefit of this approach.

Blood pressure management in intracerebral hemorrhage

Blood pressure is frequently higher in an acute ICH than an ischemic stroke. Theoretically, it has been thought that hypertension could lead to expansion of the hemorrhage. Currently, studies have not clearly demonstrated that hypertension leads to hematoma expansion. The American Stroke Association Guidelines recommend the following BP management in an acute ICH; all of these management protocols have limited (i.e., class C) evidence:

- If SBP is greater than 200 mm Hg or MAP is greater than 150 mm Hg, then consider aggressive reduction of BP with continuous intravenous infusion, with frequent BP monitoring every 5 minutes.
- If SBP is greater than 180 mm Hg or MAP is greater than 130 mm Hg and there is the possibility of elevated intracranial pressure (ICP), then consider monitoring ICP and reducing BP using intermittent or continuous intravenous medications while maintaining a cerebral perfusion pressure greater than 60 mm Hg.
- If SBP is greater than 180 mm Hg or MAP is greater than 130 mm Hg and there is no evidence of elevated ICP, then consider a modest reduction of BP (e.g., MAP of 110 mm Hg or target BP of 160/90 mm Hg) using intermittent or continuous intravenous medications to control BP and clinically reexamine the patient every 15 minutes.²

The Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial (INTERACT) pilot study, published in 2008, and the ongoing Antihypertensive Treatment in Acute Cerebral Hemorrhage (ATACH) trial have shown that it is relatively safe to acutely lower SBP to 140 mm Hg in patients presenting with SBP elevated between 150 and 220 mm Hg.^{3–5}

Blood pressure management in subarachnoid hemorrhage

BP management after subarachnoid hemorrhage (SAH) is complex because of the need to balance the risk of hypertension and risk of rebleeding with the need to maintain cerebral perfusion pressure and decrease the risk of stroke. Guidelines recommend that, before repair of the aneurysm, SBP should be less than 160 mm Hg. In the presence of vasospasm leading to delayed cerebral ischemia, it is appropriate to induce hypertension unless BP is already elevated at baseline or contraindicated due to cardiac status.⁶

Glucose control

Hyperglycemia

Blood glucose (BG) is elevated in 40% of patients with AIS, and this can be related to a medical history of diabetes, a nonfasting state, or impaired

glucose metabolism due to a stress reaction related to the stroke.^{7,8} Strong evidence now shows that a high glucose level is an independent predictor of increased infarct volume defined by magnetic resonance imaging (MRI), worse clinical outcomes, and higher risk of mortality.⁹ In addition, the presence of hyperglycemia in patients with AIS treated with IV rtPA was associated with increased incidence of symptomatic ICH and worse clinical outcomes.^{10,11}

This evidence led many stroke centers to initiate an intensive insulin therapy protocol that was modeled after those used in ICUs. However, after the Glucose-Insulin-Stroke Trial–UK (GIST-UK) failed to demonstrate any benefit of intensive insulin therapy in 933 patients with stroke,¹² most centers have reverted to the practice of lowering the BG to 140–180. This avoids the risk of inducing hypoglycemia and the placement of patients in the ICU for frequent glucose monitoring. Further studies addressing this issue are under way.

Fewer clinical studies have studied the impact of hyperglycemia on ICH strokes. Recent evidence has shown that patients with ICH who have elevated BG on admission have increased risk of mortality, and the presence of hyperglycemia is negatively correlated with outcome measures, including hematoma size, presence and severity of intraventricular extension, stroke severity, and discharge functional outcomes.^{13–15}

Patients with aSAH often have hyperglycemia on admission. The pathology of the aSAH leads to activation of the sympathetic autonomic nervous system and activation of the hypothalamic-pituitary-adrenal axis, which can result in increased glucose production. Increased cortisol levels are associated with delayed cerebral ischemia after aSAH. In addition, there is a release of cytokines, which is linked to insulin resistance, altered glucose metabolism, and hyperglycemia.^{16–18}

There are limited data addressing the benefits of glucose management after aSAH. Hyperglycemia is associated with increased incidence of vasospasm. A study to maintain tight glucose control (80–110 mg/dL) found that episodes of hypoglycemia increased vasospasm and lessfavorable clinical outcomes at 3 months.^{19,20} Current guidelines state that careful glucose management with strict avoidance of hypoglycemia may be considered as part of the general critical care management of patients with aSAH.⁶

Further clinical trials are needed to investigate optimal glucose management in all acute stroke patients. Evidence shows that hyperglycemia is associated with poorer clinical outcomes, but there is a need to establish efficacy and risk/benefit data supporting a specific target glucose level. Evidence shows it is reasonable to treat acute stroke (ischemic, intracerebral, and subarachnoid) according to the American Diabetes Association recommendations to initiate therapy to achieve glucose targets of 140–180 mg/dL in all hospitalized patients.²¹ Patients with ischemic stroke who are treated with thrombolytic therapy should be started on a standardized intravenous insulin protocol for at least the first 24 to 48 hours because of the evidence showing these patients are at high risk of intracerebral hemorrhagic transformation with elevated glucose. There are multiple subcutaneous and intravenous insulin regimens that include rapid-acting insulin for glucose greater than 180 mg/dL.^{22–24}

Management of glucose

- Normal saline instead of 5% dextrose-half-normal saline should be used for intravenous fluid replacement unless there is a medical contraindication.
- If the initial glucose level is greater than 180 mg/dL, then initiate an emergency department (ED) or ICU intravenous insulin glucose control protocol.
- If the initial glucose level is between 140 and 180, then initiate a basal bolus insulin correction protocol.
- Fasting hemoglobin A1C should be drawn the next day; if the HbA1C is greater than 6.5% in an unknown diabetic patient, formal diagnosis and diabetic education should be implemented for secondary stroke prevention.

Acute blood glucose management

ICU admission: Obtain stat blood glucose value on admission

- a. If BG remains less than 140 mg/dL and there is a history of diabetes, continue BG checks before meals and at bedtime or every 6 hours if the patient is to receive nothing by mouth or is receiving continuous enteral nutrition.
- b. If there is no history of diabetes and BG is less than 140, obtain BG every 4 hours four times. If BG remains less than 140 mg/dL, no further action is taken.
- c. If BG is greater than 140, initiate intravenous insulin glucose control orders (see Appendix).

Medical surgical admission: Obtain STAT BG value on admission

- a. If BG is 180 mg/dL or greater, initiate basal bolus insulin correction algorithm (see Appendix).
- b. If BG is 140–179 mg/dL, check BG every 4 hours four times; if BG value increases to greater than 180 mg/dL, initiate basal bolus insulin correction algorithm (Appendix).
- c. If there is a history of diabetes and BG is less than 140 mg/dL,

- Obtain BG every 6 hours (6 a.m., 12 noon, 6 p.m., 12 midnight if patient is receiving nothing by mouth or receiving continuous enteral nutrition).
- Obtain BG before meals and at bedtime if patient is eating.
- d. If there is no history of diabetes and BG is less than 140, obtain BG every 4 hours four times. If BG remains less than 140mg/dL, no further action is necessary.

Temperature control

Hyperthermia

Hyperthermia (>37.6°C) is seen in as many as one-third of all AIS cases within the first hours following stroke onset.²⁵ Patients with ICH have a higher incidence of fever after basal ganglia, lobar, and intraventricular hemorrhage, and this appears to be an independent predictor of a poorer outcome.^{26,27} Fever occurs in 41–72% of aSAH patients, and the most common predictors of elevated temperature are a poor Hunt and Hess grade and the presence of intraventricular blood.²⁸ In experimental models of cerebral ischemia, higher temperature has been associated with poor neurological outcomes, possibly related to increased metabolic needs, increased release of neurotransmitters, and increased production of free radicals.²⁹ Retrospective studies have shown that fever is independently associated with poor outcomes in stroke ^{30–34} Clinical evaluation should determine if hyperthermia is related to a secondary cause, such as pneumonia or urinary tract infection. In aSAH, fever appears to be more associated with systemic inflammatory reaction rather than infections.³⁵ The goal is to maintain normothermia and prevent hyperthermia with pharmacological and mechanical interventions.

Temperature greater than 38°C should be evaluated and treated with antipyretic agents (acetaminophen or ibuprofen). In the acute setting, delivery by mouth may not be desirable if there is dysphagia, and rectal suppository or nasogastric tube may be the preferred routes of administration in this setting. This can be a standing order in the acute stroke order set. To maintain normothermia, surface cooling devices can be used when antipyretics fail. It is important to monitor skin for injury. Aggressive temperature control with surface or endovascular cooling leads to shivering and increased metabolic needs, leading to a decrease in brain tissue oxygen.³⁶ Immediate measures should be initiated to reduce shivering, including counterwarming, meperidine, magnesium, propofol, and buspirone. Other complications related to aggressive cooling include hypotension, cardiac arrhythmias, pneumonia, and thrombocytopenia.

There is strong clinical evidence that mild-to-moderate induced hypothermia improves neurological outcomes in patients with cardiac arrest.³⁷ The utility of induced hypothermia for treatment of acute stroke patients has not been proven, and clinical trials are under way.³⁸

Oxygen saturation

There are limited data regarding the benefit of supplemental oxygen. A large controlled clinical trial in AIS found no statistical difference in 1-year mortality and neurological outcomes between patients receiving 3 L of oxygen via nasal cannula for 24 hours after stroke onset and those receiving no supplemental oxygen.³⁹ Recent emergency cardiovascular care guidelines for stroke and cardiac arrest patients recommend supplemental oxygen guided by pulse oximetry to maintain oxygen saturation of greater than 94%.⁴⁰ It is reasonable to use the least-invasive mode of supplemental oxygen.

Airway management

Endotracheal intubation and mechanical ventilation should be implemented if there is risk of airway compromise due to aspiration or in patients with severe strokes based on severity scores or changing level of consciousness. Early prevention of aspiration reduces the incidence of pneumonia. In addition, mechanical ventilation may be used as a temporizing measure to properly manage patients with elevated ICP or malignant cerebral edema.

Cardiac telemetry

Cardiac telemetry is indicated for all stroke victims for 24 hours to detect arrhythmias according to current evidence, but depending on patient history, a longer period of monitoring may be indicated; documentation should support this need to satisfy insurers.^{41,42} In patients with atrial fibrillation, telemetry should be used to ensure adequate heart rate control. Outpatient monitoring with Holter monitoring or subcutaneous implanted cardiac monitoring devices may be indicated in AIS patients with suspected paroxysmal arrhythmias or those with cryptogenic stroke, especially when hospitalization is short.⁴³

Evidence suggests the importance of performing a baseline electrocardiogram (EKG) and assessment of cardiac biomarkers on all patients with acute stroke. AIS patients are at higher risk for myocardial ischemia, congestive heart failure, and cardiac arrhythmias.^{44,45} SAH is associated with increased levels of circulating catecholamine and autonomic neural stimulation from the hypothalamus. The hypothalamic stimulation may lead only to changes in the EKG, whereas the circulating catecholamine may cause QT interval lengthening and actual myocardial damage. The prolonged QT interval has been associated with malignant ventricular arrhythmias, which can lead to hemodynamic instability.⁴⁶ Studies have shown that EKG abnormalities are more prevalent in AIS and SAH but have rarely been studied in ICH. A recent ICH study of only 31 patients showed that 81% had one or more EKG abnormalities, with QT interval prolongation being the most common. These patients showed insular cortex involvement and the presence of intraventricular blood and hydrocephalus on the initial computed tomographic (CT) head scan. A larger study would be needed to determine whether the EKG abnormalities are associated with poorer outcomes.⁴⁷

Head of the bed

Data suggest that the position of ischemic stroke patients in the hospital bed can influence cerebral perfusion pressure, middle cerebral artery mean flow velocity, and ICP. Prior studies either excluded patients who showed recanalization or did not categorize findings according to arterial recanalization.48,49 A recent study found that when the head of the bed was lowered to a horizontal position, there was an increase in mean flow velocity in patients with incompletely recanalized arteries. In cases of recanalization, patients had more stable blood flow velocities during orthostatic changes in head-of-bed positioning, whereas when recanalization was incomplete, blood flow velocities were sensitive to head-of-bed positional changes. The sample size was small in all studies, limiting generalizability.⁵⁰ The ideal head-of-bed position in the AIS patient is unknown, but data suggest supine positioning may offer advantages to cerebral perfusion. Clinicians must make a decision based on close monitoring of neurological status, hemodynamic stability, risk of increased ICP, risk of airway obstruction, and risk of aspiration.^{51,52} The head of the bed should be raised to improve venous drainage and lower ICP in patients with cerebral edema whether related to large cortical or cerebellar stroke with malignant edema or ICH leading to compression of brain or mass effect.53

ICP can also be lowered by raising the head of the bed, improving venous drainage. A side effect of this is that it could lower pressure of blood to the head, resulting in a reduced and possibly inadequate blood supply to the brain. Venous drainage may also be impeded by external factors such as hard collars to immobilize the neck in trauma patients, and this may also increase the ICP. Sandbags may be used to further limit neck movement.

Acute monitoring post-IV thrombolysis

Orolingual angioedema occurs in 1.3% to 5.1% of all patients treated with IV tPA for thrombolysis. The reaction has been observed approximately

45–90 minutes after the tPA infusion is started. The reaction leads to swelling of the tongue, lips, or oropharynx that typically is contralateral to the ischemic hemisphere. Risk of angioedema is more common in patients who are taking angiotension-converting enzyme inhibitor medications and is more often seen with infarctions occupying the frontal cortex and insular region. The treatment regimen includes intravenous ranitidine, diphenhydramine, and methylprednisone. In addition, patients may have to be intubated for airway protection.⁵⁴ The University of Cincinnati protocol is included in the Appendix.

Acute medical management

Antithrombotic therapy has demonstrated a small but statistically significant reduction in mortality and unfavorable outcomes when used within 48 hours of stroke. The Get With the Guidelines (GWTG) measure requires that antithrombotic therapy be started by the end of day 2. It appears the benefit of early antithrombotic therapy is to reduce the risk of early recurrent stroke. There is limited safety data on use of antithrombotic therapy within 24 hours of IV tPA administration. Current guidelines recommend no adjunctive therapy with antithrombotic or anticoagulant medication during the first 24 hours following IV tPA administration.¹ Antithrombotics for secondary stroke prevention are discussed further in Chapter 10.

Acute anticoagulation is not indicated for most ischemic stroke patients based on evidence that the risks outweigh benefits. There are some situations, however, in which the use of full-dose anticoagulation might be considered in the acute setting. The potential benefits would need to be weighed against the possible risk of secondary hemorrhage. In part, this risk depends on the size of the ischemic infarction.¹ These situations may include

- High risk for deep vein thrombosis (DVT)
- Identified potential cardiac source of embolus
- Extracranial carotid or vertebral dissection
- Symptomatic high-grade carotid stenosis awaiting emergency surgery

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Prevention of complications

Introduction

After the hyperacute treatment phase of stroke care, medical and nursing management focuses on prevention of complications that can occur with all types of stroke. These include aspiration pneumonia, pulmonary embolism (PE), deep vein thrombosis (DVT), urinary tract infection (UTI), malnutrition, dehydration, bowel and bladder dysfunction, joint abnormalities, seizures, and decubitus ulcers.¹ A study by Ingeman et al. enrolled 13,721 stroke patients between 2003 and 2009 and found that 25.2% of patients experienced one or more medical complication during hospitalization. Urinary tract infection (15.4%), pneumonia (9.0), and constipation (6.8%) were the most frequent complications in this population study.² Complications can hinder the progression of rehabilitation, have an impact on clinical outcomes, extend length of stay, and increase mortality.³ A geographically designated area to care for stroke patients with trained health-care providers ensures coordinated delivery of care and enhances communication among the team of caregivers. This is the optimal setting in which to prevent secondary complications.^{4,5}

Infections

Urinary tract infection and pneumonia are the most frequent complications of stroke and are a common cause of increased hospital stays and delayed rehabilitation. Infection should be suspected if the patient develops fever or a change in the level of consciousness (LOC).

Urinary tract infection

Urinary tract infection develops in approximately 16% of stroke patients and is the most common hospital-acquired infection. Urinary retention and incontinence occur in 29% to 58% of stroke patients. Bladder dysfunction is more frequent in patients with large cortical strokes. One study found that patients with hemorrhagic strokes had a higher incidence of incontinence, whereas patients with ischemic strokes had more urinary retention. Finally, cognition, aphasia, and a higher baseline National Institutes of Health Stroke Scale (NIHSS) score were independently associated with bladder dysfunction.^{6,7}

Catheter-associated UTIs (CAUTIs) are the most common cause of nosocomial blood infections and are a significant cause of increased costs of care for stroke cases. Indwelling bladder catheters are frequently inappropriately placed in every stroke patient as a routine order. Indwelling catheters should only be used in patients with decreased LOC or severe immobility or if strict urinary output monitoring is needed. Antisepticcoated and antibiotic-impregnated catheters may decrease the incidence of CAUTIs, but the evidence is limited to a few small studies. The best practice is to avoid the catheter placement or remove the catheter as soon as the patient is medically and neurologically stable to prevent iatrogenic infection. Intermittent catheterization or use of condom catheters for men has been shown to lessen the risk of UTI. Urinalysis cultures should be obtained to detect UTI in the presence of fever or change in LOC, and proper antibiotic treatment should be initiated. Many studies have looked at strategies to reduce inappropriate use of Foley catheters. These included face-to-face education of physicians and nurses in an emergency department on appropriate use of catheters. Computerized charting with decision points that prompt the need to either continue or discontinue the catheter are helpful, as are nurse-driven protocols that encourage early removal of the catheter. These strategies have resulted in a 65% reduction in catheter use and a decline in CAUTI by 81%.7,8

A bladder program should be implemented to retrain the bladder. Preventing the bladder from filling beyond 500 mL will stimulate normal physiological filling and bladder emptying. A bladder scanner must be used to assess postvoid residuals (PVRs) and determine the need for intermittent catheterization. Two or more PVRs of greater than 150 mL have been found to be an independent risk factor for UTI. Urinary incontinence is associated with poor prognosis, interferes with rehabilitation, and is the major factor in patients being discharged to nursing homes rather than to their home⁹ (Box 9.1).

Pneumonia

Pneumonia is associated with increased mortality or an unfavorable outcome in patients with ischemic and hemorrhagic stroke. Patients with infarctions of the brain stem, multiple strokes, major hemispheric lesions, or decreased LOC are at the greatest risk of dysphagia leading to aspiration. Intubated patients and those who are immobile also have a higher incidence of pneumonia.¹⁰ Indicators that alert the care team to the risk of dysphagia include an abnormal gag reflex, impaired voluntary cough, dysphonia, facial droop, and drooling. Studies have shown that nurses using an evidence-based screening tool can safely perform the initial screening and determine if it is safe to allow the patient to have liquids

BOX 9.1 BLADDER AND BOWEL TRAINING

The program should include

- 1. Provide bladder scanning and intermittent catheterization every 4–6 hours; continue intermittent catheterization as long as PVR is greater than 100 mL.
- 2. Initiate voiding strategies:
 - a. Offer a commode, bedpan, or urinal every 2 hours during waking hours and every 4 hours at night. Neurological deficits lead to frequent falls when patients try to ambulate to the bathroom.
 - b. Assist patients with toileting when impaired vision, mobility, and dexterity are present.
- 3. Provide communication boards to aphasic patients to provide quick requests.
- 4. Monitor fluid intake during the evening prior to bed.
- 5. Provide an environment conducive to having a bowel movement.
- 6. Evaluate for abdominal distension.
- 7. Evaluate for impaction every 2 days.
- 8. Integrate stool softeners, laxative, and enemas to prevent constipation.
- 9. Provide skin care to decrease incidence of skin breakdown, dermatitis, or perineal thrush.
- 10. Address psychosocial problems and decreased self-esteem associated with bowel and bladder dysfunction.

prior to the formal assessment by the speech language pathologist (SLP).¹¹ Nursing care strategies to prevent the development of pneumonia should be initiated immediately on admission of the patient with acute stroke. The presence of fever, the development of atelectasis, or a change in LOC should prompt further diagnostic evaluation and treatment. Pneumonia is associated with increased risk of short-term and long-term mortality. Studies have shown that one of three early deaths is related to pneumonia, and 1-year mortality doubles in patients with pneumonia poststroke compared to those without.²

The following are care strategies to decrease development of pneumonia:

• Nothing by mouth (NPO) until formal swallowing evaluation to assess risk of aspiration. If a speech therapist is not available, a bed-side swallowing test can be done by giving the patient 3 ounces

of water and observing for cough, dysphonia lasting 1 minute, respiratory difficulty, or drooling. If the swallowing test is failed, place an enteral feeding tube.

- Monitor airway and oxygenation and use mechanical ventilation if indicated. Hypoxia may be due to concurrent medical conditions, such as hypoventilation, atelectasis, aspiration, pneumonia, or PE.
- Minimize time on mechanical ventilation; provide frequent suctioning.
- Provide early mobilization.
- Maintain a good pulmonary toilet.
- Position the patient in semirecumbent position when feeding with a nasogastric (NG) tube or percutaneous endoscopic gastrostomy (PEG) tube.
- Consult a speech pathologist and dietician to establish a feeding plan and safety measures to decrease aspiration risk.
- Educate the patient and family regarding the risk of aspiration and the importance of following the established feeding plan and safety measures (Box 9.2).

BOX 9.2 DYSPHAGIA DETECTION AND ASPIRATION PREVENTION

Preprinted admission order sets include "swallow assessment before any oral intake" and provide further direction for those patients demonstrating a dysphagia risk.

- Maintain NPO status. No ice chips; no oral medications; no water; and no exceptions.
- SLP formal evaluation with appropriate diagnostic tool.
- Follow (SLP) recommended compensatory techniques, such as
 - Chin tuck maneuver
 - Head and neck positioning
 - Changed consistency of food (i.e., thickened liquids, pureed or semisolid foods)
 - Minimize distraction in environment when feeding
 - No use of straws
 - High Fowler's position; leave sitting up for 30 minutes
 - Place food on the unaffected side in cases of hemiparesis/ hemiplegia or hemianopia
 - Present small portions; provide time to chew and swallow
 - Evaluate for pocketing of food
 - Initiate mouth care to facilitate swallowing

Nutritional compromise

Malnutrition has been proven to interfere with recovery after a stroke. A swallow assessment should be done as soon as possible after admission to the hospital and no later than 48 hours after admission. If no dysphagia or aspiration risk is detected, oral feeding should be instituted immediately. Nutrition should be established within 48-72 hours. The Feed or Ordinary Diet (FOOD) clinical trial results showed that supplemental nutrition was not necessary in patients who could swallow, and that early feeding with a NG tube in patients with dysphagia substantially decreased the risk of pneumonia and death and resulted in better functional outcomes than when fed through the PEG. These data present a dilemma for those patients who require transfer to a long-term care facility as these institutions will not accept patients with an NG tube in place, and delay in performing the PEG procedure increases length of stay.¹⁰ Since dysphagia resolves completely in 87% of all stroke patients, the PEG may be removed at the long-term care facility. Serum albumin is a poor indicator of immediate nutritional state since it has a half-life of 18 days. The dietician should be consulted early for an evaluation of nutritional status and need.12,13

Constipation

Constipation was the third-most-identified complication in the study done by Ingeman² and was found to be more prevalent in patients with a higher NIHSS score. Patients who have to use the bedpan for defecation had a higher incidence of constipation. Constipation is one of the complications most forgotten by the health-care team. Early mobility and use of chair commodes instead of bedpans can provide a better environment to decrease incidence of constipation. It is important that the nursing team promote early mobility and encourage activity such as sitting in the chair for meals and utilizing chair commodes or bathrooms, which provide a better environment for the patient to have bowel movements² (Box 9.1).

Deep vein thrombosis and pulmonary embolism

There is no longer a need to stratify acute stroke cases for risk of development of DVT since all stroke patients are considered at high risk, and prophylaxis should be started on all patients. The incidence of DVT is 2.5%, and the incidence of PE is 1.2% in the first 3 months following a stroke.³ PE ranks as the third cause of death after a myocardial infarction or stroke.¹⁴ A PE arises from venous thrombi that develop in the deep veins of the leg or pelvis when there is immobility. In the stroke setting, this is often due to a paralyzed or paretic lower extremity. Early mobility can decrease the incidence of PE, atelectasis, pneumonia, and DVT. Patients should be mobilized as soon as they are hemodynamically stable. Early mobility is the best option for decreasing the risk of DVT. When mobility is not possible, antithrombotic agents and sequential compression devices (SCDs) are used to decrease the risk. A meta-analysis showed that antithrombotic medications such as heparin and low molecular weight heparin (LMWH) were more beneficial in decreasing the incidence of DVT in comparison to external compression devices alone. In the Prevail (Prevention of VTE after Acute Ischemic Stroke with LMWH) trial, a daily injection of 40 mg of enoxaparin was more effective than 5,000 IU of unfractionated heparin twice a day for prevention of DVT in patients with ischemic stroke. The study was only done in ischemic stroke, and bleeding complications were relatively low.¹⁵ Given the high incidence of DVTs in stroke patients, external compression devices are used in conjunction with antithrombotic medications at some centers.

Patients with hemorrhagic stroke also have high risk of developing DVT. The most recent ICH (intracerebral hemorrhage) guidelines recommend external compression devices combined with elastic stockings and initiation of low-dose subcutaneous LMWH or unfractionated heparin after demonstration of ICH stability, usually within 1 to 4 days from onset.¹⁶ It is unclear whether adding anticoagulation is safe, but two small randomized studies found no increase in bleeding when low-dose subcutaneous heparin was initiated at day 4 or at day 10 after an ICH. The incidence of DVT in patients with subarachnoid hemorrhage (SAH) can range from 1.5% to 18%; those with a higher Hunt and Hess score have a higher incidence. DVT prophylaxis generally consists of external compression devices until the aneurysm has been coiled or clipped. Prophylaxis of DVT in SAH cases with secured aneurysms typically includes SCDs and medical treatment, including unfractionated heparin.

The potential for skin damage is a concern when using SCDs in stroke patients, so close observation of the condition of the skin is recommended. Patients who have contraindications for antithrombotic treatment may need placement of an inferior vena cava filter device.^{16–19}

Musculoskeletal complications

Regarding musculoskeletal complications, the physical therapy or rehabilitation team should be consulted on admission to instruct the care team in how to perform passive and active range of motion and positioning techniques that can prevent joint contractures and atrophy.²⁰ Subluxation of the affected shoulder is common and may not be preventable. However, careful positioning and movement of the affected arm may prevent the development of a painful shoulder-hand syndrome. Special care should be taken to avoid pulling on the affected arm and shoulder when repositioning the patient in bed or when assisting with transfers.²¹

Falls

Falls are the most frequent cause of injury in stroke patients. Nurses must monitor the initial transfer from bed to upright position, and identifying orthostatic hypotension by checking lying and sitting blood pressure may help decrease the risk of falling during transfers. The most common injury is hip fracture. Hip fractures in the first 7 days poststroke are associated with a poor prognosis.²² Most fractures occur at the time of the fall and are on the paretic side.²³ Hemi-inattention or neglect syndrome, which is typically seen with right hemispheric infarcts, leads to a higher risk of falling. All of the health-care providers and the family should be made aware of the fall prevention plan.

Stroke team nurses must implement a fall prevention program that includes

- Identification of high-risk patients
- Alarm systems
- Adaptive equipment such as wedge seat cushions or enclosure beds
- Placement of call buttons and patient's belongings close to the patient
- Scheduled voiding times (many falls occur as the patient is trying to go to the bathroom)

Skin breakdown

Stroke patients are at risk for skin breakdown because of the loss of sensation and impaired circulation. Patients at risk for pressure ulcers are usually older, have a decreased LOC, and are unable to move themselves in bed due to paralysis. Other related complications, such as incontinence, can accelerate the development of skin breakdown. Pressure areas include heels, sacrum, and lateral malleoli. The following are strategies to prevent skin breakdown:

- Patients should be examined for pressure points and be massaged when turned.
- Patients should not be left in a position for longer than 2 hours.
- Patients' skin must be kept clean and dry.
- Special mattresses should be used to prevent the development of decubiti.
- Special care should be taken when repositioning, turning, or transferring patients to avoid excessive friction or excessive pressure that may lead to skin injury.

Seizures

Seizures are reported to occur in less than 10% of cases after ischemic infarcts. Seizures are often associated with large cortical or lobar strokes or in patients with hemorrhagic transformation. Seizures can occur in the acute phase of the stroke as well as later in the course. Stroke patients should only be given anticonvulsants if a seizure occurs. There is no indication for prophylactic therapy, but patients and families should be aware that seizures might occur after a stroke.²⁴

Hemorrhagic stroke cases are also a cause of clinical seizures. The incidence of seizures in ICH cases has been reported to be from 2.7% to 17% and as high as 25% in cases of SAH, for which the most common cause is a middle cerebral artery rupture. It is has been shown that if a seizure occurs at the onset of the SAH, it is a predictor of poorer clinical outcomes and increased mortality.

Anticonvulsants are recommended in patients who have a known seizure with onset of hemorrhagic stroke. If neurological deterioration occurs or a patient continues to have depressed mentation, it is advisable to monitor the patient for nonconvulsive seizures with continuous electroencephalogram. In cases of SAH, it is recommended that anticonvulsants other than phenytoin be given prophylactically for 3 to 7 days. The use of phenytoin after SAH has been associated with poorer comprehensive outcomes with prolonged treatment.^{16,25,26}

Discharge planning

The goal of discharge planning is to ensure safe transition from the acute setting to an appropriate setting where the patient can obtain optimal rehabilitation and further secondary stroke prevention. Continuing attention to the prevention of the complications discussed is essential for ensuring the best possible outcome. For patients with continued immobility, it is important to continue DVT, UTI, pneumonia, and skin breakdown prophylaxis and to continue a fall prevention program. Communication of the plan implemented in the acute care setting should be provided to the extended care or rehabilitation facility at the time of discharge.

According to the National Institute of Neurological Disorders and Stroke, 35% of stroke survivors will recover fully or only have minor impairments. However, 40% will require special care, and a skilled nursing facility will be necessary for 10%. A team involving the physicians, stroke team nurse, rehabilitation therapists, case manager, and social worker must begin looking at discharge needs immediately on admission. The average length of stay of 5.3 days, according to 2009 National Center for Health Statistics, does not provide the patient or family with sufficient time to understand the impact of the stroke. The discharge team must actively involve the family and assist them in making discharge decisions based on the patient's needs and family support systems. Discharge teams should strive to obtain optimal clinical outcomes by planning for rehabilitation and prevention of recurrent stroke and complications while being attentive to containing the financial burden for the family.

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Secondary prevention of stroke

Ischemic stroke (or transient ischemic attack)

After providing indicated acute treatments and management, the next goal of hospitalization is to determine the cause of the stroke and institute secondary prevention measures.

The risk of a recurrent ischemic event is substantial. Recurrent ischemic strokes occur in 5–18% of patients (depending on the definitions used) within 90 days of the first event.¹ There is also a significant risk of a recurrent ischemic event after transient ischemic attacks (TIAs) (see Chapter 5).² Therefore, it is imperative that a diagnostic workup be initiated during the hospitalization to identify treatable causes, and that appropriate secondary prevention strategies be offered promptly.

Causes of ischemic stroke (or TIA) can be classified as

- Cardioembolic (about 25%)
- Large-vessel atherosclerosis (about 20%)
- Small-vessel disease (about 20%)
- Other (including iatrogenic, sickle cell disease, hypercoagulable disorders, and extracranial artery dissection) (about 5%)
- Cryptogenic (30%)

An essential stroke workup consists of the following:

- Detailed medical history and family history
- Cardiac telemetry to screen for cardiac arrhythmias, particularly atrial fibrillation (AF).
- Expeditious evaluation of the extracranial (and intracranial arteries in some cases) vessels for significant stenosis, occlusion, or dissection
 - Magnetic resonance angiography (MRA), computerized tomographic angiography (CTA), and digital subtraction angiography are all capable of visualizing the extracranial and intracranial vessels.
 - T1 fat-saturated axial neck magnetic resonance imaging (MRI) is useful in the diagnosis of extracranial arterial dissection.
 - Carotid ultrasound may be used to evaluate the extracranial portion of the carotid arteries.
 - Transcranial Doppler may be used to assess intracranial vessels.

- Echocardiography with a bubble study to screen for a direct or paradoxical source of embolism
- Fasting lipid and glucose levels to screen for hyperlipidemia and diabetes
- Tests for hypercoagulable states if no obvious cause for stroke found in screening

Diagnostic considerations and management strategies for each of the common stroke etiologies are discussed further in this chapter. The best source for a detailed review of the literature and recommendations based on levels of evidence is the American Heart Association/American Stroke Association (AHA/ASA) "Guidelines for the Prevention of Stroke in Patients with Stroke or TIA," published in 2011 in *Stroke*.³

Large-artery atherosclerosis

Internal carotid artery disease

Patients with a stroke or TIA and symptoms consistent with anterior circulation ischemia should be screened for internal carotid artery (ICA) stenosis during the hospitalization. Without treatment, symptomatic patients with greater than 70% ICA stenosis carry a 26% risk of recurrent ipsilateral stroke over 2 years.⁴

Diagnosis

- Initial screening by carotid ultrasound, CTA, or MRA (see chapter on neuroimaging)
- If an intervention is being considered based on screening, a second confirmatory test should always be performed to confirm the degree of stenosis.
- A digital subtraction angiogram is the most sensitive test to determine degree of stenosis and has the added advantage of providing information regarding intracranial circulation. This is useful when the two noninvasive tests show conflicting results.

Management

Carotid artery stenting (CAS) is an alternative to carotid endarterectomy (CEA), the well-established effective treatment for symptomatic carotid stenosis. The CREST Trial was designed to compare the safety and efficacy of the two procedures. Although the overall results of the trial that randomized 2,502 symptomatic and asymptomatic patients with carotid stenosis of greater than 70% by ultrasound or 50% by angiography to CAS or CEA showed no difference in the composite primary outcome (30-day rate of stroke, death, and myocardial infarction [MI] and 4-year ipsilateral

stroke), there were other important differences in outcomes comparing the two procedures in symptomatic cases⁵:

- The 30-day rate of stroke was higher with CAS (5.5%) versus CEA (3.2%), p = 0.04.
- The 4-year rate of stroke or death was higher with CAS (8%) versus CEA (6.4%), p = 0.14.
- The 30-day rate of MI was higher in CEA (2.3%) versus CAS (1.0%), p = 0.08.
- At an age less than 70, CAS was more effective; at an age greater than 70, CEA was more effective (p = 0.02).
- If a 70–99% symptomatic ICA stenosis is identified, CEA is the established therapy.

Based on these and other trial results, the AHA/ASA guidelines recommend:

- In patients with stroke or TIA within the past 6 months and ipsilateral carotid artery stenosis of 70–99%, CEA is recommended (perioperative morbidity/mortality < 6%).
- In patients with symptomatic moderate carotid artery stenosis of 50–69%, CEA may be considered based on patient-specific factors: age, sex, comorbidities (perioperative morbidity and mortality < 6%).
- If the carotid stenosis is less than 50%, no procedure is recommended.
- CEA may be done within 2 weeks of the ischemic event if there are no contraindications.
- CAS is an alternative to CEA in patients with symptomatic 70–99% stenosis and low risk for complications associated with endovascular intervention.
- CAS may be considered in patients with symptomatic 70–99% stenosis in whom the stenosis is difficult to access surgically or in whom comorbid medical conditions increase the risk of surgery.
- CAS should be performed by operators with established periprocedural morbidity and mortality rates of 4–6%.
- For patients with symptomatic extracranial carotid occlusion, extracranial/intracranial (EC/IC) bypass surgery is not routinely recommended.

Extracranial vertebrobasilar disease

Diagnosis

- Vertebrobasilar stenosis can be visualized with CTA and MRA.
- Digital subtraction angiography provides the most accurate assessment of the degree of stenosis.

Management

- In patients with symptomatic vertebrobasilar stenosis, optimal medical therapy, including antiplatelet agents, statin therapy, and risk factor modification, is recommended. Anticoagulant treatment has not been shown to be more effective than antiplatelet agents in this setting.⁶
- Endovascular treatment may be considered in patients who are symptomatic despite optimal medical therapy.

Intracranial artery stenosis

Patients with symptomatic intracranial atherosclerotic stenosis are at high risk of subsequent stroke. In the WASID trial comparing warfarin to aspirin therapy, the overall risk of recurrent stroke in patients with intracranial stenosis was 15% in the first year, 12% in the territory of the stenosis. In the subgroup with stenosis greater than 70%, the 1-year risk of stroke in the territory of stenosis was 19%.⁶ There was optimism that an endovascular approach to these lesions might provide better outcomes.

SAMMPRIS: Stenting versus aggressive medical therapy for intracranial arterial stenosis

Stenting versus Aggressive Medical Therapy for Intracranial Arterial Stenosis (SAMMPRIS)⁷ was studied randomized 451 patients with TIA or stroke within 30 days who had 70–99% intracranial artery stenosis to aggressive medical management versus aggressive medical management with stenting. The trial was halted because the primary end points (30-day rates of stroke and death) were 14.7% in the stenting group versus 5.8% in the medical management group (p = 0.002). The 1-year primary end point occurrence rates were 20.0% in the stenting group and 12.2% in the medical management group. The lower stroke recurrence rates compared to prior studies, such as WASID, were attributed to the aggressive medical management regimen, and this approach has become increasingly common among clinicians.

Based on the SAMMPRIS trial, Food and Drug Administration (FDA) indications for the wingspan stent have been limited to patients who are between 22 and 80 years old *and* who meet *all* of the following criteria:

- Two or more strokes despite aggressive medical management;
- Most recent stroke beyond 7 days prior to planned stenting;
- Severe (70–99%) stenosis due to atherosclerosis of the intracranial artery related to the recurrent strokes; and
- Good recovery from previous stroke (modified Rankin score of 3 or less). (The Rankin scale is used to measure the degree of disability in stroke patients.)

The FDA specified that the wingspan stent system should not be used for patients with

- Early stroke (onset of symptoms within 7 days)
- TIA only

Diagnosis

- CTA and MRA visualize intracranial arterial stenosis.
- Digital subtraction angiography provides the most accurate assessment of the degree of stenosis, as well as data about the collateral circulation.

Management

- For symptomatic patients with intracranial stenosis of 50–99%, aspirin is recommended in preference to warfarin.
- Aggressive risk factor management with statins (goal low-density lipoprotein [LDL] < 70) and antihypertensives (goal systolic blood pressure [SBP] < 140); diabetes control (goal HbA1C < 7); and lifestyle modifications (tobacco cessation, exercise, weight loss) are recommended. There should be short-term combined aspirin and clopidogrel use (90 days) based on indirect evidence from the SAMMPRIS trial (see risk factor management discussion for more details).
- Intracranial stenting is only considered for those with recurrent strokes despite aggressive medical management.

Cardioembolism

Cardiogenic cerebral embolism is responsible for 20–25% of ischemic strokes. In approximately one-half of these, there is a history of AF, valvular heart disease is present in one-fourth, and left ventricular mural thrombus occurs in one-third.⁸

Diagnosis

- Patients admitted with stroke or TIA should be on cardiac telemetry for 48–72 hours.
- In patients for whom no cause for the stroke is determined during hospitalization, consideration of ambulatory longer-term cardiac monitoring can be considered to detect paroxysmal AF.
- All stroke patients should be screened for a possible cardioembolic etiology with echocardiography if this would lead to a change in management.
- Cardioembolism should be considered regardless of the presumed stroke subtype, including strokes with a lacunar appearance on imaging, since up to 25% of presumed small-vessel strokes are due to other etiologies.⁹

- If a patient is already in AF but not optimally treated, then effective long-term anticoagulation is indicated, and an echocardiogram may not be needed.
- Every patient with minimal or no risk factors should be screened for paradoxical embolus with transthoracic echocardiogram (TEE) and bubble study.

The decision to use a TTE versus a transesophageal echocardiogram (TEE) is controversial. TEE has been suggested as a cost-effective firstline diagnostic strategy by several groups.^{10,11} Others suggested that these studies did not account for increased length of hospital stay, the need for sedation, and patient discomfort that come with TEE use. A common practice is to go straight to TEE when the patient is young or without known cardiac disease or suspicion of a cardioembolic source is high (i.e., strokes in multiple vascular distributions or branch distal artery infarcts without intracranial/extracranial stenoses). In others, a TTE may be done first, followed by a TEE if there are cardiac abnormalities or suspicion of cardioembolism becomes high after receiving other study results.

Other cardiac conditions associated with stroke risk include

- Valvular heart disease/prosthetic valves
- Mural thrombus
- Heart failure with low ejection fraction
- Patent foramen ovale with atrial septal aneurysm
- Aortic arch atherosclerosis
- Infective endocarditis
- Marantic endocarditis
- Cardiac tumors

Management

Atrial fibrillation. Anticoagulation is indicated in most patients with AF.

- AF carries a 12% annual risk of recurrent stroke. Stroke recurrence rates are reduced to approximately 10% annually with aspirin therapy alone, compared to a stroke recurrence rate of 4% using anticoagulation with warfarin with a target international normalized ratio (INR) of 2.5 (range 2.0–3.0).¹²
- Warfarin alternatives: The three drugs profiled next are pharmacologically stable, have fewer drug-drug and food interactions than warfarin, and require no regular monitoring. A reliable easy-to-use reversal protocol needs to be developed.¹³
 - Dabigatran is a direct thrombin inhibitor approved for use at 150 mg BID (twice daily) in the United States by the FDA. The RE-LY Trial tested doses of 110 mg BID and 150 mg BID. Both

doses were noninferior to warfarin in preventing stroke and systemic embolism. The higher dose was superior to warfarin for preventing primary outcomes. Hemorrhagic strokes were lower than for warfarin at both dose levels. Rate of major bleeding was decreased with the lower dose and equal to warfarin at the higher dose.¹⁴ There is no way to reverse the anticoagulation with dabigatran, so patients on this drug are not candidates for intravenous tissue plasminogen activator (IV tPA).

- Rivaroxaban is a direct factor Xa inhibitor approved for use at 20 mg daily in the United States by the FDA. In the ROCKET AF Trial, patients on rivaroxaban had similar rates of strokes and major bleeding but less fatal and intracranial bleeding compared with those taking warfarin.¹⁵ Therapies to reverse the anticoagulant effect of Xa inhibitors are under investigation.
- Apixaban is a direct factor Xa inhibitor that is not currently approved for use in the United States. In the ARISTOTLE Trial, patients on apixaban at 5 mg BID had fewer overall strokes or systemic emboli and fewer major bleeding events. The apixaban group had similar ischemic stroke rates to the group on warfarin but fewer intracranial bleeds.¹⁶
- Anticoagulation initiation in patients with acute ischemic stroke is typically delayed by a few days to 2 weeks, using clinical judgment regarding the risk of hemorrhagic conversion based on the size of the infarct.
- For patients with AF at high risk for stroke who require temporary interruption of oral anticoagulation, bridging therapy with low molecular weight heparin administered subcutaneously is reasonable.³

Other causes of cardiogenic stroke are the following:

- Acute MI and left ventricular thrombus: Recommended treatment with warfarin with target INR = 2.5 (range 2.0–3.0).
- Prosthetic heart valves: Recommended treatment with warfarin with target INR = 3.0 (range 2.5–3.5). Aspirin at a daily dose of 75–100 mg may be added in cases of recurrent ischemic events on adequate warfarin therapy.
- Cardiomyopathy: In cases with left ventricular ejection fraction less than 35%, treatment with warfarin (target INR = 2.5), aspirin 81 mg daily, clopidogrel 75 mg daily, or combination aspirin 25 mg plus extended-release (ER) dipyridamole 200 mg BID may be considered to prevent recurrent cerebral ischemia.³
- Patent foramen ovale: Antiplatelet therapy is reasonable in the absence of evidence demonstrating the superiority of anticoagulation or mechanical closure.¹⁷

Arterial dissections

Among young patients with stroke, arterial dissections are relatively common. The stroke symptoms may be caused by thromboembolism from the site of the dissection or hemodynamic compromise in the artery.

Diagnosis

- MRA with fat saturation protocols or CTA may visualize the dissection.
- Digital subtraction angiography is accurate in making the diagnosis.

Management

- Antithrombotic treatment for 3–6 months is reasonable. The relative efficacy of antiplatelet therapy compared to anticoagulant therapy is not known.
- If patients fail antithrombotic treatment and have recurrent ischemic symptoms, endovascular treatment with stenting may be considered.³

Severe ascending aortic arch atheroma

Severe ascending aortic arch atheroma (≥4 mm, ulcerated, or mobile) is also a cardiac risk factor. Optimal management of this condition (i.e., antiplatelet therapy vs. short-term anticoagulants) is unknown.

Uncommon cardiac conditions

Uncommon conditions may warrant specific treatments, such as anticoagulation (marantic endocarditis), intensive antibiotic therapy (infective endocarditis), and resection (intracardiac tumors).

Other causes of ischemic stroke

Less-common causes of stroke, listed in order of prevalence, are the following:

- Iatrogenic (i.e., postsurgical or after cardiac angiography/percutaneous transluminal coronary angioplasty [PTCA])
- Hypercoagulable states (including sickle cell disease)
- Cocaine or narcotic use
- Hypotension
- Cancer
- Venous sinus thrombosis
- Temporal arteritis.
- Small-vessel ischemic disease and cryptogenic strokes

After the diagnostic workup, many patients have no specific etiology identified.¹⁸ If the patient has significant vascular risk factors and a small

infarct consistent with a lacuna (i.e., <2 cm), the etiology is often presumed to be small-vessel ischemic disease. When this is not the case, the stroke is classified as "cryptogenic." In either case, as with all stroke etiologies, modifiable vascular risk factors should be addressed, and education about these risk factors and treatments should be made available to the patient and family.

Modifiable cardiovascular risk factors

Aggressive treatment of modifiable risk factors is essential for all stroke subtypes. Risk factors with evidence-based treatment recommendations are discussed next.³

Hypertension

Hypertension is the most important modifiable risk factor for stroke. Approximately one-third of Americans have hypertension, defined as blood pressure (BP) greater than 140/90, with an even higher rate among African Americans. Less than one-half of those diagnosed with hypertension have achieved adequate control. Both systolic and diastolic hypertension are associated with increased risk of stroke, and lowering BP can decrease stroke risk by 30–40%. Management of hypertension in the acute setting must be distinguished from longer-term management goals.

Acute setting

During the acute stroke phase, mean arterial pressures (MAPs) may have a direct impact on perfusion to ischemic and oligemic brain tissue due to loss of autoregulation of the cerebral vasculature. BP is often liberalized, although randomized clinical trial data are lacking. Specifically,

- If tPA therapy was administered, maintaining BP below 180/105 for the first 24 hours after treatment is critical.
- In patients with markedly elevated BP who do not receive fibrinolysis, a reasonable goal is to lower BP by 15% during the first 24 hours after onset of stroke. The level of BP that would mandate such treatment is not known, but consensus exists that medications should be withheld unless the SBP is greater than 220 mm Hg or the diastolic BP is greater than 120 mm Hg.¹⁹
- Preadmission antihypertensive agents are often held or reduced unless they are being used for other indications such as cardiac rate control.
- Limited data suggest that autoregulation remains abnormal for the 1–2 weeks after the ischemic event; therefore, aggressive hypertension management may be delayed during this time.²⁰

Chronic setting

- All patients with stroke and TIA should be prescribed antihypertensive therapy if BPs are greater than 120/80.²¹ Randomized, controlled trials have shown that reduction of systolic BP by more than 10 mm Hg decreases stroke risk by 30% over about 5 years.²²
- The choice of medications should be tailored to the individual patient. Angiotensin-converting enzyme (ACE) inhibitors and thiazide diuretics are recommended as first-line agents for cerebrovascular disease in many cases. ACE inhibitors or angiotensin receptor blockers (ARBs) may be used first in diabetics due to their renal protective effects. Beta blockers may be used first in patients with ischemic cardiac disease.²³
- Often, the patient has been discharged from the stroke center before optimal BP control is achieved. Communication to the primary care physician regarding treatment goals is essential. Follow-up in a stroke prevention clinic may be useful in monitoring compliance and success of treatment.

Diabetes

Diabetes mellitus, affecting 8% of the adult population in the United States, is an independent risk factor for stroke.

- All stroke patients should be screened for diabetes with a fasting glucose and hemoglobin A1C level.
 - Normal fasting glucose is defined as less than 100 mg/dL.
 - Impaired fasting glucose is defined as 100–125 mg/dL.
 - Fasting glucose greater than 126 or A1C greater than 6.5% (or random glucose > 200) meets the threshold for a diagnosis of diabetes.
- Management of diabetes mellitus includes diet, exercise, oral hypoglycemic agents, and insulin.
- The goal is to maintain hemoglobin A1C levels less than 7.21

Hyperlipidemia

Hyperlipidemia (increased levels of various serum lipids) is a significant risk factor for ischemic stroke. Elevated serum triglycerides, total cholesterol, and LDL are risk factors for cardiovascular disease, and in patients with cardiac disease, high triglycerides and high density lipoprotein-cholesterol (HDL-C) with ischemic strokes.²² In a meta-analysis of the statin trials in patients with coronary disease, the larger the reduction in low density lipoprotein-cholesterol (LDL-C), the greater the reduction in stroke risk was.²⁴

In patients with TIA or stroke without a history of coronary artery disease, lowering LDL-C decreases the risk of recurrent cerebral and cardiac ischemic events.²⁵ Current recommendations include the following:

- Fasting lipids should be obtained on admission or within 24 hours of admission to guide discharge medication regimens. Because LDL levels may drop in the acute setting, repeat measurement after discharge is recommended.
- Statin treatment is recommended to reduce risk of stroke and cardiovascular events in patients with stroke or TIA who have evidence of atherosclerotic disease and whose LDL-C levels are above 100 mg/dL.
- Maximum benefit is seen when LDL-C is reduced by 50% or to a target of less than 70 mg/dL.
- Strategies for lowering LDL-C include lifestyle modification, emphasizing a reduction in saturated fat and cholesterol intake; weight reduction to ideal body weight; increase in physical activity; as well as medical therapy.

One of the reported metrics for the Get With the Guidelines stroke database is the percentage of patients with stroke or TIA who are discharged on statin therapy. Some data suggest that patients with stroke or TIA benefit from statin therapy regardless of cholesterol levels.²⁶

Tobacco use

- Patients who are currently tobacco users should be strongly encouraged to quit smoking.
- Even a reduction in smoking is helpful.
- Limiting second-hand smoke exposure is also recommended.
- Exposure to tobacco has been shown to increase the rate of atherosclerosis accumulation.²⁷
- Counseling should be provided to all stroke patients who use tobacco. Support groups should be offered if available. Successful tobacco cessation requires repeated interventions.
- Pharmacotherapies, including nicotine products and oral smoking cessation medications, should be offered to all patients who are attempting to quit smoking.²⁸

Alcohol consumption

Heavy drinking or chronic alcoholism is a significant risk factor, and alcohol use should be stopped or reduced to no more than two drinks per day for men and one drink per day for women.³

Obesity

Obesity, defined as a body mass index (BMI) greater than 30 kg/m², is an independent risk factor for stroke. Weight management through balanced calorie intake, exercise, and behavioral counseling should be encouraged in all patients with stroke and TIA. A goal BMI of 18.5–24.9 kg/m² is recommended.³

Treatment

Exercise

Thirty minutes of moderate-intensity exercise on most days is recommended for secondary stroke prevention.³

Antiplatelet therapy for secondary stroke prevention

Aspirin is well-established antiplatelet therapy for secondary stroke prevention.

- Therapy should be started within 48 hours of stroke and is typically started on the day of presentation, after determining that there is no intracerebral hemorrhage (ICH) on the head computed tomography (CT).
- Early aspirin treatment leads to an absolute risk reduction in stroke recurrence of 1% in the first 2 weeks after stroke.²⁷
- In the long term, among stroke and TIA patients, aspirin in doses from 50 to 325 mg daily reduces the rate of recurrent stroke by 22% annually, regardless of dosing.²⁹
- If the patient receives an acute reperfusion intervention, aspirin should be held until 24-hour CT results showing no hemorrhage have been obtained.

Alternative agents to be considered include clopidogrel (Plavix) and an aspirin/ER dipyridamole combination (Aggrenox).

- Clopidogrel has been shown to reduce stroke recurrence rates comparably to aspirin.
 - However, clopidogrel decreased the risk of the combined end point of MI, stroke, and vascular death by an additional 8.7% compared with aspirin, primarily due to an increased benefit in patients with peripheral vascular disease.³⁰
 - For patients allergic to aspirin, clopidogrel is a reasonable choice.
 - Of note, combination therapy using both aspirin and clopidogrel offers no benefit over clopidogrel or aspirin alone but provides added risk of life-threatening and major bleeding events.^{31,32}
 - Whether there may be a role for this combination immediately after an acute stroke, analogous to acute coronary stent placement, has not been investigated.
- The aspirin/ER dipyridamole combination has been shown to lead to a 23% relative risk reduction in stroke recurrence rates compared with aspirin.³³

The PROFESS Trial that randomized patients to aspirin/ER dipyridamole combination versus clopidogrel 75 mg showed no net difference in the rate of recurrent stroke or major hemorrhage.³⁴ The decision regarding which antiplatelet agent to initiate after a first stroke must be individualized. Aspirin, ER dipyridamole, and clopidogrel are all acceptable options.³

Discharge instructions after ischemic stroke

In-depth education regarding secondary prevention is difficult in the acute care setting. However, at discharge it is ideal to give the patient and family a written summary of the individual risk factors, the target for improving each risk factor, and the strategy for doing so. This should also be communicated to the primary care physician, who will usually resume care of the patient (Appendix).

Intracerebral hemorrhage

- Uncontrolled hypertension is, by far, the most common risk factor for ICH. As in the case of ischemic stroke, the management of hypertension is usually handled differently in the acute versus chronic settings. See Chapter 8 and the "Guidelines for the Management of Spontaneous Intracerebral Hemorrhage"³¹ for a discussion of acute BP management.
- Strict hypertension management should be achieved within 4–6 weeks after the initial ICH.
- Amyloid angiopathy, trauma, vascular malformations, or an underlying tumor are other underlying causes for ICH, depending on the location and appearance of the ICH. The workup is dictated by the clinical scenario
- Considerations include the imaging appearance of the ICH and the patient's history. For example, lobar hemorrhages warrant gradient echo (GRE) MRI imaging to look for evidence of prior microhemorrhages. Diagnosis of amyloid angiopathy may have an impact on the decision to use anticoagulation therapy, although definitive evidence on this topic is not available.³²
- A heterogeneous or atypical appearance of the ICH warrants an MRI to look for vascular malformations during the hospitalization. If unrevealing, another MRI should be performed after 1–2 months to reassess the appearance after blood resorption
- Clinical factors adding to suspicion of a vascular malformation, such as an MRI appearance of blood vessel feeders, a lobar location of ICH, younger age, and no history of hypertension, should raise the suspicion for an underlying vascular malformation and lead to further screening by MRA or cerebral angiogram.

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chapter eleven

Stroke rehabilitation

Introduction

Considerable emphasis continues to be placed on stroke prevention by state and national organizations, yet stroke incidence remains stable and is rising in some subgroups. Concurrently, thrombolytic treatment constitutes a major focus of many stroke programs and stroke center certifications, yet only a minority of patients access these treatments. As a result of these trends, 40% of stroke patients retain moderate functional impairments, and 15–30% exhibit severe disability in the weeks and months following their acute hospitalizations. Moreover, the number of community-dwelling stroke survivors exhibiting significant impairments that undermine independence is expected to rise exponentially.

In response to this growing need, a number of stroke rehabilitative treatments have been developed, and a burgeoning evidence base supporting stroke rehabilitation efficacy has emerged. Although it is widely acknowledged that the acute phase constitutes a critical rehabilitative period, primary stroke center guidelines are meager in addressing the provision of rehabilitative services. For example, the most recent primary stroke center guidelines do not suggest specific therapy approaches that could be incorporated into acute rehabilitation, despite ample evidence supporting particular treatment strategies. In addition, even the eligibility criteria to become a primary stroke center do not include the word *rehabilitation*, and therapy services do not have to be available on site for a facility to be eligible to become a primary stroke center. Ironically, these oversights occur despite the fact that stroke recovery and rehabilitation constitute the most time-intensive aspect of the entire stroke continuum. Formal rehabilitative efforts may occur for months, while the adjustment and recovery process is frequently a lifelong undertaking. Given this, we encourage integration of rehabilitative personnel into the stroke team continuum and hierarchy, including placing rehabilitative personnel in key positions on the stroke team.

Despite these shortfalls, every stroke center needs access to a multidisciplinary rehabilitation team to maximize recovery for each patient. Ideally, patients should be evaluated by the rehabilitation team as soon as the patients are medically stable, with the following five major goals in mind:

- 1. Preventing, recognizing, managing, and minimizing the impact of preexisting medical conditions
- 2. Training for maximal functional independence
- 3. Facilitating optimal psychosocial adaptation and coping by both the patient and family
- 4. Promoting community reintegration, resumption of prior life roles, and return to home, family, recreational, and vocational activities
- 5. Enhancing quality of life

Because of the diversity of functional domains encompassed by these goals, rehabilitation relies heavily on a "team approach" that ideally includes the physiatrist, speech therapist, physical therapist, occupational therapist, neuropsychologist, social worker, recreation therapist, and others working with the patient to set goals and reintegrate the patient into the desired community. In our experience, a physiatrist is, perhaps, the most key player as he or she is the team leader who organizes the various components of a rehabilitation plan for a particular patient. Ironically, in our experience, regular, yearly contact with the physiatrist is also often the most ignored component of a comprehensive rehabilitation plan. Despite the high possibility of patients developing secondary sequelae in the months and years after stroke, many stroke centers do not have outpatient physiatry services for follow up with patients.

While physical rehabilitation (e.g., occupational therapy, spasticity management) is perhaps the most recognized facet of stroke rehabilitation, other aspects of the rehabilitative process, such as management of depression and cognitive impairments, are equally important. These deficits continue long after the usual rehabilitation process has been completed and may involve professionals such as neuropsychologists. Participation in a stroke support group by both the patient and the caregiver can also be helpful in connecting to community resources. An online directory of stroke support groups—as well as extensive informational and educational resources for patients and their care partners—is available through the National Stroke Association (http://www.stroke.org).

In this chapter, we outline the common components and goals of inpatient stroke rehabilitation. Secondarily, we also review the typical stroke motor recovery patterns, rehabilitation strategies for specific motor impairments, and some new stroke rehabilitation techniques.

Acute inpatient rehabilitation

During acute stroke inpatient rehabilitation, the patient is ideally involved in physical rehabilitation as soon as medical stabilization occurs. Acute rehabilitation is ideally directed not only at restoration of function and compensation for lost function but also at preventing secondary complications, including the occurrence of another stroke. All of these aspects of care should be included in the clinical pathway. Programs in acute rehabilitation should not only include physical, occupational, and speech therapy, but also evaluate

- Medical problems
- Mental status
- Sensation
- Skin integrity
- Frequency of turns and position changes (to prevent development of pressure sores)
- Edema
- Venous thromboembolism prophylaxis
- Rest and sleep
- Endurance/cardiorespiratory status

In the acute setting, the rehabilitation team may also provide

- Psychological support to patient or family
- Educational programs on stroke, stroke prevention, and personal care
- Functional mobility skills assessment and exercises such as
 - Bed mobility
 - Transfers
 - Wheelchair or assistive device mobility

As stated previously, care in this setting is ideally directed by a physiatrist with training and expertise in neurorehabilitation and carried out by a team, with weekly meetings for case discussion. Therapy duration is usually 3 hours per day. Recent animal and human research studies suggested that, for brain plasticity and subsequent improvements to occur, rehabilitation must be task specific, repetitive, and motivating to the patient. Thus, many centers are identifying pathways whereby additional "bedside" therapy is provided by other hospital staff. The goal of these therapies is to provide additional practice attempts and to harness the considerable brain plasticity thought to be available during the acute stage. The acute rehabilitative team will also make the recommendation for what type of postacute rehabilitation should occur. If your hospital does not have a rehabilitation unit, then it is important to establish close relationships with one or more rehabilitation facilities so that patients can be transferred and task-specific, repetitive rehabilitation started in a timely fashion.

Stroke rehabilitation outcomes

Several factors may influence the specific outcome of an individual patient involved in a poststroke stroke rehabilitation program. Potentially important factors may include the following:

- Type, distribution, pattern, and severity of physical impairment
- Cognitive, language, communication, and learning ability
- Number, type, and severity of comorbid medical conditions
- Depression or other psychological impairment
- Coping ability and style
- Nature and degree of family and other social supports
- Type and quality of specific rehabilitation training programs

The strongest and most consistent predictor of discharge functional ability is admission functional ability. The strongest predictors of adverse outcomes are coma at onset, persistent incontinence, poor cognitive function, severe hemiplegia, prior stroke, visuospatial neglect, cardiovascular disease, and large cerebral lesion. With these factors noted, it is important to remember that it may be difficult to apply these predictors to a particular patient given the heterogeneous nature of stroke and the impact that premorbid factors can have on outcome.

New approaches to motor and functional training

A variety of rehabilitative strategies have been developed to target motor, affective, and cognitive impairments. A review of all of these approaches and domains is beyond the scope of this chapter. Instead, we highlight recent developments in motor therapies because (a) motor impairments are the most frequently encountered and, arguably, the most disabling of all stroke sequelae; and (b) many strategies in other domains (e.g., speech) are based on innovations in motor therapies (e.g., constraint-induced aphasia therapy, the use of contextual interference).

Traditionally, stroke patients have been encouraged to use their lessaffected hands. This has been based on the premise that motor recovery in the more affected hand is not plausible, and thus, compensatory strategies (e.g., one-handed shoe tying, writing with the less-affected hand) should be taught. During compensatory training, the patient is encouraged to make use of residual abilities to develop new ways of achieving old goals and perform routine tasks such as transferring. Reduced lengths of stay have also forced therapists to focus on compensatory strategies using the less-affected limb.¹ Motor training programs have traditionally consisted of positioning, passive and active range-of-motion exercises, and progressive resistance exercises. However, many conventional training approaches offer negligible efficacy or are no more efficacious than another.

Task-specific training approaches

Numerous studies have shown that movement patterns and anatomical regions used more frequently become represented over larger cortical surface areas. Based on this finding in both animals and humans, a number of task-specific training regimens have been developed and are being tested as new approaches to rehabilitation.

Constraint-induced movement therapy

For decades, it had been observed that individuals with neurologic insults, although capable of using their more affected limbs, often chose not to use them.^{2,3} However, following success in early conditioned response studies,⁴ researchers⁵ forced use of patients' more affected limbs by requiring sling wear on their less-affected limbs during nearly all waking hours of a 2-week period. Following the intervention, patients demonstrated significant but small improvements in 19 of the 21 motor tasks on the Wolf Motor Function Test. Subsequent researchers⁶⁻⁸ have combined a restriction schedule identical to the one used by Wolf and colleagues with a 6-hour/day training protocol during which patients perform functional tasks using the more affected limb. Training sessions occur on all weekdays of the same 2-week period that restriction of the less-affected limb occurs. The studies have shown that this constraint-induced movement therapy (CIT) causes substantial increases in use and function of the more affected limb in patients with chronic disability (>1 year after cerebrovascular accident [CVA]) and, more recently, in more subacute stroke cases.9 However, CIT is no more efficacious than time-matched upper extremity (UE) training in the acute stage^{10,11} and can be taxing and costly to administer at any point after stroke.

Modified constraint-induced therapy: An outpatient, reimbursable alternative

A recent survey¹² found that many stroke patients would not want to participate in CIT and would prefer a therapy protocol lasting for more weeks with shorter activity sessions or fewer hours wearing the restrictive



Figure 11.1 A stroke survivor performs home-based activity under the guidance of an occupational therapist during modified constraint induced therapy.

devices. Given CIT shortcomings, shorter forced-use protocols have been developed.^{13,14} The most notable example has been an outpatient therapy called modified constraint-induced therapy (mCIT), which combines one-half-hour practice sessions with restriction of the unaffected arm 5 days/week for 5 hours/day, both during a 10-week period. Besides being reimbursable by most managed care programs, mCIT increased affected arm use and function in randomized, controlled studies with acute,¹⁵ subacute (>3 months but <12 months poststroke),^{16,17} and chronic¹⁸ stroke patients. Currently, mCIT is used as a care model in many outpatient and inpatient rehabilitative facilities in the United States and is successfully reimbursed using existing current procedural terminology codes.

Following stroke, the size of the cortical representation of the more affected hand is known to decrease,^{19,20} possibly due to limb nonuse.²¹ However, as stated previously, in task-specific protocols like mCIT, in which the more affected limb is used in behaviorally relevant ways, the size of the cortical areas representing the limb increases.^{22–25} As such, increased functional affected arm use via mCIT participation has been shown to cause cortical reorganization.²⁶

Bilateral training

Research suggested that patients who are unable to voluntarily move their affected arms unilaterally can often move the arm when bilateral practice strategies are used (i.e., both arms are moved at the same time and in the same direction). This is a critical discovery as it means that patients who normally would be barely able to move their affected arms can often practice meaningfully when bilateral training strategies are employed. This repetitive practice conveys increased strength, range of motion, and performance of discrete unilateral and bilateral movement in the affected limb.²⁷

Bilateral training may also be optimal for stroke patients because they receive proprioceptive and visual feedback from the unaffected limb that they do not receive during unilateral practice. Indeed, when practicing bilaterally, a patient can use the unaffected extremity's neurologically intact afferent and efferent signals, and the look and feel of movement within that limb, to promote similar movement in the affected limb. For example, a stroke patient could simultaneously practice a reaching movement, such as reaching for a cup, with the affected and unaffected limb. This would provide sensory input, as the patient feels what it is like to reach for the cup with the unaffected limb. However, the visual input of seeing the unaffected arm reach for the cup may also provide a model with which the patient can better move the affected limb and become more successful.

A number of clinical models are available to facilitate bilateral training. Bilateral arm training with rhythmic auditory cueing (BATRAC) uses a robot to deliver controlled, bilateral, rhythmic movements to the upper limbs and has been shown to be effective. Although BATRAC serves as a useful tool to study bilateral training under optimal conditions, most therapy clinics do not have robotic arms and cannot afford to invest in these types of strategies.

In this laboratory, we have used the NuStep to administer bilateral training for the arms and legs. As shown in Figure 11.2, the NuStep is a commonly available machine that has traditionally been used to rehabilitate or strengthen geriatric patients. However, the NuStep also allows the patient to oppositionally, bilaterally practice with both legs and both arms simultaneously. Importantly, whereas many approaches to affected limb function in stroke require intense supervised practice, specialized training, or additional personnel, the NuStep is easy to use and requires minimal supervision, and widespread application can be quickly realized. The device has the advantage that the patient can engage in bilateral practice that simulates walking while remaining seated. Pilot data suggest that the NuStep is a promising clinically practical approach to improving balance and ambulation in stroke patients, even many years after injury. Importantly, the resistance on the device can be adjusted, resulting in




marked improvement in cardiovascular conditioning in most of our subjects. Currently, we are investigating the use of an auditory component (e.g., a metronome), as auditory cueing is believed to increase learning of naturally rhythmic behaviors such as walking. The possibility also exists that layering in electrical stimulation (described next) may expand the number of patients who may engage in bilateral training.

Electrical stimulation

Despite growing evidence suggesting that motor relearning after stroke is activity dependent, large segments of stroke survivors are not able to take part in such strategies due to the severity of their hemiparesis. Cyclic neuromuscular electrical stimulation (NMES) involves stimulation applied to the affected muscle(s) to elicit muscle contraction(s). It is presumed that the afferent input improves motor control with repeated use. Several studies have shown increased affected arm function following cyclic surface NMES use.²⁸ However, cyclic surface NMES therapy may be suboptimal because patients are not responsible for volitionally activating their muscles (i.e., their participation is passive).

Given that repeated, *volitional*, affected limb use facilitates neuroplasticity and improves function, it would seem advantageous to use a device focused on that type of activity. Surface electromyography (EMG)-triggered neuromuscular stimulation (ETMS) incorporates concepts of repeated limb use, biofeedback, and electrical stimulation. When using ETMS, the patient attempts to activate the affected musculature (for the purposes of this discussion, the affected extensors). If the intended muscles are activated such that a preset threshold is reached (as detected by EMG in the device), the musculature will be electrically stimulated by the device, and full extension is realized. If the threshold is not reached, the threshold is automatically lowered, and the patient tries again. Thus, the patient is provided with biofeedback that "reteaches" active muscle contraction through the reward of stimulation. ETMS appears to restore more movement in the affected wrist in both subacute and chronic stroke patients.^{29–32}

A new electrical stimulation approach incorporates a hand orthosis that provides synchronized activation of finger and wrist flexors and extensors during practice of functional, valued activities.^{33,34} The device, called the Bioness H200, can be self-administered in the home, making it more accessible to a larger number of stroke patients than traditional modalities, which typically require therapist supervision in a structured environment. The device is used during performance of valued activities, which overcomes a shortcoming of passive electrical stimulation modalities. Studies conducted by this team^{35–37} have demonstrated significantly increased paretic upper extremity use and function and have determined the neural biomarkers³⁸ and optimal duration³⁹ associated with this approach, all in moderately impaired stroke patients. Again, this is a potentially important development since many therapies (e.g., CIT, mCIT) are only efficacious on minimally impaired patients. The device is depicted in Figure 11.3.

Mental practice

Imagined movement practice—known as *mental practice* (MP)—is an inexpensive, easily administered technique during which physical skills are cognitively rehearsed. Because therapy frequency and duration are becoming increasingly limited, cost-effective rehabilitation strategies that require low supervision and setup are needed. MP is a noninvasive, cost-effective technique in which physical skills are cognitively rehearsed in the absence of overt physical movements. Since the 1890s, studies have reported improved motor function when MP is combined with physical practice.^{40,41} The same musculature is activated during MP as during physical practice of the same task.^{42–44} The genesis of new and improved brain imaging techniques has also revealed that the same neural structures subserve both physical and imagined movements.^{45,46} Consequently, MP, when combined with physical practice, has been shown to be efficacious for relearning of motor skills in rehabilitative settings^{47–51} and has been



(a)

(b)



(c)

Figure 11.3 One example of electrical stimulation is the Bioness H200, which allows patients to receive stimulation while participating in a functional activity such as reaching for objects.

shown to accelerate motor skill learning. The use of MP in stroke rehabilitation is supported by evidence showing that repeated MP activates the same neural areas as physical practice of the same tasks.^{53–57} Furthermore, we have shown that repetitive MP use causes cortical reorganizations,⁵⁸ and that addition of mental to physical practice significantly increases affected arm use and function.^{59–64}

Despite billions of dollars spent annually on stroke rehabilitation,⁶⁵ conventional rehabilitation strategies targeting the affected leg have no evidence,⁶⁶ or negative evidence,^{67,68} supporting their use. Automated devices (e.g., robotics) have been developed to augment lower-extremity function^{69,70}; however, because of their cost, size, and level of sophistication, these devices are limited to use in just a few clinics and hospitals. A recent multicenter trial also demonstrated that body-weight-supported treadmill training is no more effective than a home exercise program.⁷¹

MP has been suggested as a promising technique, and pilot data suggest that its use may also increase affected leg function.^{72–76} To date, however, its impact has mostly been examined in case studies and small exploratory studies with varied procedures and outcome measures.

Conclusion

There is an increasing prevalence of stroke survivors with disabilities in the United States. Acute stroke care units confer larger benefits than thrombolytics or medications,^{77,78} with the largest functional gains observed when a rehabilitative component is included.^{79,80} As a result of all of these factors, stroke rehabilitation is expected to play an even greater role in returning patients to a normal level of function and quality of life. Ideally, stroke rehabilitation is integrated meaningfully and early into the acute stroke care continuum and emphasizes a team approach that includes input from therapy staff, neuropsychologists, social workers, and most important, the patient's family and the patient. Stroke center guidelines should meaningfully reflect the integration of rehabilitation to maximize full patient recovery.

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Appendix A: Emergency tools for stroke team nurses

The clinical tools and order sets in this appendix were developed by the stroke teams at Saint Luke's Neuroscience Institute, Kansas City, Missouri, and the University of Cincinnati in Cincinnati, Ohio. They are based on current evidence and are updated on a regular basis by each of the teams. Their purpose is to improve efficiency and to standardize care.

The first four tools are faxed to a referring hospital in the regional network when the decision has been made to administer intravenous tissue plasminogen activator (IV tPA) before transporting the patient to Saint Luke's Stroke Center.

Tissue Plasminogen Activator (Alteplase – Activase [®]) What You Should Know

What is Alteplase - Activase *?

Alteplase – Activase ® is a medication that dissolves blood clots. It is called a thrombolytic agent or more commonly referred to as the "clot buster." It is an intravenous or IV medication usually given through a catheter inserted into a vein the arm.

What type of stroke is IV Alteplase – Activase® used for?

It was approved by the FDA in 1996 to treat ischemic type strokes. About 8 out of 10 brain attacks/strokes are ischemic. These types of strokes are most often caused by blood clots that block the flow of blood to the brain causing tissue death. Alteplase – Activase ® is given to help dissolve the clot quickly and restore the blood flow to the brain tissue.

The other common type of brain attack is called a hemorrhagic stroke. This brain attack/stroke is due to bleeding from a blood vessel into the brain. Alteplase – Activase ® is not used with this type of brain attack because it could increase the amount of bleeding and possibly cause more damage to the brain.

A CT scan or MRI of the head is done to confirm there is no bleeding in the brain before Alteplase – Activase ® is given.

When is Alteplase – Activase * used? Alteplase – Activase ® has been approved to treat brain attacks in the first three hours following the onset of symptoms. If given promptly, 1 in 3 patients who receive Alteplase – Activase * resolve their symptoms or have major improvement in their stroke symptoms. What are the risks of Alteplase - Activase *?

Bleeding (hemorrhage), in the brain or in other parts of the body, is the most common risk that can occur. In 6 out of 100 patients, bleeding may occur into the brain and cause further injury. For 1 of these 6 patients it may cause death or long term serious disability. Advanced age and more severe stroke symptoms are associated with an increased risk of bleeding.

Should everyone receive Alteplase – Activase [®] therapy?

Unfortunately the answer is no. Persons who cannot be treated within four hours and thirty minutes of their first symptom, patients with certain medical conditions, and patients with certain types of strokes will not qualify for this treatment.

Inform your physician if you have had any of the following:

- î Recent heart attack
- î Serious head trauma within the last three months
- î Bleeding from the stomach or urinary tract within the last 21 days
- î Major surgery within previous 14 days
- î Bleeding disorders
- î Use of blood thinners, such as warfarin
- î Uncontrolled high blood pressure

Adapted from OS St Francis Medical Center, Form No 9660041

AHA Guidelines for the Early Management of Patients with Ischemic Stroke, 2005

Early Treatment Confirmed as Key to Stroke Recovery The Lancet, March 2004;363:768-774.

Saver, J. Hemorrhage After Thrombolytic Therapy for Stroke Stroke. 2007;38:2279-2283

Demaerschalk, B. Thrombolytic Therapy for Acute Ischemic Stroke, The Likelihood of Being Helped Versus Harmed. ETC> Stroke. 2007;38:2215-2216

(0.9 mg/kg Alteplase – Activase ® Dosing (Ma			x 90 mg	g)		10% IV bolus - 90% infusion				
(LB)	(KG)	WASTE	10% BOLUS	90% INFUSION	TOTAL IV DOSE	(LB)	(KG)	WASTE	10% BOLUS	90% INFUSION	TOTAL IV DOSE
88	40.0	64.0	3.6	32.4	36.0	156	70.9	36.2	6.4	57.4	63.8
89	40.5	63.6	3.6	32.8	36.4	157	71.4	35.8	6.4	57.8	64.2
90	40.9	63.2	3.7	33.1	36.8	158	71.8	35.4	6.5	58.2	64.6
91	41.4	62.8	3.7	33.5	37.2	159	72.3	35.0	6.5	58.5	65.0
92	41.8	62.4	3.8	33.9	37.6	160	72.7	34.5	6.5	58.9	65.5
93	42.3	62.0	3.8	34.2	38.0	161	73.2	34.1	6.6	59.3	65.9
94	42.7	61.5	3.8	34.6	38.5	162	73.6	33.7	6.6	59.6	66.3
95	43.2	61.1	3.9	35.0	38.9	163	74.1	33.3	6.7	60.0	66.7
96 97	43.6 44.1	60.7 60.3	3.9 4.0	35.3 35.7	39.3 39.7	164 165	74.5 75.0	32.9 32.5	6.7 6.8	60.4 60.8	67.1 67.5
97	44.1	59.9	4.0	35.7	39.7 40.1	165	75.5	32.5 32.1	6.8	61.1	67.9
99	44.0	59.5	4.0	36.5	40.1	167	75.9	31.7	6.8	61.5	68.3
100	45.5	59.1	4.1	36.8	40.9	168	76.4	31.3	6.9	61.9	68.7
101	45.9	58.7	4.1	37.2	41.3	169	76.8	30.9	6.9	62.2	69.1
102	46.4	58.3	4.2	37.6	41.7	170	77.3	30.5	7.0	62.6	69.5
103	46.8	57.9	4.2	37.9	42.1	171	77.7	30.0	7.0	63.0	70.0
104	47.3	57.5	4.3	38.3	42.5	172	78.2	29.6	7.0	63.3	70.4
105	47.7	57.0	4.3	38.7	43.0	173	78.6	29.2	7.1	63.7	70.8
106	48.2	56.6	4.3	39.0	43.4	174	79.1	28.8	7.1	64.1	71.2
107	48.6	56.2	4.4	39.4	43.8	175	79.5	28.4	7.2	64.4	71.6
108	49.1	55.8	4.4	39.8	44.2	176	80.0	28.0	7.2	64.8	72.0
109	49.5	55.4	4.5	40.1	44.6	177	80.5	27.6	7.2	65.2	72.4
110	50.0	55.0	4.5	40.5	45.0	178	80.9	27.2	7.3	65.5	72.8
111	50.5	54.6	4.5	40.9	45.4	179	81.4	26.8	7.3	65.9	73.2
112 113	50.9	54.2	4.6 4.6	41.2 41.6	45.8	180 181	81.8 82.3	26.4	7.4 7.4	66.3 66.6	73.6 74.0
113	51.4 51.8	53.8 53.4	4.6	41.6	46.2 46.6	182	82.3 82.7	26.0 25.5	7.4	67.0	74.0
115	52.3	53.0	4.7	42.0	47.0	183	83.2	25.1	7.4	67.4	74.9
116	52.7	52.5	4.7	42.7	47.5	184	83.6	24.7	7.5	67.7	75.3
117	53.2	52.1	4.8	43.1	47.9	185	84.1	24.3	7.6	68.1	75.7
118	53.6	51.7	4.8	43.4	48.3	186	84.5	23.9	7.6	68.5	76.1
119	54.1	51.3	4.9	43.8	48.7	187	85.0	23.5	7.7	68.9	76.5
120	54.5	50.9	4.9	44.2	49.1	188	85.5	23.1	7.7	69.2	76.9
121	55.0	50.5	5.0	44.6	49.5	189	85.9	22.7	7.7	69.6	77.3
122	55.5	50.1	5.0	44.9	49.9	190	86.4	22.3	7.8	70.0	77.7
123	55.9	49.7	5.0	45.3	50.3	191	86.8	21.9	7.8	70.3	78.1
124	56.4	49.3	5.1	45.7	50.7	192	87.3	21.5	7.9	70.7	78.5
125 126	56.8 57.3	48.9	5.1 5.2	46.0	51.1 51.5	193 194	87.7 88.2	21.0 20.6	7.9 7.9	71.1 71.4	79.0
126	57.3 57.7	48.5 48.0	5.2	46.4 46.8	52.0	194	88.6	20.6	8.0	71.4	79.4 79.8
127	58.2	47.6	5.2	40.0	52.4	196	89.1	19.8	8.0	72.2	80.2
129	58.6	47.2	5.3	47.5	52.8	197	89.5	19.4	8.1	72.5	80.6
130	59.1	46.8	5.3	47.9	53.2	198	90.0	19.0	8.1	72.9	81.0
131	59.5	46.4	5.4	48.2	53.6	199	90.5	18.6	8.1	73.3	81.4
132	60.0	46.0	5.4	48.6	54.0	200	90.9	18.2	8.2	73.6	81.8
133	60.5	45.6	5.4	49.0	54.4	201	91.4	17.8	8.2	74.0	82.2
134	60.9	45.2	5.5	49.3	54.8	202	91.8	17.4	8.3	74.4	82.6
135	61.4	44.8	5.5	49.7	55.2	203	92.3	17.0	8.3	74.7	83.0
136	61.8	44.4	5.6	50.1	55.6	204	92.7	16.5	8.3	75.1	83.5
137	62.3	44.0	5.6	50.4	56.0	205	93.2	16.1	8.4	75.5	83.9
138	62.7	43.5	5.6	50.8	56.5	206	93.6	15.7	8.4	75.8	84.3
139	63.2	43.1 42.7	5.7 5.7	51.2 51.5	56.9	207 208	94.1 94.5	15.3	8.5 8.5	76.2	84.7 85.1
140 141	63.6 64.1	42.7 42.3	5.7	51.5 51.9	57.3 57.7	208	94.5 95.0	14.9 14.5	8.5 8.6	76.6 77.0	85.1 85.5
141	64.1 64.5	42.3 41.9	5.8	52.3	57.7 58.1	209	95.0 95.5	14.5 14.1	8.6	77.3	85.9
142	65.0	41.9	5.9	52.5	58.5	210	95.9 95.9	14.1	8.6	77.7	86.3
143	65.5	41.1	5.9	53.0	58.9	212	96.4	13.3	8.7	78.1	86.7
145	65.9	40.7	5.9	53.4	59.3	213	96.8	12.9	8.7	78.4	87.1
146	66.4	40.3	6.0	53.8	59.7	214	97.3	12.5	8.8	78.8	87.5
147	66.8	39.9	6.0	54.1	60.1	215	97.7	12.0	8.8	79.2	88.0
148	67.3	39.5	6.1	54.5	60.5	216	98.2	11.6	8.8	79.5	88.4
149	67.7	39.0	6.1	54.9	61.0	217	98.6	11.2	8.9	79.9	88.8
150	68.2	38.6	6.1	55.2	61.4	218	99.1	10.8	8.9	80.3	89.2
151	68.6	38.2	6.2	55.6	61.8	219	99.5	10.4	9.0	80.6	89.6
152	69.1	37.8	6.2	56.0	62.2	220	100.0	10.0	9.0	81.0	90.0
153	69.5	37.4	6.3	56.3	62.6	>220	100.0	10.0	9.0	81.0	90.0
154	70.0	37.0	6.3	56.7	63.0						
155	70.5	36.6	6.3	57.1	63.4						

Saint Luke's Stroke Network

lsch	emic Stroke Management with IV t-PA (Activase – Alteplase) Recommendations
Date:	Time:Patient Name:
	gist Recommending:
	Treatment Recommendations
	Last time patient was known to be well (please document)
	CT shows no hemorrhage
	Labs completed Platelets greater than 100,000 and INR less than or equal to 1.7
	Place two peripheral IV's, one must be 18 or 20 gauge AC or proximal to AC in upper arm.
	Insert Foley prior to t-PA administration
	Strict NPO
	Place oxygen per nasal cannula and titrate to maintain O2 sat greater than 95% with oxygen
	If patient intubated please place OG tube
	Prior to administration of IV t-PA BP must be less than 185/110 – see Blood Pressure Management in
0.	section C below.
	atment Dosing Recommendations if patient is Candidate for IV t-PA (DOC 1 to check which
	endation) For Patient Safety Never hang more t-PA than ordered dose . Use charts to figure
	dose to give and waste .
1.	Dosing with 0.9 mg/kg (maximum dose – do not exceed 90 mg)
	a. 0.9 mg/kg XWt(kg) =Total mg
	IV Bolus = 10% of total mg (mg) over 1 minute. Time administered
	Infusion dose = 90% of total mg (mg) per pump over 60 minutes. Time infusion
	started
	agement recommendations – Pre (≤ 180/110) and Post t-PA Treatment (≤ 180/105)
1.	IV Bolus Management
	Labetalol 10mg over 2 minutes, if goal not met, then repeat labetalol 20 mg X 1 over 2 minutes.
	Do not use if heart rate less than 60 Meteorolol 5 and 1/ holus, report every 5 minutes for movimum of 20mg, Hold if SDD less than
	Metoprolol 5 mg IV bolus, repeat every 5 minutes for maximum of 20mg. Hold if SBP less than 140 or DBP less than 80 and HR less than 60
2	Initiate (or titrate existing) continuous infusion
2.	Nicardipine infusion, 5 mg/h, titrate up by 2.5 mg/h at 5 to 15-minute intervals, maximum dose
	15 mg/h; when desired blood pressure attained, reduce to 3 mg/hr
	Labetalol 2mg/min infusion as initial dose & titrate by increasing 1 mg/minutes every 5 minutes up to
	8 mg/min
Post t-P	A Treatment recommendations
1.	Monitor & document blood pressure and neuro checks every 15 minutes during and after t-PA
	administration. If any significant deterioration turn off t-PA and obtain STAT CT Head Scan
	Monitor for deterioration in neurological status from baseline; for example LOC, motor
	Monitor for sudden onset of headache, nausea and/or vomiting
	Monitor for sudden elevation in BP
	After t-PA infusion, may start NS at 80 ml per hour
	rt Preparation
	Titrate BP medication to maintain BP less than 180/105 after t-PA administration and during transport
	Stop BP medication infusion if SBP less than 140 or DBP greater than 80
3.	Obtain emergency contact number for family, caregiver or bystander (preferably cell phone) to obtain further information or obtain consent for further treatment. (Cell Phone)
	Send copy CT Head Scan.
5.	Send patient records with documentation of allergies, current medications and IV t-PA dosages, past medical history
6.	Review with transport team t-PA IV Bolus given, total IV infusion mg and expected time of infusion completion
7.	Give transport team Transport Protocol

Note: Doc 1 initiates this documentation and faxes to referring hospital for continued documentation

Saint Luke's Stroke Network Saint Luke's Brain and Stroke Institute Please Give To Transporting EMS Team

		Stroke Management Transport Protocol
Dat		Time: Transport Unit Name:
A.		or to Departure
А.		Two peripheral IV's, one 18 or 20 gauge AC or proximal to AC in upper arm is preferred
		Foley to Dependent Drainage
		Strict NPO
		Place oxygen per nasal cannula and titrate to maintain O2 sat greater than 95%
		If patient intubated please place OG tube to suction
		Obtain emergency contact number for family, caregiver or bystander (preferably cell phone) to obtain further information or obtain consent for further treatment
		If t-PA has been started, document BP is less than 180/105 before departure. BP =
В.	Rev	iew with RN Prior to Departure
	1.	Type of stroke 🛛 Ischemic stroke 🖾 Hemorrhagic 🗔 Unknown
	2.	Initial physical assessment, Vital, ABC's, Neurologic Exam focusing on LOC and focal deficits
C.	Acu	te Management according to type of Stroke – Call Medical Control for management & patient concerns
	1.	For all stroke types: Target HR 60-120; target O2 sat greater than 95%
	2.	Ischemic with IV t-PA (Please refer to section E for post treatment recommendations).
		a. Confirm current t-PA Treatment Times and Amount of Infusion
		ii. IV t-PA Bolus Time started
		iii. t-PA infusion mgTime startedTime to completeTime t-PA stopped
		Maintain Target BP before treatment less than 180/105 – Follow BP protocol in sections F-2 . Call medical control if does not meet parameters.
	3.	Ischemic Stroke – Not eligible for thrombolytic therapy or type of stroke unknown
		a. Target BP < 220/120 follow BP management protocol
	4.	Hemorrhagic Stroke - Target BP less than 160/90 - follow BP management protocol in section F below.
D.	Dur	ing Transport
		Maintain HOB 15 - 30 degrees
		Continuous cardiac and pulse monitoring
	3.	Obtain and Record Vitals and Neurologic checks every 15 minutes
E.		t t-PA Treatment Recommendations
	1.	Monitor & document blood pressure and neuro checks every 15 minutes during and after t-PA
		administration. If deterioration in neurological status - turn off t-PA and notify Medical Control
		Monitoring for deterioration in neurological status from baseline; for example LOC, motor changes Monitor for sudden onset of headache, nausea and/or vomiting
		Monitor for sudden elevation in BP
		Titrate BP medication to maintain BP less than 180/105 after t-PA administration and during transport
		Stop BP medication infusion if SBP less than 140 or DBP less than 80
	2.	After t-PA infusion completed start LR or NS at 80 ml per hour to infuse remaining t-PA in the tubing
F.		Isport Treatment or Complication
		Hypotension- STOP t-PA infusion, HOB flat, Turn off any drips, administer 500ml fluid bolus (NS)
		Recheck for response
	2.	Hypertension— Check with medical control. If available give Labetalol 10 mg IV over 2 minutes, Recheck
	3.	in 5 minutes for response, may repeat one time Neurologic Deterioration —STOP t-PA, assess ABC's, obtain full set of vitals, Finger stick glucose (treat
		accordingly)
		Airway edema —STOP t-PA infusion and treat according to allergic reaction protocol
		Nausea and Vomiting – treat according to protocol
		Bleeding— Apply direct pressure to all bleeding cutaneous sites
Afte	er an	y treatment call St. Luke's to update and await further orders

Note: This is the hand-off documentation: Referring ED to EMS

Emergency department tools used by the stroke team nurses

Saint Luke's Hospital Kansas City, MO 64111

Emergency Department Stroke (Ischemic/ICH/SAH/TIA) Flowsheet

Code Stroke Documentation, Asso	essment and Treatment
DATE: Time: Time Last Known Well Date: Time: EMS Notification prior to arrival Yes No Transporting Service Transporting Service No Family contact number	IV t-PA Inclusion : If all boxes checked patient is a candidate No Hemorrhage on CT Onset < 4.5 hours
Discuss BP treatment with physician if BP > 185/110 and patient within tPA treatment window. Strict NPO until Nurse Dysphagia Screening completed unless NIHSS 0 ED Nurse Date Time CNN Date Time Additional Documentation :	acute treatment plan, benefits, and risks. Eligible for tPA but not given or started > 3 hrs from onset of symptoms: Patient returned to baseline Patient Stabilization Concerns prior to tPA Unable to determine eligibility – further assessment needed Patient/Family initial refusal Additional time to educate patient/family Fluctuating symptoms Coagulopathy Correction Time Initiated Vitamin K (phytonadione) Prothrombin Compliment FFP Platelets Factor VII Ventriculostomy Right Left Level of drain

Saint Luke's Hospital
Kansas City, MO 64111
Emergency Department Stroke (Ischemic/ICH/SAH/TIA) Flowsheet

NIH STROKE SCALE	Description	Date Time	Date Time	Date Time	Glasgow Co	ma Scal	е	
1a. Level of Consciousness	Alert-0		1		TIME	$ \top \top \top$		
	Drowsy – 1					++++	_	
	Stuporous – 2				EYE OPENING 1-4			
	Coma – 3					++++	_	
1b. LOC Questions	Answers Both Correctly – 0				VERBAL 1-5			
(Month, Age)	Answers One Correctly – 1						-	
	Answers Neither Correctly - 2				BEST MOTOR 1-6			
1c. LOC Commands	Performs Both Correctly – 0					+ $+$ $+$ $+$	-	
(Open, close eyes, make fist,	Performs One Correctly – 1				GCS TOTAL			
let go)	Performs Neither Correctly - 2							
2. Best Gaze	Normal – 0				If GCS ≤ 5 or notify M	FN 1-800-366	-679	1
(Eyes open-patient follows	Partial Gaze Palsy – 1				Reference #		-	
examiner's fingers/face)	Forced Deviation – 2							
3. Visual	No Visual Loss – 0				ABCD Score			
(Introduce visual stimulus (or	Partial Hemianopia – 1						-	
threat) to patient's visual field	Complete Hemianopia – 2				Risk Factor	Point	Sc	ore
quadrants)	Bilateral Hemianopia – 3				Age >60 years - 1 point	1		
4. Facial Palsy	Normal – 0				Ager of Jouro Thoma			
					BP: Systolic	1		
(Show teeth, raise eyebrows,	Minor Paralysis – 1				BP 140 or			
and squeezes eyes shut)	Partial Paralysis – 2	1			Diastolic≥90		1	
	Complete Paralysis – 3	-	-				-	
5a. Motor Left Arm	No Drift – 0	1			Clinical Features	2	1	
(Elevate extremity to 90 and	Drift – 1	1			Unilateral weakness		1	
score drift/movement)	Some Effort Against Gravity – 2				with or without speech		1	
	No Effort Against Gravity – 3				impairment			
	No Movement – 4				Speech impairment	1		
	Amputation, Joint Fusion – UN				without unilateral			
5b. Motor Right Arm	No Drift – 0				weakness			
(Elevate extremity to 90 and	Drift – 1						-	
score drift/movement)	Some Effort Against Gravity – 2				Duration of TIA ≥ 60	2		
	No Effort Against Gravity - 3				minutes			
	No Movement – 4				Diabetes history	1		
	Amputation, Joint Fusion – UN				Diabetes history	'		
6a. Motor Left Leg	No Drift – 0					Total Score		
(Elevate extremity to 30 and	Drift – 1					Total Ocore		
score drift/movement)	Some Effort Against Gravity – 2				Interventional Radiology			
score drift/movement)					□Ischemic Clot □Aneury	om Other		
	No Effort Against Gravity – 3					smillottier.		
	No Movement – 4				□Right □Left or □Both			
	Amputation, Joint Fusion – UN				Vessel Involved			
6b. Motor Right Leg	No Drift – 0				□ICA □T Occlusion □MCA			
(Elevate extremity to 30 and	Drift – 1				□ACA □Vertebral □Basila	r		
score drift/movement)	Some Effort Against Gravity – 2				□AComA □PCom □PCA			
	No Effort Against Gravity – 3				Treatmen			
	No Movement – 4				□IA t-PA			
	Amputation, Joint Fusion – UN				Mechanical retrieval			
7. Limb Ataxia	Absent-0				□ Stent placed			
	Present In One Limb – 1							
	Present In Two Limbs – 2							
8. Sensory	Normal-0							
(Pinprick to face, arm, trunk and leg	Mild to moderate Loss – 1				□Other			
compare side to side)	Severe to Total Loss – 2				Additional Documentation	n:		
9. Best Language	No Aphasia – 0							
(Name items, describe a picture,	Mild to Moderate Aphasia – 1							
read sentences)	Severe Aphasia – 2	1						
1000 0011010003/	Mute-3							
10. Dysarthria	Normal Articulation – 0	+		+				
Evaluate Speech Clarity by	Mild to Mod Dysarthria – 1							
	Near to Unintelligible – 2	1						_
patient repeating – Mama,		1			I			
tip-top , fifty-fifty, thanks,	Intubated/Other Barrier – UN	1						
huckleberry, baseball player)		-	-				_	
11. Extinction and Inattention	No Neglect – 0							
(Use prior testing to identify	Partial Neglect – 1							
neglect or double simultaneous	Complete/Profound Neglect – 2	1						
stimuli)								
	TOTAL SCORE-							
	Initials							
Initial Signature	Initial Signature	Initia	al Sig	nature				
	<u> </u>	—						

Patient Label:

Ka	aint Luke's Hospital Insas City, MO 64111 emic/ICH/SAH/TIA) Documentation Set
Nursing Code Stroke Docur	mentation, Assessment and Treatment
Strict NPO until Nurse Dysphagia Screening completed Page iSTAT Page Code Neuro Nurse Page Code Stroke Obtain appropriate order set Trine Last Known Well Market Nown Well Market Nown Vell Mar	Ask physician to sign order or place in computer. IV t-PA Inclusion Checklist: If all boxes checked patient is a candidate. \n> No Hemorrhage on CT _ Onset < 4.5 hours
IV t-PA prior to transfer Yes No O.9 mg/kg Bolus Total dose Time Given **Begin q. 15 minute vitals and neuro checks – record on Frequent Assessment section page 2. Ambulation Prior to Admission Independent Independent w/assistance Unable to ambulate Unknown Currently on Warfarin Currently on antiplatelets HOB Flat if Ischemic Elevated 30 for ICH/SAH	IV t-PA Dose:
Baseline Neuro Exam: NIHSS GCS Times: Labs drawn Reviewed	└── └── └── └── └── └── └── └── └── └──
Times: To CT Back to ED Reviewed Discuss BP treatment with physician if BP > 185/110 and patient within tPA treatment window. □ Two IVs if candidate for IV t-PA (20 g or larger bore IV required for CT Perfusion or CT Angiog Peripheral BG Istat Results: INR CR Platelets □ Patient received Dye Allergy Treatment T	gram)
ED Nurse Date Time CNN Date Time	The Golden Hour of Stroke Treatment Door to Physician Evaluation≤ 10min Door to Stroke Team Notification≤ 15min Door to CT≤ 25min Door to CT & Labs reviewed≤ 45min Door to needle (IV t-PA)≤ 60min
Code Neuro Nurse (CNN) Documentation :	



Saint Luke's Hospital Kansas City, MO 64111

ER Record

Code Stroke (Ischemic/ICH/SAH/TIA) Documentation Set

NIH STROKE SCALE	Description	Date Time	Date Time	Date Time	Glasgow Coma Scale
1a. Level of Consciousness	Alert-0	inne	- mie	Time	TIME
	Drowsy – 1				
	Stuporous – 2				EYE OPENING 1-4
	Coma – 3				
1b. LOC Questions	Answers Both Correctly – 0				VERBAL 1-5
(Month, Age)	Answers One Correctly – 1				VERDINE 10
	Answers Neither Correctly - 2				
1c. LOC Commands	Performs Both Correctly – 0				BEST MOTOR 1-6
(Open, close eyes, make fist, let go)	Performs One Correctly – 1				
	Performs Neither Correctly – 2				GCS TOTAL
2. Best Gaze	Normal – 0				
(Eyes open-patient follows	Partial Gaze Palsy – 1				If GCS ≤ 5 or notify MTN 1-800-366-6791
examiner's fingers/face)	Forced Deviation – 2				Reference #
3. Visual	No Visual Loss – 0				From Assessment Cost Millifer Costing
(Introduce visual stimulus	Partial Hemianopia – 1				Freq Assessment – See NIH for Scoring
(or threat) to patient's visual field	Complete Hemianopia – 2				TIME
quadrants) 4. Facial Palsy	Bilateral Hemianopia – 3 Normal – 0				BLOOD PRESSURE
	Minor Paralysis – 1				BLOOD PRESSURE
(Show teeth, raise eyebrows, and					
squeezes eyes shut)	Partial Paralysis – 2 Complete Paralysis – 3				LOC 0-3 (1a)
5a. Motor Left Arm	Complete Paralysis – 3 No Drift – 0				┨┠─────┼┼┼┼┼┼
(Elevate extremity to 90° and score	Drift-1				LOC Commands 0-2 (1c)
drift/movement)	Some Effort Against Gravity – 2				
diit/iiovenent)	No Effort Against Gravity – 3				FACIAL PALSY 0-3 (4)
	No Movement – 4				
	Amputation, Joint Fusion – UN				MOTOR (5) RA
5b. Motor Right Arm	No Drift – 0	<u> </u>			ARMS 0-4
(Elevate extremity to 90° and score	Drift-1				LA
drift/movement)	Some Effort Against Gravity - 2				MOTOR (6) RL
	No Effort Against Gravity – 3				LEGS 0-4
	No Movement – 4				LLG3 014
	Amputation, Joint Fusion – UN				
6a. Motor Left Leg	No Drift – 0				BEST LANGUAGE 0-3 (9)
(Elevate extremity to 30° and score	Drift – 1				
drift/movement)	Some Effort Against Gravity - 2				PUPIL SIZE R
	No Effort Against Gravity – 3				REACTION
	No Movement-4				
	Amputation, Joint Fusion – UN				
6b. Motor Right Leg	No Drift – 0				Interventional Radiology
(Elevate extremity to 30° and score	Drift – 1				🗆 Ischemic Clot 🗆 Aneurysm 🗆 Other
drift/movement)	Some Effort Against Gravity – 2				□ Right □ Left or □ Both
	No Effort Against Gravity – 3				
	No Movement – 4				Vessel Involved
	Amputation, Joint Fusion – UN				□ ICA □ T Occlusion □ Middle Cerebral Artery
7. Limb Ataxia	Absent-0				□ Anterior Cerebral Artery □ Vertebral □ Basilar
	Present In One Limb – 1				-
	Present In Two Limbs – 2				AComA D PCom
8. Sensory	Normal – 0				Treatment
(Pinprick to face, arm, trunk and	Mild to moderate Loss – 1				□ IA t-PA
leg compare side to side)	Severe to Total Loss – 2				
9. Best Language	No Aphasia – 0				Mechanical clot retrieval
(Name items, describe a picture, read sentences)	Mild to Moderate Aphasia – 1	1			□ Angioplasty
reau sentences)	Severe Aphasia – 2				Stent placed
10. Dysarthria	Mute – 3 Normal Articulation – 0				Coiling
Evaluate Speech Clarity by patient	Mild to Mod Dysarthria – 1	1			
repeating - Mama, tip-top,	Near to Unintelligible – 2				Other
fifty-fifty, thanks, huckleberry,	Intubated/Other Barrier –UN				
baseball player)	mubaleu/Other banter-UN				No treatment
11. Extinction and Inattention	No Neglect – 0	+			Departure From Specials
(Use prior testing to identify neglect	Partial Neglect – 1				
or double simultaneous stimuli)	Complete/Profound Neglect – 2				Family informed of procedure results and destination Time Via
,	TOTAL SCORE-Initials				
Initial Signature	TOTAL SCORE–Initials		al Signa		MICU # NSICU # Other

Appendix B: Management of subarachnoid hemorrhage

Order sets for the management of subarachnoid hemorrhage



Saint Luke's Care Saint Luke's Health System

Physician Orders Physician Order Write Down and Read Back for all Verbal Orders ANOTHER MEDICATION SIMILAR IN FORM AND ACTION MAY BE DISPENSED PER MEDICAL STAFF POLICY THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT ED Subarachnoid Hemorrhagic Stroke Orders Date: Time[.] Orders that are checked will be implemented - Initial the bottom of the page if any checks made. Additions, Deletions or Modifications (including strike through) of orders (line items) must be individually initialed. Admit to NSICU 2. IN NO IV tPA indicated due to subarachnoid hemorrhage Time last known well 3. 4. Nursing: I Triage Level: 2 NIH Stroke Scale Assessment BP and neurological assessment with pupil exam q. 15 minutes and place print out on chart (from ER computer) AND Use Frequent Neuro Assessment Form (SYS-194) NPO - no meds by mouth until dysphagia screen completed by Code Neuro Nurse S Foley if patient intubated, otherwise avoid ☑ HOB up 30° if SBP > 90 🛛 Urine for UA and microanalysis and if positive send for reflex culture IF foley placed-obtain upon insertion Notify Neurosurgery/APN I Hunt-Hess score upon admission. Scale and scoring below 1 = Asymptomatic, mild headache, slight nuchal rigidity 2 = Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy 3 = Drowsiness / confusion, mild focal neurologic deficit 4 = Stupor, moderate-severe hemiparesis 5 = Coma, decerebrate posturing Score = 🗆 1 🛛 2 🗔 3 🗔 4 🗔 5 ☑ Maintain O₂ at 2 – 4 L / NC, titrate to sat > 95% 5. IV Fluids: Normal Saline @ 100 mL/hr 6. Labs and Diagnostics: Stat non-contrast CT head scan--If not already done □ CTA Cerebral Angiogram/Arteriogram ⊠ ECG – If not already done STAT Stroke Panel (BMP, CBC, Coag screen, Troponin I, Mg)-If not already done STAT HCG / UCG for all women (10-50 years old) unless they have had surgical hysterectomy or are post menopausal Target SBP Parameter: less than 140 7. Hypertension Management Labetalol 10 mg IV over 1 min. May repeat every 15 min to a max dose of 40 mg. Do not use if HR less than 60 If blood pressure controlled, may give Labetalol 10 mg IV q 1 hour PRN. If unable to reach target start nicardipine. □ Nicardipine 5 mg/hr IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/hr q. 15 min to maximum of 15 mg/hr. If evidence or suspicion of elevated ICP discuss with neurosurgeon consideration of ICP monitoring and keep MAP > 80mmHg OR CPP > 60 mm Hg Other BP Target range 8. ☐ ICU Intensive Glucose Control order (SYS-1299) If glucose greater than 140 Physician Initials: (Continued) DANGEROUS ABBREVIATIONS A LLERGIES / INTOLERANCES - DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, g.o.d. or QOD, U or u, IU

Apply Patient Label In This Box Never_use zero after decimal Do Not Cover Up Below Barcode Height. point (1.0 mg) _____ 🗆 kg 🗆 grams Weight_ Always_use zero before decimal Latex Allergy Yes D No D point (0.5 ma)



Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

ANOTHER MEDICATION SIMILAR IN FORM AND ACTION MAY BE DISPENSED PER MEDICAL STAFF POLICY THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT

Orders that are checked will be implemented. Additions, Deletions or Modifications (including strike through) of orders (line items) must be individually initialed.

ED Physician Risk Stratification based on patient history of anti-coagulation

9. Correct Coagulopathy Immediately in ED unless contraindicated

Discontinue any antithrombotic or anticoagulant medication

- If patient is on warfarin and INR is elevated (greater than 1.4) Give Vitamin K (phytonadione) 10 mg IV in 50 mL D5W over 1 hour, begin STAT if INR less than 1.7. If INR greater than 1.4
 - Give 2 units of FFP.
 - Check INR 30 minutes after infusion complete
 - ☑ If INR remains greater than 1.4 Repeat above and notify physician

10. Medications

- Sondansetron (Zofran) 4-8 mg IV q. 6 hours PRN nausea
- acetaminophen 650 mg PR q. 6 hours for temp greater than 99.5° F
- Blevetiracetam (Keppra) 1000 mg IV Loading Dose X 1
- □ fentanyl 25-50 mcg IV q.1 hr PRN pain

Other

11. Initiate Subarachnoid Hemorrhage Inpatient Mgt (SYS-1291)

Date: _____ DANGEROUS ABBREVIATIONS

- DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, q.o.d. or QOD, U or u, IU ____Time:

Apply Patient Label In This Box

Height	
Weight	_ 🗆 kg 🗆 grams

Latex Allergy Yes D No D

Physician Signature:

ALLERGIES / INTOLERANCES



 Never use zero after decimal point (1.0 mg)
 Do Not Cover Up Below Barcode

 Always use zero before decimal point (0.5 mg)
 Do Not Cover Up Below Barcode





Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

		White Down	
	ANOTHER MEDIC ATION SIMIL AR	IN FORM AND ACTION MAY BE DISPENSED PER M	MEDIC AL STAFF POLICY
	THIS DOCUMENT IS INTENDE	D TO BE INDIVIDU ALIZED AS NEEDED FOR ANY S	PECIFIC PATIENT
	Suba	rachnoid Hemorrhage Initial Care	
Date	:: Time:		
	ers that are checked will be implemented		
lele	tions or modifications (including strike	through) of orders (line items) must	be individually initialed.
1.	□ eICU®		
2.	Allergies:		
3.	Nursing:		
	 ☑ Vital signs q. 15 min 4, then q. 30 min ☑ Neurological assessment q. 15 min 4 		
	ECG upon admission if not done in ED		
	Initiate SAH Clinical Path, IPOC or app		
	Keep HOB elevated at 30 degrees		
	□ Strict bedrest □ Normothermia order set (SYS-1300). F	Physician to complete and sign	
		RN to complete if not done.	
	1 = Asymptomatic, mild headache, slig		
		uchal rigidity, no neurologic deficit other t	han cranial nerve palsy
	3 = Drowsiness / confusion, mild foc 4 = Stupor, moderate-severe hemipare		
	5 = Coma, decerebrate posturing	-313	
	Score = 0 1 0 2 0 3 0 4 0 5		
	Complete NIHSS per SAH clinical path		, and at discharge)
	 Initiate Stroke Risk Reduction Plan she Instruct patient about Aneurysm preca 		
	Provide patient/family with designated		
	Provide a copy of the Completed Strok		structions to all patients
4.	Ventriculostomy: drain at	cm H ₂ O level – clamp for travel or p	osition changes
5.	\square Titrate O ₂ to keep SpO ₂ > 95%. Notify	physician if O ₂ > 6 L/NC	
6.	□ Foley to DD. Obtain C&S upon insertion		
	Urine sent for UA and microanalysis.	Reflex urine culture if positive	
7.	⊠ I & O		
8.	DVT Precautions: SCD's	Graduated compression	stockings
9.	Place NG tube		
0.	Stroke Dysphagia Screening (SYS-30		
	If NIHSS = 0, choose diet and follow order \Box Low fat low Chol 2 gram Na (Simpl	y healthy) OR Consistent Carb 75 g (18	00 ADA) if diabetic
	If NIHSS greater than 0, RN to complete Str		
	Abnormal: 🛛 🛛 Keep NPO, including me		
		pathology (SLP) for swallow evaluation/n	
		n Na (Simply healthy) OR Consistent C diet modifications on all patients	arb 75 g (1800 ADA) if diabetic
Phys	ician Initials:		(Continued)
	rgies / Intoler ances	DANGEROUS ABBREVIATIONS	(continued)
(LLL)		– DO NOT USE!	
		MS, MSO4, MgSO4, q.d. or QD,	
		q.o.d. or QOD, U or u, IU	Apply Patient Label In This Box
		<u>Never</u> use zero after decimal point (1.0 mg)	Do Not Cover Up Below Barcod
	ht 🗆 kg 🗆 grams		
		Always_use zero before decimal	
-	Allergy Yes 🗆 No 🗆	<u>Always</u> use zero before decimal point (0.5 mg)	

Physician Orders Write Down and Read Back for all Verbal O ation similar in form and action may be dispensed per medical staff policy ent is intended to be individualized as needed for any specific patient
ATION SIMILAR IN FORM AND ACTION MAY BE DISPENSED PER MEDICAL STAFF POLICY
NATIO NATING TO BE NUMBER TO DED TO DECEMPT FOR ANY ORFOLD DATENT
Subarachnoid Hemorrhage Initial Care (Continued)
implemented – Initial the bottom of the page if any checks made. Addition g strike through) of orders (line items) must be individually initialed.
Irs and PRN.
lium Chloride via Central Line ONLY if not already infusing from ED.
is than 140
B/P parameter
n. May repeat every 15 min to a max dose of 40 mg. Do not use if HR less than 6 give Labetalol 10 mg IV g. 1 hour PRN
on as initial dose; titrate to ordered BP parameters by increasing 2.5 mg/hour er
g/hr
al line placement
agement Point of Care Glucose STAT upon admission
G Value upon Admission
s less than 140 mg/dL and a history of diabetes, continue blood glucose checks at NPO or receiving continuous enteral nutrition.
and BG less than 140 obtain BG q. 4 hour 4. If BG remains less than 140mg/dL
Intravenous Insulin Glucose Control orders SYS-1299
on: Obtain STAT BG value upon admission
or greater, initiate Basal Bolus Insulin Correction Algorithm (SYS-1140)
check BG q. 4 hours X 4, if BG value increases to >180 mg/dL, initiate Basal Bo
i thm (SYS-1140) liabetes and BG less than 140 mg/dL
obtain BG q. 6 hours: 6-12-6-12 if NPO or receiving continuous enteral nutrition.
obtain BG AC and HS if patient is eating,
d BG less than 140 obtain BG q. 4 hour x 4.If rises above 140 refer to above treatments
urs if on 3% Sodium Chloride infusion
on 3% Sodium Chloride infusion
than 320 OR Na greater than or equal to 160 meq/L, HOLD 3% Sodium Chlo
screen,Troponin I, Mg) STAT if not done in ER
⊠HGA _{tc}
⊠ Phosphorus every day
CT Head Scan non-contrast in AM: Indication: hemorrhage evaluation
eurysm
(Continued)
DANGEROUS ABBREVIATIONS –
DO NOT USE!
MS, MSO4, MgSO4, q.d. or QD,
q.o.d. or QOD, U or u, IU Apply Patient Label In This Bo
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Saint Luke's Care Saint Luke's Health System

Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

ANOTHER MEDICATION SIMILAR IN F	FORM AND ACTION MAY BE DISPENSED PER MED	ICAL STAFF POLICY			
THIS DOCUMENT IS INTENDED TO	D BE INDIVIDUALIZED AS NEEDED FOR ANY SPEC	IFIC PATIENT			
Subarachnoid	Hemorrhage Initial Care (Continued)				
Orders that are checked will be implemented. Ad (line items) must be individually initialed.	ditions, deletions or modifications (including strike through) of orders			
17. Smoking Cessation					
Give smoking cessation education to pa	tient (if has used tobacco in the past 12	months)			
18. Scheduled medications:					
⊠docusate (Colace) 100 mg PO BID, capsule o	r liquid depending on patient's ability to	take PO			
⊠ famotidine 20 mg IV or PO BID (pharmacist may adjust for CrCl of less than 50 mL/min)-If patient not already on proton pump inhibitor)					
	⊠levetiracetam (Keppra) 500 mg IV or PO q 12 hours 7 days				
⊠ nimodipine 60 mg PO/NG q 4 hours times 21	,				
notify pharmacy if 30 mg PO/NG q 2 hours	, i				
total administration of nimodipine to be 21	days				
CHOOSE ONE:					
□ pravastatin 40 mg PO/NG q HS times 21 days	3				
OR .					
Continue home statin	PO/NG q HS				
9. PRN Medications:					
🖾 docusate sodium/sennosides 50/8.6 mg (Senokot-S) 2 tabs q HS PRN, give if no BM in last 24 hours.					
⊠ bisacodyl suppository 10 mg PR q day PRN if no BM in last 24 hours and unable to take PO					
Electrolyte Replacement orders (SYS-1059) -physician to complete and sign					
acetaminophen 650 mg PO, elixir, or supp q. 4 hrs PRN T greater than 99.5 F (depending on patient's ability to take PO)					
□ ondansetron 4-8 mg IV q. 6 hrs PRN nausea					
hydrocodone and acetaminophen 5/325 mg 1		t can take PO			
Morphine sulfate 2.5 - 5 mg IV q. 2 hrs PRN r					
□ Fentanyl 25-50 mcg IV q. 1 hr PRN severe p	ain				
20. Consultation	_				
		_Date/Time:			
Hospitalist for management of HTN, glucose					
Notified: Name:Date/		5.4.7			
Pulmonary (for Ventilated Patients ONLY)		Date/Time:			
Rehabilitation Medicine (enter on computer –	do not call office)	_(indication)			
PT and OT to evaluate and treat					
Social Services Worker for discharge plannin	9				
Physician Signature:	Date:	Time:			
Allergies / Intolerances	DANGEROUS ABBREVIATIONS – DO NOT USE!				
	MS, MSO4, MgSO4, q.d. or QD,				
-lah4	q.o.d. or QOD, U or u, IU	Apply Patient Label In This Box			
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Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

_		e (SAH) Post Aneurysm Clipping /	Coming Orders	
Dat				
	lers that are checked will be implemented Nodifications (including strike through) of o			
1.	Neurosurgeon: Dr		_	
2.	Diagnosis: Subarachnoid Hemorrhage			
3.	⊠ Vital signs q. 15 min 4, then q. 30 min 2			
Neurological assessment q. 15 min 4, then q. 30 min 2, then q. 1 hour				
	For Coiling ⊠ Check groin site and distal puls Keep □ R □ L leg straight for		hit routine.	
4	Complete NIHSS per SAH clinical path (A			
	_	$_$ cm H ₂ O level – clamp for travel or positic	n changes	
	Allergies:		in onunges	
	HOB elevated at 30 degrees			
	☐ HOB elevated at 30 degrees ☑ Mobility according to clinical path, progress patient activity daily			
8.	. ⊠I&O			
	⊠ Foley to DD. Remove as soon as possible after surgery.			
9.	D. In NG tube to administer medications if patient NPO			
10.). Itrate O ₂ to keep SpO ₂ > 95%, notify physician if O ₂ > 6 L/NC			
11.	Initiate SAH Clinical Path AND Stroke Ris	k Reduction Plan sheet		
2.	Provide patient/family with designated stro	ke education materials		
13.	Stroke Dysphagia Screening (SYS-300)			
	If NIHSS = 0, choose diet and follow orders below:			
	Low fat, low Chol, 2 gram Na (Simply healthy) OR Consistent Carb 75 g (1800 ADA) if diabetic			
	Abnormal: Keep NPO, including meds	If NIHSS greater than 0, RN to complete Stroke Dysphagia Screening Abnormal: Keep NPO, including meds and		
	☐ Order speech language pathology (SLP) for swallow evaluation/management.			
	Normal: Low fat, low Chol, 2 gram N Consult SLP for further diet	a (Simply healthy) OR Consistent C	arb 75 g (1800 ADA) if diabetic	
1/1	IV and Fluid Management	modifications on all patients		
1-4.	□ IV Fluids			
	□ Saline lock IV flush q. 8 hrs and after usage			
	PICC line placement			
15.	DVT Prophylaxis:			
	Graduated compression stockings			
Phy	vsician Initials:		(Continued)	
ALL	ergies / Intolerances	DANGEROUS ABBREVIATIONS		
		- DO NOT USE!		
		MS, MSO4, MgSO4, q.d. or QD,		
		q.o.d. or QOD, U or u, IU <u>Never</u> use zero after decimal	Apply Patient Label In This Bo Do Not Cover Up Below Barcod	
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	x Allergy Yes □ No □	1.		

		Saint Luke's Care Saint Luke's Health System	
	ician Order	Physician Orders	
Physi	cian Order	Write Down	and Read Back for all Verbal Orders
	ANOTHER MEDICATION SIMILAR	N FORM AND ACTION MAY BE DISPENSED PER MEDIC	CAL STAFF POLICY
	THIS DOCUMENT IS INTENDED	TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECI	FIC PATIENT
	Subarachnoid Hemorrhage (SA	AH) Post Aneurysm Clipping / Coiling Or	ders (Continued)
1		Initial the bottom of the page if any che	
Modification	s (including strike through) of orde	rs (line items) must be individually initia	aled.
10 Lab and	Disersation		
16. Lab and	-		
1	trolytes serum q. day i upon admission if not completed in El	n	
	Scan Head non-contrast in AM: Indica		
🖾 Tran	scranial Doppler daily 2 weeks		
□ Othe	ər:	Other:	
17. Consul			
Card	bitalist for management of HTN, glucos		te/Time:
	mailstroi management of HTN, glucos	Notified: Name:	Date/Time:
	onary (Ventilator Management)		
1	abilitation Medicine (enter on computer	- do not call office)	(indication)
1	INDERCENT IN THE INDERCENT IN THE INPUT INTERPORT INTERPOR		
	3P Parameter: Keep SBP greater than	and less than	
°	pertension or to maintain BP paran		
		q. 15 min to a max dose of 40 mg. Do not	use if HR less than 60
1	ood pressure controlled, may give Lab	etalol 10 mg IV q. 1 hr PRN	
	o response to Labetalol, start:		
1	rdipine 5 mg/nr iv infusion as initi iinutes to a max dose 15 mg/hr	al dose; titrate to ordered BP paramet	ers by increasing 2.5 mg/nour q.
	potension or to maintain B/P param	eters	
СНОО	SE ONLY ONE:		
1	nylephrine 20 mcg/min IV infusion as i	nitial dose; titrate to ordered BP parameters	s with max dose of 300 mcg/min
	ninenhrine 2 mcc /min IV infusion as i	nitial dose; titrate to ordered BP parameter	s with may dose of 30 mcg/min
	oactive drips started consult anest		
	e Blood Glucose (BG) Management		
	t BG upon Admission		
	If < 140 mg/dL and has NO history of	f diabetes,	
		ns < 140 mg/dL, no further action necessar	у
b.	If <140 mg/dL and HAS history of dia	ibetes,	
	 BG AC and HS if eating OR BG q. 6 hrs if NPO or receiv 	ing continuous enteral nutrition	
C.		ntensive Glucose Control orders (SYS-1299). Physician to complete and sign
21. Smokin	g Cessation:		
□ Nico	tine transdermal 14 mg patch daily if p	patient smokes less than 10 cigarettes per o	lay
1		patient smokes 10 or more cigarettes a day	
		nt if has used tobacco in the past 12 month	
Physician In	itials:	1	(Continued)
ALLERGIES / IN	TOLERANCES	DANGEROUS ABBREVIATIONS	
		- DO NOT USE! MS, MSO4, MgSO4, q.d. or QD,	
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Height		<u>Never</u> use zero after decimal	Do Not Cover Up Below Barcode
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Saint Luke's Care Saint Luke's Health System

Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

	ANOTHER MEDICATION SIMILAR IN	FORM AND ACTION MAY BE DISPENSED PER MEDIC	CAL STAFF POLICY	
	THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT			
Subarachnoid Hemorrhage (SAH) Post Aneurysm Clipping / Coiling Orders (Continued)				
	ers that are checked will be implemented. A items) must be individually initialed.	dditions, Deletions or Modifications (ii	ncluding strike through) of order	
2.	Scheduled medications:			
	 ☑ Docusate (Colace) 100 mg PO BID, capsul ☑ Famotidine 20 mg IV or PO BID (pharma inhibitor 			
	⊠ nimodipine 60 mg PO/NG q. 4 hrs times 21 days			
	notify pharmacy if 30 mg PO/NG q. 2	hrs needed due to hypotension		
	total administration of nimodipine to be	e 21 days		
	Dexamethasone 6 MG IV q. 6 hrs			
	Fosphenytoin loading dose of 15 mg/kg IV			
	Fosphenytoin 100 mg IV q. 8 hrs if << 80 kg	OR Fosphenytoin 200 mg IV q. 12	hrs ≥ 80 kg. Pt wt =kg	
	Levetiracetam 500 MG IV or PO q. 12 hrs			
	Albumin 5% 250 mL IV q. 8 hrs			
	CHOOSE ONE:			
	Pravastatin 40 mg PO/NG q. HS times 21 c	lays OR LI Continue home statin	PO/NG q H	
	Bisacodyl suppository 10 mg PR q. day if r Electrolyte Replacement orders (SYS-1) Acetaminophen 650 mg PO, elixir, or suppo Ondansetron 4-8 mg IV q. 6 hrs PRN naus Hydrocodone and acetaminophen 5/325 m Morphine sulfate 2.5-5 mg IV q. 2 hrs PRN Fentanyl 25-50 mcg IV q. 1 hour PRN sev	159). Physician to sign sitory q. 4 hrs PRN for T greater than 99.5 ea or emesis g 1 tab PO q. 4 hour PRN mild pain if pati I moderate pain		
4.				
5.				
hvs	sician Signature:	Date:	Time:	
	RGIES / INTOLEBANCES	DANGEROUS ABBREVIATIONS		
eigh	t InfoLerrances	- DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, q.o.d. or QOD, U or u, IU <u>Never</u> use zero after decimal point (1.0 mg)	Apply Patient Label In This Bo Do Not Cover Up Below Barcoc	
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University of Cincinnati stroke team Guideline for Management of Subarachnoid Hemorrhage/Vasospasm



General information

Document Title:Management of Subarachnoid Hemorrhage/VasospasmPurpose:Define treatment options for managing subarachnoid
hemorrhageObjectives:1. Establish parameters for treatment
2. Manage complications of subarachnoid hemorrhage
3. Optimize treatment of vasospasmAuthors:Mario Zuccarello, MD; Andrew Ringer, MD; Opeolu Adeoye MD
Kristine Atchley ACNP-BC; Erin Silva ACNP-BCOriginal Date:May 29, 2006; updated February 28, 2013

Content of document

Patients with subarachnoid hemorrhage (SAH) sustain the primary insult at the initial time of bleed. Secondary damage to the brain occurs as a result of complications associated with the initial insult. These guidelines are developed to provide team members with information on

- a) Managing patients after subarachnoid hemorrhage
- b) Suggesting interventions to treat the associated complications

- I. Assessment
 - A. Clinical assessment includes

WFNS. Glasgow Coma Scale (GCS). Cranial nerve exam (pupillary response, extraocular movements, facial symmetry, corneal and gag reflexes); motor strength; motor tone; sensory assessment; and vital signs. Note any seizure activity.

Other assessment scores: Hunt and Hess score, Fisher CT grade

- B. Diagnostic assessment of subarachnoid hemorrhage may include
 - 1. Brain imaging: CT (computed tomography), MRI (magnetic resonance imaging)
 - 2. Cerebral vascular imaging: angiogram, CTA (computed tomographic angiography), MRA (magnetic resonance angiography), MRV (magnetic resonance venography)
 - 3. Neuromonitoring options: Intracranial pressure (ICP); LICOX brain tissue oxygen (PbtO₂); EEG (electroencephalogram)
 - 4. Neurovascular monitoring options: Transcranial Dopplers (TCDs); cerebral blood flow studies
- II. Initial Management
 - A. Emergency department (ED) or on intensive care unit (ICU) admission

Implement initial general resuscitation protocols. Appropriate interventions include:

- 1. Airway management:
 - a. Supplemental O_2 to maintain $SaO_2 \ge 95\%$
 - b. Intubate for WFNS = 4 or an inability to protect airway: Use RSI Protocol. Titrate ventilator to maintain $PaO_2 \ge 100 \text{ mm Hg}$ and $PaCO_2$ normalized.
- 2. Circulation
 - a. Establish minimum of 2 large-bore intravenous lines.
 - b. Place nasogastric (NG) tube/Foley if indicated.
 - c. Draw initial assessment labs (CBC [complete blood count], renal profile, cardiac enzymes, TEG profile, aspirin (ASA) and Plavix assay).
 - d. Place central intravenous catheter and arterial line if indicated during initial care. If patient has limited peripheral access or multiple infusions or blood draws requiring additional access, may consider central intravenous catheter placement.
- 3. Diagnosis/assessment
 - a. Arrange for appropriate diagnostic imaging.
 - b. Obtain baseline physical exam/assessment, including WFNS and GCS.

- 4. Hemodynamic management:
 - a. Avoid hypotension and hypertension (i.e., goal systolic blood pressure [SBP] < 140 mm Hg).
 - b. Goal mean arterial pressure $(MAP) \le 70 \text{ mm Hg}$.
- 5. *Sedatives and analgesics* as indicated for mechanical ventilation. Preferred agents based on desired goal:
 - a. For *sedation*: use propofol for mechanically ventilated patients.
 - b. For *analgesia*: use fentanyl.
- 6. Management options for signs of intracranial hypertension or herniation:
 - a. Hyperventilation (temporary)
 - b. Mannitol
 - c. Hypertonic saline per NSICU protocol
 - d. Consider placement of ICP (intracranial pressure) monitor/ventriculostomy
 - i. Preferred device: *ventriculostomy*. If in ED, transfer to ICU or operating room (OR) for placement.
- 7. Management options for signs of hydrocephalus or intraventricular hemorrhage:
 - a. Insert *ventriculostomy*. If in ED, transfer to ICU or OR for placement.
 - b. Keep ventriculostomy open to drain as specified by neurosurgery.
 - c. Monitor ICP every hour; goal ICP ≤ 20 mm Hg.
- 8. Seizure prophylaxis:
 - a. Keppra with initial dose of 1,000 mg PO/IV BID
 - b. Other antiepileptics may be indicated based on clinical situation.
- 9. Calcium channel blockers:
 - a. Start nimodipine 60 mg PO q 4 hours × 21 days or while hospitalized.
- B. Intensive care unit: preoperatively:
 - 1. Review all initial care needs from Section II.A.
 - a. Place arterial line/central lines if clinically indicated.
 - b. Initiate analgesia; if mechanically ventilated, monitor for effects on MAP.
 - 2. Respiratory management:
 - a. Maintain $SaO_2 \ge 95\%$ with supplemental O_2 as needed.
 - b. If intubated, goal $PaCO_2 = 35-45$ mm Hg.
 - c. If $PbtO_2$ monitor placed, titrate ventilator to maintain $PbtO_2 \ge 20$ mm Hg.

- 3. Neurological examinations:
 - a. Nursing documentation of hourly vital signs and GCS. NIHSS (National Institutes of Health Stroke Scale) documentation every 12 hours. Cranial nerve exam documented every shift.
- 4. Hemodynamic management:
 - a. Maintain MAP ≤ 70 mm Hg or SBP < 140 mm Hg with antihypertensive agents until etiology of SAH determined and causative aneurysm is secured. Agents for blood pressure control include intravenous labetalol as needed or nicardipine drip.
 - b. Administer fluids. *Avoid hypervolemia;* fluid balance goal is a range of 0–500 mL positive every 24 hours. Goal is euvolemia.
 - c. Fluid choice: normal saline with or without 20 meg KCl.
- 5. Ventriculostomy/acute phase ICP management:
 - a. Keep ventriculostomy open to drain as specified by neurosurgery.
 - b. Monitor ICP every hour; goal ICP ≤ 20 mm Hg.
 - c. Neurosurgical resident should be notified for ICP elevation.
 - d. Management options for signs of intracranial hypertension or herniation:
 - i. Hyperventilation (temporary)
 - ii. Mannitol
 - iii. Hypertonic saline per NSICU protocol
- 6. Seizure prophylaxis:
 - a. Keppra at 1,000 mg PO/IV BID.
 - b. Other antiepileptics may be indicated based on clinical situation.
- 7. General care issues:
 - a. Glucose: Initiate treatment for hyperglycemia. Goal glucose < 180 mg/dL.
 - b. Sodium: Maintain in normal range (135–146 mEq/L).
 - c. Magnesium: Maintain $\geq 1.8 \text{ mg/dL}$.
 - d. Hematologic: reverse coagulopathy (FFP/cryoprecipitate/ platelets/vitamin K).
 - e. Temperature: Goal is normothermia. Culture per NSICU protocol for fever ≥ 101.5°F.
 - f. Nutrition: Address within first 24–48 hours after admission. Keep NPO (nothing by mouth) while awaiting imaging and neurosurgical plan.
 - g. Nimodipine 60 mg PO q 4 hours \times 21 days while hospitalized.
- 8. Prepare for any neurosurgical procedures.

- III. Aneurysm Treatment/Neurosurgical Management
 - A. Surgical clipping or endovascular treatment of a ruptured aneurysm will occur as early as feasible.
 - B. Goal is to secure aneurysm within 24 hours of presentation to UCMC (University of Cincinnati Medical Center).
 - 1. Complete obliteration of the aneurysm is the goal of treatment.
 - 2. Determination of aneurysm treatment is a multidisciplinary decision made by cerebrovascular specialists based on characteristics and condition of the patient and aneurysm.
 - C. If the patient presents in a delayed fashion or is found to have vasospasm on admission, treatment of the aneurysm may be delayed until it is deemed to be safe by the cerebrovascular specialist.
- IV. ICU Care after Aneurysm Is Secured
 - A. General management:
 - 1. Hemodynamic management:
 - a. *Do not initiate vasospasm treatment empirically.* Vasospasm treatment is based on the clinical exam, TCD results, and radiographic findings.
 - b. Maintain MAP 70–100 mm Hg. Track medication effects on MAP.
 - c. Closely monitor fluid status. (Refer to Table A.1.)
 - i. Daily body weights and fluid balance calculations with a goal range of 0–500 mL positive every 24 hours
 - ii. Fluid choice: normal saline with or without 20 mEq KCl
 - 2. Respiratory management:
 - a. Maintain $SaO_2 \ge 95\%$ with supplemental O_2 as needed.
 - b. If intubated, goal $PaCO_2 = 35-45$ mm Hg.
 - c. If $PbtO_2$ monitor placed, titrate therapies to maintain $PbtO_2 \ge 20$ mm Hg.
 - 3. Neurological examinations:
 - a. Nursing documentation of hourly vital signs and GCS while in the NSICU. NIHSS documented every 12 hours. Cranial nerve exam documented every shift.
 - 4. Ventriculostomy/ICP management:
 - a. Keep ventriculostomy open to drain at 5–10 mm Hg or as specified by neurosurgery.
 - b. Monitor ICP every hour; goal ICP \leq 20 mm Hg.
 - c. Neurosurgical resident to be notified for ICP elevation.
 - d. Management options for ICP elevations and herniation:
 - i. Ensure patent drainage from EVD
 - ii. Mild hyperventilation

- iii. Hypertonic saline per NSICU protocol
- iv. Mannitol
- 5. Seizure prophylaxis:
 - a. Continue Keppra for total of 3 days unless GCS less than 8, then total of 7 days, unless clinically indicated to continue treatment.
 - In patients with GCS < 8, continuous EEG monitoring × 72 hours.
 - c. Other antiepileptics may be indicated based on clinical situation.
- 6. *Corticosteroids*: There is no indication for corticosteroids after aneurysmal clipping.
- 7. *TCDs*:
 - $a. \ Baseline as soon as possible or at days 1-3 posthemorrhage.$
 - b. Surveillance every Monday, Wednesday, and Friday unless patient has exam change or severely elevated TCDs.
- 8. General care issues:
 - a. Glucose: Treat hyperglycemia. Goal glucose < 180 mg/dL.
 - b. Sodium: Maintain in normal range (135–146 mEq/L).
 - c. Magnesium: Maintain $\geq 1.8 \text{ mg/dL}$.
 - d. Temperature: Goal is normothermia. Culture per NSICU protocol for fever ≥ 101.5°F.
 - e. Nutrition: Address by 24–48 hours after admission.
 - f. Lower-extremity Dopplers in nonambulatory patients per NSICU protocol: screening at 72 hours, then weekly.
 - g. Nimodipine 60 mg PO q 4 hours \times 21 days or while hospitalized.
 - h. Early physical, occupational, and speech therapy consultations.
 - i. Consult to social services.
 - j. Consult other ancillary departments, such as nutrition services, wound care, diabetes education, and so on as indicated.
- 9. Multidisciplinary management:
 - a. Patients admitted to the NSICU with aneurysmal SAH have a multidisciplinary team, including neurosurgery and neurocritical care, who evaluate patients before and after surgery or endovascular procedures.
 - b. Patients undergoing endovascular procedures will also be evaluated prior to and postprocedure by a physician from interventional radiology.
 - c. Consultations to other services for pre- or postoperative evaluation, such as internal medicine, cardiology,

pulmonology, will be made on an as-needed basis per the patient's clinical status and past medical history.

- V. Vasospasm Treatment Algorithms
 - A. See separate algorithm sheets for specific management guidelines. The algorithms are based on the neurological exam.
 - 1. Aneurysmal subarachnoid hemorrhage with stable neuro-logic exam
 - a. Continue to monitor TCDs and perform per protocol.
 - b. Treatment will be guided by any changes in exam in conjunction with TCD data, including Lindegaard Index.
 - c. Patients with elevations in TCD readings will not be treated based on readings alone; rather, treatment will be guided based on neurological exam. *Do not initiate vasospasm treatment empirically.*
 - d. Patients with elevated TCD readings and stable neurological exam can consider more frequent assessment with TCD or blood flow studies.
 - e. For patients who are severely neurologically impaired and difficult to assess for neurological change, consider angiogram if velocity > 200, interval rise > 50% since last study, or Lindegaard Index > 4:1. Can consider other blood flow studies.
 - 2. Aneurysmal subarachnoid hemorrhage with new neuro-logic deficit
 - f. Algorithm 1.
 - g. Consider full differential (Table A.2).
 - h. Contact neurosurgery immediately for any acute neurological worsening.
 - i. Treatment goal for patients with symptomatic vasospasm is induced hypertension with euvolemia/hypervolemia.
 - j. Careful consideration in treatment of vasospasm in those with preexisting cardiac disease. In patients with heart disease, if vasospasm is suspected, may consider early angiogram.
 - 3. Specific vasospasm management issues:
 - a. Refer to tables in vasospasm management algorithm.
 - b. Options to increase volume (Table A.3).
 - c. Options to increase blood pressure (Table A.4).
 - d. Options to increase cardiac index (Table A.5).
 - e. Cerebral angioplasty or selective intra-arterial vasodilator therapy will be considered in patients with symptomatic cerebral vasospasm who are not responding rapidly to other treatment measures.



Tables for vasospasm algorithms

Tuble A.1 General Assessment of volume Status		
Physical exam	Vital signs; daily weight	
Daily fluid balance	Intakes & Outputs; UOP	
Urine studies	Specific gravity; FENA; osmolality	
Serum studies	Renal panel: Na, BUN (blood urea nitrogen), Cr; osmolality	
Chest X-ray	Pulmonary edema	
Cardiopulmonary status	CVP (central venous pressure); PCWP Systolic pressure variability if mechanically ventilated	

eral Assessment of	Volume Status
	eral Assessment of
Differential diagnosis	Diagnostic workup
--	--
Rebleed/new infarct/acute HCP	Head CT
Vasospasm	TCDs, angiogram
↑ICP	Refer to ICP treatment algorithm
Seizure	$\sqrt{\text{AED}}$ level, EEG
Metabolic abnormality	$\sqrt{\text{Renal panel, LFTs, NH}_3}$
Hypotension	Fluid bolus, \sqrt{CBC} (? sepsis/hemorrhage)
Infection	$\sqrt{\text{Temp}, \text{WBC}}$ (white blood cells), cultures
Medication overdose	\sqrt{MAR} (medication administration record), consider Narcan
Hyper-/hypothermia	Normalize temperature
Hyper-/hypoglycemia	$\sqrt{\text{FSBS}}$
Respiratory issues (hypoxia, hyper-/hypocarbia)	ABG (arterial blood gas), \uparrow FlO ₂ , assess need for intubation

Table A.2 Differential and Preliminary Diagnostic Workup for a New Neurological Deficit in Patients with Aneurysmal Subarachnoid Hemorrhage

Table A.3 Options to Increase Volume

Agent	Volume
NS or normosol	500–1,000 mL
5% albumin	250–500 mL
3% saline (if Na low)	250 mL
Blood (if hemoglobin < 10)	1–2 units packed red blood cells

Intervention	Considerations
Fluid bolus	Refer to Table A.3 for recommendation
Review medications	Eliminate or decrease those that lower BP
	Sedation/analgesia: propofol, fentanyl Calcium channel blockers: nimodipine; beta-blockers Antiepileptics: phenytoin
Vasopressors	Norepinephrine Vasopressin if serum sodium normal (not more than 0.04 units/min)

Table A.5	Options	to Increase	Cardia	ic Index
-----------	---------	-------------	--------	----------

Dobutamine Norepinephrine Milrinone (long T½)

References

General

Connolly ES Jr., et al. Guidelines for the management of subarachnoid hemorrhage: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2012;43:1711–1737.

Use of a scale to classify severity

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Appendix C: Management of intracerebral hemorrhage

Order sets for the management of intracerebral hemorrhage



Saint Luke's Care Saint Luke's Health System

Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

ANOTHER MEDIC ATION S				
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THIS DOCUMENT IS I	NTENDED TO B	E INDIVIDU AL	IZED AS NEEDED FOR ANY SPE	CIFIC PATIENT
ED	Intra-Cerebr	al Hemorrha	agic Stroke Orders	
Date: Time:				
Orders that are checked will be implemented of the implemented of the second of the se				
1. 🛛 Triage Level: 2				
2. 🛛 Time last known well:				
 MOB up 30° if SBP 90 NIH Stroke Scale Assessment BP and neurological assessment AND use Frequent Neuro Assess NPO – no meds by mouth until dy ECG – If not already done Maintain O₂ at 2 – 4 L/NC, titrate 1 ICH Score within one hour of ED Measurements Glasgow Coma Score 3-4 Glasgow Coma Score 5-12 ICH volume ≥ 30 mL Intraventricular hemorrhage 	icog screen, 0-50 years (UA and mic with pupil ey- ment Form (sphagia scr to sat greate arrival Points 2 1 1 1	old) unless roanalysis. kam q. 15 m (SYS-194) een comple	they have had surgical hy Reflex urine culture if po ninutes and place print ou	rsterectomy or are post menopausa ssitive tt on chart (from ER computer)
Infrantentorial origin	1		-	
Age <u>> 80</u>	1			
	SCORE			
 □ Labetalol 10 mg IV over 1 min. Ma If blood pressure controlled, may □ Nicardipine 5 mg/hr IV infusion as of 15 mg/hr ∞ No IV tPA indicated due to Intra-cer 	ay repeat q. give Labeta s initial dose rebral hemor	15 min to a lol 10 mg IV e; titrate to c rhage	/ q. 1 hour PRN. If unabl desired effect by increasing	e to reach target start nicardipine ng 2.5 mg/hr q. 15 min to maximur
, ,,			entormages needing surg	gical screening
8. Admit to hospitalist Admit to	neurosurge	on		
Physician Initials:				(Continued)
A LLERGIES / INTOLER ANCES		– [MS, MSO q.o.d. <u>Never_</u> use p	S ABBREVIATIONS DO NOT USE! 4, MgSO4, q.d. or QD, or QOD, U or u, IU e zero after decimal oint (1.0 mg) e zero before decimal oint (0.5 mg)	Apply Patient Label In This Bo Do Not Cover Up Below Barcoc



Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

		ANOTHER MEDIC ATION SIM	LAR IN FORM AND ACTION M	AY BE DISPENSED PER MED	DIC AL STAFF POLICY
THISDOCUMENT IS INTENDED TO BE INDIVIDU ALIZED AS NEEDED FOR ANY SPECIFIC PATIENT					
ED Intra-Cerebral Hemorrhagic Stroke Orders (Continued)					
orde	ers (line items) mi	ust be individually initia	led.		ons (including strike through) o
	-		on patient history of a	nti-coagulation	
9.	If transferring t Discontin If patient is on Give Vita If patient is on Give Pro	ue any antithrombotic varfarin min K (phytonadione) varfarin or [rivaroxabar	s INR management with t and/or antiplatelet medic 10 mg IV in 50 mL D5W o n (Xarelto *) with Prothrom ncentrates (Profilnine ® \$	cation over 1 hour abin Time over 18 secor	
		Weight < 70 kg	Weight 70-100 kg	Weight >100 kg	
	INR < 3	2000 units	3000 units	4000 units	
	INR	3 3000 units	4000 units	5000 units	
10.	Administer PCC * Note PCC do If patient Check P If INR re Medications Ondansetro	T/INR 1 hour after PC mains greater than 1.4 n (Zofran) 4 mg IV q. 6	Factor VII (NovoSeven @ C is administered notify physician to cons hours PRN nausea		administered over 2-5 minutes.
11.		hen 650 mg PR for T >	hage Initial Care Orders (S	VE 1200)	
	ician Signature:			Date:	Time:
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Allergies / Intoler ances		MS, MSO4, M q.o.d. or G	INT USE! IQT USE! IQSO4, q.d. or QD, IQD, U or u, IU ro after decimal	Apply Patient Label In This Bo Do Not Cover Up Below Barcoc	
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ale)	Anergy res L		l		
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SLCSYS1288



Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

<u> </u>				I MAY BE DISPENSED PER MEDIC /		
⊢				ZED AS NEEDED FOR ANY SPECIFIC	PATIENT	
		oral Hemo	rrhagic Strol	e Initial Care Orders		
Date						
	ers that are checked will be implemented – Ir difications (including strike through) of o					
1.	Diagnosis: Intracerebral Hemorrhagic S	troke	🖾 No tP/	A indicated due to Intracerel	oral hemorrhage	
2.	Vital signs and neuro checks					
	Telemetry / MedSurg: q. 2 hrs 4 and 1 ICU: Vital signs q. 1 hr and neuro check	s q. 2 hrs	4 and the	•		
3.	Complete Baseline NIHSS assessment logic deterioration and on day of dischar		hission, befo	ore ICU discharge or within 2	24 hrs of procedures, with any neuro-	
4.	ICH Score within one hour of ED arrival					
	Measurements P	Points	Score]		
	Glasgow Coma Score 3-4	2		1		
	Glasgow Coma Score 5-12	1				
	ICH volume <u>> 30 cc</u>	1				
	Intraventricular hemorrhage	1				
	Infrantentorial origin	1]		
	Age <u>> 80</u>	1]		
	S	CORE		J		
5.	Telemetry monitoring for 72 hrs then dis	scontinue	if no signific	ant rhythm abnormalities		
6.	Allergies:					
7. 8.	Notify physician managing care in hospi and no orders have been written	ital for fur	rther coagu	lopathy management if patie	ent received treatment in ED	
9.	☑ Keep HOB elevated at 30 degrees ☑ Mobility according to clinical path or nur	sing plan	of care, pro	gress patient activity daily		
10.	⊠ I&O					
11.	 ☑ Foley to DD if present ☑ Discontinue Foley at 24 hrs unless contr 	raindicate	d per Foley	Cath Insertion and MGT or	ders (SYS-1310)	
12.	\Box Maintain O_2 at 2-4 L/NC, titrate to O_2 sat	t > 95%				
13.						
14.						
15.						
16.	Provide facility specific stroke education	n material	s to patient	and family per nursing plan	of care	
17.	X Stroke Dysphagia Screening (SYS-300) If NIHSS = 0, choose diet and follow orders below:					
Phys	Physician Initials: (Continued)					
Heigh Weigł	A LLERGIES / INTOLER ANCES DANGEROUS ABBREVIATIONS - DO NOT USE! MS, MSO4, MgSO4, q, d, d, or QD, q, o, d, or QOD, U or u, IU Neight Ikg I grams Always, use zero after decimal point (1.0 mg) Always use zero before decimal point (0.5 mg)					
	Latex Allergy Yes Do					



	ANOTHER MEDICATION SIMILAR IN FORM AN	ID ACTION MAY BE DISPENSED PER MEDICAL ST	d Read Back for all Verbal Orders
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	Intracerebral Hemorr	hagic Stroke Initial Care Orders (Continued)	
	ers that are checked will be implemented – Initia difications (including strike through) of orde		
18.	. IV and Fluid Management ☑ May Intiate Order Set: Intradermal Lidocaine Orders (SYS-1148) Physician to complete and sign □ NS at 80 mL/hr □ IV Fluidto run @mL per hr □ Saline Lock IV and flush q. 8 hrs and PRN		
9.	DVT Prophylaxis: SCDs (Place immediately on admission) Graduated compression stockings (knee hig	jh)	
20.	Lab and Diagnostic Tests Storke Panel (BMP, CBC, Coag screen, Trop Coagulation panel q 6 hours for 24 hours, n Coagulation panel q 6 hours for 24 hours, n UA cad UA Reflex on admission UA C&S on patients with a Hx of Foley from Next AM Lab: Stasting Lipid Profile St HbArto D BMP q, day x 4 days C T head scan non-contrast in AM Indication C T head scan STAT non-contrast if patients O Other:	tify physician if INR is 1.4 or fibrinogen is the facility, if not done in ED and CBC are called a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility if not done in ED and the facility is a control of the facility i	< 100mg/dL hemorrhage evaluation
:1.	Consults: Deurosurgeon Pulmonary if intubated for ventilator manage Rehabilitation Medicine Physician Refrabilitation Medicine Physician Refrabilitation Medicine Physician Refract and Refraction Physician Social Services for discharge planning O ther:	ement (indication) (indication)	
2.	BP Management Target SBP less than 160 Ta Labetalol 10 mg IV over 1 min. May repeat co If blood pressure controlled, may give Labet Nicardipine 5 mg/hr IV infusion as initial dos	I. 15 min to a max dose of 40 mg. Do not use talol 10 mg IV q. 1 hr PRN. If unable to reach	target start nicardipine.
:3.	 b If < 140 mg/dL and HAS history BG q. 6 hrs if NPO or re c. If ANV BG 140 mg/dL, initiatel Telemetry/ MedSurg Admission: a. If < 140 mg/dL and has NO histo BG q. 4 hrs x 4. If BG right or the second second	emains < 140 mg/dL, no further action neces of diabetes, OR CU Intensive Glucose Control orders (SYS-1 CU Intensive Glucose Control orders (SYS-1 bry of diabetes emains < 140 mg/dL, no further action neces 9, repeat BG q. 4 hrs of diabetes,	299). Physician to complete and sign sary
hys	sician Initials:		(Continued)
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itex	«Allergy Yes □ No □		L



Saint Luke's Care Saint Luke's Health System

Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders

ANOTHER MEDICATION SIMIL	AR IN FORM AND ACTION MAY BE DISPENSED PER ME	DICAL STAFF POLICY			
THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT					
Intracerebral Hemorrhagic Stroke Initial Care Orders (Continued)					
Orders that are checked will be implemente (line items) must be individually initialed.	d. Additions, deletions or modifications	(including strike through) of orders			
24. Smoking Cessation: Choose one below	, if smoker				
5	aily if patient smokes less than 10 cigarettes	per day			
	aily if patient smokes 10 or more cigarettes a				
	atient (if has used tobacco in the past 12 mo	nths)			
25. Scheduled Medications:					
	psule or liquid depending on patient's ability D (pharmacist may adjust dose for CrCl < 50				
26. PRN Medications:					
Give Laxative or fleets enema if no BM					
⊠ Milk of Magnesia 30 mL PO daily PRN ⊠ Bisacodyl suppos 10 mg PR daily for o					
Selectly suppose to high reading to a					
	elixir, or suppository PRN for temperature				
to take PO) Discuss with physician ot Ondansetron (Zofran) 4mg IV q. 6 hrs I	ner options if unable to maintain temp < 99.5				
	Q q. 4 hrs PRN moderate pain (if patient can t	take PO)			
Choose only ONE	_				
Morphine sulfate 2-5 mg IV q. 4 hrs F pain	RN for severe pain OR Fentanyl 2	25-50 mcg IV q. 2 hrs PRN for severe			
27. Initiate Order Set: Electrolyte Replacer	nent (SYS-1059). Physician to complete and	sign			
28. Discharge Day Orders ⊠ Provide patient with designated stroke ⊠ Complete NIHSS assessment any time ⊠ Complete Stroke Risk Reduction Plan		tients			
29. Were any of the following conditions pres					
Pressure Ulcers Catheter-Associated Urinary Tract II]Yes 🗌 No]Yes 🗍 No			
Vascular Catheter-Associated Unitary Tracting					
Surgical Site Infection, if yes which		Yes 🗆 No			
Physician Signature:	Date: 1	lime:			
ALLERGIES / INTOLERANCES	DANGEROUS ABBREVIATIONS				
	- DO NOT USE!				
	MS, MSO4, MgSO4, q.d. or QD,				
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University of Cincinnati stroke team

Guideline for management of intracerebral hemorrhage/intraventricular hemorrhage



General information

Document Title:	Management of ICH/IVH		
Purpose:	Define treatment options for managing ICH/IVH		
Objectives:	 Establish parameters for workup and treatment Manage complications of intracerebral and intraventricular hemorrhage 		
Authors:	Mario Zuccarello, MD; Andrew Ringer, MD; Opeolu Adeoye MD Kristine Atchley ACNP-BC; Erin Silva ACNP-BC		
Original Date:	March 12, 2013		

Content of document

Patients with intracerebral and intraventricular hemorrhage (ICH/IVH) sustain the primary insult at the initial time of bleed. Secondary damage to the brain occurs as a result of complications associated with the initial insult. These guidelines are developed to provide team members with information on

- a) Managing patients after ICH/IVH
- b) Suggesting interventions to treat the associated complications

- I. Assessment
 - A. Clinical assessment includes: Glasgow Coma Scale (GCS). NIHSS (National Institutes of Health Stroke Scale). Cranial nerve exam (pupillary response, extraocular movements, facial symmetry, corneal and gag reflexes); motor strength; motor tone; sensory assessment; and vital signs. Note any seizure activity.
 - B. Diagnostic assessment of ICH/IVH may include
 - 1. Brain imaging: computed tomography (CT), magnetic resonance imaging (MRI)
 - 2. Cerebral vascular imaging: angiogram, computerized tomographic angiography (CTA), magnetic resonance angiography (MRA), magnetic resonance venography (MRV)
 - 3. Neuromonitoring options: intracranial pressure (ICP); electroencephalogram (EEG)
- II. Initial Management
 - A. Emergency department or on intensive care unit (ICU) admission: Implement initial general resuscitation protocols. Appropriate interventions include
 - 1. Airway management:
 - a. Supplemental O_2 to maintain $SaO_2 > 95\%$.
 - b. Intubate for respiratory distress, GCS ≤ 8, inability to protect airway: Use RSI Protocol. Titrate ventilator to maintain PaO₂ >100 mm Hg and PaCO₂ normalized.
 - 2. Circulation:
 - a. Establish minimum of two large-bore intravenous lines.
 - b. Place nasogastric (NG) tube/Foley if indicated.
 - 3. Draw initial assessment labs:
 - a. CBC (complete blood count), renal profile, and cardiac enzymes, TEG profile, aspirin (ASA), and Plavix assay
 - 4. Hemostasis
 - a. Discontinue any anticoagulant/antiplatelet agents.
 - b. Reverse coagulopathy and rapidly correct INR (international normalized ratio) for those patients on oral anticoagulants.
 - c. Anticoagulation reversal protocol:
 - i. Goal INR ≤ 1.5 .
 - ii. Administer vitamin K 10 mg IV q 8 hours × 3 doses.
 - iii. Can consider administering FFP 10 mg/kg, recheck INR immediately.
 - iv. If not at goal INR after infusion completed, repeat FFP as needed.
 - v. For fibrinogen < 150, give 10 pk cryoprecipitate.

- vi. For platelets < 50,000 or current Plavix or Aggrenox use, administer two 5 packs of platelets.
- vii. In patient who needs urgent surgery and INR is not rapidly correcting can consider factor V11a $40 \ \mu g/kg \times 1$.
- viii. Current heparin use and partial thromboplastin time (PTT) > 50, protamine 50 mg IV × 1.
 - ix. Patients with severe thrombocytopenia or other coagulation factor deficiency should receive appropriate factor replacement or platelets.
- 5. *Place arterial line if indicated during initial care.* If patient has limited peripheral access or multiple infusions or blood draws requiring additional access, may consider central intravenous catheter placement.
- 6. Diagnosis/assessment:
 - a. Obtain baseline exam including GCS and NIHSS.
 - b. Perform immediate CT head without contrast.
 - c. Can consider early CT with contrast or CTA to assess for patients who may be at high risk for hematoma expansion.
 - d. Arrange for other appropriate diagnostic imaging, such as magnetic resonance imaging (MRI) of the brain, or if vascular lesion suspected, possible MRA or CTA of the brain.
- 7. Hemodynamic management:
 - a. Blood pressure control: Target mean arterial pressure (MAP) 110 mm Hg, blood pressure (BP) < 160/90 mm Hg.
 - i. If systolic blood pressure (SBP) > 200 mm Hg or MAP is > 150 mm Hg, consider aggressive reduction of BP with continuous intravenous infusion and frequent BP monitoring.
 - ii. If SBP > 180 mm Hg or MAP is > 130 mm Hg and there is evidence of elevated ICP, reduce BP using intermittent or continuous intravenous medications. Maintain CPP \ge 60 mm Hg.
 - iii. If SBP > 180 mm Hg or MAP > 130 mm Hg and there is no evidence of elevated ICP, then modest reduction of BP using intermittent or continuous intravenous medications with target of MAP 110 mm Hg and BP 160/90 mm Hg.
- 8. *Sedatives and analgesics* as indicated for mechanical ventilation. Preferred agents based on desired goal:
 - a. For sedation: Use propofol for mechanically ventilated patients.
 - b. For analgesia: Use fentanyl.

- 9. Management options for signs of intracranial hypertension or herniation:
 - a. Hyperventilation (temporary)
 - b. Mannitol
 - c. Hypertonic saline per NSICU protocol
 - d. Consider surgical options.
 - e. Consider placement of ICP monitor/ventriculostomy.
 - i. Preferred device: ventriculostomy. If in emergency department (ED), transfer to ICU or operating room (OR) for placement.
- 10. Management options for signs of hydrocephalus or intraventricular hemorrhage:
 - a. Insert ventriculostomy. If in ED, transfer to ICU or OR for placement.
 - b. Keep ventriculostomy open to drain as specified by neurosurgery.
 - c. Monitor ICP every hour; goal ICP < 20 mm Hg.
- 11. Seizure prophylaxis:
 - a. Not indicated unless clinical seizure activity is noted on admission or any seizure activity noted on electroen-cephalogram (EEG).
- B. Intensive care unit
 - 1. Review all initial care needs from Section IIA.
 - a. Place arterial line/central lines if clinically indicated.
 - b. Initiate analgesia if mechanically ventilated; monitor for effects on MAP.
 - 2. Respiratory management:
 - a. Maintain $SaO_2 > 95\%$ with supplemental O_2 as needed.
 - b. If intubated, goal $PaCO_2 = 35-45$ mm Hg.
 - 3. Neurological examinations:
 - a. Nursing documentation of hourly vital signs and GCS. NIHSS documentation every 12 hours. Cranial nerve exam documented every shift.
 - 4. Hemodynamic management:
 - a. Blood pressure control: Target MAP 110 mm Hg, BP 160/90 mm Hg.
 - b. See previous protocol for treatment if outside target range.
 - c. Administer fluids. Avoid hypervolemia or hypovolemia; fluid balance goal is a range of 0–500 mL positive every 24 hours. Goal is euvolemia.
 - d. Fluid choice: normal saline with or without 20 mEq KCl.
 - 5. Ventriculostomy/acute-phase ICP management:
 - a. If IVH is present, consider ventriculostomy placement for evidence or symptoms of hydrocephalus.

- b. Keep ventriculostomy open to drain as specified by neurosurgery.
- c. Monitor ICP every hour; goal ICP < 20 mm Hg.
- d. Neurosurgical resident to be notified for ICP elevation.
- e. Management options for signs of intracranial hypertension or herniation:
 - i. Hyperventilation (temporary)
 - ii. Mannitol
 - iii. Hypertonic saline per NSICU protocol
 - iv. If ventriculostomy in place, ensure patency.
 - v. Neurosurgery to consider surgical treatment options.
- 6. Seizure prophylaxis:
 - a. In patients with GCS < 8, continuous EEG monitoring × 72 hours.
 - b. Antiepileptics may be indicated based on clinical situation, including presence of clinical seizure activity or evidence on EEG.
- 7. DVT (deep vein thrombosis) prophylaxis:
 - a. SCDs (sequential compression devices) bilateral lower extremities.
 - b. On day 2, if hematoma stability has been documented on follow-up head CT, may start subcutaneous low molecular weight heparin or unfractionated heparin.
 - c. Perform screening lower-extremity ultrasounds per NSICU protocol or when indicated for symptomatic DVT.
- 8. General care issues:
 - a. Glucose: Initiate treatment for hyperglycemia. Goal glucose < 180 mg/dL.
 - b. Sodium: Maintain in normal range (135–146 mEg/L).
 - c. Magnesium: Maintain > 1.8 mg/dL.
 - d. Temperature: Goal is normothermia. Culture per NSICU protocol for fever > 101.5°F.
 - e. Nutrition: Address within first 24–48 hours after admission. Keep NPO (nothing by mouth) while awaiting imaging and neurosurgical plan.
 - f. Early physical, occupational, and speech therapy consultations.
 - g. Consult to social services.
 - h. Consult other ancillary departments, such as nutrition services, wound care, diabetes education, and so on as indicated.
- 9. Multidisciplinary management:
 - a. Patients admitted to the NSICU with aneurysmal subarachnoid hemorrhage (SAH) have a multidisciplinary

team, including neurosurgery and neurocritical care, who evaluate patients before and after surgery or endovascular procedures.

- b. Patients undergoing endovascular procedures will also be evaluated prior to and postprocedure by a physician from interventional radiology.
- c. Consultations to other services for pre- or postoperative evaluation, such as internal medicine, cardiology, pulmonology, will be made on an as-needed basis per the patient's clinical status and past medical history.
- III. Surgical treatment/management:
 - A. Patients with cerebellar hemorrhage who have poor or declining neurological status due to brain stem compression or hydrocephalus will be evaluated for early surgical intervention.
 - B. Open craniotomy for BG hematomas is not recommended; surgical evacuation of lobar ICH has to be decided case by case until more studies will clarify the role of surgery. Minimally invasive ICH evacuation can be applied in selected cases, but its widespread use requires further validation in large clinical trials.

References

Morgenstern LB, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2010;41:2108–2129.

Appendix D: Management of ischemic stroke

Order sets for the management of acute ischemic stroke

Saint Luke's Care Saint Luke's Health System

Physician Orders

Write Down and Read Back for all Verbal Orders

ANOTHER NEDICATION SIMIL	AR IN FORM AND ACTION MAY BE DISPENSED PER MED	I and Read Back for an verbar Orders
	ncy Department Ischemic Stroke Orders	ICAL STAFF POLICY
	Stroke t-PA-Neuro Intervention Orders	
 Date: Time:		
Orders that are checked will be implemented (line items) must be individually initialed.	d. Additions, deletions or modifications (including strike through) of orders
1. 🛛 Triage Level: 2 🛛 🖾 Time last known	well	_
 Imaging: ⊠ Stat CT head non-contrast 		
 STAT Stroke Panel (BMP, CBC only, Co: STAT hCG if female patient 10 – 50 years STAT ECG 		
4. ⊠BP q. 15 minutes		
 S. ⊠ Titrate oxygen to maintain saturation ≥ 95 	5%	
May repeat q. 15 minutes to a max de OR	teplase – Activase ®. May give: 20 mg over 2 minutes to reach target BP 11 ose of 40 mg administered. Do not use if hear al dose: titrate to BP 185/110 by increasing 2.5	rt rate < 60
7. ED Physician Risk Stratification in colla	boration with Neurology	
 Order Cerebral Arteriogram 	(kg) =Total mg usemg Dosemg per infusion y 15 minutes if IV t-PA administered. Rec ologist to evaluate for further treatment/c al Arteriogram with possible intervention n STAT	
 Maintain BP 180/105 duri 11. 	ng intervention if patient has received t-PA	
 Initiate Saint Luke's Brain and Stro Orders PO.191. 	oke Institute Plaza – Ischemic Stroke	Patient Management (Admission)
Physician Signature:	Date:	Time:
Allergies / Intolerances	DANGEROUS ABBREVIATIONS	
Height kg grams Weight kg grams Latex Allergy Yes No	- DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, q.o.d. or QOD, U or u, IU <u>Never</u> use zero after decimal point (1.0 mg) <u>Always</u> use zero before decimal point (0.5 mg)	Apply Patient Label In This Box Do Not Cover Up Below Barcode

SLCSYS1036



Physician Order

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Saint Luke's Hospital Kansas City, MO 64111

Physician Orders

Write Down and Read Back for all Verbal Orders

ANOTHER MEDICATION SIMILAR IN FORM AND ACTION MAY BE DISPENSED PER MEDICAL STAFF POLICY								
THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT								
Saint Luke's Brain and Stroke Institute Plaza Ischemic Stroke Patient Management Orders								
Date: Time:								
Orders that are checked will be implemen deletions or modifications (including strike th								
1. Diagnosis: Ischemic Stroke								
	hrs ute intervention: q. 15 min X 2 hrs, q. 30 mi ain vital signs q. 1 hr and neuro check q. 2 hr							
 Complete Baseline NIHSS assessment any neurologic deterioration and on day or 		within 24 hrs of procedures, with						
4. Telemetry monitoring for 72 hrs then disco	ontinue if no significant rhythm abnormalities.							
5. ⊠ Allergies: ☐ If patient received pre imaging treat initial imaging	ment for dye/contrast allergy give Hydro	ocortisone 200 mg IV 3 hrs post						
 G. ☐ Keep HOB flat as tolerated if not at risk of ⊠ Mobility according to clinical path or nurs ⊠ Out of bed for all meals 	aspiration/airway obstruction for first 24 hrs. ing plan of care, progress patient activity dail							
7. ⊠I&O								
 B Foley to DD if present B Discontinue Foley at 24 hrs unless contra 	aindicated per Foley Cath Insertion and MGT	orders (SYS-1310)						
9. ⊠ Maintain O ₂ at 2-4 L/NC, titrate to O ₂ sat >	95%							
10. Initiate Stroke Clinical Path AND Stroke R	isk Reduction Plan sheet							
11. 🖾 Provide facility specific stroke education	on materials to patient and family per nur	sing plan of care						
12. ⊠ Stroke Dysphagia Screening (SYS-300) If NIHSS = 0, choose diet and follow orders below:								
if diabetic ⊠ Consult SLP for fu	rther diet modifications on all patients with a	normal screen						
13. IV and Fluid Management ⊠ May Initiate Order Set: Intradermal Lic □ NS at 80 mL/hr □ IV Fluidto run @ □ Saline Lock IV and flush q. 8 hrs and PRI	docaine Orders (SYS-1148) Physician to co mL per hr N	mplete and sign						
	nL/min dose = 30 mg) SQ q. 24 hrs – Hold 2							
Physician Initials:		(Continued)						
ALLERGIES / INTOLERANCES Height Weight kg grams Latex Allergy Yes No	DANGEROUS ABBREVIATIONS - DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, q.o.d. or QOD, U or u, IU <u>Never</u> use zero after decimal point (1.0 mg) <u>Always</u> use zero before decimal point (0.5 mg)	Apply Patient Label In This Box Do Not Cover Up Below Barcode						
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Physician Orders

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			d Stroke Institute Plaza nagement Orders (Conti	nued)				
			-	ks made. Additions, deletions or				
difications (including strike								
Select appropriate	treatmer	it:						
□ NO intervention	□IV t-PA	Only	□ IA or Device: with IV	□ IA or Device: without				
□ Target BP 220/120	BP Tar	get Parameters:	t-PA	IV t-PA				
OR	Keep SB	P <u>180</u> and DBP	BP Target Parameters					
□Кеер	105		Keep SBP 180 and DB	P Keep SBPand				
	Keep SB	> c	105	DBP				
SBP		Management	Keep SBP	Keep SBP >				
and		below to keep BP	See BP Management	See BP Management				
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Keep		ete NIHSS at	at target	at target.				
SBP		post treatment	Complete NIHSS at	Complete NIHSS at 24 hrs				
See BP Management		e for bleeding	24 hrs post treatment	post treatment				
orders below to keep		cations and notify	Observe for bleeding	Observe for bleeding				
BP at target		an if needed	complications and not	ify complications and notify				
Ŭ		id scan STAT	physician if needed	physician if needed				
		ntrast if patient's	CT head scan STAT	CT head scan STAT non-				
	neurolo	ogical status	non-contrast if patient	's contrast if patient's neuro-				
		rates and notify	neurological status deteriorates and notify	logical status deteriorates				
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		coagulants or	No anticoagulants or	⊠24 hr ICU stay post				
		atelets for 24 hrs	anti-platelets for 24 hr	intervention				
	after IV		after IV t-PA	Check groin site and dis-				
		CU stay post	24 hr ICU stay post	tal pulses q. 15 min 4, q.				
	interve	ntion	intervention	30 min 6 then routine				
			Check groin site and dis	Keep □ R □ L leg straight				
			pulses q. 15 min 4, q					
			30 min 6 then routine					
			Keep R L leg straight					
			for hrs					
BP Management Order a. Antihypertensive:	rs:		•					
	V over 1 m	inute or 20 mg IV	over 2 minutes to reach	target BP. May repeat q. 15 min t				
			ne. Do not use labetalol					
	nr IV infusior	n: Ttitrate to target E	P by increasing 2.5 mg/hr	q. 15 min to max dose of 15 mg/hr				
b. Vasopressors:	ta 40 maan/		······································	DD Mau dana 250 man/min				
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c. Other BP Manager		2 1109/1111	allon. Infate to target obr	. Max dooc oo mog, min				
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Saint Luke's Hospital Kansas City, MO 64111

Physician Orders

Write Down and Read Back for all Verbal Orders

	IDED TO BE INDIVIDUALIZED AS NEEDED FOR AN	Y SPECIFIC PATIENT
	nt Luke's Brain and Stroke Institute Plaza Stroke Patient Management Orders (Conti	nued)
	ted – Initial the bottom of the page if any check orders (line items) must be individually initialed	
0. ⊠Acute Blood Glucose (BG) Man		
Stat BG upon Admission	-	
ICU Admission :	NO history of dishetes	
a. If 140 mg/dL and has BG g. 4 hrs	4. If BG remains 140 mg/dL, no further ac	tion necessary
b. If 140 mg/dL and HAS		· · · · · · · · · · · · · · · · · · ·
 BG AC and H 	5	
	f NPO or receiving continuous enteral nutritio ., initiate ICU Intensive Glucose Control order	
complete and sign	, millate ICO milensive Glucose Control order	s (313-1299). Physician to
Telemetry/ MedSurg Admissi	on:	
a. If 140 mg/dL and has		
	4. If BG remains 140 mg/dL, no further ac s to 140-179, repeat BG g. 4 hrs 4	tion necessary
b. If <180 mg/dL and HAS		
BG AC and H	IS if eating OR	
	f NPO or receiving continuous enteral nutritio	
C. II ANY BG ≥ 180 mg/dL,	initiate Basal Bolus Insulin orders (SYS-1140). F	Physician to complete and sign
Nicotine transdermal 14 mg	g patch daily if patient smokes less than 10 ciga	
	g patch daily if patient smokes 10 or more cigan to patient if has used tobacco in the past 12 mo	
Bigive smoking cessation education	to patient in has used tobacco in the past 12 mo	nuns
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		Saint Luke's Hospital Kansas City, MO 64111		
	Physician Order	Physician Orders	Write Down	and Read Back for all Verbal Orders
	ANOTHER MEDICATION SIMILAR IN	N FORM AND ACTION MAY BE		
	THIS DOCUMENT IS INTENDE	D TO BE INDIVIDUALIZED AS	NEEDED FOR A	NY SPECIFIC PATIENT
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	lers that are checked will be impler rders (line items) must be individually in		ons or modifica	ations (including strike through)
24.	Discharge Day Orders ⊠Provide patient with designated s ⊠Complete NIHSS assessment ar ⊠Complete Stroke Risk Reduction	ly time on day of discharge		to all patients
25.	Additional Orders			
26.	Were any of the following conditions Pressure Ulcers Catheter-Associated Urinary Tra Vascular Catheter-Associated In Surgical Site Infection, if yes whi	ict Infection fection	[Yes □No Yes □No Yes □No Yes □No Yes □No Yes □No
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University of Cincinnati stroke team Protocol for acute ischemic stroke management



Authors: Pooja Khatri, MD, MSC; Sharyl Martini, MD; Opeolu Adeoye, MD, MSc

Date: April 1, 2013

Acute stroke management is progressing rapidly. Our team offers several options for acute stroke therapy, including clinical trials, and is available for immediate consultation 24 hours per day and 7 days per week.

- 1. When should the stroke team be called?
 - Contact the stroke team at **513-844-7686** as soon as a *potential* candidate for treatment is identified (preferably before the computed tomographic [CT] scan is completed).
 - Potential treatment candidates are:
 - Patients of any age with a suspected ischemic stroke who were "last seen normal" within 6 hours of symptom onset
 - Patients with a suspected transient ischemic attack (TIA) or minor ischemic stroke patient who were last seen normal within 12 hours of symptom onset (to screen for the POINT Trial)
- 2. Sequence of events by emergency department (ED) (Faster Treatment = Better Clinical Outcomes)
 - Determine last seen normal time within 10 minutes of ED arrival.
 - Patients are eligible for intravenous tissue plasminogen activator (IV rtPA) up to *4.5 hours* from onset.
 - Select stroke patients are eligible for intra-arterial therapies up to *6 hours* from onset.

- TIA/minor stroke patients may be eligible for the POINT Trial up to 12 *hours* from onset.
- Perform brief neurological exam and activate stroke team (513-844-7686) within 15 minutes of ED arrival.
- Perform noncontrast CT scan rapidly to rule out intracranial hemorrhage within 25 minutes of ED arrival.
- Draw bloods for lab tests (CBC [complete blood count], renal, coagulations, pregnancy, fingerstick glucose).
 - Note: Fingerstick glucose should be obtained promptly to determine IV rtPA eligibility. tPA should not be delayed to wait for other lab results unless there is clinical suspicion for potential abnormalities.
- Establish two intravenous lines.
- Record blood pressure.
 - Gently treat (usually labetalol 10 mg to start, assuming no clinical contraindications) if blood pressure (BP) is > 185/110 if potential IV rtPA candidate.
- Perform electrocardiogram (EKG).
- Review eligibility criteria for IV rtPA (details in the following material).
- Interpret CT scan to rule out intracerebral hemorrhage (ICH) or significant ischemic changes within 45 minutes of arrival.
- Store IV rtPA in emergency department Pyxis for ready accessibility.

• Start IV rt-PA bolus if eligible within 60 minutes of arrival.

- 3. Treatment:
 - Mix IV rtPA: 0.9 mg/kg dose (maximum 90 mg). Administer 10% as bolus over 1–2 minutes and remainder as infusion over 60 minutes.
 - Do *not* use the cardiac dose.
 - Do not exceed the 90 mg maximum dose.
 - Use rtPA = Activase[®] = alteplase. Do not use other thrombolytic agents.
 - Do not give aspirin, clopidogrel, heparin, warfarin, or other oral anticoagulants for the first 24 hours after IV rt-PA.
 - Monitor the patient carefully, especially BP.
 - Treat BP > 180/105 (details in the following).
 - Repeat head CT stat if increased BP, headache, nausea, vomiting, or decline in neurological status.
 - Call stroke team MD with any questions or concerns at 513-844-7686.
- 4. Adjunctive/additional therapy:
 - Potential IV rtPA treatment candidates **should not** receive antiplatelet (aspirin, clopidogrel) or anticoagulant (heparin, warfarin, or other novel oral agents such as dabigatran) medications on

arrival to the emergency department if potential reperfusion treatment candidates.

- However, patients who have taken antiplatelet medications prior to arrival in the emergency department **are** still considered candidates, and those taking anticoagulant medications **may** still be considered candidates for thrombolytic therapy.
- No concomitant antiplatelet or anticoagulant medications during the first 24 hours after symptom onset. At 24 ± 6 hours, a noncontrast CT scan or magnetic resonance imaging (MRI) must be performed (to rule out any intracranial hemorrhage) before starting an antiplatelet or anticoagulant medication.
- Endovascular reperfusion therapies (as primary treatment of IV rtPA-ineligible patients or adjunctive therapy among IV rtPA patients with severe strokes) will be considered in select patients as per the stroke team's clinical judgment and rapidly evolving evidence. Considerations will be based on, but not limited to, the following criteria:
 - Age < 85 years
 - Time from stroke onset < 6 hours
 - IV rtPA ineligibility
 - Location and severity of acute thrombus (if known), including basilar artery or intracranial internal carotid thrombus (ICAT)
- 5. Criteria for IV rtPA eligibility (2013 American Heart Association/ American Stroke Association [AHA/ASA] Clinical Guideline, Tables A.10 and A.11)
- 6. Blood pressure control
 - Pretreatment:
 - For IV rtPA candidates: BP should be brought to < 185/110 mm Hg if possible. This must be done without aggressive antihypertensive treatment for the patient to remain eligible for IV rtPA. If blood pressure remains > 185/110 mm Hg with nonaggressive measures, then the patient is not eligible for IV rtPA.
 - Blood pressure management prior to IV rtPA administration:
 - Up to two of the following agents may be used for nonaggressive treatment:
 - Labetalol 10 to 20 mg IV over 1 to 2 minutes; may repeat × 1 (up to maximum total dose of 40 mg)
 - Nicardipine infusion, 5 mg/hour; titrate up by 2.5 mg/ hour at 5- to 15-minute intervals (up to maximum dose of 15 mg/hour; when desired blood pressure attained, reduce to 3 mg/hour)

- Enalaprilat 0.625 to 1.25 mg IV (up to maximum dose of 1.25 mg)
- Hydralazine 10 mg IV over 1 to 2 minutes; may repeat × 1 (up to maximum dose of 20 mg)
- Nitropaste 1 to 2 inches (up to maximum dose of 2 inches)
- If IV rtPA not planned, then permissive hypertension up to 220/120 mm Hg may be reasonable.
- Post-tPA treatment:
 - During/after treatment with rtPA or other acute reperfusion intervention, BP must be aggressively maintained at < 180/105 mm Hg.
 - Monitor BP every 15 minutes for first 2 hours, then every 30 minutes for next 6 hours, then every hour for the next 16 hours.
 - Monitor blood pressure every 15 minutes during the antihypertensive therapy. Observe for hypotension.
- Blood pressure management during/after administering IV rtPA:
 - If systolic BP > 180–230 mm Hg or diastolic BP > 105–120 mm Hg:
 - Labetalol 10 mg IV followed by continuous intravenous infusion 2–8 mg/min; or
 - Nicardipine 5 mg/hour IV; titrate up to desired effect by 2.5 mg/hour every 5–15 minutes; maximum 15 mg/hour.
 - If BP not controlled or diastolic BP > 140 mm Hg:
 - Consider intravenous sodium nitroprusside.
- 7. Management of intracranial hemorrhage after thrombolysis:
 - If an intracranial hemorrhage is suspected, the stroke team treating MD (513-844-7686) should be contacted *immediately*.
 - Suspect intracranial hemorrhage if there is any acute neurological deterioration (new headache, acute hypertension, seizure, or nausea and vomiting).
 - If hemorrhage is suspected, then do the following:
 - Discontinue rt-PA infusion until ICH is ruled out.
 - Immediately perform CT scan.
 - Draw blood for international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count, and fibrinogen and type and screen.
 - Prepare for administration of 6 to 8 units of cryoprecipitate.
 - Prepare for administration of 6 to 8 units of platelets.
 - If intracranial hemorrhage present:
 - Consider administering 6–8 units cryoprecipitate followed by 6–8 units platelets.
 - Consider emergent neurosurgical consultation.
 - Notify patient's family or next of kin.

8. Management of angioedema after thrombolysis:



- Post IV rtPA stroke monitoring
 - Admit patient to ICU and follow post-tPA order set, including
 - Frequent monitoring of BP and neuro status: q 15 min \times 2 hours, q 30 min \times 6 hours, then q 1 hour \times 16 hours
 - Call stroke MD if BP >180/105 mm Hg, decline in neuro status, or new headache, nausea, or vomiting
 - NPO until swallowing assessed
 - DVT (deep vein thrombosis) prophylaxis with intermittent stocking compression devices (sequential compression devices, SCDs)

- Consider transfer to a neuroscience ICU for patients needing specialized monitoring and management, including
 - Severe (National Institutes of Health Stroke Scale [NIHSS] score ≥ 10) stroke with risk of malignant middle cerebral artery (MCA) syndrome requiring anticipation and consideration of decompressive hemicraniectomy by neurosurgery
 - Cerebellar stroke with risk of malignant edema requiring anticipation and consideration of posterior decompression by neurosurgery
 - Fluctuating neurological symptoms requiring specialized BP management
 - Posterior circulation syndrome that may require more aggressive endovascular measures in upcoming hours

Appendix E: Additional tools for stroke team nurses

The following tools are used by the stroke center nurses.

Table A.10 Inclusion and Exclusion Characteristics of Patients with Ischemic Stroke Who Could Be Treated with IV rtPA within 3 Hours from Symptom Onset

Inclusion criteria

Diagnosis of ischemic stroke causing measurable neurological deficit Onset of symptoms <3 hours before beginning treatment Aged ≥ 18 years

Exclusion criteria

Significant head trauma or prior stroke in previous 3 months Symptoms suggest subarachnoid hemorrhage Arterial puncture at noncompressible site in previous 7 days History of previous intracranial hemorrhage Intracranial neoplasm, arteriovenous malformation, or aneurysm Recent intracranial or intraspinal surgery Elevated blood pressure (systolic > 185 mm Hg or diastolic > 1000 mm Hg) Active internal bleeding Acute bleeding diathesis, including but not limited to Platelet count < 100,000/mm³ Heparin received within 48 hours, resulting in abnormally elevated aPTT greater than the upper limit of normal Current use of anticoagulant with INR > 1.7 or PT > 15 seconds

Current use of direct thrombin inhibitors or direct factor Xa inhibitors with

elevated sensitive laboratory tests (such as aPTT, INR, platelet count, and ECT;

IT; or appropriate factor Xa activity assays)

Blood glucose concentration < 50 mg/dL (2.7 mmol/L)

CT demonstrates multilobar infarction (hypodensity > 1/3 cerebral hemisphere)

Relative exclusion criteria

Recent experience suggests that under some circumstances—with careful consideration and weighting of risk to benefit—patients may receive fibrinolytic therapy despite 1 or more relative contraindications. Consider risk to benefit of IV rtPA administration carefully if any of these relative contraindications are present:

Only minor or rapidly improving stroke symptoms (clearing spontaneously) Pregnancy

Seizure at onset with postictal residual neurological impairments

Major surgery or serious trauma within previous 14 days

Recent gastrointestinal or urinary tract hermorrhage (within previous 21 days) Recent acute myocardial infarction (within previous 3 months)

 Table A.10 (continued)
 Inclusion and Exclusion Characteristics of Patients

 with Ischemic Stroke Who Could Be Treated with IV rtPA within 3 Hours
 from Symptom Onset

The checklist includes some FDA-approved indications and contraindications for administration of IV rtPA for acute ischemic stroke. Recent guideline revisions have modified the original FDA-approved indications. A physician with expertise in acute stroke care may modify this list.

Onset time is defined as either the witnessed onset of symptoms or the time last known normal if symptom onset was not witnessed.

In patients without recent use of oral anticoagulants or heparin, treatment with IV rtPA can be initiated before availability of coagulation test results but should be discontinued if INR is > 1.7 or PT is abnormally elevated by local laboratory standards.

In patients without history of thrombocytopenia, treatment with IV rtPA can be initiated before availability of platelet count but should be discontinued if platelet count is < 100,000/mm³.

aPT indicates activated partial thromboplastin time; CT, computed tomography; ECT, ecarin clotting time; FDA, Food and Drug Administration; INR, international normalized ratio; IV, intravenous; PT, partial thromboplastin time; rtPA, recombinant tissue plasminogen activator; and TT, thrombin time.

Table A.11Additional Inclusion and Exclusion Characteristics of Patientswith Acute Ischemic Stroke Who Could Be Treated with IV rtPA within3 to 4.5 Hours from Symptom Onset

Inclusion criteria

Diagnosis of ischemic stroke causing measurable neurological deficit Onset of symptoms with 3 to 4.5 hours before beginning treatment

Relative exclusion criteria

Aged ≥ 80 years

Severe stroke (NIHSS > 25)

Taking an oral anticoagulant regardless of INR

History of both diabetes and prior ischemic stroke

INR indicates international normalized ratio; IV, intravenous; NIHSS, National Institutes of Health Stroke Scale; and rtPA, recombinant tissue plasminogen activator.



Saint Luke's Care Saint Luke's Health System Physician Orders

Physician Order

Write Down and Read Back for all Verbal Orders ANOTHER MEDICATION SIMILAR IN FORM AND ACTION MAY BE DISPENSED PER MEDICAL STAFF POLICY THIS DOCUMENT IS INTENDED TO BE INDIVIDUALIZED AS NEEDED FOR ANY SPECIFIC PATIENT **Transient Ischemic Attack Initial Management Orders** Time: Orders that are checked will be implemented – Initial the bottom of the page if any checks made. Additions, Deletions or Modifications (including strike through) of orders (line items) must be individually initialed. Diagnosis: Transient Ischemic Attack. ABCD² Score_ **Risk Factor** Point Score Age ≥60 years - 1 point 1 **BP**: Systolic $BP \ge 140$ or Diastolic ≥ 90 1 **Clinical Features** 2 Unilateral weakness with or without speech impairment 1 Speech impairment without unilateral weakness Duration of TIA ≥ 60 minutes 2 Total Score:_ 10-59 minutes 1 Score 0-2 Observation Status **Diabetes history** ≥ 3 Inpatient Status 1 2. Telemetry for 72 hours; discontinue if no significant rhythm abnormalities S and Neuro checks q. 2 hrs × 4 then q. 4 hrs Complete Baseline NIHSS assessment upon admission and with any neurologic change or deterioration O2 sat on admission, Maintain O2 sat greater than 95% (2-4 L/NC). 🛛 Initiate TIA Clinical Path or Individualized Plan of Care if available 🛛 Initiate Stroke Risk Factor Reduction Plan. 3. Give patient/family designated stroke education materials Advance patient mobility daily as tolerated Out of bed for all meals 4 Stroke Dysphagia Screening (SYS-300) If NIHSS = 0, choose diet and follow orders below: Low fat, low Chol, 2 gram Na (Simply healthy) OR Consistent Carb 75 gram (1800 ADA) if diabetic If NIHSS greater than 0, RN to complete Stroke Dysphagia Screening Abnormal: Keep NPO, including meds and Consult speech language pathology (SLP) for swallow evaluation/management. Low fat, low Chol, 2 gram Na (Simply healthy) OR Consistent Carb 75 gram (1800 ADA) if diabetic Normal: Consult SLP for further diet modifications on all patients with a normal screen Insert saline lock. Saline flush q. 8 hours and after usage □IV Fluid _ to run at _ _ mL/hour VTE Prophylaxis: Choose one below or provide contraindication □Heparin 5000 units SQ q. 12 hours □SCDs 8. Lab and Diagnostic Tests CT Scan Head w/o contrast, if not performed in the ED Indication: cerebral ischemia Secg upon admission if not done in ED Stroke Panel if not drawn in ED or transferring hospital (BMP, CBC, Coag screen, Troponin I, Mg) Next AM Lab: SFasting Lipid Profile HbA1c CMP CK ChCG Qual if female 10-50 yrs old B12 TSH □ Other SLHS preferred diagnostics testing. Indication: TIA - cerebral flow work-up MRI Head with/without contrast Date:___ MRA Head without contrast Date:_ (Do Not Order if CTA done in ER) MRA Neck with/without contrast Date:___ _ (Do Not Order if CTA done in ER) Cerebral arteriogram Echo (TTE) with agitated saline TEE (in GI Lab) If above testing contraindicated or not available Carotid Duplex Scan Physician / Initials: (Continued) ALLERGIES / INTOLERANCES DANGEROUS ABBREVIATIONS-DO NOT USE! MS, MSO4, MgSO4, q.d. or QD, q.o.d. or QOD, U or u, IU Apply Patient Label In This Box Height _ Never use zero after decimal Do Not Cover Up Below Barcode Weight ____ _____ □ kg □ grams point (1.0 mg) Latex Allergy Yes No Always use zero before decimal point (0.5 mg) © Saint Luke's Care 2012

SLCSYS1278





Saint Luke's Care Saint Luke's Health System

Code Stroke (Ischemic/ICH/SAH/TIA) Nursing Flowsheet DYSPHAGIA SCREENING

Assessment and Screening	Documentation		Risks and Protocol			
	Yes	No				
Patient has been NPO until this screening:						
Alert, able to participate, able to maintain oxygen saturation above 90% with or without oxygen and not intubated?	Yes	No □	If "No" – Patient <u>at risk</u> for dysphagia • Keep NPO/NO ORAL MEDICATIONS • Obtain orders for IV/NG medications/ nutrition • When patient is alert, reassess and continue with screening process			
History free of previous problems with swallowing or being on dysphagia diet prior to admission?	Yes	No □	If all answers "Yes" Proceed to swallow challenge 			
 Patient has: Strong clear voice (no dysphonia) Manages own secretions, no suctioning required Understandable speech (i.e. no dysarthria) Absent facial droop Normal voluntary cough Normal gag reflex 			If any "No" answers – Patient <u>at risk</u> for dysphagia. • Keep NPO - NO ORAL MEDICATIONS • Consult SLP (Speech and Language Pathologist) for dysphagia assessment and management • Follow all SLP recommendations for management • Obtain orders for IV/NG/medications/ nutrition, if SLP unable to see within 24 hours • Mark assessment abnormal box below and initiate orders.			
Dysphagia Screening:	Dyspha	gia Evalua	tion: Check all observed signs:			
 Sit patient upright at 90 degrees with head in neutral position. Ensure patient wearing all assistive devices (dentures, hearing aids, glasses, etc.). After each water challenge, instruct patient to say "Aah" and observe for dysphagia signs for one minute. Document observed signs in box at right. Stop water challenge and make patient NPO if dysphagia signs noted at any step in challenge. 	Voice change after swallow (wet/gurgling voice) Coughing or throat clearing after swallow Holding water in mouth Leakage of water from mouth Delayed/difficult/painful swallow Tearing with swallowing effort Shortness of breath Additional concerns (specify)					
Water challenge:						
 Give 5 mL of water (with a cup) Give 10 mL of water (with a cup) Give 20 mL of water (with a cup) 	Mark appropriate findings below and initate orders. Place completed form in Physican Orders section.					
ORDERS per lso	hemic St	roke Adm	ission Orders			
 Swallow function normal Place on Low fat, low Chol, 2 gram Na (Simpl Consistent Carb 75 gram (1800 ADA) diet if E Order SLP consult for evaluation for diet modified 	G is ≥ 120	or	Assessment abnormal Keep NPO Order SLP consult for bedside swallow screening evaluation and management			

Nurse's Signature: _____

__ Date: _____ Time: _____

Patient Label:



PROCEDURE

Saint Luke's Care Saint Luke's Health System

reque	nt Assess. Flowsheet Neur	ologica	l Fre	quen	t Ass	essn	nent	Flov	wshe	et		F	Prin	t Fo	rm	
DATA	INITIALS															
	DATE															
	TIME															
	TEMP															
VITAL SIGNS	BP															
SIG	PULSE															
ITAI	RESP.															
>	0 ₂ SAT															
	PEDAL	R														
PROCEDURE CHECK	PULSES	L														
EC ED	EXT. COLOR															
о С С	EXT. TEMP															
	GROIN DRESSING															
	LOC 0-3															
ΝT	LOC 0-2 QUESTIONS															
SME	LOC 0-2 COMMANDS															
ESS	MOTOR:	RA														
NEUROLOGICAL ASSESSMENT	ARMS 0-4	LA														
∀ / ⁄	MOTOR:	RL														
GIC	LEGS 0-4	LL														
LOO	FACIAL PALSY 0-3															
JRC	VERBAL 0-3															
NEL	VISUAL FIELD *															
	SENSORY 0-2															
	EYE OPENING 1-4															
щ	VERBAL 1-5															
CAL	BEST MOTOR 1-6															
AS	GCS T	GCS TOTAL														
GLASGOW COMA SCALE	RAMOTOR															
N N	RL MOTOR															
GO	LA MOTOR/															
LAS	LL MOTOR															
Ū	PUPIL SIZE & REACTION	R														
		L														
Pupil Scale		PUPIL REACTION Reactive		* Code K				e Key	ey on Back							
				Init.		Signature				Init.		Signature			,	
		– Nor	- Nonreactive													
		Slu	ggish													

Patient Label:
Saint Luke's Care Saint Luke's Health System

Neurological Frequent Assessment Flowsheet

FREQUENT NEUROLOGICAL ASSESSMENT KEY						
	ss) sponsive to verbal stimuli; is able slightly impaired; arouses when s		king; responds appropriately.			
If the patient scores	If the patient scores either 2 or 3 in this section of the neuro check, proceed to the Glasgow Coma Scale					
2 Stuporous; aroused with dif reverts to original state whe	ficulty, often painful stimuli must be en not stimulated.	e applied; arousal usually incor	nplete; responds inadequately;			
3 Comatose; unresponsive t	o all stimuli or responds with ref	lex motor or autonomic effect	S.			
LOC QUESTIONS						
1 Patient answers one quest	the month (<u>only initial answer is</u> tion correctly. understand or answers incorrec	,				
LOC COMMANDS						
 Patient grips hand <u>and</u> clo Patient does one correctly Patient does neither correctly 						
MOTOR: ARM (Right & Left)						
sciousness or comprehension is 0 No drift (Limb holds at 90 1 Drift (Limb holds position, 2 Cannot resist gravity (Limb	The patient is examined with arms outstretched at 90 if sitting or at 45° if lying down. Request full effort for <u>10 seconds</u> . If con- sciousness or comprehension is abnormal, <u>cue</u> patient by actively lifting arms into position as the request for effort is verbally given. No drift (Limb holds at 90 if sitting, at 45 if lying down for full 10 seconds). Drift (Limb holds position, but drifts before 10 seconds; does <u>not</u> touch the bed). Cannot resist gravity (Limb falls to the bed <u>before</u> the full 10 seconds, but some effort against gravity). No effort against gravity (Limb falls, no effort against gravity, some voluntary movement observed).					
cuepatient by actively lifting leg0No drift (Leg holds 30 for f1Drift (Leg falls to intermedia	MOTOR: LEG (Right & Left) While supine, the patient is asked to maintain the leg at 30 for five seconds. If consciousness or comprehension is abnormal, cue patient by actively lifting leg into position while the request for effort is verbally given. 0 No drift (Leg holds 30 for five seconds). 1 Drift (Leg falls to intermediate position by the end of five seconds). 2 Cannot resist gravity (Leg falls to bed by five seconds but some effort against gravity).					
4 No movement.			- ,			
U Untestable due to amputat		1	1			
FACIAL PALSY	VERBAL	VISUAL FIELD	SENSORY			
Ask the patient to show	0 No Aphasia 1 Mild to Moderate	LFC Left Field Cut	0 Normal			
teeth, raise eyebrows, squeeze and shut eyes.	1 Mild to Moderate 2 Aphasia	RFC Right Field Cut B Blind	1 Partial Loss			
0 Normal 2 Partial	3 Global Aphasia (Mute)	Dinid	2 Severe Loss			
1 Minor 3 Complete	,					
	GLASGOW CO	MA SCALE				
EYE OPENING	VERBAL RESPONSE	BEST MOTO	R RESPONSE			
 Spontaneous To Speech To Pain None 	 5 Oriented 4 Confused Conversation 3 Inappropriate Words 2 Incomprehensible Sounds 1 None 	5 Localized 2	Abnormal Flexion Abnormal Extension None			
CODE KEY	SKIN TEMPERATURE	PU	LSES			
✓ Within Normal Limits	W Warm	CHARACTERISTIC	LOCATION			
* See Nursing Process Notes	C Cool	 Bounding Easily Felt Barely Palpable Present with Doppler Absent 	DP Dorsalis pedis (Pedal) POP Popliteal PT Posterior Tibia			
Print Form		Patient Label:				

	Saint Luke's Hospital Kansas City, MO 64111
Clinical Path	Clinical Path – Stroke (Ischemic and Intra-cerebral Hemorrhagic)
	troke and Intra-cerebral Hemorrhagic Stroke a, Retinal artery occlusion, Brain mass/tumor/abscess
Initiated Date:	Perfusion fusion, ineffective - Cerebral related to cerebral ischemia or cerebral hemorrhage Modified:Resolved: ebral perfusion will remain adequate as evidenced by:
Expected Outcome:	 Neuro function in expected range (IER) – LOC/motor/sensory/visual/cognitive Patient free of signs and symptoms of increased ICP Vital Signs remain in prescribed range Patient will not demonstrate seizure activity
Nursing Assessment/ Intervention (Practice Interventions)	 Assess neuro status/function trends – LOC, motor (movement, muscle tone, drift) sensory, pupils size, shape, symmetry, reactivity), cognition – every 4 hours Assess VS-BP, HR, respiratory rate and pattern, temp – every 4 hours Maintain body alignment in midline and avoid neck flexion or head rotation – ongoing Plan nursing care, procedure for energy conservation to minimize increased ICP – every 12 hours
	 Prevent accumulation of tracheobronchial secretions – every 4 hours Administer prescribed medications/fluids (volume expanders, vasoactive medication, anticoagulants, sedative, analgesics, etc.) to maintain hemodynamic parameters and optimize cerebral perfusion – as ordered
Problem: Communication Nursing Diagnosis: Impaired Initiated Date:	Verbal Communication related to neurological impairment Modified:Resolved:
Key Outcome Statement: Ab	lity to receive, interpret, and express messages will improve as evidenced by:
Expected Outcome:	 Communicates understanding of messages received Use of non-verbal, verbal and written communication to exchange messages with others
Nursing Assessment/	1. Assess ability to comprehend/communicate – daily
Intervention (Practice Interventions)	 Speak slowly, using short sentences – ongoing Provide communication board – once
(Fractice Interventions)	 Frome communication board = once Teach patient to use nurse call system – once
	 Teach Patient/SO use of communication tools/techniques – once Verify effectiveness of communication tool – PRN
	dge ge Deficit related to unfamiliarity with information / resources
	tient/SO demonstrates knowledge and / or skills needed to practice health behaviors
Expected Outcome:	 Patient/SO verbalized understanding of procedures and disease process Patient/SO verbalizes/demonstrates ability to care for self/patient Patient/SO sets realistic goals
Nursing Assessment/ Intervention (Practice Interventions)	 Assess patient/SO current knowledge level – daily Provide individualized instruction on specific aspect of care focusing on a single concept or idea – daily
	3. Review, reinforce and modify teaching methods as needed – daily
	 Assess readiness and ability to learn – daily Calleboard with Datient/OO to develop readictic learning chief in the second second
	 Collaborate with Patient/SO to develop realistic learning objectives – daily Evaluate Patient/SO ability to verbalize/demonstrate understanding of information/ instruction taught – once
Dath internet diate (diath and	

Path intermediate/discharge goals reviewed with patient/SO and mutually agreed upon. Date/Time: ______ RN Signature: _____

SAINT LUKE'S HOSPITAL OF KANSAS CITY Saint Luke's Care

The suggested plan represents the initial desired course of treatment and goals of recovery. These are representative or average guidelines only and should be reviewed periodically by the attending physician and other involved disciplines. Deviations are generally expected and reviewer the plane about he period on surgested. revisions to the plan should be made as warranted.

Patient Label

Clinical Path	Saint Luke's Hospital Kansas City, MO 64111 Clinical Path – Stroke (Ischemic and Intra-cerebral Hemorrhagic)
Initiated Date:	I Perfusion Peripheral Tissue Perfusion related to reduction / interruption of venous / arterial blood flow Modified:
Expected Outcome:	 Peripheral perfusion IER (color/temp/capillary refill/pulses) Skin intact Absence or peripheral edema Absence of localized extremity pain Sensation level IER Motor function IER
Nursing Assessment/ Intervention (Practice Interventions)	 Assess peripheral perfusion, i.e. peripheral pulses, color, temperature, capillary refill every 8 hours Inspect skin for tissue breakdown or ulcers – every 12 hours Assess pain level – every 12 hours Assess sensation and motor function – every 8 hours Maintain SCD's-collaborate with physician – once Assess for signs of peripheral embolus – ongoing Implement appropriate wound care; consider need for multidisciplinary consult i.e. skin nurse, pharmacy – once
Expected Outcome:	 Lung sounds IER Respiratory rate, rhythm IER Absence of onset of new fever/cough
Nursing Assessment/ Intervention (Practice Interventions)	 Assess pulmonary status – every 8 hours Keep HOB elevated 30 ° at all times – continuously * If not contraindicated* Maintain patient airway – ongoing Assess LOC, cough reflex, gag reflex and swallowing ability – every 8 hours and before each feeding Complete Stroke Dysphagia Screening: Keep patient NPO if demonstrates risk of

- before each feeding
 Complete Stroke Dysphagia Screening: Keep patient NPO if demonstrates risk of aspiration with nurse screening- once. Collaborate with speech therapy to assess swallow ability within 24 hours of admission.
 Position upright 90 degrees or as far as possible for at least 15 minutes before and after feeding with meals
 Implement oral care protocol once
 Suction set-up to bedside daily
 Verify placement of enteral tube prior to feeding/administering meds daily
 Collaborate with physician for peptic ulcer prophylaxis once

Patient Label



Clinical Path	Saint Luke's Hospital Kansas City, MO 64111 Clinical Path – Stroke (Ischemic and Intra-cerebral Hemorrhagic)
Initiated Date:	ced Nutrition, Less Than Body Requirements related to biologic / physiologic factors Modified: Resolved: utrient intake meets metabolic needs as evidenced by:
Expected Outcome:	 Patient/SO/Family/ caregiver expresses understanding of nutritional deficit/plan Fluid and food intake IER Blood glucose IER Patient/SO/caregiver demonstrates ability to maintain adequate nutritional intake
Nursing Assessment/ Intervention (Practice Interventions)	 Record percent of meal eaten – TID Assess weight - as ordered but at least weekly Collaborate with dietitian on nutritional assessment, counseling and/or plan – once Assess blood glucose - per orders and PRN Assess abdomen, bowel sounds, and bowel elimination – every 12 hours Assess Patient/SO/family/caregiver ability to maintain adequate nutrition – daily Assess Patient/SO/family understanding of nutritional deficit/plan – daily
Initiated Date:	y I Physical Mobility related to neurological impairment Modified:Resolved: bbility is maintained or increased as evidenced by:
Expected Outcome:	 Patient will demonstrate balance/strength development and ambulation activities IER No evidence of complications from impaired mobility Participates in establishing activity goals Patient/SO verbalizes and demonstrates understanding of safety measures and physical limitations
Nursing Assessment/ Intervention (Practice Interventions)	 Assess and record amount of activity – daily Assess level of assistance needed – every 12 hours Teach Patient/SO sign/symptoms of complications from impaired mobility – daily Collaborate with PT/OT and rehab to assess progress with mobility – daily Assess for complications of immobility – every 12 hours Collaborate with PT and patient to assess and set goals related to balance, strength and mobility – daily VTE prophylaxis – per physician orders Create Safe environment Provide mobility/SO safety measures relevant to patient's level of mobility. Use safe

Teach Patient/SO safety measures relevant to patient's level of mobility. Use safe patient handling equipment.

Patient Label



Saint Luke's Hospital Kansas City, MO 64111 Clinical Path – Stroke (Ischemic and Intra-cerebral Hemorrhagic)

		Intensive-Close Monitoring Phase	
		DATE: to	
	NURSING DIAGNOSIS/ OUTCOME STATEMENT	INITIAL Bolded Outcomes must be met before moving to W1 W2 next phase of care .	Explain unmet EOs Indicate time and nursing diagnosis with key word
	Nursing Diagnosis: Ineffective cerebral tis - sue perfusion related to cerebral ischemia or cerebral hemorrhage	Intervention : Neuro Assessment ICU without intervention q. 2 hr 4 then q. 4 hrs ICU post intervention/tPA q. 15 2 hr, q. 30 6 hr, and q. hr 16 (M/S) q. 2 hr 4 then q. 4 hr NIHSS or GCS if Hemorrhage(TJC) HOB according physician orders Initiate Physician Pre-printed Stroke Orders if not done (SLH Measure)	
NEURO/COGNITIVE PSYCH	Outcome Statement: Cerebral perfusion will be adeguate as evidenced by: Nursing Diagnosis: Impaired Verbal Com - munication related to neurological impairment Outcome Statement: Ability to receive, interpret & express information will	Expected Outcomes: Change in neuro status recognized and communicated. Change in neuro status recognized and communicated. Stat CT and NIHSS per physician orders Intervention : Establish communication tools/ techniques with Patient/SO Expected Outcomes for patient: Optimal use of verbal and nonverbal communication	
CARDIO / PULMONARY	improve AEB: Nursing Diagnosis: Ineffective peripheral tissue perfusion related to reduction/interruption of blood flow	Intervention: Vital Signs ICU without intervention q, 1 hr ICU post endovascular intervention/tPA q, 15 2 hr, q, 30 6 hr, q, hr 16 (M/S) q, 2 hr 4; q, 4 hrs if stable Maintain BP within range ordered per physician orders SaO ₂ monitoring per physician orders (TJC-1) Telemetry	
CARI	Outcome Statement: Tis- sue Perfusion will remain adequate as evidenced by:	Expected Outcomes: BP within desired range according to orders SPO_2 95 % on 5L NC Cardiac rhythm stable with HR 60 and 120	
	Nursing Diagnosis: Risk for aspiration related to impaired swallowing	Intervention: Complete Stroke Dysphagia Screening if NIHSS 0 on admission (TJC-7) Enter consult Speech Language Pathology as indicated on screening (TJC-10)	
RITION	Outcome Statement : Patient will not aspirate as evidenced by:	Expected Outcomes: Lung sounds IER/ Moves sputum out of airway SOA not present	
GI/GU/NUTRITION	Nursing Diagnosis : Imbalanced nutrition , less than required related to disease process	Intervention: Admission blood glucose Blood Glucose management per physician orders Avoid Foley or remove after 24 hours if not in ICU Bowel Management per physician orders	
	Outcome Statement : Nutrient intake meets needs as evidenced by: Nursing Diagnosis:	Expected Outcomes: Blood Glucose < 140 Patient/SO expresses understanding of nutritional plan Intervention:	
SKIN/ MUSCULO SKELETAL	Impaired Physical Mobility related to disease Outcome Statement: Mobility is maintained or	Rehab Team consult entered (TJC- 10) Expected Outcomes: Dangle as tolerated	
	increased as evidenced by Nursing Diagnosis: Defi- cient knowledge related to unfamiliarity with information/ resources	Intervention: Stroke Education Book Review and educate about Stroke Risk Reduction Plan (TJC-8) Initiate discharge assessment & needs planning Explain to Patient/SO admission procedures and unit visiting hours Tobacco cessation material provided if applicable	
ΤEA	Outcome Statement: Patient/SO demononstrate knowledge/skills needed to practice health behaviors as evidenced by:	Expected Outcomes: Patient/SO verb understanding of ADM/visiting hours Patient/SO verb understanding of Stroke Education Book	
	DL KEY: Expected	A in a indicates intervention done A "o" in a or on a line indicates the item was not pertinent	"Initials" on a line means Expected Outcome done and
SYMBC	Outcome Interventions	A " " in a or on a line indicates the item was not done as expected	findings as expected
SYMBC			findings as expected



NursingDagnosis intervention seeperlasion metaded to seeperlasion metaded to seeperlasion metaded to seeperlasion metaded to seeperlasion metaded to monitoring Intervention q.1 tr CLUMS on the metaded to the commendation of the commendation to the seesement CLU post intervention q.1 tr CLUMS on the metaded to monitoring Intervention q.1 tr CLUMS on the metaded to monitoring Outcome Statement: dedquate as evidenced by monitoring Expected Outcomes: munication related to munication			Saint Luke's Hospital	
DURSING DIAGNOSIS UTTCOME STATEMENT NUTTAL Bolded Outpactations must be met before moving to WTW2 next phases of care Explain unmet EOs Indicate time and nursing diagnosis infectore certain sis as seperfusion related to certain storemain corrections (BIATA CT and NIHSS per physician orders for changes in neuro status Uttome Statement: Cerebral perfusion will be departed as evidenced by: Intervention (LCL-so) (Estematic Strate-sol multi-24 hour Strate-sol (Estematic Strate-sol multi-24 hour Strate-sol (Estematic St	(Sumpour and	ath – Stroke (Ischemic and Intra-cerebral Hemorrhagic)	
OUTCOME STATEMENT MUTAL Bolded Outcomes must be met before moving to MITAL metry hase of care . Explain undECS Indicate time and number ECS Indicate time ECS Indicate time ECS Indicate time and number ECS Indicate time ecs Indicate time ecs Indicate time ecs Indicate time ecs Indicate time ECS Indicate tintervention Indicate Indicate Indicate Indicate Indi				
Open Participant Provided		OUTCOME STATEMENT	INITIAL Bolded Outcomes must be met before moving to	Indicate time and nursing
Outcome Statement: Expected Qutcomes: Ability to receive, interpret and express information will improve as evidenced by: Depticipation of the second optimal patient communication stabilished Nursing Diagnosis: Intervention: Telemetry monitoring Q, as ordered to keep > 95%. ICH patients may wean Q ₂ as ordered to keep > 95%. ICH patients may wean Q ₂ Doubload flow VTE prophulaxis per orders (TJC-1) Durting Diagnosis: Expected Outcomes: 	E PSYCH	Ineffective cerebral tis - sue perfusion related to cerebral ischemia or cerebral hemorrhage	Neuro assessment ICU-post intervention q.1 hr ICU/MS no intervention q.4 hr if no deterioration NIHSS score before ICU DC or within 24 hrs of procedure (SLH Measure) STAT CT and NIHSS per physician orders for changes in neuro status	
Outcome Statement: Expected Outcomes: Ability to receive, interpret and express information will improve as evidenced by: Expected Outcomes: Nursing Diagnosis: Intervention: Termentry monitoring Outcome Statement: Termentry monitoring O ₄ as ordered to keep 9 5%. ICH patients may wean O ₂ after 48 hours if Spoce 9 5%. Nursing Diagnosis: Expected Outcomes: Diod flow VTE prophylaxis per orders (TJC-1) Outcome Statement: Expected Outcomes: Synthetic experimention: Follow SLP recommendations if aspiration risk. Outcome Statement: Expected Outcomes: Patient/SO/AO not present/ Moves sputum out of airway Evaluate q. shift for signs of aspiration Outcome Statement: Expected Outcomes: Patient/SO/AO not present/ Moves sputum out of airway Evaluate q. shift for signs of aspiration Outcome Statement : Expected Outcomes: Nursing Diagnosis : Intervention: Review lipid panel (LD > 100 discus with physician orders blood Glucose management per physician orders Blood Glucose management per physician orders Discome Statement : Expected Out comes: Nursing Diagnosis: Expected Out comes: Nurang Diagnosis: Expected Out comes:	COGNITIVE	Cerebral perfusion will be adequate as evidenced by:	Change in neuro status recognized and communicated. (<u>lschemic Stroke only</u>) Antithrombotic started within 24 – 48 hours (TJC- 5)	
OPDOUND Ability to receive, interpret and express information improve as evidenced by:	NEURO/ C	Impaired Verbal Com - munication related to neurological impairment	Intervention : Reinforce patient/SO communication tools/techniques	
Opposed Ineffective peripheral to reduction/interruption of VTE prophylasis per orders (TUC-1) Dutcome Statement: Tile- sue Perfusion will remain adequate as evidenced by: Expected Outcomes: SPO2, 95 % on 5L NC Mursing Diagnosis: Risk evidenced by: Intervention: Follow SLP recommendations if aspiration risk. Patient will not aspirate as evidenced by: Expected Outcomes: Supervention: Follow SLP recommendations if aspiration risk. Nursing Diagnosis: It evidenced by: Evaluate q. shift for signs of aspiration out of airway Patient/SOC/Family understands in dragement per physician orders less than required related to disease process Intervention: Breath sounds IER/ SOA not present/ Moves sputum out of airway Outcome Statement : nutrient intake meets needs as evidenced by: Breath sounds IER/ SOA not present/ Moves sputum out of airway Outcome Statement : nutrient intake meets needs as evidenced by: Breath sounds IER/ SOA not present/ Moves sputum out of airway Outcome Statement : nutrient intake meets needs as evidenced by: Expected Outcomes: Patient/SO Understands nutritional deficit and feeding plan Outcome Statement : nutrient intake meets needs as evidenced by: Expected Outcomes: Patient/SO understands nutritional deficit and feeding plan Outcome Statement : needs as evidenced by: Expected Outcomes: Patient/SO transfer plans and expectations out of ICU Discuss stroke Type, etiology/dx tests utilizing education book Out		Ability to receive, interpret and express information will improve as evidenced by:	SLP consult and recommendations available (TJC- 10) Optimal patient communication established	
adequate as evidenced by:	ARDIO / MONARY	Ineffective peripheral tissue perfusion related to reduction/interruption of blood flow	Telemetry monitoring O ₂ as ordered to keep > 95%. ICH patients may wean O ₂ after 48 hours if SpO ₂ > 95% VTE prophylaxis per orders (TJC- 1)	
Oppose For aspiration related to Follow SLP recommendations if aspiration risk. Dutcome Statement : Expected Outcomes: Expected Outcomes: Patient will not aspirate as evidenced by: Breath sounds IER/ SOA not present/ Moves sputum out of airway Nursing Diagnosis : Intervention: Imbalanced nutrition , less than required related to disease process Intervention: Outcome Statement : Blood Glucose management per physician orders Blood Glucose management per physician orders if not in ICU Assess bladder incontinence and retention when Foley removed Outcome Statement : Expected Outcomes: Nutrient intake meets needs as evidenced by: Patient/SO understands nutritional deficit and feeding plan Uffore output adequate Intervention: Impaired Physical Mobility related to disease Out of bed for all meals Very Outcome Statement: Expected Outcomes: Mobility is maintained or increased as evidenced by: — Patient/SO knowledge level, readiness/ability to learn Mobility with information/ resources Defi- Discuss stroke type, etiology/dx tests utilizing education book Ducome Statement: Expected Outcomes: Assess Patient/SO framily: Out of bed for all meals Discuss stroke type, etiology/dx tests utilizin	D II	sue Perfusion will remain adequate as evidenced by:	BP within desired range according to orders SPO ₂ 95 % on 5L NC Cardiac rhythm stable with HR 60 and 120	
Symbol Preview lipid panel (LDL > 100-discuss with physician) (TJC 6) Outcome Statement : Expected Outcomes: Nutrient intake meets needs as evidenced by: — Patient/SO understands nutritional deficit and feeding plan Nursing Diagnosis: Intervention: Impaired Physical Mobility related to disease Out output adequate Mursing Diagnosis: Intervention: Mobility is related to disease Out of bed for all meals Out own Statement: Expected Outcomes: Mursing Diagnosis: Defi- increased as evidenced by Intervention: Nursing Diagnosis: Defi- cient Knowledge related to unfamiliarity with information/ resources Intervention: Outcome Statement: Fige-terd Outcomes: Patient/SO knowledge level, readiness/ability to learn Discuss stroke type, etiology/dx tests utilizing education book Discuss with Patient/SO framify: — Demonstrates safety measures, verbalizes rehab team practice health behaviors as evidenced by: SYMBOL KEY: Expected A in a indicates intervention done "Initials" on a line means	NOIL	for aspiration related to impaired swallowing Outcome Statement : Patient will not aspirate as	Follow SLP recommendations if aspiration risk. Evaluate q. shift for signs of aspiration Expected Outcomes: Breath sounds IER/ SOA not present/ Moves sputum out of airway	
Outcome Statement : Expected Outcomes: 	GI / GU / NUTR	Imbalanced nutrition , less than required related	Intervention: Bowel management per physician orders Blood Glucose management per physician orders Remove Foley per physician orders if not in ICU Assess bladder incontinence and retention when Foley removed	
Opposite Impaired Physical Mobility Rehab Team POC in place (TJC- 10) Impaired Physical Mobility Out of bed for all meals Out of bed for all meals Outcome Statement: Expected Outcomes: — Participates in ADLs Mobility is maintained or — No vidence of skin breakdown — Intervention: Versing Diagnosis: Deficient knowledge related to Movidence of skin breakdown Cient knowledge related to Movidence of skin breakdown Discuss with Patient/SO knowledge level, readiness/ability to Intervention: resources Discuss with Patient/SO transfer plans and expectations out of ICU Discuss Stroke Risk Reduction Plan (TJC-8) Expected Outcomes: Cutcome Statement: Practiced Networks as evidenced by: — Demonstrates safety measures, verbalizes rehab team plan evidenced by: Outcome Statement: Practiced Just comes - Patient/SO/Family: — Demonstrates safety measures, verbalizes rehab team plan evidenced by: SYMBOL KEY: Expected A in a indicates intervention done "Initials" on a line means		Nutrient intake meets needs as evidenced by:	Expected Outcomes: Patient/SO understands nutritional deficit and feeding plan Demonstrates normal bowel function	
Nursing Diagnosis: Defi- cient knowledge related to unfamiliarity with information/ resources Intervention: Assess Patient/SO knowledge level, readiness/ability to learn Discuss with Patient/SO transfer plans and expectations out of ICU Discuss stroke type, etiology/dx tests utilizing education book Outcome Statement: Patient/SO demonstrates knowledge/skills needed to practice health behaviors as evidenced by: Expected Outcomes-Patient/SO/Family: 	AUSCULO SKELETAL	Impaired Physical Mobility related to disease Outcome Statement: Mobility is maintained or	Rehab Team POC in place (TJC- 10) Out of bed for all meals Expected Outcomes: — Participates in ADLs	
Policient's adventer and the second control as a faith of the		Nursing Diagnosis: Defi- cient knowledge related to unfamiliarity with information/	Tolerates out of bed for meals 15-30 min each time Intervention: Assess Patient/SO knowledge level, readiness/ability to learn Discuss with Patient/SO transfer plans and expectations out of ICU Discuss Stroke type, etiology/dx tests utilizing education book	
	TE	Patient/SO demonstrates knowledge/skills needed to practice health behaviors as	Expected Outcomes -Patient/SO/Family: Demonstrates safety measures, verbalizes rehab team plan Verbalizes understanding diagnostic tests/type of stroke	
Outcome A "o" in a or on a line indicates the item was not pertinent Expected Outcome done and Interventions A " "in a or on a line indicates the item was not done as expected findings as expected	SYMBC	DL KEY: Expected Outcome	A in a indicates intervention done A "o" in a or on a line indicates the item was not pertinent	Expected Outcome done and
Init. Signature PATIENT LABEL	Init.	Signature		

	linical Path	Saint Luke's Hospital Kansas City, MO 64111	
	Clinical F	Path – Stroke (Ischemic and Intra-cerebral Hemor	
		Transitional-Rehabilitation F	lase
	NURSING DIAGNOSIS/ OUTCOME STATEMENT	DATE:to INITIAL Bolded Outcomes must be met before moving to W1 W2 next phase of care.	Explain unmet EOs Indicate time and nursing diagnosis with key word
н	Nursing Diagnosis: Ineffec- tive cerebral tissue perfusion related to ischemia or cerebral hemorrhage	Intervention: Neuro Assessment q. 4 hours STAT CT and NIHSS per physician order if changes in neuro status	
VEURO / COGNITIVE PSYCH	Outcome Statement: Cerebral perfusion will be adequate as evidenced by: Nursing Diagnosis:	Expected Outcomes -Patient: No deterioration in neuro status Intervention: Patient/SO:	
URO/COG	Impaired Verbal Com- munication related to neurological impairment Outcome Statement:	Reinforce use of communication tools/techniques Follow SLP POC Expected Outcomes:	
Ž	Ability to receive, interpret and express information will improve as evidenced by: Nursing Diagnosis	Intervention:	
DIO / NARY	Ineffective peripheral tissue perfusion related to reduction/interruption of blood flow	DC Telemetry monitoring if no arrhythmias at 72 hours O ₂ sat q. shift and PRN VTE prophylaxis per orders (TJC-1)	
CARDIO / PULMONARY	Outcome Statement: Tissue Perfusion will remain adequate as evidenced by:	Expected Outcomes: BP within desired range according to orders No supplemental O₂ required Cardiac rhythm stable with HR ≥ 60 and ≤ 120	
	Nursing Diagnosis: Risk for aspiration related to impaired swallowing Outcome Statement:	Intervention: Follow SLP recommendations if aspiration risk Evaluate q. shift for signs of aspiration Expected Outcomes:	·
NOILIS	Patient will not aspirate as evidenced by: Nursing Diagnosis: Imbal-	Lung sounds IER/SOA not present/Moves sputum out of airway Patient/SO/Family acknowledge risk for aspiration Intervention:	
GI/GU/NUTRITION	anced nutrition, less than required related to disease process	Establish long term nutrition plan with team and family Bowel management program per physician orders Blood Glucose management per physician orders Assess bladder incontinence and retention	
	Outcome Statement: Nutri- ent intake meets needs as evidenced by:	Expected Outcomes: Expected Outcomes: Expected Outcomes: East at least 50% of diet or able to tolerate tube feeding Demonstrates normal bowel function Urine output adequate	
	Nursing Diagnosis: Impaired Physical Mobility related to disease	Intervention: Follow Rehab Team POC for progression with mobility Up in chair for all meals Ambulating as tolerated	·
SKIN/ MUSCULO SKELETAL	Outcome Statement: Mobility is maintained or increased as evidenced by	Expected Outcomes: Ne evidence of complication from impaired mobility Demonstrates maintenance/ improvement of strength/ balance Patient/SO/Family participates in establishing activity goals	
		Demonstrates increasing independence with ADLs / self care Tolerates Up in chair for all meals 30 – 60 minutes each time	
EACHING	Nursing Diagnosis: Deficient knowledge related to unfamiliarity with information/ resources Outcome Statement:	Intervention: Review stroke cause/lest results Discuss with Patient/SO rehab/discharge needs Discuss Stroke Risk Reduction Plan (TJC-8) Expected Outcomes Patient/SO/Family Verbalizes Under-	
TEACI	Patient/SO demonstrates knowledge/skills needed to practice health behaviors as evidenced by:	standing of: Relevant safety measures (mobility/ nutrition) Rehab needs & next level of care Stroke etiology/test results	
SYMBOL	KEY: Expected Outcome Interventions	Personal Stroke risk factors A v in a indicates intervention done A °o" in a or on a line indicates the item was not pertinent A °o" in a or on a line indicates the item was not done as expected	"Initials" on a line means Expected Outcome done and findings as expected

Init. Signature

PATIENT LABEL



	llinical Path	Saint Luke's Hospital Kansas City, MO 64111	
		ath – Stroke (Ischemic and Intra-cerebral Hemorrhagic) Intensive-Close Monitoring Phase	
	NURSING DIAGNOSIS/	DATE: to	
	OUTCOME STATEMENT	INITIAL Bolded Outcomes must be met before moving to W1 W2 next phase of care .	Explain unmet EOs Indicate time and nursing diagnosis with key word
E PSYCH	Nursing Diagnosis: Ineffective cerebral tissue perfusion related to ischemia or cerebral hemorrhage	Intervention : NIH Stroke Scale Score on day of discharge (SLH Measure) (Ischemic Stroke) Discharge on antithrombotic per physicon order (TDC-2) Ischemic Stroke) Discharge on anticoagulant if AFib per physician order (TDC-3)	
VEURO/COGNITIVE PSYCH	Outcome Statement: Cerebral perfusion will be adequate as evidenced by: Nursing Diagnosis: Impaired Verbal Com - munication related to neurological impairment	Expected Outcomes: No deterioration in neuro assessment Intervention : Reinforce Patient/SO use of communication tools/techniques	
ШN	Outcome Statement: Ability to receive, interpret and express information will improve as evidenced by:	Expected Outcomes for Patient: Improved ability to communicate with team/SO	
CARDIO/ PULMONARY	Nursing Diagnosis: Ineffective peripheral tissue perfusion related to reduction/interruption of blood flow	Intervention: VTE prophylaxis per orders (TJC- 1)	
PULI	Outcome Statement: Tissue Perfusion will remain adequate as evidenced by:	Expected Outcomes: BP within desired range according to orders SpO ₂ 95% on <u>RA</u> Cardiac rhythm stable with HR 60 and 120 or IER	
NO	Nursing Diagnosis: Risk for aspiration related to impaired swallowing Outcome Statement : Patient will not aspirate as evidenced by:	Expected Outcomes: Lung sounds IER/ SOA not present/Moves sputum out of airway Patient/SO/Family acknowledge risk for aspiration	
GI/GU/NUTRITION	Nursing Diagnosis : Imbalanced nutrition , less than required related to disease process Outcome Statement :	Intervention: Discharge on cholesterol medication if LDL > 100 per physician orders (TIC- 6) Expected Outcomes:	
GI/G	Nutrient intake meets needs as evidenced by:	Lette du clower at least 50% of diet or able to tolerate tube feeding Demonstrates normal bowel function and adequate urine output Intervention:	
	Impaired Physical Mobility related to disease	Discuss with patient/SO/Family mobility/activity goals for discharge. Multidisciplinary Team finalize discharge assessment and discharge planning needs	
MUSCULO	Outcome Statement: Mobility is maintained or increased as evidenced by	Expected Outcomes: Ne evidence of complication from impaired mobility Patient/SO/Family understands activity goals Patient/SO/Family involved in and understands discharge planning Up in the chair 60 - 90 min for all meals	
S M	Nursing Diagnosis: Deficient knowledge related to unfamiliarity with information/resources	Intervention: CompleteStrokeRiskPlan at discharge (TJC-8&SLH Measure) Discuss plan to prevent stroke (meds/risks) (TJC-8) Give discharge F/U plan- Dr appt/rehab/lab test (TJC-8 & SLH Measure) Educate on stroke S&S (ACT FAST) (TJC-8) Patient/SO/Family discuss rehab plan and patient disposition Discuss Stroke Risk Reduction Plan (TJC-8)	
TEACHING	Outcome Statement : Patient/SO demonstrates knowledge/skills needed to practice health behaviors as evidenced by:	Expected Outcomes: Patient/SO verbalizes discharge plan (risk factors, meds, discharge F/U needs, stroke warning signs (ACT FAST) (TJC- 8)	
YMBC	DL KEY: Expected Outcome Interventions	A in a indicates intervention done A "o" in a or on a line indicates the item was not pertinent A " " in a or on a line indicates the item was not done as expected	"Initials" on a line means Expected Outcome done and findings as expected

			Saint Luke's Care Saint Luke's Health System	n Date	of Discharge:
Dis	charge Instructions	TIA and S	Stroke Education / Dischar	ge Packet	
FOLLOW UP CARE	 Review and follow another TIA/Strok If you use tobacc quit in the last tw or call Tobacco Q Call 911 If you experient 	roke Sca t ately severe evere stroke a Plan to De w v recomme e. co, we want relve monti Line 1-80 uce sudden	stroke crease Another TIA/Stroke" indations on Risk Factor Re to advise you to quit. If you is and need help staying tob 00-QUITNOW (1-800-784-8669 onset of these stroke warnin	are interested acco free, plea २) g signs:	in quitting or if you have se talk to your physician
	o Difficulty speaki o Trouble with visi o Trouble walking, o Severe headach After discharge you ne Clinic/Physici	ng or unders on – loss of dizziness, l e without kr ed to call fo an	or blurring oss of balance or coordination iown cause or a follow-up appointment to Phone #	see the doctor Date	·
	Date	Time	Test/Treatment INR		Script for test given to patient YES □ NA
	□ Call		to schedule outpatient PT/O	T/Speech	
ACTIVITY	No restrictions No de Return to work Other: May resume sexual active		days if had a stroke		
DIET	\Box No salt added if being	treated for h	low the food guide pyramid. igh blood pressure or heart dis recommended by speech thera		

Original with Patient Signature: To Chart

Copy: To Patient Patient Label:



Saint Luke's Care Saint Luke's Health System

Date of Discharge: _____

PAIN	 A. Pain control is important to your recovery. Do not hesitate to take the pain medication. Take pain medications as prescribed and take them before your pain becomes severe. If your level of pain is not allowing you to gradually increase your activity as directed, contact your physician. B. Please refer to more specific information about your pain medications that you received from the hospital or pharmacist. Other:								
		Stroke S	Support Groups						
CES	Saint Luke's East 816-932-5100 When: Second Thursday of every month, 6 - 7:30 p.m. Hospital Saint Luke's East Hospital Legacy Conference Room 100 N. E. Saint Luke's Blvd. Lee's Summit, MO 64086								
COMMUNITY RESOURCES	Saint Luke's Hospital- Plaza campus	816-932-2020	When: Second Thursday of every month, 5 - 7 p.m. Center for Health Enhancement 4200 Wornall Road Kansas City, MO 64111						
NUNIT	American Stroke 913-648-6727 www.strokeassociation.org Association								
COMN	American Stroke Foundation	913-649-1776	www.americanstroke.org						
	Other Resources: (include Name or Resource and contact) National Stroke Association: www.stroke.org Free – Stroke Connection Magazine, call 1(888) 478-7653 Free – Stroke Smart Magazine, call 1(800) 787-6537 HealthStream® Research Online may call you for a brief survey about your hospital stay. We value your input and care about you.								
l have l have	 e all of my personal belonging e been screened for current vie e been given discharge instructions (Kra information about needed vie a copy of my discharge me 	accinations. mes core education or str vaccinations if appropriate dications	roke booklet), a,						
	 my personal stroke risk factors and understand how to decrease my risk for future strokes aderstand and agree with this plan of care: 								
Patie	nt/SO Signature								
Disch	narge RN Signature		Date/Time						

Original with Patient Signature: To Chart

Copy: To Patient Patient Label:



Saint Luke's Care Saint Luke's Health System

Risk Factor Reduction Plan to Decrease Another Stroke or TIA Take this sheet to your physician to show treatment recommendations

My Risk Risk Factors		Goals	My Numbers	My Plan	RN Initials/Date
Checked items are your risks	You are at risk for future strokes	Medication to prevent clots that lead to another stroke.	You have been placed on 		
□ □NA	High Blood Pressure	Less than 120/80	Last blood Pressure	Last blood Pressure	
□ Atrial Fibrillation Medications to thin blood as directed Scheduled lab draw measure thinning of blood if ordered		Last INR Next INR to be drawn (date)	□Take Coumadin daily and know INR (target 2-3) □Take dabigatran (Pradaxa) twice daily □Take rivaroxaban (Xarelto) daily □Other blood thinners		
□ □NA	Abnormal lipids (fats in the blood)	 Cholesterol less than 200 Triglycerides less than 150 HDL (Good) greater than 40 LDL (Bad) less than 100 If Diabetic less than 70 	Cholesterol Triglycerides HDL LDL	⊡Medicine ⊠Low fat diet ⊠Exercise	
□ □NA	Smoking	Stop smoking	packs per day	packs per day UCessation aids UCessation education	
	Diabetes	Goal Hemoglobin A1C 7% ≥9% - Blood sugars are	Hemoglobin A1C	⊡Medicine / insulin ⊠Check blood sugar	
		high ≥ 7% - Blood sugars high sometimes 7% -Great control Blood sugar less than 120	Last Blood Sugar	as directed ⊠Diabetic Diet	
□ □NA	Alcohol use	Less than 2 drinks a day for men Less than 1 drink a day for women	Average number of drinks per day prior to stroke	Alcohol intake within recom- mended limits	
□ □NA	Weight Management	BMI less than 25% Waist circumference: Men less than 40 inches Women less than 35 inches	Weight	⊠Work to achieve targets	
	Physical Activity 30 minutes daily. Stroke patients should have approval by physician prior to beginning.		Aerobic exercise prior to stroke Yes No	Work to increase to daily aerobic exercise	
		ACT <u>F.A.S.T</u> – Call 911 if Stro	oke Warning Signs O	ccur	
<u>F</u> ace	Ask the person to smile. Does one side of the face droop?		<u>S</u> peech	Ask the person to say a simpl sentence.	
<u>A</u> rms	Ask the person to Does one arm drif		<u>T</u> ime	If the person shows symptoms, time is ir Call 911.	

RN Initial: ____ Signature: _____RN Initial: ____ Signature: ____

RN Initial: _____ Signature: ______ RN Initial: _____ Signature: _____

Discharge Review RN Signature: ____

_Date: _____

Original with Patient Signature: To Chart Copy: To Patient

Patient Label:

___ Time: __

Clinical Medicine/Neurology

The Stroke Center Handbook Second Edition



Organizing Care for Better Outcomes

Marilyn M. Rymer, MD • Debbie Summers, MSN • Pooja Khatri, MD

Since publication of the first edition of this book, new treatments have become available in acute intervention for stroke and new evidence has been uncovered regarding prevention and neurorehabilitation. Designed for the entire team at any stroke center, including physicians, nurses, therapists, and administrators, **The Stroke Center Handbook: Organizing Care for Better Outcomes, Second Edition** delivers a timely update of the latest developments in this critical area.

Exploring best practice management protocols, the content covers initial presentation, rapid response and emergency interventions, clinical and administrative procedures, and neurocritical care. Highlights of the second edition include

- The significant contribution of telemedicine to stroke treatment
- Improved organization of EMS systems to route patients directly to stroke centers
- New devices available for intra-arterial thrombectomy
- The impact of CT and MR perfusion and angiography on case selection for advanced therapy
- New pharmaceutical therapies for the prevention of stroke in patients with atrial fibrillation
- Advances in stroke rehabilitation techniques

The authors of the book are pioneers in developing stroke centers and regional networks that have provided diagnosis and treatment to thousands of patients. Their expertise enables practitioners to establish and efficiently manage a facility and to participate more effectively as part of a multidisciplinary care team—providing stroke victims with the best possible outcome.



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