# Stroke Essentials for Primary Care

# Current Clinical Practice

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David Alway · John Walden Cole
Editors

# Stroke Essentials for Primary Care

## A Practical Guide

Humana Press

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# Series Editor Introduction

A little inclination sometimes only nudges a physician to learn more when seeing a patient with a particular problem, and the right resource makes that inclination to learn easy to carry out. *Stroke Essentials for Primary Care: A Practical Guide*, by Drs. David Alway and John Cole is an excellent, easy-to-read practical resource for the practicing physician who takes care of patients with stroke. This resource is important because stroke is *the* most common serious acute neurologic condition seen in primary care. Stroke is the third most common cause of death in the United States, and among those who survive many are left with significant disability. Approximately 5.8 million individuals in the United States have a history of stroke, with 8% of persons over 65 years of age reporting a history of a stroke.[1] Talk to any older adult, and they will readily tell you that having a stroke is one of the things they are most afraid of, as it is common enough that most people have a family member or a close friend who has had a stroke and they are afraid of the loss of function, and potential loss of independence that too often occurs after a stroke. Almost half a million patients present each year with transient ischemic attacks, and 25% of those patients go on to have an additional event within the first 90 days after initial presentation.[2] Initial diagnosis and management are essential in achieving optimal outcomes, and risk factor management is essential in decreasing the incidence and recurrence of stroke.

*Stroke Essentials for Primary Care: A Practical Guide* starts with a discussion of differential diagnosis, and then covers each of the common types of stroke in depth. These types of strokes – ischemic, intracerebral hemorrhage, and subarachnoid hemorrhage –make up over 95% of strokes that present to primary care and are discussed in detail. The first chapter on each of these topics discusses the details of initial presentation and management. The next chapter discusses aspects of prevention of initial stroke, reoccurrence of stroke, and long-term management. The last five chapters of the book cover specific topics

[1] Prevalence of Stroke – United States, 2005. MMWR 2007; 56(19):469–474

[2] Solenski NJ. Transient Ischemic Attacks: Part I Diagnosis and Evaluation. Am Fam Physician 2004;69:1665–74

to be aware of in the presentation of stroke. In summary, *Stroke Essentials for Primary Care: A Practical Guide* offers a concise, practical overview of initial diagnosis and management as well as long-term follow-up of patients who present with acute stroke and should be a useful resource for all primary care physicians.

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# Preface

This book focuses the reader on the *essential knowledge* required to evaluate and treat stroke patients. The first chapter assumes an emergency room setting and helps to orient the reader to the distinguishing features of presentation and initial evaluation of stroke types. We then devote two chapters each to the major stroke types (ischemic stroke, intracerebral hemorrhage, and subarachnoid hemorrhage). The first chapter focuses on acute presentation and evaluation (in-hospital evaluation and management) for a particular stroke type. The second such chapter focuses on prevention and long-term complications (out-patient/follow-up issues). The five remaining chapters review special topics that may apply to specific populations: stroke in the young adult, headaches as they relate to stroke, hypercoagulable states, carotid artery disease, and cerebral venous thrombosis. Where appropriate, most chapters include a quick summary of their content and conclusions, allowing for rapid review when necessary.

We hope you find this text useful for rapid access to essential stroke information.

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# Chapter 1
# Identifying Stroke and Stroke Type

**David Alway**

This chapter is a quick review of the typical presenting symptoms, signs, and CT imaging characteristics of the major stroke types (ischemic stroke, transient ischemic attack (TIA), intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), and cerebral venous thrombosis). It should be used to familiarize yourself with the common emergency room presentations of the major stroke types. Detailed discussions of these stroke types, and their management, will be presented in later chapters (2–7 & 12).

## Identifying Stroke

The hallmark of all stroke types is a relatively sudden onset of neurological dysfunction which may involve any or all of the following: weakness, numbness, vision loss, diplopia, dysarthria, gait disorder, aphasia, lightheadedness, vertigo, or disturbed level of consciousness. Knowing a stroke is a stroke is difficult in perhaps 5–10% of cases, and no one, based on history and physical examination alone, is able to identify stroke and stroke type at all times. The many mimics of stroke to keep in mind include partial complex seizures, hypotensive episodes, the re-experience of old stroke symptoms in the setting of infection or metabolic derangement, multiple sclerosis, isolated cranial nerve dysfunction, nerve root disease, migraine with aura, CNS infections, etc. Assessment is especially difficult in cases where symptoms were transient and the patient now has a normal neurological exam. Here the timing of symptoms, what the patient and witnesses report, and the past medical history of the patient can help distinguish a TIA, for example, from alternatives. We must also consider the possibility of dual diagnoses. A patient who has experienced a cortical ICH may well present with signs and symptoms of both hemorrhage and, because of blood-induced cortical irritation, seizures.

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## Typical Symptoms/Signs of Ischemic Stroke and Transient Ischemic Attack

Ischemic stroke is the most common stroke type, representing about 85% of all strokes. Ischemic stroke patients will typically present with the sudden onset of weakness, numbness, vision loss, diplopia, dysarthria, gait disorder, vertigo, aphasia, or disturbed level of consciousness. The location of the stroke will determine which particular pattern of symptoms occurs and will be covered in chapter 2. Ischemic stroke typically involves an absence of function. For example, an ischemic stroke patient will often report loss of vision in a single eye or in an entire hemifield. Rarely would an ischemic stroke patient experience positive visual phenomena, such as bright lines or objects in vision, or scintillations. These are the hallmarks of alternative diagnoses such as the visual aura of migraine, occipital lobe seizures, or retinal detachment. Ischemic stroke patients may experience numbness in part of the body. Rarely will they acutely experience extra sensations (paresthesias or pain) such as may occur with nerve root disease. Ischemic stroke patients may experience weakness or paralysis on one side of the body. Rarely will they experience extra, involuntary movements. All these examples of 'extra' sensations or movements are referred to as 'positive' phenomena, and they suggest a diagnosis other than ischemic stroke.

So long as the examination is performed within hours of symptom onset, and assuming the brainstem has not been markedly damaged, patients with ischemic stroke are more likely to have a preserved level of consciousness (compared with other stroke types) – meaning they will likely appear awake and be able to cooperate, to some degree, with a neurological exam. From the practical standpoint, if a patient has experienced the sudden onset of focal neurological dysfunction, without positive phenomena, has a head CT that is negative for evidence of blood, and alternative explanations for symptoms, based on the history, are not forthcoming, the patient is assumed to be suffering from an ischemic stroke until proven otherwise. In the event that the patient's symptoms resolve quickly within an hour and the head CT is negative, this may represent a TIA. Symptoms which last longer than an hour, even if they appear to resolve completely, may be due to small ischemic strokes, perhaps only evident on MR imaging.

Head CT results: The early head CT (within 1 h) in ischemic stroke is often normal, but is very useful to exclude the presence of blood or other intracranial lesions. Within 1–3 h there can be regions of discernible hypodensity or loss of gray–white matter differentiation, which may be the earliest imaging evidence of ischemia (see Figs. 1.1 and 1.2). Other findings may be a very bright artery in a non-contrast head CT (typically the middle cerebral artery) relative to another portion of the same artery or the opposite hemisphere artery (see Fig. 1.3) or regions of sulcal effacement or ventricular compression due to swelling. In the case of a TIA, the head CT is expected to be normal.

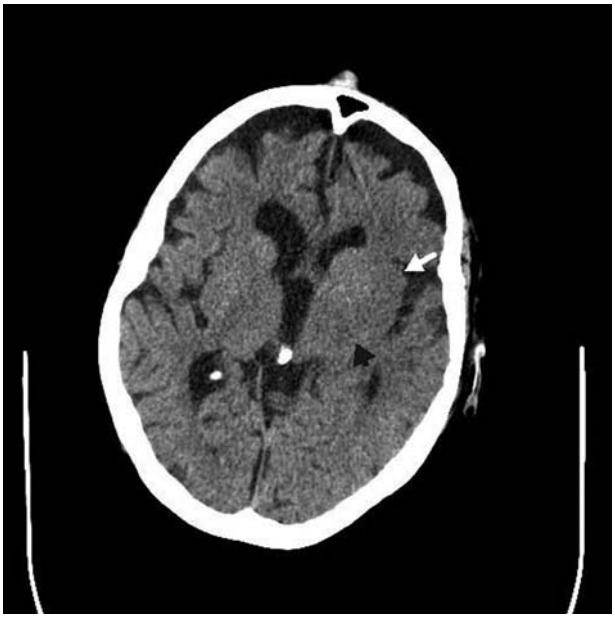

**Fig. 1.1** Early head CT (within a few hours) demonstrating loss of gray–white differentiation in the insula

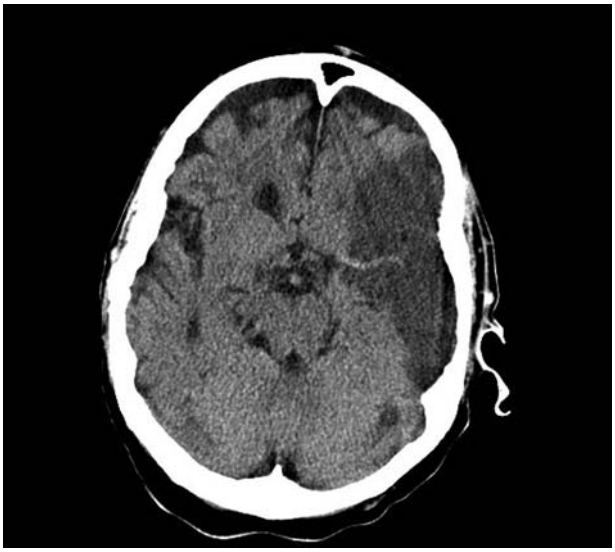

**Fig. 1.2** Later head CT (day 2) revealing hypodensity within the left MCA distribution

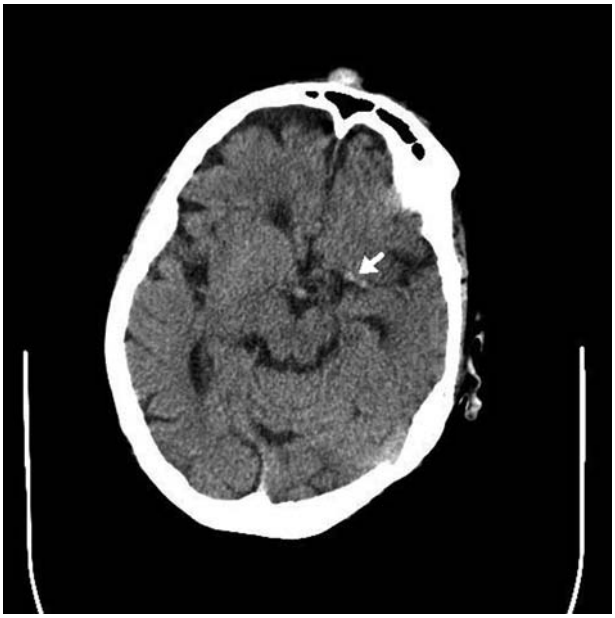

**Fig. 1.3** Hyperdense left MCA in an acute ischemic stroke case

## Typical Signs/Symptoms of Intracerebral Hemorrhage

Intracerebral hemorrhages (ICH) represent about 10% of all strokes, and in this text, the term refers to bleeding within the substance of the brain, including bleeding into the ventricles. (We are excluding, in this definition, bleeding over the surface of the brain [epidural, subdural] or into the subarachnoid space [SAH]). A patient suffering from an ICH may present with the sudden onset of neurological dysfunction, just as with other stroke types, including weakness, numbness, vision loss, diplopia, dysarthria, gait disorder, vertigo, aphasia, or disturbed level of consciousness. In addition, headache is more common with ICH, occurring in 40% of patient presentations. Nausea and vomiting due to increased intracranial pressure may also occur. The rapidity of neurological worsening is typically more marked than with ischemic strokes. For example, if the hemorrhage is large enough, pressure effects on the whole brain and brainstem may lead to a markedly diminished level of consciousness. In addition, a bleed that begins small (2 cm in diameter) may enlarge over the first few hours to become massive. This will lead to a rapid worsening of symptoms, including a rapid reduction in the patient's level of consciousness.

Head CT results: Head CT will reveal blood in the parenchyma (bright signal, see Fig. 1.4), possibly extending into the ventricular system as well.

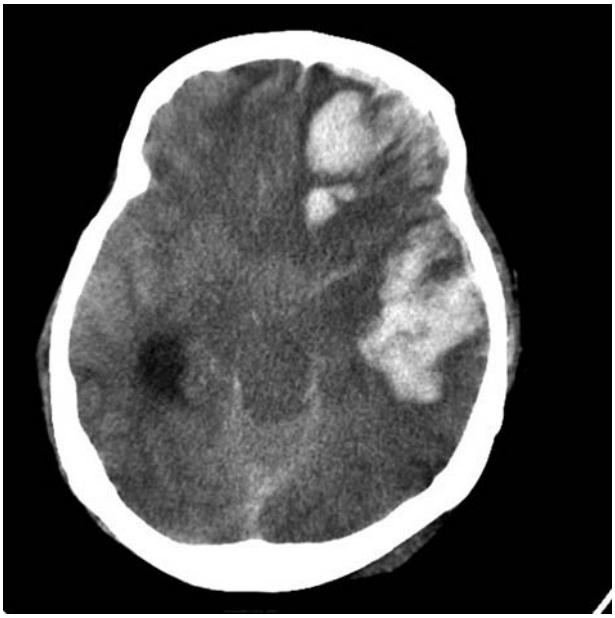

**Fig. 1.4** Large intracerebral hemorrhage

## Typical Signs/Symptoms of Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) represents about 5% of all strokes. SAH typically presents with a sudden severe headache which may be followed by a diminished level of consciousness or complete unconsciousness. The headache is often the most painful the patient has ever experienced, but lesser headaches may still occur with SAH. Due to blood irritation of meninges, patients may also experience neck stiffness, back pain, and photophobia. At times the blood of the SAH has an effect (mass or otherwise) on part of the brain to produce focal neurological symptoms or seizures. If the SAH is massive, the presentation may simply be the sudden loss of consciousness and collapse of the patient. In many cases, a detailed review of the patients' history will reveal a lesser headache or other focal neurological symptom in the days or weeks prior to the presenting event. This is felt to be due to a smaller bleed (sentinel bleed), most typically from an intracranial aneurysm.

Head CT findings: Blood in the subarachnoid space. Figure 1.5 shows a massive SAH, but imaging can be much more subtle. The head CT is 95–99% sensitive for subarachnoid blood, if performed within 12 h of the event. Sensitivity declines with each passing day after the event. Lumbar puncture may be needed if head CT is normal but suspicion remains high for SAH. (Testing of CSF fluid should include cell count in the first and

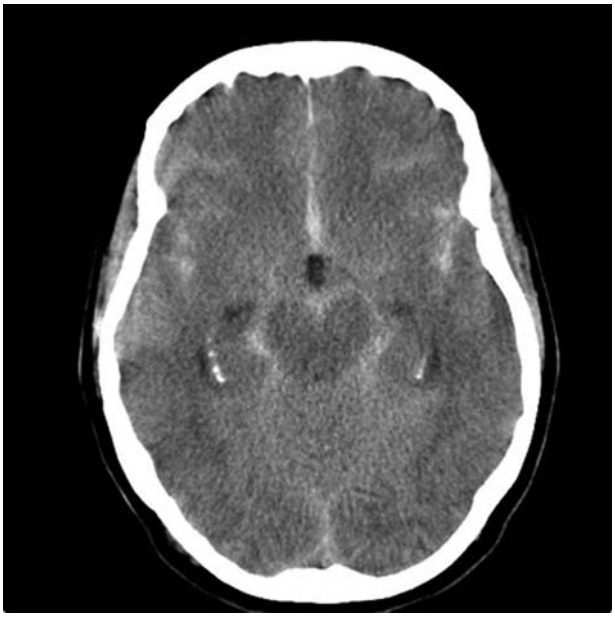

**Fig. 1.5** A massive subarachnoid hemorrhage

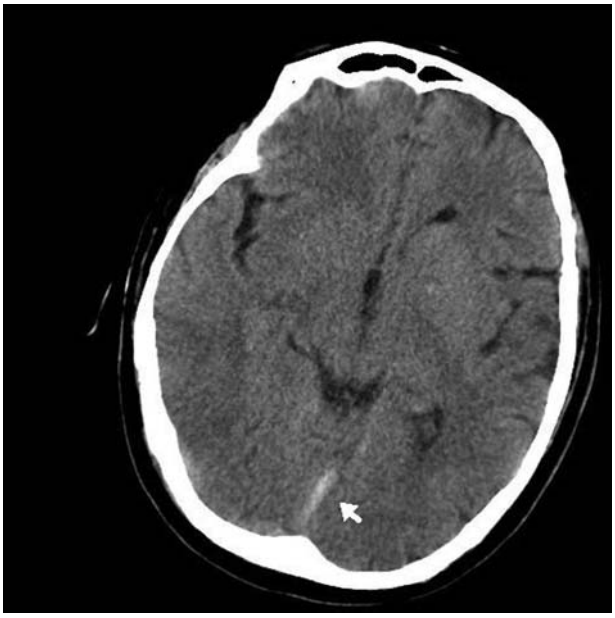

**Fig. 1.6** Increased signal within the straight sinus due to a thrombus

**Table 1.1** Chart comparing features of transient ischemic attack (TIA), ischemic stroke, intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), and cerebral venous thrombosis. LOC = level of consciousness

| | TIA | Ischemic stroke | ICH | SAH | Cerebral venous thrombosis |
|---|---|---|---|---|---|
| Decreased LOC | Uncommon in history, absent after minutes | Possible, but uncommon | Common (50%) | Common if large | Variable, more likely if large vessel with increased ICP |
| Headache | Usually absent | 10%, especially with arterial dissection | Common (40%) | Universal, unless patient unconscious | Variable, common if large vessel with increased ICP |
| Focal symptoms and signs | Absent after minutes | Almost always | Very common | Common if large | Variable |
| Seizures | Absent | Uncommon | 6–7% | 10–25% | Common |
| Nausea/vomiting | Absent | Uncommon | 40–50% | Common | Common, if large vein involved |
| Head CT | Normal | Normal in first few hours, then hypodense regions | Blood in parenchyma | Blood in subarachnoid space | Normal or hypodense, but common to have hemorrhagic infraction. May see thrombus in cerebral vein |

last collected tubes and testing for xanthochromia. Xanthochromia testing, performed on a centrifuged sample, may be positive if the lumbar puncture was performed 6 h or later after the SAH.)

## Typical Signs/Symptoms of Cerebral Venous Sinus Thrombosis

Cerebral venous thrombosis is a much rarer diagnosis, and its frequency has been difficult to estimate. It may present with focal neurological symptoms if a small cortical vein is occluded. This can lead to both ischemia (though not in an arterial distribution) and hemorrhage into the region of ischemia. This may also result in seizures. If larger cortical veins or sinuses are obstructed, there may or may not be focal neurological symptoms. In these cases, patients may present with global symptoms such as headache, nausea, and vomiting. In addition, symptoms may be of longer duration and do not necessarily have to occur suddenly. Clinical suspicion must be high (known hypercoagulable state, history of prior venous thromboses, SLE or other connective tissue disease known or suspected) to keep this rarer stroke type in mind.

Head CT results: Head CT may be normal. Often, though, especially if the radiologist is asked specifically to look for such, there will be evidence of thrombosis within one of the cerebral veins (see Fig. 1.6). The larger the vein involved (such as the saggital sinus) the more likely it is to be seen on imaging (Table 1.1).

# Chapter 2
# Ischemic Stroke and Transient Ischemic Attack – Acute Evaluation and Management

**W. Alvin McElveen and David Alway**

Stroke is the third leading cause of death in the United States behind heart disease and all forms of cancer. Each year 750,000 Americans will have a new or recurrent stroke. Stroke is also the most common medical cause of disability. It is the most highly incident and prevalent neurological condition managed in the hospital setting.

## Pathophysiology of Ischemic Stroke

Ischemic stroke is most often due to a lack of blood flow to all or part of the brain, resulting in the deprivation of neurons of vital glucose and oxygen. This deprivation, if severe and prolonged, results in the interruption of normal cellular processes and eventual cell death with breakdown of the neuronal cell membrane. Ischemia can also result from oxygen deprivation alone (hypoxic–ischemic damage, as may occur in patients who experience a cardiac arrest, respiratory collapse, or both) or glucose deprivation alone (as may occur with insulin overdoses in diabetic patients). A very low (or no) blood pressure can produce a distinct pattern of 'watershed' infarcts, which are typically regions of infarcted tissue between the major cerebral arterial territories. More commonly, ischemic stroke involves only a portion of the brain due to an occlusion of a large or small artery. It also may develop rapidly in multiple arterial territories in the event of multiple emboli or a single embolus which breaks up as it travels.

When an artery is occluded and the brain is deprived of blood flow, there is an almost immediate inhibition of the natural function of the neurons feed by that artery. The neurons cease to perform their normal function, and patients will experience symptoms relevant to the area of the brain involved (weakness, numbness, vision loss, etc.). There is a gradient of blood flow around the location of a large arterial occlusion. So, for example, at the center of the region of ischemia, blood flow may be less than 10 mL/100 g/min. This represents the

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ischemic 'core' of the infarcted region – and these neurons may undergo irreversible cell death in as little as 2 h, if blood flow is not reinstated [1]. As one moves away from this ischemic core, blood flow tends to improve, but is still not considered adequate to maintain survival. This region represents the ischemic penumbra. It is a region surrounding the ischemic core and is considered 'at risk' brain territory. While the existence of an ischemic penumbra in every stroke patient may be in debate, the concept holds that very early intervention (recanalization of the relevant artery within 1 h) is likely to result in no stroke at all while later recanalization (after 2 h) may result in a smaller infarct than otherwise would have occurred.

Transient ischemic attacks (TIAs) involve the same pathophysiology as ischemic strokes, but with an early (usually within 10 min) restoration of blood flow to the brain, and thus, no actual infarction. TIAs were previously defined as stroke symptoms that subsided within 24 h, however, MRI studies with diffusion-weighted imaging revealed that over half of patients whose symptoms lasted greater than 60 min actually had areas of infarction despite resolution of symptoms.

Even if the amount of infarcted tissue does not increase over time, infarcted tissue changes during the coarse of hospitalization, leading to edema or possibly to hemorrhage. Edema of the region of infarction can peak as early as 24 h or as late as 4 days after ischemic onset [2]. Over time, an initially large area of infarction can increase in size leading to increased intracranial pressure, or local pressure effects that can cause obstructive hydrocephalus, further infarction due to pressure on adjacent arteries, or herniation of brain into other compartments. Young patients with large infarctions are the most likely to develop problems related to edema formation.

Areas of infarction may also undergo hemorrhagic transformation, meaning that hemorrhage occurs in the infarcted region. This typically is less problematic than edema formation and may simply be 'petechial' hemorrhages, which are of no clinical significance. Frank hemorrhage with associated clinical deterioration and mass effect occurs in as many as 10% of ischemic stroke patients – typically within the first 2 weeks of the ischemic event. Bleeding disorders (including anticoagulation use), poor blood pressure control, and large infarctions are more prone to such hemorrhages.

## Early Stroke Recognition and Identification of Stroke Type

For many years the management of ischemic stroke largely involved supportive care and physical therapy. Management of risk factors for prevention of secondary stroke was and remains an important aspect of stroke management. With the advent of tissue plasminogen activator (rt-PA) for the acute treatment of ischemic stroke in 1996, the management of stroke changed dramatically. The move toward treatment algorithms founded on evidence-based medicine has also altered the care of the stroke patient. Several states have adopted