Does This Dizzy Patient Have a Serious Form of Vertigo?

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CLINICAL SCENARIOS: COMMON CAUSES OF VERTIGO

Patient 1

A 52-year-old woman was admitted to the hospital because of nausea, a constant spinning sensation, and vomiting of 24 hours' duration. Any movement of her head made these symptoms worse. On examination, she had bilateral horizontal spontaneous nystagmus. Two days later, after symptomatic improvement, she was dismissed. At follow-up 2 weeks later, her symptoms and nystagmus had completely resolved.

Patient 2

A 70-year-old woman had a 4-month history of an intermittent whirling sensation when turning her head and especially when rolling over in bed. On examination, a left-side-down head-hanging maneuver elicited rotatory nystagmus, with the fast component to the left ear (Figure). There was a latency of about 3 seconds before the onset of nystagmus, which lasted approximately 10 seconds.

WHY EVALUATE VERTIGO?

"Vertigo" is defined in Webster's dictionary[2] as a disturbance "in which the external world seems to revolve around the individual or in which the individual seems to revolve in space." Vertigo is an illusion of motion[3] and is one of several forms of dizziness. The word *dizziness* is derived from the old English word *dysig*, meaning foolish or stupid. The modern usage of the word includes "a whirling sensation in the head with a tendency to fall," "mentally confused or dazed," and "giddiness."[2]

In one study[4] from a general internal medicine outpatient clinic, dizziness was the third most frequent complaint of patients. In a national survey reported in 1989,[5] it was the 13th most frequent reason for visits to internists in the United States. Dizziness is often a diagnostic problem in the emergency department.[6] Among patients seen in an emergency department,[6] in an outpatient clinic,[7] and in two subspecialty dizziness clinics,[8,9] vertigo was the most frequent category of dizziness.

Most patients with dizziness can be classified as having one of the following syndromes:

1. Impaired perfusion of the central nervous system or near syncope (eg, orthostatic hypotension, cardiac presyncope).
2. Disequilibrium, a sensation of imbalance when standing or walking[7] (eg, multiple sensory deficits).
3. Psychogenic dizziness (eg, major depression, anxiety disorder, and somatization disorder).
4. Vertigo (eg, Meniere's disease and vestibular neuronitis).[8]

Usually dizziness can be classified on the basis of information obtained from the medical history and physical examination. In this article, we concentrate on the evaluation of vertigo, the most common category of dizziness. Serious forms of vertigo are due to conditions associated with increased mortality or long-term disability. Vertigo severe enough to impair daily functioning and lasting for more than a month would be included as a serious form of vertigo.

The importance of recognizing a patient's complaint of dizziness as vertigo is that it narrows the list of possible causes. Customarily the causes of vertigo are divided into central causes (lesions of the central nervous system) and peripheral causes (lesions of the vestibular labyrinth or nerve or both) (Table 1). Because of the importance of detecting lesions or diagnosing syndromes that can be treated and because of the need to determine prognosis, physicians should attempt to make a specific diagnosis for patients with vertigo.

Most cases of vertigo are due to lesions of the vestibular nerve or labyrinth.[6-9] In two dizziness clinics, the most common cause of vertigo was benign paroxysmal positional vertigo.[8,9]

PATHOPHYSIOLOGY OF VERTIGO AND NYSTAGMUS

Origins of Vertigo

Before discussing how the medical history and physical examination can help in diagnosing the various disorders that cause vertigo, a brief explanation of the pathophysiology of vertigo and its causes is necessary. The maintenance of the sense of balance and spatial orientation depends on input from the vestibular labyrinth, visual system, and proprioceptive nerves arising from tendons, muscles, and joints.[10] The vestibular nuclei, which are in the medulla and lower pons, receive input from the vestibular labyrinth via the vestibular branch of cranial nerve VIII and from the cerebellum.[11] The vestibular nuclei, in turn, send efferent fibers to the cerebellum, the medial longitudinal fasciculus, and the vestibulospi-

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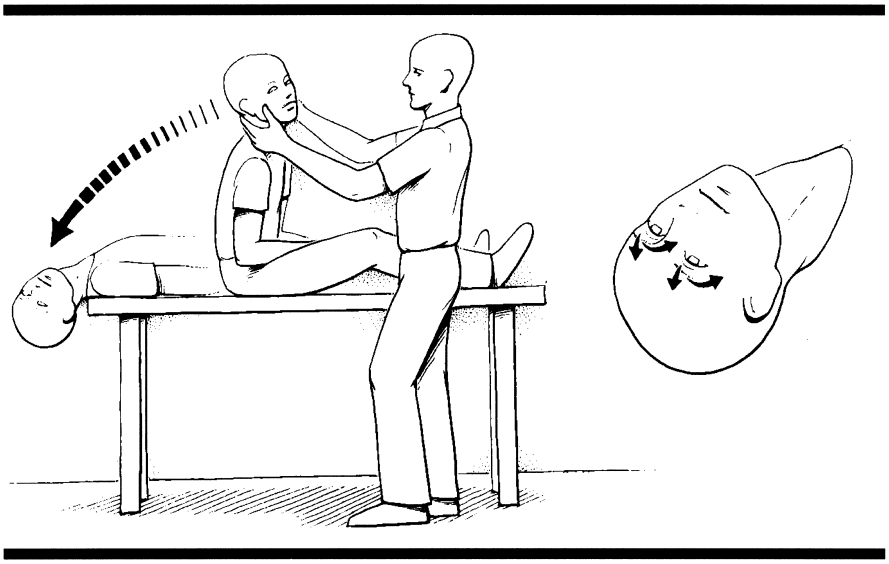

How to test for positional nystagmus (reprinted by permission of the *Western Journal of Medicine*[1]).

Table 1.—Common Causes of Vertigo

Peripheral
Benign paroxysmal positional vertigo
Vestibular neuronitis
Recurrent vestibulopathy
Classic Meniere's disease
Head trauma (labyrinthine concussion)
Acoustic neuroma
Otosclerosis
Herpes zoster oticus
Cholesteatoma
Perilymph fistula
Aminoglycoside ototoxicity
Central
Vertebrobasilar transient ischemic attacks
Cerebellar or brain stem stroke
Brain tumors
Multiple sclerosis
Vertebrobasilar migraine

nal tract. Visceral manifestations of vertigo (such as nausea and vomiting) are caused by altered input to the dorsal nucleus of the vagus nerve from the vestibular nuclei. Conscious awareness of vertigo resides in the superior temporal gyrus of the cerebral cortex[10] and involves a mismatch between input to the cerebral cortex from the visual, proprioceptive, and vestibular systems.[12] Lesions in various locations, including the inner ear, brain stem, and cerebellum, may all be manifested as vertigo.

Origins of Nystagmus

Nystagmus is the objective accompaniment of vertigo and is defined best as a "rhythmical oscillation of the eyes, with a fast movement in one direction and a slow movement in the other."[13] The fast component may be horizontal, vertical, rotatory, or any combination of these.[14]

There are two clinically relevant kinds of nystagmus in evaluating vertigo: (1) Spontaneous nystagmus is elicited by having the patient look straight ahead, up, down, to the right, and to the left. This type of nystagmus is not influenced by head position.[15] It is normal to have a few beats of nystagmus with extreme lateral gaze.[14] (2) Positional nystagmus is elicited by a head-hanging maneuver (Figure).[14]

Altered input passing from the vestibular nuclei to the nuclei of the extraocular muscles through the medial longitudinal fasciculus and related pathways in the reticular formation produces nystagmus. This input may be modified by information arising from the cerebral cortex and the cerebellum.[14] For example, the fast component of spontaneous nystagmus depends on interaction between the vestibular system and the cerebral cortex.[16]

HOW TO ELICIT THE SYMPTOMS AND SIGNS OF VERTIGO

First, Distinguish Vertigo From Other Causes of Dizziness

Patients often have difficulty describing symptoms of dizziness, and even those who have disorders that produce vertigo may not clearly describe a hallucination of movement. As Olsson and Atkins[17] pointed out, "A person is so rarely conscious of his own vestibular system, he has a great deal of trouble describing his symptoms to a doctor." Thus, clues must be gathered from the medical history and physical examination to classify the dizziness properly.

Dizziness when standing may be due to vertigo, decreased cerebral perfusion,[18] or disequilibrium.[7] If the patient reports having symptoms of dizziness primarily while standing, the blood pressure should be checked with the patient in the supine position and also after standing for 5 minutes. If there is an orthostatic decrease in blood pressure, the symptom is likely due to impaired central nervous system perfusion.

Unsteadiness while walking, especially in elderly patients, is often due to disequilibrium (a feeling of imbalance). The cause is usually multifactorial. On examination, the findings of decreased visual acuity and signs of peripheral neuropathy or abnormal vestibular function support a diagnosis of disequilibrium.[7,8]

Dizziness when turning, and especially when rolling over in bed, is usually due to vertigo.

Psychogenic dizziness is a diagnosis of exclusion that should be considered especially in patients with psychiatric illnesses, such as major depression, anxiety disorder, and a somatization disorder. In this setting, the patient should be asked to hyperventilate for 2 minutes and then asked whether the feeling associated with hyperventilation is exactly the same as the dizzy symptom. The physician should initially hyperventilate along with the patient; this approach encourages the patient and demonstrates the desired rate and depth of breathing for the test.[19] If hyperventilation reproduces the symptom, the dizziness is often psychogenic. However, the usefulness of hyperventilation in diagnosing psychogenic dizziness is unclear. In a study by Kroenke et al[7] of 100 ambulatory patients with a chief complaint of dizziness, symptoms of dizziness were reproduced by hyperventilation in 21; however, only one of these patients had hyperventilation as the primary cause of dizziness. Most of them had dizziness inducible by other maneuvers in addition to hyperventilation. Further studies of the hyperventilation maneuver in the evaluation of patients with suspected psychogenic dizziness are needed. In this study of 100 patients, only 16% had pure psychogenic dizziness, but 24% had other causes of dizziness exacerbated by psychiatric illness.[7]

Second, Take a Proper History From Patients With Vertigo

After it is clear that the patient is describing vertigo, further questions help elicit clues about its specific cause.

Ask When the Dizziness Occurs.—It is probably more important to ask a patient about the circumstances in which the dizziness occurs than to ask for a description of the dizziness. Dizziness related to early-morning activities is somewhat helpful in distinguishing between peripheral and central vertigo. Matutinal vertigo (vertigo on first arising in the morning) is usually due to a peripheral vestibular disorder.[20]

Ask About Other Otologic Symptoms.—Associated otologic symptoms can be helpful in identifying a peripheral cause of vertigo. Hearing loss and

Table 2.—Accuracy of Signs and Symptoms for Diagnosing Peripheral Vertigo in an Emergency Department*

	No. of Patients With Peripheral Vertigo (Not an Emergency)	No. of Patients With Other Causes of Dizziness That Might Be an Emergency	Total	Predictive Value	Likelihood Ratio
Positive cluster of signs and symptoms†	23	4	**27**	Positive 85% (23/27)	7.6
Lack of one or more elements in cluster	31	67	**98**	Negative 68% (67/98)	0.6
Total	**54**	**71**	**125**	. . .	. . .

*Data from Herr et al.[6]
†Positive cluster includes positive results on head-hanging maneuver plus either vertigo or vomiting.

Table 3.—Accuracy of Signs and Symptoms for Detecting Serious Causes of Dizziness in an Emergency Department*

	No. of Patients With Serious Causes of Dizziness†	No. of Patients With Nonserious Causes of Dizziness	Total	Predictive Value	Likelihood Ratio
Absence of vertigo or age >69 y or neurological deficit	33	50	**83**	Positive 40% (33/83)	1.5
Presence of vertigo or age ≤69 y or no neurological deficit	5	37	**42**	Negative 88% (37/42)	0.3
Total	**38**	**87**	**125**	. . .	. . .

*Data from Herr et al.[6]
†Serious causes of dizziness include medication side effects, seizures, stroke, and cardiac arrhythmia.

vertigo are common in patients with otosclerosis.[21] Episodes of hearing loss with vertigo, tinnitus, and a sensation of fullness in the ear occur in patients with Meniere's disease.[22] Patients with acoustic neuromas usually present with hearing loss rather than vertigo. Most of these patients notice dizziness but complain of unsteadiness rather than vertigo.[23]

Ask About Other Neurological Symptoms.—Symptoms of neurological disease, such as weakness, difficulty with speech, or diplopia, in addition to vertigo suggest a central cause.

Ask About Symptom Patterns.—Patients with vestibular neuronitis[24] (also called "labyrinthitis"), benign paroxysmal positional vertigo,[24] and recurrent vestibulopathy[25] (also called "benign recurrent vertigo"[26] and "vestibular Meniere's disease"[27]) have normal hearing. Patients with benign paroxysmal positional vertigo[24] (also called "benign paroxysmal positional nystagmus"[28] and "cupulolithiasis"[29]) have intermittent episodes of vertigo with head turning.[1,24] Vestibular neuronitis is characterized by a relatively sudden onset of severe, constant vertigo (made worse by head movement) that resolves after days or weeks.[24,30] Patients with recurrent vestibulopathy have intermittent episodes of constant vertigo lasting for minutes or hours.[25,26] Vertigo (with or without hearing loss) in a patient who has recently received aminoglycoside antibiotics may be due to the toxic effect these agents have on the vestibular labyrinth.[31]

How to Examine Patients With Vertigo

Findings on physical examination can help physicians detect abnormalities that can be used to determine the cause of vertigo.

Perform a Brief Neurological Examination.—Look for cranial nerve palsies, weakness, reflex changes, ataxia, decreased sensation in the feet, and abnormalities of gait and station. Vertical nystagmus is associated with lesions of the vestibular nuclei or of the cerebellar vermis.[14] Neurological findings other than pathological nystagmus suggest that the lesion is central.

Examine the Ears.—Briefly check hearing sensation with a mechanical wristwatch or tuning fork. Cholesteatoma, a complication of chronic otitis media that can present with hearing loss, drainage from the ear, and vertigo, may be found[32]; the usual treatment for this is surgery. Alternatively, vesicles associated with herpes zoster oticus (also called "Ramsay Hunt syndrome") may be present; patients with this condition often have facial palsy and deafness together with vertigo.[33]

Check for Spontaneous Nystagmus.—Patients with vestibular neuronitis usually have spontaneous horizontal nystagmus or a mixture of spontaneous horizontal nystagmus and rotatory nystagmus.[30] Patients with disorders of the central nervous system may also have spontaneous nystagmus.[34] In most of the patients examined by Silvoniemi,[30] Lachman and Stahle,[35] and Aantaa and Virolainen,[36] nystagmus was readily apparent, but in some, detection required Frenzel glasses or electronystagmographic monitoring with the patients' eyes closed. Patients with vestibular neuronitis may also have positional nystagmus.[30] Patient 1 had vestibular neuronitis.

Perform a Head-Hanging Maneuver.—Most physicians test for positional nystagmus with a method first outlined by Dix and Hallpike[24] and more recently by Mohr.[1] The head-hanging maneuver begins with the patient in a sitting position, with gaze fixed on the examiner's forehead (Figure). The examiner firmly grasps the patient's head and has the patient quickly lie supine, with the head turned about 30° to one side and about 30° below the level of the examining table. Next, the patient sits up, and the maneuver is repeated with the head turned to the opposite side. In 1979, Baloh et al[37] observed that if the maneuver was performed slowly (during a period of 20 seconds), nystagmus was not induced; thus, they recommended performing the position change in about 2 seconds. After each head-hanging maneuver, the physician should observe the patient's eyes for 5 to 15 seconds to determine whether nystagmus has been induced.[1] Overall, it takes about 3 to 5 minutes to explain the head-hanging maneuver to the patient, to perform the position changes, and to observe for nystagmus.

Benign paroxysmal positional vertigo is the most common cause of vertigo[8,9] and can usually be suspected on the basis of the history alone. Features of this syndrome include vertigo that occurs only with positional changes and an associated positional nystagmus that is usually rotatory, with a vertical or horizontal component. Also, the nystagmus usually begins 5 to 15 seconds after the head-hanging maneuver, lasts 2 to 30 seconds, and, if the patient is repeatedly returned to the provocative position, occurs less and less until it cannot be induced.[1,24] Positional nystagmus cannot always be elicited in a patient with a history otherwise compatible with the diagnosis of benign paroxysmal positional vertigo.[38-40] Its occurrence during a head-hanging maneuver occasionally makes a vague description of dizziness clearer. Rarely, patients with central nervous system lesions may present with positional vertigo and nystagmus and with no other neurological abnormality.[41] Patient 2 had benign paroxysmal positional vertigo.

Learning how to check for positional nystagmus usually requires practice. Always explain to the patient what you are

going to do before performing a head-hanging maneuver. Specifically, ask the patient to keep the eyes open if he or she becomes vertiginous; many patients close their eyes if vertigo develops. The head-hanging maneuver should be performed quickly but not so rapidly as to injure the patient. Be observant because the nystagmus may last only a few seconds.

ACCURACY OF THE SYMPTOMS AND SIGNS OF VERTIGO

Data are available on three clinically relevant questions about the accuracy of the clinical examination in patients with vertigo.

1. Can positional nystagmus identify patients with benign paroxysmal positional vertigo? The answer is "not very well." Only 198 of 255 patients with positional vertigo examined in a dizziness clinic had positional nystagmus during initial and subsequent examinations (sensitivity, 78%).[38] In an epidemiologic study of positional vertigo, only 13 of 26 patients tested had positional nystagmus (sensitivity, 50%).[40]

2. Can matutinal vertigo distinguish peripheral causes from central causes of vertigo? Again, the answer is "not very well." In a study of 100 neurology patients (48 of whom had matutinal vertigo), matutinal vertigo had a sensitivity of 51% and a specificity of 69% for peripheral disorders,[42] and in an epidemiologic study, symptoms of vertigo when rolling over in bed generated a sensitivity of 40% for benign paroxysmal positional vertigo.[40]

3. Can any set of symptoms and signs distinguish urgent causes from nonurgent causes of dizziness? Symptoms and signs can help identify patients in need of an urgent evaluation, as shown in Tables 2 and 3, which are from a study of 125 emergency department patients with the complaint of dizziness.[6] Patients who had the highly specific cluster of positive results on the head-hanging test and either vertigo or vomiting almost always had a nonurgent peripheral vertigo (a finding with high specificity, if positive, tends to rule in the target disorder). In Table 3, the high sensitivity (87%) of the absence of vertigo or age older than 69 years or the presence of a neurological deficit for a serious cause of dizziness meant that younger patients with vertigo but no neurological deficit were unlikely to have an urgent cause of dizziness (a finding with high sensitivity, if negative, tends to rule out the target disorder).

These reassuring results of the accuracy of the clinical examination come from a single study in an emergency department with rates of peripheral vertigo and serious disease characteristic of such settings; they need independent confirmation in different settings. Although the nonurgent causes of dizziness may not require immediate hospitalization, some of the causes of peripheral vertigo (eg, acoustic neuroma) deserve further diagnostic study.

THE BOTTOM LINE

The following are our recommendations on useful symptoms and signs in the evaluation of patients with dizziness:

1. In patients with suspected vertigo, ask whether they have dizziness when changing body position (rolling over in bed, looking up at the ceiling, or bending over to tie shoelaces) and perform a head-hanging maneuver to check for positional nystagmus.

2. In combination with other data (including a brief neurological examination) in an emergency department setting, the presence of positional nystagmus can be useful in identifying serious causes of dizziness.

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thiamine. However, if a fingerstick glucose test reveals no hypoglycemia, thiamine should be administered before glucose, if possible. In addition, patients with suspected thiamine deficiency should also receive concurrent magnesium as this acts as a cofactor for transketolase activity.[2]

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Clinical Trials Comparing Surgical vs Nonsurgical Therapy

To The Editor.—Dr Howard and colleagues[1] correctly emphasize that studies comparing surgical vs nonsurgical therapy pose distinctive statistical challenges because the complications of surgery usually occur early, whereas the complications of nonsurgical therapy generally occur at a constant rate over time. However, other inherent differences between surgical and nonsurgical therapies can invalidate comparisons between them despite the most careful statistical adjustments.[2]

Drug therapy is constant throughout the duration of a study since the chemical compound does not change, whereas surgical technique evolves continuously. The operation being used by the end of a study can differ markedly from the one with which the study began. Thus, increased experience with surgery improves results and decreases complication rates, whereas increased use of drugs usually unveils more complications. In one study, the mortality of coronary bypass surgery decreased from 3 (12%) of 25 patients in 1968 to 2 (1.5%) of 134 patients in 1973.[3] Furthermore, drug therapy in collaborative studies is standardized among all participants and is unrelated to physician skill, whereas the quality of surgical therapy varies unavoidably. Crossovers occur from medical to surgical therapy, but the reverse cannot occur. Drugs also usually have an indistinguishable placebo. Moreover, when surgery is an alternative, imperceptible bias often occurs before randomization. Physicians are likely to refer their sickest patients for the therapy they feel is best, while allowing lower-risk patients to be randomized.[4] This tendency probably explains why randomized studies that compare nonsurgical with surgical therapy frequently enroll low-risk subsets that usually fare better than historical controls in both arms of treatment.

For these reasons, it is often impossible to make enough statistical adjustments in studies that compare surgical with nonsurgical therapy. The implications are substantial because an increasing number of invasive procedures (comparable to surgical therapy) are being carried out by nonsurgeons, which leads to the possibility of more randomized studies that are unavoidably flawed.

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In Reply.—We agree that the comparison of medical vs surgical treatments is complex. However, Dr Bonchek's overall conclusion that "it is often impossible to make enough statistical adjustments in studies that compare surgical with nonsurgical therapy" seems to be suggesting that randomized clinical trials (RCTs) may be inappropriate to establish the relative efficacy in challenging clinical situations. We suggest that while RCTs of medical vs surgical trials present challenges, the objective comparison of treatments by RCTs remains central to the scientific advancement of medicine.

Many of the concerns raised by Bonchek to discount the value of RCTs may be overstated. For example, he suggests that improvement in surgical technique may imply a differential efficacy at the beginning and end of a study. We agree. However, any investigator would agree that it is likely inappropriate to conduct an RCT if the surgical procedure is rapidly evolving. Furthermore, most medical vs surgical clinical trials have an implicit or explicit 1-sided hypothesis in which surgery will not be performed unless it is clearly superior to medical management. For studies such as the Asymptomatic Carotid Atherosclerosis Study,[1] in which surgery was found superior, an improving efficacy of surgery will only strengthen the findings as surgery continues to improve. Bonchek's suggestion that drug therapy is standardized whereas surgery varies from surgeon to surgeon seems to show a lack of appreciation for variations in medical practice and patient compliance. His concerns regarding crossovers fail to acknowledge that any crossover will only serve to bias results toward the null hypothesis. As such, in RCTs that find a significant result, the relative superiority of a treatment is understated. That drugs have a placebo effect and that patients participating in clinical trials may not be completely generalizable are problems common to any RCT.

Although Bonchek raises some valid concerns, these concerns represent relatively minor issues when compared with the great benefit gained by objectively contrasting the relative merit of alternative treatments. While there are flaws in any study that should be reflected by the cautious interpretation of the results, the RCT remains the "gold standard" approach for contrasting alternative treatments. The careful planning of a study focuses on minimizing potential biases. However, even in the presence of these concerns, surely the scientific community is not ready to base important medical decisions on subjective clinical impressions.

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1. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA.* 1995;273:1421-1428.

CORRECTIONS

Incorrect Figure Key.—In the Special Communication entitled "Assessing Differences in Clinical Trials Comparing Surgical vs Nonsurgical Therapy: Using Common (Statistical) Sense" published in the November 5, 1997, issue of THE JOURNAL (1997;278:1432-1436), the key to Figure 2 was reversed. The dotted line should have indicated "Surgically Treated"; the solid line should have indicated "Medically Treated."

Incorrect Wording.—In The Rational Clinical Examination entitled "Does This Dizzy Patient Have a Serious Form of Vertigo?" published in the February 2, 1994, issue of THE JOURNAL (1994;271:385-388), the wording in a table was incorrect. On page 387, Table 3, the sentence that reads, "Presence of vertigo or age ≤69 y or no neurological deficit," should read, "Presence of vertigo and age ≤69 y and no neurological deficit."