

# Vertigo, Vertebrobasilar Disease, and Posterior Circulation Ischemic Stroke

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## KEYWORDS

• Vertigo • Vertebrobasilar disease • Posterior circulation ischemic stroke • Dizziness

## KEY POINTS

- Patients with dizziness frequently present to the emergency department. The cause of the complaint ranges from the benign to the life threatening.
- Ischemia of structures supplied by the posterior circulation may present in a more subtle fashion than those affecting the anterior circulation.
- Posterior circulation ischemia rarely presents as a single symptom.
- The classic presentation of those with posterior circulation ischemia has been described as crossed findings with cranial nerve findings on the side of the lesion and long tract (eg, motor or sensory) findings on the contralateral side.
- Data regarding the cause and treatment of posterior circulation stroke has lagged behind that of anterior circulation stroke.

An estimated 7.5 million patients with dizziness are seen each year in ambulatory care settings.<sup>1</sup> It is one of the most common principal complaints in the emergency department (ED), responsible for 2.5% of all ED visits.<sup>2</sup> Benign paroxysmal positional vertigo, thought to be caused by loose particles in the semicircular canals, is the most common cause of vertigo, with an incidence estimated to be 107 cases per 100 000 population per year.<sup>3</sup> Dizziness in older individuals is associated with a variety of cardiovascular, neurosensory, and psychiatric conditions and with the use of multiple medications.<sup>4</sup> Among patients older than 60 years, 20% have experienced dizziness severe enough to affect their daily activity.<sup>5</sup> In a study of 1000 outpatients, dizziness was the third most common complaint. *Vertigo* is defined more clearly as a sensation of disorientation in space combined with a sensation of motion. Dizziness and imbalance are most commonly seen with peripheral processes but they can be the only clinical manifestations of a central life threat.

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This article focuses on diagnosis and treatment when stroke and other central neurologic processes are in the differential.

## **PATHOPHYSIOLOGY**

A complaint of dizziness is an imprecise term. The ED physician may think that these patients will be difficult to interview and that the condition will be problematic to diagnose and treat. But in reality, most of these patients have an organic basis for symptoms that can be successfully identified and treated. The diagnostic process is consistently based on 2 basic concepts: deciding whether patients have true vertigo and, if vertigo exists, deciding whether the cause is a central or peripheral neurologic entity.<sup>6</sup>

The maintenance of equilibrium and awareness of the body in relationship to its surroundings depend on the interaction of 3 systems: visual, proprioceptive, and vestibular. The eyes, muscles, joints, and otic labyrinths continuously supply information about the position of the body. Visual impulses, mediated through the higher brain centers, provide information about body position in space. Impulses from proprioceptors of the joints and muscles supply data about the relative positions of the parts of the body. Impulses from the neck are of special importance in relating the position of the head to the rest of the body. The sense organs of the visual, vestibular, and proprioceptive systems are connected with the cerebellum by way of the vestibular nuclei in the brainstem. Any disease that interrupts the integration of these 3 systems may give rise to symptoms of vertigo and disequilibrium.

## **DIAGNOSTIC APPROACH**

### ***Differential Considerations***

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Patients use the term *dizzy* to describe a variety of experiences, including sensations of motion, weakness, fainting, light-headedness, unsteadiness, and depression. To clarify the picture, it is often helpful to have patients describe the sensation without using the word *dizzy*. True vertigo may be defined as a sensation of disorientation in space combined with a sensation of motion. There is a hallucination of movement either of the self (subjective vertigo) or the external environment (objective vertigo). Descriptions of light-headedness or feeling faint are more consistent with presyncope. The differential diagnosis for these patients should include hypovolemia, dysrhythmias, myocardial infarction, sepsis, drug side effects, and pulmonary embolism. For some patients, dizziness is simply a metaphor for malaise, representing a variety of other causes, such as anemia, viral illness, or depression.

If patients have true vertigo, the clinician must determine whether the cause is a peripheral lesion (eg, of the inner ear) or a central process, such as cerebrovascular disease or a neoplasm. In most cases, peripheral disorders are benign and central processes are more serious. Occasionally, as in the case of a cerebellar hemorrhage, immediate therapeutic intervention is indicated. Acute suppurative labyrinthitis is the only cause of peripheral vertigo that requires urgent intervention.

### ***Pivotal Findings***

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#### ***History***

The medical history is the most important source of information. A first key question is, does true vertigo exist? Do patients have a sensation of disorientation in space or a sensation of motion? The sensation of spinning usually indicates a vestibular disorder. Some nausea, vomiting, pallor, and perspiration accompany almost all but the mildest forms of vertigo. The presence of these symptoms without vertigo should suggest a different cause. The labyrinth has no effect on the level of consciousness.

Patients should not have an associated change in mentation or syncope. A sensation of imbalance often accompanies vertigo, but true instability, disequilibrium, or ataxia makes a central process more likely.<sup>7</sup> The symptom of imbalance in most cases should warrant a workup for stroke.

Because nystagmus accompanies acute vertigo, it is often helpful to ask members of the patients' family if they have noted any unusual eye movements during the dizzy spells. This question is especially important in children unable to offer a concise history.<sup>8</sup> Occasionally, patients may be able to describe a flickering or oscillating visual field immediately after a change in position, such as rolling over in bed. In addition, interviewing family and other witnesses can often uncover evidence suggesting seizures, syncope, or imbalance unrelated to feelings of vertigo.

The *time of onset* and the *duration of vertigo* are important clues to the cause. Episodic vertigo that is severe, lasts several hours, and has symptom-free intervals between episodes suggests a peripheral labyrinth disorder. Vertigo produced primarily by a change in position also suggests a peripheral disorder. Vestibular neuritis and benign positional vertigo fit this pattern.

The presence of *auditory symptoms* suggests a peripheral cause of the vertigo, as in middle and inner ear problems, or a peripheral cause that progresses centrally, such as an acoustic neuroma. The abnormally hearing ear is usually the side of end-organ disturbance. Progressive unilateral hearing loss of several months duration may be the earliest symptom of an acoustic neuroma. Tinnitus occurs in most patients with acoustic neuroma and, along with vertigo, is what often prompts patients to seek medical attention. Another peripheral process, Meniere disease, can present with the characteristic triad of hearing loss, vertigo, and tinnitus.

*Are there associated neurologic symptoms?* Patients or family members should be questioned about the time of onset of ataxia or gait disturbances. Ataxia of recent and sudden onset suggests cerebellar hemorrhage or infarction in the distribution of the posterior inferior cerebellar artery or the superior cerebellar artery. The salient feature of chronic cerebellar disorders is a slowly progressive ataxia. True ataxia may be difficult to discern from the unsteadiness that occurs when patients with significant vertigo attempt to walk.

Vertiginous symptoms are common after head injuries. The presence of recent head or neck trauma should be explored because vertiginous symptoms are common after both.<sup>9,10</sup> Head injuries can cause vertigo occasionally from intracerebral injury and more commonly from labyrinth concussion. Neck injuries can cause vertigo from strain of muscle proprioceptors. In addition, a vertebral artery injury has been seen resulting from activities, such as chiropractic manipulation and even hair shampooing with marked hyperextension in a salon.<sup>11</sup>

It has clearly been shown that isolated vertigo can be the only initial symptom of cerebellar and other posterior circulation bleeds, transient ischemic attacks (TIAs), and infarction.<sup>12-14</sup> One study showed that emergency physicians often did not make the correct diagnosis in patients with validated strokes or TIAs that presented with only vertigo.<sup>7</sup> Identifying these individuals is without a doubt the most important and difficult challenge for clinicians taking care of patients with vertigo. Most patients with isolated dizziness do not have TIA or stroke but the risk of not identifying the few that do is significant for ultimate patient outcomes. Stroke has been seen in 3.2% of patients with a dizziness syndrome but only 0.7% of those with isolated dizziness had a stroke.<sup>7</sup> A recent study also showed less than 1 in 500 patients discharged with a diagnosis of dizziness or vertigo experienced a major vascular event in the month after discharge.<sup>15</sup> In addition, a cross-sectional study of the National Hospital Ambulatory Medical Care Survey suggests significant overuse of computed tomography (CT) in

low-risk patients.<sup>16</sup> Risk-factor assessment and symptom patterns can be extremely helpful in deciding which patients warrant imaging, neurologic consultation, and admission. Older age, male sex, and the presence of hypertension, coronary artery disease, diabetes mellitus, and atrial fibrillation puts patients at a higher risk for having an acute stroke as the cause of their symptoms. In addition, frequent episodes lasting only minutes or prolonged episodes of a day or more are more often associated with central processes.<sup>7,12,13</sup> One retrospective study showed that emergency physicians often failed to chart the triggers and the duration of dizziness, information that could potentially lead to increased likelihood of a more serious cause of symptoms.<sup>17</sup> Also, as previously mentioned, the symptom of imbalance raises the likelihood of TIA and stroke.

### ***Past medical history***

Many medications have direct vestibulotoxicity. The most commonly encountered are the aminoglycosides, anticonvulsants, alcohols, quinine, quinidine, and minocycline. In addition, caffeine and nicotine can have wide-ranging autonomic effects that may exacerbate vestibular symptoms. The history of past and present illnesses should be explored, with specific questioning about the existence of diabetes, drug or alcohol use, and the risk factors mentioned earlier.

### ***Physical Examination***

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#### ***Vital signs***

In some cases, pulses and blood pressure should be checked in both arms. Most patients with subclavian steal syndrome, which can also cause vertebrobasilar artery insufficiency, have pulse or systolic blood pressure differences between the two arms.

#### ***Head and neck***

Carotid or vertebral artery bruits suggest atherosclerosis. The neck is auscultated along the course of the carotid artery from the supraclavicular area to the base of the skull.

Vertigo can be caused by impacted cerumen or a foreign object in the ear canal. Accumulation of fluid behind the eardrum as a result of a middle ear infection may cause mild vertigo, as can occlusion of the eustachian tubes associated with an upper respiratory tract infection. A perforated or scarred eardrum may indicate a perilymphatic fistula, especially if the history includes previous trauma.

The examination of the eyes is key in assessing patients with vertigo or disequilibrium. The focus is on any pupillary abnormalities indicating third cranial nerve or descending sympathetic tract involvement or optic disk signs of early increased intracranial pressure. Extraocular movements should be assessed carefully. Relatively subtle ocular movement abnormalities can be the only clue to a cerebellar hemorrhage. A sixth cranial nerve palsy ipsilateral to the hemorrhage may result from early brainstem compression by the expanding hematoma. Internuclear ophthalmoplegia is recognized when the eyes are in a normal position on straight-ahead gaze but on eye movement the adducting eye (cranial nerve III) is weak or shows no movement while the abducting eye (cranial nerve VI) moves normally, although often displaying a coarse nystagmus. This finding indicates an interruption of the medial longitudinal fasciculus on the side of the third cranial nerve weakness.

Abnormal nystagmus is the cardinal sign of inner ear disease and the principal objective evidence of abnormal vestibular function. In nystagmus, patients have difficulty maintaining the conjugate deviation of the eyes or have a postural control imbalance of eye movements.

The abnormal jerk nystagmus of inner ear disease consists of slow and quick components. The eyes slowly drift in the direction of the diseased hypoactive ear, then quickly

jerk back to the intended direction of gaze. Positional nystagmus, induced by rapidly changing the position of the head, strongly suggests an organic vestibular disorder. A central nervous system cause of positional nystagmus should be considered when the pattern of nystagmus is persistent down beating, pure torsional, or when it is refractory to repositioning maneuvers. The characteristics of nystagmus are one of the most valuable tools for distinguishing peripheral from central causes of vertigo.

### ***Positional testing***

If nystagmus is not present at rest, positional testing can be helpful in determining its existence and characteristics. In the Hallpike maneuver, patients are moved quickly from an upright seated position to a supine position and the head is turned to one side and extended (to a head-down posture) approximately 30° from the horizontal plane off the end of the stretcher. The eyes should be observed for nystagmus, and patients should be queried for the occurrence of symptoms. This test should be repeated with the head turned to the other side. Positive elicitation of symptoms and signs to one side or the other generally indicates a vestibular pathologic condition on that same side. This test should be performed with caution if vertebrobasilar insufficiency is suggested because sudden twisting movements theoretically might dislodge atheromatous plaques.

### ***Neurologic examination***

The presence of cranial nerve deficits suggests a space-occupying lesion in the brainstem or cerebellopontine angle. The corneal reflex is a sensory cranial nerve (V) and motor cranial nerve (VII) circuit. Its diminution or absence can be one of the early signs of an acoustic neuroma. Vertigo caused by eighth cranial nerve involvement is likely to be accompanied by a unilateral hearing loss. Patients cannot hear a tuning fork when it is held alongside the affected ear but they can hear it when it is held against the mastoid process. The involvement of the eighth cranial nerve suggests an acoustic tumor. Seventh cranial nerve involvement causes facial palsy that affects the side of the face. In supranuclear facial paralysis, the forehead is spared because these muscles receive bilateral cortical innervation.

Patients should be evaluated specifically for evidence of cerebellar dysfunction. This examination must be performed in bed and standing because truncal ataxia may be occult on testing of the limbs in bed and may become obvious only when patients have to sit, stand, or walk unaided. Dysmetria is the inability to arrest a muscular movement at the desired point. Dysmetria should be assessed using finger-to-finger/finger-to-nose pointing and heel-to-shin testing, and dysdiadochokinesia (an inability to perform coordinated muscular movement smoothly) is assessed with rapid alternating movements. The gait must be evaluated when patients give a history suggesting ataxia, although examination may be impossible during an attack of vertigo. Any marked abnormality (eg, consistent falling or a grossly abnormal gait) should suggest a central lesion, especially in patients whose vertiginous symptoms have subsided. The main features of a cerebellar gait are a wide base (separation of legs), unsteadiness, irregularity of steps, tremor of the trunk, and lurching from side to side. The unsteadiness is most prominent on arising quickly from a sitting position, turning quickly, or stopping suddenly while walking. Patients with gait ataxia cannot perform heel-to-toe walking.

### ***Ancillary Testing***

Most routine laboratory testing is not helpful in the evaluation of patients with vertigo. A finger-stick blood glucose test should be performed in most cases because

hypoglycemia can present as vertigo.<sup>18</sup> Blood counts and blood chemistries are sometimes helpful when it is difficult to distinguish whether dizziness is vertigo or near syncope. An electrocardiogram should be obtained if there is any possibility of myocardial ischemia or dysrhythmia.

### **Radiologic imaging**

If cerebellar hemorrhage, cerebellar infarction, or other central lesions are suggested, emergent CT or magnetic resonance imaging (MRI) of the brain is indicated. MRI, when available, has become the diagnostic modality of choice when cerebellar processes other than acute hemorrhage are possible. MRI is particularly useful for the diagnosis of acoustic neuromas and for sclerotic and demyelinating lesions of the white matter, as seen in multiple sclerosis. Acute vertigo by itself does not warrant urgent CT or MRI in all patients, particularly patients in whom a clear picture of peripheral vertigo emerges. But as mentioned earlier, many studies strongly support the use of imaging in patients of advanced age or at risk for cerebrovascular disease.<sup>7,12,13,19</sup> CT, although often useful for identifying hemorrhage, is insensitive for acute stroke presentations, especially for infarction within the posterior fossa. MRI with MR angiography (MRA) is considered a much more sensitive modality and should be performed quickly in patients with changing neurologic signs and symptoms, suggesting impending posterior circulation occlusion. The use of these modalities is discussed in more depth in the subsequent sections.

Audiology and electronystagmography are helpful in the follow-up evaluation of patients with vertigo. Audiology can locate the anatomic site of a lesion causing vertigo. Electronystagmography is a collection of examinations that, when abnormal, suggest vestibular dysfunction but do not yield the specific diagnosis.

**Posterior circulation stroke** The clinical presentations of patients with posterior circulation ischemia can be vague and may overlap significantly with many frequently seen ED complaints. As described previously, approximately 7.5 million individuals with dizziness are seen in ambulatory care settings in the United States annually.<sup>1</sup> Importantly, less than 25% of cases are thought to result from a central cause.<sup>20</sup> As with many other ED scenarios, the emergency physician is faced with the challenge of identifying the few patients with concerning pathologic conditions from the many with less serious causes of illness.

### **Epidemiology**

Posterior circulation strokes (PCS) account for approximately 20% to 30%<sup>21-23</sup> of all strokes, and the clinical manifestations of the disease can be varied. Although some patients may experience mild, intermittent brainstem symptoms (thought to arise from fluctuations in posterior circulation blood flow), others may unfortunately become locked-in, which is a result of basilar artery or bilateral vertebral artery occlusion in which patients retain full awareness and cognition but only have movements of their eyelids or eyes. Similarly, the mortality of PCS ranges from as low as 3.6% (at 30 days)<sup>24</sup> to more than 90% for those who are locked-in.<sup>25</sup>

### **Anatomy**

Classically, the posterior circulation consists of the vertebral arteries, the basilar artery, the posterior cerebral arteries, and their branches. The vertebral arteries arise from the subclavian arteries and course through the vertebral foramina of the sixth through second cervical vertebrae before traversing the foramen magnum. At the level of the pontomedullary junction, the vertebrals join to create the basilar artery. Distally,

the basilar artery divides into the posterior cerebral arteries and, finally, the posterior communicating artery in the circle of Willis. Although there is significant variability in the exact arterial anatomy of the posterior circulation, these arteries supply the brainstem, the thalamus, the hippocampus, the cerebellum, and portions of the occipital and temporal lobes.<sup>26</sup>

### ***ED Presentation***

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Ischemia of structures supplied by the posterior circulation may present in a more subtle fashion than those affecting the anterior circulation. For example, in contrast to disease affecting the middle cerebral artery, obvious speech, motor, and sensory deficits are often not the dominant presenting feature. In one large series of patients with confirmed PCS, dizziness, dysarthria, headache, nausea/vomiting, and blurred vision were the most common presenting symptoms, whereas unilateral limb weakness, gait ataxia, unilateral limb ataxia, dysarthria, and nystagmus were the most common presenting signs.<sup>27</sup> In another series of patients with posterior circulation events, vertigo, unsteadiness, dysarthria, and nausea/vomiting were the most common presenting symptoms, whereas the most common neurologic signs included facial palsy, ataxia, focal weakness, and nystagmus.<sup>28</sup> Posterior circulation ischemia rarely presents as a single symptom. Rather, depending on the location of the ischemia, patients present with a constellation of symptoms and signs; less than 1% of patients in one series had a single presenting symptom or sign.<sup>24</sup> Patients may also present with significant alterations in their level of consciousness ranging from mild lethargy to frank coma if areas, such as the reticular activating system, are involved.<sup>27,29</sup>

The classic presentation of those with posterior circulation ischemia has been described as crossed findings with cranial nerve findings on the side of the lesion and long tract (eg, motor or sensory) findings on the contralateral side. Signs that may indicate vertebrobasilar ischemia include internuclear ophthalmoplegia, unreactive pupils, skew deviation, hemianopia, and cortical blindness.<sup>27</sup>

The exact constellation of symptoms and findings depends on the precise location of the infarct; commonly described syndromes include Wallenberg syndrome (nystagmus, vertigo, ataxia, hoarseness, dysphagia); Anton syndrome (somnolence, memory defects, confusion, visual hallucinations with bilateral loss of vision with unawareness or denial of blindness, vertical gaze paralysis, skew deviation of the eyes); Weber syndrome (ipsilateral oculomotor palsy with contralateral hemiplegia); and Dejerine-Roussy syndrome (contralateral hemisensory loss of all modalities).

Importantly, patients presenting with symptoms consistent with a TIA of the posterior circulation are thought to be at a high risk for early recurrent ischemic events. Although the risk of subsequent early stroke on patients with carotid artery stenosis has been well documented, only in recent years has a similar phenomenon been reported in those with stenosis of the vertebrobasilar system. The development of contrast-enhanced MRA and contrast CT angiography (CTA) has enabled investigators to noninvasively study the posterior circulation, and the risk of recurrent PCS (after initial stroke or TIA) is thought to be as high as 30.5%.<sup>22,30</sup>

### ***ED Diagnosis***

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As with many other conditions, the ED physician must maintain a high level of suspicion to identify patients with evidence of posterior circulation ischemia. Patient complaints may be vague, and neurologic abnormalities may be subtle; the astute clinician must rely heavily on a thorough history and physical examination with particular focus on the cranial nerves, eye movements, and cerebellar findings.

In many centers, noncontrast CT of the brain remains the initial diagnostic test of choice in patients in whom PCS is suspected. Although CT imaging allows for the rapid acquisition of images and is sensitive for the presence of acute hemorrhage, artifacts from surrounding bone may limit the clarity with which one can see the posterior fossa. Alternatively, MRI is unaffected by neighboring bone and it offers superior resolution of brainstem structures.<sup>31</sup> If readily available, MRI, including MRA and diffusion-weighted and perfusion-weighted images, is the study of choice for patients with PCS.<sup>32</sup>

Importantly, stroke-related impairment scales, such as the National Institute of Health Stroke Scale (NIHSS), which are often used to judge the severity of stroke symptoms, may underestimate the degree of impairment in patients with posterior circulation disease. Although the NIHSS is regarded as easy to administer and has been widely validated, it is weighted more toward motor and sensory symptoms than cranial nerve deficits and other signs of PCS.<sup>33,34</sup> Similarly, the Face Arm Speech Test score,<sup>35</sup> which was developed to assist with the identification of patients with stroke, and the ABCD2 score,<sup>36</sup> which aims to identify patients with TIA at a high risk for recurrent stroke, have both been shown to be less effective in the diagnosis and identification of PCS.<sup>37</sup>

### ***Evaluation and Management of Posterior Circulation Ischemia***

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Despite the frequency with which PCS patients are encountered in the ED, clinical research regarding the optimal investigation and management of PCS has lagged behind that of stroke attributable to disease of the anterior circulation. The reasons for this are multifactorial but largely stem from an (incorrect) thought that strokes of the posterior circulation were solely related to hemodynamically significant lesions in the vertebral or basilar arteries (or their penetrating branches), a relative inability to study the posterior circulation noninvasively (eg, before the routine use of MRI/MRA and CTA), and before the development of interventional approaches to the vessels of the posterior circulation.<sup>32</sup>

Traditionally, patients presenting with symptoms suggestive of posterior circulation ischemia were treated differently from those with disease of the anterior circulation. Although patients with right arm weakness and expressive aphasia might have undergone brain imaging as well as vascular studies (eg, carotid ultrasound, echocardiography), patients with abnormalities of cerebellar testing and diplopia may only have brain imaging before a diagnosis of vertebrobasilar insufficiency is made. Furthermore, rather than searching for the cause and mechanism of brain ischemia, physicians only debated whether or not to introduce anticoagulation (warfarin) or at what intensity the anticoagulation should be maintained. Conversely, therapeutic considerations for those with anterior circulation strokes included angioplasty, anticoagulants, antiplatelet agents, and carotid artery surgery.<sup>24,32</sup>

In 2004, Caplan and colleagues<sup>24</sup> published data from the *New England Medical Center Posterior Circulation Registry* that challenged this paradigm and argued that mechanisms of anterior and PCS were more alike than dissimilar. In their series of more than 400 patients with posterior ischemia, 40% were thought to have an embolic source (24% cardioembolic, 14% artery-artery, and 2% cardiac and artery-artery), and 32% resulted from large artery stenosis. In comparison, data collected during a similar time from patients with anterior circulation ischemia revealed that 38% had a cardioembolic source and 9% were caused by larger artery occlusive lesions. Other causes of posterior circulation ischemia include arterial dissection, migraine, fibromuscular dysplasia, coagulopathies, and drug abuse.<sup>38</sup> Similarly, other large series suggest that the rates of vascular risk factors, such as hypertension, diabetes,



smoking, and dyslipidemia, are also comparable between those with disease of the anterior and posterior circulation.<sup>21</sup>

As a result, leaders in stroke management began to emphasize the importance of using a diagnostic approach to PCS similar to that which had traditionally been reserved for anterior circulation stroke (ACS). Similarly, it was suggested that therapy for patients with PCS should be tailored to the specific cause of the stroke, as is the practice with ACS.<sup>24,39</sup>

### **Management of PCS**

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As with research regarding the cause of PCS, data regarding the treatment of PCS has lagged behind that of ACS.<sup>40</sup> Patients with posterior circulation disease were often specifically excluded from trials (eg, PROACT II, which demonstrated an improved outcome in patients with middle cerebral artery occlusion who were treated with intra-arterial thrombolysis<sup>41</sup>) or were less likely to be enrolled because of specific inclusion criteria (eg, the landmark National Institute of Neurologic Disorders and Stroke study documenting the effectiveness of intravenous thrombolytic therapy for acute stroke<sup>42</sup>).

#### **Pharmacologic therapy**

To a great extent, medical therapy for PCS has not been rigorously tested in controlled trials. Aspirin and other antiplatelet drugs have been used to treat posterior circulation disease but controlled trials of their efficacy are lacking<sup>43</sup> and recommendations for their use are largely based on expert opinion, anecdotal evidence, and studies of carotid or ACS.<sup>44–47</sup> Although patients with PCS have traditionally been anticoagulated with heparin, the data supporting this treatment has been taken from uncontrolled trials showing some benefit relative to historical controls.<sup>32</sup> Although treatment with warfarin has been shown to be effective in the prevention of subsequent cerebrovascular events in patients with embolic stroke of cardiac origin, there are no recent prospective controlled trials directly addressing the potential benefit of these therapies in preventing PCS.

Similarly, intravenous thrombolysis is used in many centers for the treatment of acute ischemic stroke of both the anterior and posterior circulations despite a relative lack of data regarding its safety or efficacy for posterior events; only 5% of patients from the National Institutes of Neurologic Diseases and Stroke (NINDS) study had PCS,<sup>42</sup> the European Cooperative Acute Stroke Study (ECASS) I and II trials only included patients with ACS,<sup>48,49</sup> and the Alteplase Thrombolysis for Acute Noninterventional Therapy in Ischemic Stroke and the ECASS III trials did not report on the number of patients with PCS.<sup>50–52</sup>

The few studies in the literature that examined intravenous thrombolysis in patients with PCS are limited by the small sample size (9–12 patients)<sup>40,53,54</sup> or were restricted to those with basilar artery occlusion.<sup>55,56</sup> In 2011, however, Sarikaya and colleagues<sup>57</sup> published data on 883 consecutive patients with acute ischemic stroke (788 ACS, 95 PCS) who were treated with intravenous thrombolysis. They report a lower rate of intracerebral hemorrhage in those patients with PCS treated with thrombolytics compared with those with ACS, although favorable outcome and mortality rates were similar in the two populations.

At some centers, intra-arterial catheter-directed thrombolysis is also performed for patients with stroke who are outside the 3-hour window dictated by the NINDS trial. Once again, controlled prospective data are lacking and expert opinion argues that there is insufficient data to guide the choice between intravenous and intra-arterial thrombolytic therapy for vertebrobasilar ischemia.<sup>38</sup>

### **Endovascular therapy**

The successful use of percutaneous transluminal angioplasty in coronary and renal arteries led to attempts to use similar techniques for diseases of the posterior cerebral circulation. Unfortunately, however, complications, such as plaque disruption resulting in distal emboli and arterial vasospasm, have limited the utility of this practice. One retrospective study of 21 patients who had failed medical therapy, had poor collateral flow, were deemed high risk, and who subsequently underwent endovascular balloon angioplasty with stent placement reported favorable outcomes (although long-term follow-up was lacking).<sup>58</sup> Small vessel caliber and angulation of the vertebral vessels make endovascular treatment technically challenging. Generally, because of the high risk of complications, angioplasty of the basilar artery is advised only for patients with severe symptoms who have failed traditional medical therapy.<sup>43</sup> Randomized prospective trials of endovascular therapy are lacking.

### **Admission criteria**

With few, if any, exceptions, all patients with PCS should be admitted to the hospital, preferably under the care of a stroke specialist. Patients may require intensive care unit level care if their stroke is of a large volume; if there is thought to be a significant area of brain tissue at risk for further injury; if critical brainstem function is threatened; if they have received acute intervention, such as thrombolytics; or if they have significant comorbid medical conditions. Examinations aimed at identifying the cause of the stroke should be undertaken and risk-factor modification should be addressed.

## **SUMMARY**

Dizzy patients present a significant diagnostic challenge to the emergency clinician. Discrimination between peripheral and central causes is important and will inform subsequent diagnostic evaluation and treatment. Isolated vertigo can be the only initial symptom of a PCS. The sensation of imbalance especially raises this possibility. Research involving strokes of the posterior circulation has lagged behind that of the anterior cerebral circulation. Investigations of the last 20 years, using new technologies in brain imaging in combination with detailed clinical studies, have revolutionized our understanding of the clinical presentation, causes, treatments, and prognosis of posterior circulation ischemia.<sup>38</sup> Traditional teaching, which emphasized differences in the cause, diagnosis, and treatment of ACS and PCS, must be reconsidered, and the entities may be more similar than initially thought. The approach to patients with PCS should be no different from those with strokes elsewhere in the brain: the immediate goals are to correctly identify stroke as a diagnostic possibility, determine the time of onset, proceed with rapid imaging, and involve neurologic expertise. The ED physician must maintain a high index of suspicion for PCS because patients may present in a more subtle fashion than those with ACS. Further research examining optimal medical and interventional therapy for patients with PCS is needed.

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