An Update on Transient Ischemic Attacks

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Abstract: *Each year in the United States 200,000–500,000 people have a transient ischemic attack (TIA). These episodes of brief neurologic deficits were thought to be fairly benign, but this view is changing. In 2002, a new definition for TIA was proposed, and a more intensive diagnostic workup recommended to look for a probable cause of the transient neurologic deficit. Implementation of prevention strategies is essential because the risk of a stroke following a TIA is approximately 30% within 5 years. These changes have important implications for nursing care and patient advocacy. In addition, patient and family education must be addressed by the entire healthcare team.*

Current estimates are that between 200,000 and 500,000 people have transient ischemic attacks (TIA) each year in the United States (Johnston, 2002). Approximately 5 million adults in the United States have had a transient ischemic attack (TIA), and many are undiagnosed (Johnston et al., 2003). These episodes of a sudden, brief neurologic deficit used to be thought of as fairly benign events. In 2002, a new pathophysiologically based definition was proposed for TIAs. The current evaluation, including laboratory and diagnostic tests, is aggressive and intensive. These changes have important implications for nursing care, patient advocacy, and pertinent patient and family teaching.

Many changes have occurred in the last two decades regarding TIAs. This article provides a historical perspective on the evolution of the definition of TIA and reviews the associated pathophysiology and symptoms. The laboratory and diagnostic tests a neuroscience nurse can expect in the evaluation of a patient following a TIA are identified. Nursing interventions a neuroscience nurse needs to use in the emergency department (ED) and during inpatient stay for TIA also are addressed. Patient and family teaching regarding modifiable risk factors associated with a TIA are an important component of care.

Historical Perspective

The definition of TIA as a time-based event was discussed in the 1950s and 1960s when the proposed temporal criteria varied widely (Albers et al., 2002). A National Institutes of Health committee on the classification of cerebrovascular disease suggested in 1958 that a TIA could last several hours, but typically ranged from a few seconds to 10 minutes; the upper duration was 1 hour (Winn, 2004).

In 1964, Acheson and Hutchinson reported their observations of 82 patients with what was then referred to as episodes of transient cerebral ischemia. In this study, which took place before computed tomography (CT), there was a predominance of males over females and a mean age of 56 years (Acheson & Hutchinson, 1964). Forty of the episodes were classified as TIA, using the time frame of less than 1 hour (Acheson & Hutchinson). The authors noted that little was known about the natural history of TIA at that time.

The 1975 classification of cerebrovascular diseases defined TIA as lasting up to 24 hours (National Institute of Neurologic Diseases and Stroke ad hoc Committee on Cerebrovascular Diseases, 1975). Dyken and colleagues (1977) reported on a large cooperative study of hospital frequency and characteristics of TIAs. TIAs were then acknowledged as a warning sign of an impending stroke. This study, funded by a National Institute of Neurological Diseases and Stroke contract in 1972, gathered data on 1,323 patients with TIA-like symptoms at six major medical institutions (Dyken et al., 1977). Sixty-six percent of patients who had carotid artery symptoms and 63% of those who had vertebral artery symptoms were male; the median age was 63 years and 7% were African American (Dyken et al.). The median duration of the TIA was 14 minutes for those with carotid artery symptoms and 8 minutes for those who had vertebral artery symptoms. The majority of TIAs (90%) in the study were reported to have cleared within 10 minutes.

Levy (1988) reported on 1,343 hospitalized patients included in a database of patients with TIA (defined as acute neurologic changes resolving within 24 hours of onset), reversible ischemic neurological deficit (defined as resolving between 24 hours and 4 weeks of onset), and ischemic stroke. Fifty six percent of the patients were male with a mean age of 66 years (Levy, 1988). In 382 patients, TIA was diagnosed and of these 191 (50%) had episodes that lasted less than 30 minutes, and 9% had symptoms that lasted 30–60 minutes. These authors suggested less than 24 hours as the longest permissible duration for TIA. Understanding the natural history of TIAs was becoming more important as the pilot studies using intravenous (IV) tissue plasminogen activators

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(t-PA), such as Activase, for ischemic stroke were taking place. Differentiating between patients for whom early treatment would bring favorable clinical outcomes or those for whom it could cause harm became more important with a potential therapy on the horizon.

Werdelin and Juhler (1988) reported on 78 patients admitted to hospital with their first episode of presumed ischemic origin to decide whether the differential diagnosis of stroke versus TIA could be made earlier than 24 hours. Within the first hour, 50% of TIA patients had recovered; 90% recovered within 4 hours. CT scans obtained on half of the patients with TIA showed infarction in two, multiple infarctions in one, and no infarction in the remainder of the cases (Werdelin & Juhler).

TIA is still defined as a "neurological deficit lasting less than 24 hours that is attributed to focal cerebral or retinal ischemia."

The 1990s brought a flurry of algorithms and clinical guidelines to assist healthcare practitioners in managing TIAs. Brown and colleagues (1994) published a cost-effective, scientifically based algorithm for the evaluation and treatment of TIA and minor ischemic stroke. That same year the stroke council of the American Heart Association (AHA) published guidelines for the management of TIAs (Feinberg et al., 1994). In 1999 these guidelines were updated to include prevention strategies (Wolf et al., 1999). That same year a supplement to the AHA guidelines, which addressed risk-factor modification and provided an update of TIA medical and surgical management, was published (Albers, Hart, Lutsep, Newell, & Sacco, 1999).

TIA is still defined as a "neurological deficit lasting less than 24 hours that is attributed to focal cerebral or retinal ischemia" (Johnston, 2002, p. 1687). The 24-hour time limit is arbitrary, was adopted before the widespread availability of neurodiagnostic studies, and remains debatable (Albers et al., 2002; Dyken et al., 1977; Fisher, 2002; Levy, 1988). Disadvantages of time-based TIA definitions include the following:

- the suggestion that TIA symptoms are benign
- the promotion of diagnosis based on a temporal course rather than pathophysiology
- delays in interventions for acute cerebral ischemia because the definition inaccurately predicts the presence or absence of ischemic brain injury
- overlooking the distinction between angina and myocardial infarction (MI); angina is a symptom of MI (Albers et al., 2002).

Because of these limitations a new diagnostic description was proposed: A TIA is a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of acute infarctions (Albers et al., 2002). The advantages of this new definition are that it is based on the presence or absence of a biologic end point, indicates that transient ischemic symptoms can cause permanent brain injury, encourages use of neurodiagnostic tests to identify brain injury and its cause, more accurately reflects the presence or absence of ischemic brain injury, and is more consistent with the distinction of angina as a symptom of MI (Albers et al.). The current consensus is that TIA is not a benign event and should not be ignored (Daffertshofer, Mielke, Pullwitt, Felsenstein, & Hennerici, 2004).

Pathophysiology

The pathophysiology of TIA is transient ischemia to the brain, which can have various causes. The etiology of TIA is thought to be either from atrial fibrillation, carotid artery disease, large artery disease, small artery disease (Johnston, 2002), or other causes. Some less common causes include hypercoagulable states, illicit drug use (Feinberg et al., 1994), and fibromuscular dysplasia (Slovut & Olin, 2004).

The various symptoms produced by TIAs are classified according to the anterior or posterior blood circulation to the brain (Bader & Littlejohns, 2004; Hickey, 2003). Anterior circulation symptoms include those associated with either the carotid, anterior cerebral, or middle cerebral artery. Posterior circulation includes the posterior cerebral artery and vertebrobasilar system. Posterior circulation supplies glucose and oxygen to the brainstem and cerebellum. An overview of the symptoms corresponding with each arterial distribution can be found in Table 1.

Assessment

A neurological assessment is most commonly based on both subjective and objective data. Often there are no objective findings by the time the patient with TIA is seen by a healthcare professional (Johnston, 2002). Therefore, a careful medical history is crucial, as the diagnosis is often based on clinical history alone (Bader & Littlejohns, 2004; Johnston, 2002). The patient or an accompanying family member may report any of the symptoms outlined in Table 1. Careful questioning may be needed to elicit recall of the timing and exact nature of the event.

Nonischemic causes of the attack must be ruled out during the initial evaluation. Many types of seizures can mimic TIA (Schulz & Rothwell, 2002). Medication reported to be associated with TIA include sildenafil (Viagra) (Morgan, Alhatou, Oberlies, & Johnston, 2001) and risperidone (Risperdal; U.S. Department of Health and Human Services [DHHS], 2003). There are many other possible causes that should be considered; Table 2 contains a list compiled from existing literature.

The recommended initial diagnostic evaluation for TIA patients more than 50 years old includes both laboratory and

neurodiagnostic studies (Bader & Littlejohns, 2004). Any diagnostic evaluation, regardless of patient age, must be individualized. Recommended laboratory studies include

- complete blood count with platelet count
- chemistry profile (with fasting cholesterol level and glucose tolerance)
- prothrombin time and activated partial thromboplastin time
- erythrocyte sedimentation rate (ESR) with syphilis serology
- lipid profile.

Diagnostic studies include an electrocardiogram (ECG; Albers & Easton, 2001; Feinberg et al., 1994); noncontrast cranial CT, particularly in hemispheric TIAs (Albers & Easton; Feinberg, et al.); and noninvasive arterial imaging (e.g., ultrasound, magnetic resonance angiography; Albers & Easton; Bader & Littlejohns; Feinberg et al.). Patients also need to be evaluated for asymptomatic coronary artery disease (Adams et al., 2003).

Although there are recommendations for the diagnostic evaluation of patients following TIA, there is no current directive as to whether the evaluation needs to be done on an inpatient or outpatient basis (Brown et al., 1994; Feinberg et al., 1994; Moore, 2001). There is agreement that the workup needs to be completed within 24 hours; therefore patients will need to be hospitalized if it is not possible to complete the diagnostic studies within that time. For example, the wait for outpatient CT may be several days in some settings.

Table 1. TIA Symptoms According to Cerebral Circulation

Type of Circulation	Symptoms
Anterior	
Carotid artery	Contralateral motor and sensory loss
	Amaurosis fugax (transient blindness) or transmonocular blindness caused by emboli to retinal artery
Anterior cerebral artery (ACA)	Confusion
	Personality change
	Incontinence
	Contralateral motor or sensory loss leg greater than arm
Middle cerebral artery (MCA)	Contralateral motor or sensory loss (arm greater than leg)
	Contralateral motor loss in lower face
	Contralateral visual field loss
	Language loss (dominant hemisphere)
	Spatial-perceptual loss (nondominant hemisphere)
Posterior	
Posterior cerebral artery (PCA)	Contralateral motor or sensory loss
	Ipsilateral visual field loss
	Cortical or bilateral blindness
	Dysarthria
	Dysphagia
	Diplopia
	Quadriparesis
Vertebrobasilar	Altered brainstem and cerebellar functions
	Cranial nerve deficits for cranial nerves III–XII
	Ataxia
	Bilateral blindness or hemianopia
	Confusion
	Diplopia
	Bilateral limb weakness
	Bilateral paresthesias
	Slurred speech
	Vertigo

Secondary Prevention

By definition, a patient who has experienced TIA has no residual neurological deficit, however, approximately 30% of patients who have had a TIA go on to have a stroke within 5 years (Albers et al., 1999). The goal of treatment is to prevent another TIA or stroke by identifying and treating the probable cause. Medical therapy for preventing a recurrence of TIA or stroke may include antiplatelet agents or anticoagulation therapy. Patients with a critical percentage of carotid stenosis may benefit from carotid endarterectomy or stenting. Those with a patent foramen ovale (PFO) will need to have the foramen closed.

Anticoagulants

The treatment of choice for patients with atrial fibrillation (AF) and a recent TIA is anticoagulation. Microscopic emboli are thought to form as the blood pools in the heart when a patient experiences AF. The drug of choice is warfarin sodium (Coumadin) with an international normalized ratio goal of 2.5 (Albers et al., 1999; Wolf et al., 1999). In patients without AF, antiplatelet therapy is recommended.

Antiplatelet Therapy

There is much ongoing debate in the medical literature about antiplatelet agents following TIA. The most commonly recommended drug regimens include acetylsalicylic acid (aspirin), 50–325 mg daily (Albers et al., 1999; Wolf et al., 1999); ticlopidine (Ticlid), 250 mg twice daily (Albers & Easton, 2001; Albers et al., 1999); clopidogrel bisulfate

(Plavix), 75 mg daily (Albers et al.); or extended-release dipryridamole 400 mg with Aspirin 50 mg (Aggrenox), daily (Albers & Easton; Albers et al; Sarasin, Gaspoz, & Bounameaux, 2000). Various combinations of these regimens have been tried and the best combinations among types of cerebrovascular events and their subgroups is a source of debate and the subject of several ongoing clinical trails (Tran & Anand, 2004).

Research by Sarasin and colleagues (2000) analyzed the cost effectiveness of antiplatelet regimens in secondary prevention of TIA. Among patients who had experienced TIA but were not candidates for carotid surgery, Aggrenox was the most cost effective. The authors suggest that the incremental costs associated with this regimen were offset by the savings afforded through the avoidance of additional stroke-related costs (Sarasin et al., 2000).

All antiplatelet aggregates are available in oral formulations. Recently the FDA issued a warning about patients who experience choking, gagging, tablets stuck in their throats, and dysphagia while taking certain oral medications (DHHS, 2004). Nurses need to be hypervigilant when administering oral medications to patients who have experienced a TIA that may have included some of these same symptoms. The FDA recommends that patients take all pills with a full glass of water.

Carotid Endarterectomy (CE)

The most common surgical therapy following TIA is CE for eligible candidates. The benefit of carotid endarterectomy for symptomatic patients with severe (70%–99%) carotid stenosis has been known since the early 1990s (North American Symptomatic Carotid Endarterectomy Trial [NASCET], 1991). Patients with asymptomatic carotid artery stenosis of greater than 60% also benefit from CE (Asymptomatic Carotid Atherosclerosis Study, 1995).

More recently the Carotid Endarterectomy Trialists Collaboration pooled and analyzed data from 5,893 patients with 33,000 years of follow-up. Results of the trial suggest that patients who undergo CE for carotid stenosis for more than 70% of the vessel diameter reduce their stroke risk from 26% to 9% (Rothwell, Eliasziw, Gutnikov, Warlow, & Barnett, 2004). In patients with 50%–70% stenosis, the surgery reduces the risk of stroke over 5 years from 22% to 16% (Barnett et al., 1998; Rothwell et al.). The greatest benefit of the surgery is seen in males with recent stroke or TIA symptoms, patients 75 years or older, and in patients with hemispheric rather than ocular symptoms (Rothwell et al.; Winn, 2004). Endarterectomy is most beneficial when performed within the first 2 weeks after a cerebrovascular event (Rothwell et al.; Winn).

Patients with severe carotid artery stenosis who are at high risk due to comorbidities may benefit from angioplasty and carotid stenting. Protective carotid artery stenting with the use of an emboli-protection device may be indicated for certain patients (Yadav et al., 2004).

Table 2. Nonischemic Causes of TIA Symptoms

Anticardiolipin-antibody syndrome
Anxiety
Arrhythmia
Arterial dissection
Arterial venous malformations
Carpal tunnel syndrome
Cerebral venous thrombosis
Cervical disk disease
Compressive neuropathy
Conversion disorder
Giant aneurysm
Hyperviscosity
Hypoglycemia
Inner-ear dizziness, vertigo, or Meniere's disease
Medications
sildenafil (Viagra)
risperidone (Risperdal)
Migraine
Neoplasms
Neuropathy
Ocular disorders
Peripheral vascular disease
Polycythemia
Seizures
Akinetic seizure
Parietal-lobe epilepsy
Focal motor seizures
Severe postural hypotension
Subacute bacterial endocarditis
Subdural hematoma
Temporal arteritis
Thrombocythemia
Transient global amnesia
Vasovagal syncope

Patent Foramen Ovale

PFO may cause TIA by permitting emboli to form, escape from the heart, and travel to the brain. Closure can be accomplished using open heart surgery or using a transcatheter closure (Alameddine & Block, 2004; Sommer & Levchuk, 2004)

Nursing Interventions

The patient who has experienced TIA is at risk for altered cerebral tissue perfusion. Important nursing interventions include performing baseline and subsequent serial assessments of neurological status for any further signs and symptoms of altered cerebral tissue perfusion that may indicate a recurrence of TIA or a developing stroke. The nurse must also educate the patient and family about the laboratory and diagnostic tests being performed during the initial evaluation (Hickey, 2003; Hinkle, 1997).

Most patients with TIA and their families also have a knowledge deficit related to risk factors for TIA and stroke. A nationwide survey of 11,400 adults reported that 8.2% of those surveyed were able to identify the

definition of TIA and only 8.6% were able to recognize at least one common symptom (Johnston et al., 2003). In another study of 215 hospitalized women, all respondents named stress as the number one risk factor for stroke (Kattapong et al., 1998).

Patient and family education should proceed according to a baseline assessment of knowledge deficit related to risk factors and secondary prevention. The degree of readiness to change must also be assessed (Miller & Spilker, 2003). Assessment needs to be directed first toward the recognition of nonmodifiable risk factors including advancing age, male gender, Hispanic or African American race, and heredity (Albers & Easton, 2001; Albers et al., 1999). Individuals in these groups need to be more vigilant about controlling modifiable risk factors.

Recommendations for prevention of modifiable TIA risks include

- Hypertension should be treated aggressively to maintain systolic blood pressure (BP) below 140 mm Hg and diastolic BP below 90 mm Hg (Albers et al., 1999; Wolf et al., 1999).
- Diabetes mellitus, if present, must be controlled (Albers et al., 1999; Wolf et al., 1999).
- Cigarette smoking must be eliminated (Feinberg et al., 1994; Wolf et al.).
- Coronary artery disease, cardiac arrhythmias, congestive heart failure, and valvular heart disease should be treated (Feinberg et al., 1994).
- Excessive alcohol use or any illicit drug use should be eliminated (Wolf et al., 1999).
- Use of oral contraceptive should be discontinued, or at minimum a low-estrogen version should be used (Feinberg et al., 1994).
- Cholesterol levels need to be monitored and hyperlipidemia should be treated for reduction of coronary artery disease (Wolf et al., 1999).
- Physical inactivity must be corrected and the benefits of an exercise program as well as a healthy diet leading to weight loss in overweight individuals should be explained (Albers et al., 1999; Wolf et al., 1999).
- Hormone replacement therapy in postmenopausal women is not recommended for prevention of stroke (Brass, 2004).

All educational interventions need to be appropriate for individual educational levels and learning styles (Hickey, 2003; Moore, 2001). Educational materials in a variety of media are available to meet the needs of patients and families. Printed materials about specific medications are available from most hospital pharmacies, many in both English and Spanish. Videotapes or DVDs on medications and procedures are also available for visual learners. Patients who are computer savvy may prefer to read information that is available online from organizations such as the American Stroke Association (ASA) and National Stroke Association.

Table 3. Sources of TIA Clinical Trial Information

Stroke Trial Directory, Washington University in St. Louis, School of Medicine, www.strokecenter.org/trials/
A list of ongoing and concluded stroke trial information organized alphabetically by study name. Trials are listed as ongoing until results are presented. Links to publications are provided in the reference list for each therapeutic agent.

The Center Watch, www.centerwatch.com
Clinical trial listing service designed for both patients and researchers that offers a large collection of resources on clinical trials. Patient resources include background information on clinical research, research headlines, and an index of government-funded clinical research studies being conducted by NIH. An e-mail notification service is available that alerts subscribers to new postings in up to eight therapeutic areas.

American Stroke Association, a division of the American Heart Association
www.americanheart.org/presenter.jhtml?identifier=1165
Contains a Stroke Trials Directory with descriptions of completed and ongoing stroke therapeutic trials.

PubMed, www.ncbi.nlm.nih.gov/PubMed/
The Medline service of the National Library of Medicine provides citations to completed studies and trials.

Clinical Research Studies at the U.S. National Institutes of Health, clinical studies.info.nih.gov/
Includes some of the research studies at the Clinical Center of the NIH in Bethesda, MD.

Each patient and family should receive printed materials outlining the warning signs of stroke. These are available from the ASA in both English and Spanish (ASA, 2000). Printed patient teaching materials regarding stroke risks, modifiable risk factors, and carotid endarterectomy are also available through the ASA's Get with the Guidelines Program (www.americanheart.org).

In their capacity as healthcare professionals, advocates, educators, and role models, neuroscience nurses can best educate and advocate for patients and families by staying informed about current research findings related to TIAs. Table 3 provides sources of information about ongoing clinical trials that can assist in answering questions about options, especially prevention, for patients who have had TIA.

Summary

Healthcare professionals want the best outcomes for people who have had TIA. Achieving better outcomes includes being knowledgeable about the proposed definition of TIA and the aggressive work-up recommended. Patient and family members need to be knowledgeable about anticoagulant and antiplatelet agents, surgical procedures, and risks so that all can work together to prevent a recurring TIA and possible future stroke.

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