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CHAPTER 1

History taking

Beep…Beep…Beep
CODE STROKE in the Emergency Department,
Acute zone bed 10.

Welcome to the code stroke; let’s get started.

The initial assessment of the code stroke patient involves identifying whether the clinical presentation is compatible with an acute stroke diagnosis or a stroke mimic. The first two chapters of this book will help answer this question. Like a good detective, you need to gather the important clues, ignore distractions and red herrings, and eliminate the other suspects—all in a timely manner. This chapter will provide you with a stepwise approach to:

- Taking an appropriate and focused history by gathering relevant clinical information from multiple sources.
- Identifying the common symptoms associated with (and not associated with) acute stroke.

Chapter 2 will discuss various stroke mimics and how to clinically differentiate them.

Early stroke symptom recognition is important to facilitate rapid transfer to a stroke center. Regional Emergency Medical Services (EMS) have protocols in place to identify and prioritize potential stroke cases,
and try to minimize transportation time to the most appropriate stroke center. The mnemonic **FAST**, which stands for **F**ace (sudden facial droop), **A**rm (sudden unilateral arm weakness), **S**peech (sudden speech difficulty), and **T**ime to call EMS, is being used to promote public awareness. Most prehospital stroke screening tools involve some combination of these cardinal symptoms.

It has been estimated that nearly **two million neurons die each minute that elapses during the evolution of an average acute ischemic stroke**. Each hour without treatment the brain loses on average as many neurons as 3.6 years of normal aging. This is captured by a commonly used phrase **“time is brain.”**

### Ideal stroke treatment targets

- Door-to-needle time for intravenous tissue plasminogen activator (tPA): <30 min
- Door-to-groin puncture time for endovascular therapy: <60 min

*Disability decreases with quicker treatment; therefore, aim for the fastest assessment for potential brain-saving or lifesaving treatment.*

For the resident physician or medical student on call, the first task is a simple one: write down the time you first received the code stroke page. There are many other time-related parameters that you may need to document throughout the code stroke, including time of patient arrival, time of the first CT scan slice, and time of tPA administration. This becomes important later when calculating *door-to-CT scan* time or *door-to-needle* time. After all, the quicker a stroke patient is treated, the more likely they are to have a functionally independent outcome.
Regional variations exist in terms of code stroke triage in the emergency department (ED). Depending on the hospital, the pager may notify you where the stroke patient is in the ED (or on the inpatient hospital ward), or you may need to call the number on the pager to confirm you received the page, ask the location of the stroke patient, and their estimated time of arrival if they are not already in the ED.

Sometimes the ED charge nurse will have some additional information for you. This prenotification clinical information can vary in terms of how detailed it is. Sometimes it is very detailed with a high pretest probability for stroke, such as:

*We have a 76-year-old woman from home with a witnessed onset at 1500 hours of aphasia and right face, arm and leg weakness.*

At other times, the clinical information is vague and undifferentiated, such as:

*“85-year-old man with confusion.”* This could be a number of neurological or non neurological conditions (more on stroke mimics to come in Chapter 2).

Not all activated code strokes are from the ED. Inhospital strokes (i.e., a patient admitted to the ward) also occur, though with less frequency. Your approach to the patient should be the same. Often, the patient's medical comorbidities or recent surgery precludes the use of tPA.

Once the code stroke is activated, many different people are set in motion (even before the stroke resident/staff make their way to the patient). The first step is a rapid assessment and rushing the patient to the CT scanner as quickly as possible. In some hospitals, prior to the CT scan, the nurses will insert two cubital fossa IV lines, complete a 12-lead ECG, and draw urgent bloods that are sent stat for: CBC, electrolytes,
creatine, coagulation profile, random blood glucose level, troponin and type and screen. This blood work will help with treatment decisions and contraindications to tPA.

You have now made your way to the stroke patient in the ED. Like any acute situation in medicine, do not forget the basics: ABCs—Airway, Breathing, Circulation. Quickly eyeball the patient and check the vital signs from the monitors or from EMS or the triage nurse. Make sure that the patient is protecting their airway and there are no immediate life-threatening issues. Luckily, this is typically not the case, although some patients have a depressed level of consciousness either from a devastating intracranial event or another systemic issue. If the patient looks unstable, do not hesitate to request help from an ED physician, or rapid response/ICU.

**Important initial questions to ask**

Make every effort to speak directly to the paramedics, the patient, patient’s family, and any eyewitness to obtain the most reliable medical history. There are 6 key questions to ask first, before we get a more detailed history and understand exactly what happened (specific symptoms and chronology):

1. Clarify the time the patient was “last seen normal” and the exact time of onset of symptoms, or the time the patient was found with symptoms.
2. What are the main neurological deficits? Did they improve or worsen en route?
3. Relevant past medical history and medications (do they have known atrial fibrillation? Are they taking anticoagulant medications? Do they have an allergy to contrast dye?).
(1) The most important initial question to clarify with the patient, family, or witness is the stroke onset time and the patient’s “last seen normal time,” as it starts the clock on eligibility for acute treatment, i.e., thrombolytic therapy with tPA and/or endovascular therapy. Sometimes the exact time of onset is unknown/uncertain or difficult to obtain, but try to really pin it down. Use clock time (i.e., 23:00), rather than “2 h ago,” or “30 min ago.” If the patient woke up with symptoms (i.e., a wake-up stroke), when were they last seen well? Did they get up in the middle of the night to use the washroom and were they normal then? If the patient woke up with symptoms in the morning without previous awakenings, we must use their last seen normal time which is typically when they went to bed. A common reason for ineligibility for tPA is arrival at the hospital too late, beyond the time window for treatment (although this is an evolving area of clinical research, and advanced imaging may enable the use of tPA outside the traditional time window).

(2) Now we need to clarify the neurological deficits.

Clinical features in favor of an acute arterial stroke diagnosis:

- Sudden onset of persistent focal neurological symptoms
- Symptoms compatible with a vascular territory (see Chapter 4—stroke syndromes).
Specifically, what are the neurological symptoms? Are they acute? Are they stable, fluctuating, worsening, or improving? Acute stroke is a dynamic condition and it is important to ask EMS if the symptoms have improved compared to their initial assessment.

Was there a loss of consciousness or evidence of seizure (rhythmic activity, bitten tongue, bruising, incontinence)? Focal deficits can occasionally follow a seizure (postictal) and are transient (called Todd’s paresis). Are there associated fever or infectious symptoms, or other systemic symptoms such as palpitations, chest pain, or shortness of breath?

Time course and duration of symptoms is important. Migraine auras by definition last between 5 and 60 min in adults; however, typically they last 20–30 min. Seizures on average occur for 30 s–3 min. Syncope is brief, lasting seconds. More on stroke mimics in the next chapter.

**What is a transient ischemic attack (TIA)?**

**Definition:** a clinical syndrome characterized by the sudden onset of focal neurological symptoms that resolve within 24 h (although typically lasting minutes) AND no infarction is visualized on brain imaging.

The symptoms are transient as blood flow is temporarily blocked and then restored. Perfusion is dependent on many local and systemic factors (migration of clot, collateral circulation, cardiac output, blood pressure, etc.).

These patients are at risk of recurrent stroke—especially within the first week of symptom onset—and require timely assessment and management.

**Clinical pearl:** Given the increasing availability of MRI with diffusion weighted sequences, many clinical events previously thought to be TIAs are in fact small ischemic strokes.
Clinical pearls—We will review examples of neurological symptoms typically *not* associated with stroke

**Recurrent/stereotyped episodes of aphasia**
Aphasia is a cortical phenomenon and repeated ischemia to the same cortical area can be caused by TIAs if there is significant intracranial occlusive disease. However, one should also consider focal seizures (ictal aphasia). Another less likely etiology is migraine aura which may occur without headache.

**Isolated dysphagia**
When dysphagia is acute in onset, stroke should be considered, although isolated dysphagia is rare. Often, clarification of the history reveals a subacute or chronic presentation in which case the differential diagnosis is broad and includes neurological and non neurological etiologies.

**Lower motor neuron (“peripheral”) facial weakness (i.e., Bell’s Palsy)**
This pattern of weakness involves the forehead and is usually due to a lesion in the ipsilateral facial nerve (seventh cranial nerve). Rarely, a lesion in the brainstem facial nucleus or fascicle can also result in a lower motor neuron CN 7 palsy, but is almost always accompanied by a nuclear sixth nerve palsy or other symptoms in this scenario.

**Isolated anisocoria**
You cannot attribute isolated anisocoria to a stroke without associated ptosis to suggest a Horner’s syndrome (associated with carotid artery dissection), or ptosis with some deficits in the rectus muscles innervated by the third cranial nerve to suggest a third nerve palsy (assuming the patient is not comatose).
(3) What is their past medical history? Do they have a previous history of stroke/TIA?

Vascular risk factors include:
- Previous TIA/stroke
- Atrial fibrillation
- Hypertension
- Diabetes
- Dyslipidemia
- Coronary artery disease or congestive heart failure
- Valvular heart disease
- Smoking
- Obstructive sleep apnea
- Alcohol abuse
- Other less common factors: migraine, oral contraceptive agents, hormone replacement therapy, antiphospholipid antibody syndrome, infection, cancer
- Rare genetic conditions such as (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL)) or Fabry disease.

Any recent surgery or invasive procedures? Recent gastrointestinal bleeding, genitourinary bleeding, or other adverse bleeding events? Any known kidney or liver disease or malignancy? Any recent myocardial infarction or recent TIA/strokes? Any prior intracranial hemorrhage? History of seizures? Recent headaches, neck pain, whiplash or trauma? Known allergies to drugs or X-ray contrast dye?

(4) What is their baseline functional status? What is their occupation? What is their cognitive baseline, and what are their goals of care/DNR status?

(5) Were they hyper/hypotensive en route? Does the cardiac rhythm strip show an irregularly irregular rhythm or abnormalities relating to myocardial infarction (ST elevation)? Are they hypoglycemic?
Severe hypoglycemia or hyperglycemia can result in focal neurological signs and altered consciousness that can mimic stroke and blood glucose should always be checked on arrival at ED or obtained from EMS. Look at the rhythm strip from EMS and telemetry monitor in ED as it may identify atrial fibrillation.

(6) Did EMS bypass a hospital en route to your stroke center? This is a practical question as it may be relevant to local hospital repatriation policies at some centers.

The history is extremely important. This cannot be stressed enough. You may not get all of it initially, but try to hit the high-yield questions before you move on to quickly examine the patient.

In summary, the most important questions are:
- Clarify the stroke onset time and/or last seen normal time
- What are the main new deficits
- Baseline functional status
- Is the patient on anticoagulation or have a past medical history of bleeding
- Vital signs and glucose

It is not possible to reliably predict an ischemic from a hemorrhagic stroke type based on history or examination alone, which is why patients are not recommended to take antiplatelets or anticoagulants at onset of symptoms before a CT head is done (approximately 15% of stroke events in North America are hemorrhagic). **Neuroimaging is necessary to differentiate ischemic from hemorrhagic stroke.**

Clinical clues for a hemorrhagic etiology include:
- Patient on anticoagulation
- Head trauma
- Progressive neurological deterioration
Decreased level of alertness
Thunderclap headache
Nausea/vomiting
Brain tumor
Bleeding diathesis
Vascular malformation/aneurysm

Improvement or recovery shortly after the onset of neurological deficit argues against a hemorrhagic etiology. Hemorrhagic TIA mimics exist, but are rare. However, remember that these clinical clues are not specific.

A word about…. Time is brain

As noted earlier, on average, approximately two million neurons are lost per minute in the setting of an acute stroke. This, however, is more variable depending on the patient's physiologic factors (e.g., hemodynamics, collateral blood supply) and can range from 35,000 up to 27 million neurons per minute. All of this means that when a code stroke is activated, a team has to assemble and carry out a series of defined tasks and execute them with precision. A series of interventions have been described in order to facilitate rapid registration, clinical assessment, neuroimaging, and decision-making with regard to acute stroke treatment.

It goes without saying that a cohesive team that is able to function well, communicate effectively, and rapidly assess and transition the patient in the emergency department from triage to the CT scanner is a key ingredient. Having an effective partnership with local EMS providers, and understanding the systems of care and patterns of referral are important. Some interventions that have been described to improve assessment times and door-to-needle
and/or to groin puncture times and acute stroke management include the following:

- Engagement with EMS providers/systems of care
- Stroke center prenotification about the arrival of the patient, ideally with some personal health information
- Splitting up the tasks among the code stroke team members. Tasks to be split include: eliciting the history from EMS and family, examining the patient, looking up previous medical records in the electronic medical records system, checking previous and current blood work, and talking to family members to obtain a more detailed history and contraindications to thrombolytic therapy
- Rapid triaging of the patient with IV insertion, blood work draw, rapid CT order entry, and transfer of the patient directly to the CT scanner as quickly as possible
- Delivery of thrombolytic agent to the CT scanner with the ability to administer on the CT scanner table
- Availability of CT angiogram to assess for proximal occlusion and systems in place to proceed directly to the angiosuite or transfer the patient to an endovascular therapy-capable center
- Rapid neurologic assessment pre- and post-CT
- Rapid imaging protocols, optimized image transition from the scanner to the electronic medical system with appropriate advanced imaging, and rapid radiology interpretation.
- Patient disposition—transfer to appropriate monitored setting

Taken together, such interventions in the setting of teamwork can truly improve the workflow processes required to honor the phrase “time is brain.”
As part of a process of continuous quality improvement, hospital-based stroke teams should rehearse their code stroke protocols, identify and correct local process or system issues that introduce delays to treatment, and monitor their local door-to-needle treatment times and other metrics in order to maximize efficiency. Regular education, case conferences, and feedback to team members about performance and patient outcomes are recommended.

In the context of the current COVID-19 pandemic caused by the SARS-CoV-2 virus, the ability to deliver timely and efficacious care must be balanced with the risk of infectious exposure to the clinical team. Therefore, we proposed modifications to routine hyperacute processes to account for COVID-19. Specific infection prevention and control recommendations were considered by adding clinical screening criteria. In addition, we recommended nuanced considerations for the healthcare team (using appropriate personal protective equipment), thereby modifying the conventional code stroke protocol in order to achieve a “protected” designation.

**Summary**

The history is an important part of the code stroke assessment. Based on the history gathered, you will have a low or high pretest probability for a stroke prior to the CT scan. Remember the six important questions to ask, specifically the time last seen normal, as it starts the clock on potential acute stroke therapy. Sometimes the onset of symptoms is vague, but try your best to clarify it. Vascular risk factors and a history of risk factors such as atrial fibrillation or nonadherence to antithrombotic therapy (ask when the last dose of anticoagulation was taken) are important information to gather.
Further reading

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