DIAGNOSING DIZZINESS IN THE EMERGENCY DEPARTMENT

Why "What do you mean by 'dizzy'?" Should Not Be the First Question You Ask

by

David Edward Newman-Toker, M.D.

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Abstract

Dizziness is a complex neurologic symptom reflecting a perturbation of normal balance perception and spatial orientation. It is one of the most common symptoms encountered in general medical practice. Considering the dual impact of symptom-related morbidity (e.g., falls with hip fractures) and direct medical expenses for diagnosis and treatment, dizziness represents a major healthcare burden for society. However, perhaps the dearest price is paid by those individuals who are misdiagnosed, with devastating consequences.

Dizziness can be caused by numerous diseases, some of which are dangerous and manifest symptoms almost indistinguishable from benign causes. The risk appears highest among patients with new or severe symptoms, particularly those seeking medical attention in acute-care settings such as the emergency department. Nevertheless, even acute dizziness is more often caused by benign inner ear or cardiovascular disorders. Thus, a major challenge faced by frontline providers is to efficiently identify those patients at high risk of harboring a dangerous underlying disorder.

Unfortunately, diagnostic performance in the assessment of dizzy patients is poor. In part, this simply reflects the generally high rates of medical misdiagnosis encountered in frontline settings. However, misdiagnosis of dizziness is disproportionately frequent. Although possible explanations are myriad, I propose that an important cause stems from the pervasive use of an antiquated, oversimplified clinical heuristic to drive diagnostic reasoning in the assessment of dizzy patients. In this dissertation, I contend that the commonly-applied bedside rule that dizziness symptom quality, when grouped into one of four dizziness "types" (vertigo, presyncope, disequilibrium, or ill-defined dizziness),

predicts the underlying cause, is false and potentially misleading. The argument supporting this theory is developed in the chapters that follow.

Chapter 1 focuses on why dizziness diagnosis presents a significant challenge worthy of our concerted attention. Chapter 2 describes a multi-institutional survey of emergency physicians confirming that the "quality-of-symptoms" approach to dizziness is the dominant paradigm for diagnosis. Chapter 3 describes a cross-sectional study of emergency department dizzy patients demonstrating how this approach is fundamentally flawed. Chapter 4 concludes with a discussion of why this flawed paradigm might have garnered and maintained such widespread acceptance for over three decades.

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Dissertation Readers Serving on the Final Oral Examination Committee

David S. Zee, MD (*Mentor, Committee Chair, Non-Departmental Voting Member*)

Frederick L. Brancati, MD, MHS (*Advisor, Departmental Voting Member*)

Harold P. Lehmann, MD, PhD (*Non-Departmental Voting Member*)

Scott L. Zeger, PhD (*Departmental Voting Member*)

Richard E. Rothman, MD, PhD (*Optional Member*)

Preface

Publication of this dissertation marks the culmination of seven years of study and effort devoted to changing the way physicians approach the diagnostic assessment of dizzy patients. It also signals the start of a career dedicated to reducing misdiagnosis in frontline healthcare settings, using the tools of clinical investigation, medical informatics, and physician education. It is my belief that, by changing the way providers seek, solicit, and synthesize a patient's illness-related symptoms for the purpose of diagnosis, we can improve diagnostic accuracy, without sacrificing efficiency. Thus, it is my hope that this dissertation represents the first major milestone in a career-long journey intended to help bring science to the "art" of bedside diagnosis.

Even the inception of this journey would not have been possible without the substantial and ongoing support of others. I am deeply indebted to all of my mentors, collaborators, co-authors, employees, and funding agencies who have made possible the research described in this dissertation. Many of these important individuals are named below, although there are many more who have contributed than those I am able to list in these pages. I am also profoundly thankful for the knowledge and skills imparted to me by all of my former teachers, mentors, and role models throughout the various stages of my education and post-graduate career.

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Dedication

This dissertation is for my grandparents who remain with us in spirit (Fay Toker, Philip Toker, and Victor Monroe Harkavy). In particular, this work is dedicated to the loving memory of Victor ("Grandaw" as I knew him): grandfather, engineer, role model, true *mensch*, and wellspring of personal inspiration for me.

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- Eric B. Bass, MD, MPH
- David R. Cornblath, MD
- Steven N. Goodman, MD, MHS, PhD
- Gabor D. Kelen, MD
- Guohua Li, MD, DrPH
- Albert W. Wu, MD, MPH
- Scott L. Zeger, PhD

Co-Authors & Collaborators

Chapter 2

- Victoria Stanton, BA, MSII
- Yu-Hsiang Hsieh, PhD
- Carlos A. Camargo, Jr., MD, DrPH
- Jonathan A. Edlow, MD
- Paris Lovett, MD, MBA
- Joshua N. Goldstein, MD, PhD
- Stephanie Abbuhl, MD
- Michelle Lin, MD
- Arjun Chanmugam, MD, MBA
- Richard E. Rothman, MD, PhD

Chapter 3*

- Lisa M. Guardabascio, MD
- Matthew E. Stofferahn, BM, MSIV
- Richard E. Rothman, MD, PhD
- Yu-Hsiang Hsieh, PhD, MS
- David S. Zee, MD

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Elizabeth Elliott, ScD
Adjunct Assistant Professor of Epidemiology
Johns Hopkins Bloomberg School of Public Health

Adam Stubblefield, PhD
Assistant Research Professor of Computer Science
Johns Hopkins University

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Julie R. Newman-Toker, MD
Private practice, Baltimore, MD

Joshua M. Sharfstein, MD
Baltimore City Health Commissioner

Jakob Scherer
Diatouch, Inc.

Elisabeth Scherer
Diatouch, Inc.

** Research Assistants (in alphabetical order): Christopher Belknap, Allison Berken, Emmanuella Eastman, Katina Fox, Michael Green, Kareem Idrees, Julia Loux, Jessica Lozier, Maura McTague, Avantika Mishra, Jamia Saunders, Skylar Turner*

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Chapter 1

Why is Dizziness So Hard to Diagnose?

A Review from Biology to Bedside

Introduction

This introductory chapter is divided into three parts. Part I, "The Nature of Dizziness," describes terminology, physiology, and phenomenology of dizziness as a prelude to discussion of its clinical importance. Part II, "The Impact of Dizziness," describes the clinical significance and costs of dizziness, both as a symptom and as a marker of dangerous underlying disease. Part III, "Mis-Diagnosing the Dizzy Patient," outlines critical issues in diagnosis of dizzy patients, and offers possible explanations for the high rate of misdiagnosis seen in this patient population.

In my concluding remarks, I outline the theory I propose — that reliance on the standard bedside heuristic "dizziness symptom quality predicts etiology" (Figure 1.1) will place providers at significant risk for critical misdiagnosis in frontline healthcare settings.

Part I: The Nature of Dizziness

What is Dizziness? (The Terminology of Dizziness)

Dizziness is a complex neurologic symptom that reflects a perturbation of normal balance perception and spatial orientation. Traditionally, dizziness is categorized as one of four "types" based on symptom quality:[1] (1) *vertigo* (illusion of spinning or motion), (2) *presyncope* (feeling of impending faint), (3) *disequilibrium* (loss of balance or equilibrium when walking), and (4) *other ill-defined dizzy sensations* (lightheadedness,

wooziness, giddiness, etc.) sometimes known as *vague* or *nonspecific dizziness*. In considering the clinical terminology used to describe dizziness, it should be noted that precise definitions are not uniformly agreed upon, even among clinical experts whose sole focus is dizziness. Some important linguistic variations are considered below.

Dizziness is construed narrowly by some, and broadly by others. Some authors insist *dizziness* itself is not even a medical term, and recommend it be used only by laypeople.[2] Others cast a wide net around *dizziness*, including not only the four traditional categories, but anyone with generalized weakness or fatigue,[3] patients with syncope and falls,[4] or those with feelings of drowsiness,[5] unreality,[6] depersonalization,[7] or confusion.[8]

Vertigo (Type 1) is often thought of as the most clearly-defined type. However, otologists and neuro-otologists cannot agree on a precise meaning for vertigo — they are evenly divided whether it should describe any illusory sense of motion, or only a frank "spinning" or "turning" sensation.[9] Even those restricting "vertigo" to a spinning sensation may disagree. Some say it refers only to an external sense of the world spinning,[6] while others include as a subset those with spinning "inside the head." [10] This nuanced distinction is muddied further by use of the qualified terms "*objective* vertigo" and "*subjective* vertigo" to describe world-referenced and self-referenced motion, respectively.[11-14]

Presyncope (Type 2), strictly speaking, refers only to a feeling of impending faint or loss of consciousness.[1] However, it is common to find definitions for presyncope that reference other dizziness sensations. These definitions often include Type 4 sensations (e.g., "a feeling of lightheadedness,"[15] "an extreme form of lightheadedness"[16] or "the sensation of near-fainting (dizziness, lightheadedness, wooziness), without actual loss of

consciousness" [17]). Some authors extend the definition even further to encompass "vertigo," (Type 1) "unsteadiness," (Type 3) or "weak spells." [18]

Disequilibrium (Type 3) was originally defined as a "loss of balance without head sensation." [19] Some refer to disequilibrium as "imbalance," [20] "postural instability," [21] or (postural) "unsteadiness." [22, 23] More importantly, the category often no longer expressly excludes the co-morbid presence of "[dizzy-in-the] head sensations" [21, 24] that might fall into Types 1, 2, or 4.

Ill-defined dizzy sensations (Type 4) are sometimes referred to as "giddiness," [21] "non-specific dizziness," [25] or simply "other" dizziness.[24] Some remove "to-and-fro" or "rocking" sensations from this category and place them with vertigo (Type 1).[1, 20, 21] Many authors remove sensations of *lightheadedness* from this category and group them with presyncope (Type 2), considering them synonymous with a feeling of near faint.[7, 15, 24, 26-28] Along the same lines, some construe lightheaded sensations to represent milder symptoms in a continuum that extends from lightheadedness to presyncope to syncope,[16] or one of several possible symptoms experienced by a patient during a near faint.[17, 29] However, others maintain that lightheadedness is clearly distinct from presyncope.[1, 2, 12, 19, 30, 31] Some authors have even suggested lightheadedness may sometimes be a mild manifestation of vertigo (Type 1),[1] and, amidst all the confusion, others deliberately avoid using the term.[32, 33]

Despite the lack of consensus on terminology, it is standard practice to classify symptoms of dizziness according to the four-type schema described above. Although classifying dizzy sensations in this way is well accepted (*Chapter 2*), I contend that it may ultimately prove unhelpful in diagnosing the dizzy patient (*Chapter 3*), particularly

with respect to identifying dangerous disorders in frontline healthcare settings (*Chapter 4*). Anecdotally, the everyday clinical experience of both generalists and specialists confirms that the "language of dizziness" is nebulous and fraught with difficulty, both for the patient and for the physician. Clinicians are trained to inquire about symptom quality by asking "What do you mean by 'dizzy'?," [34] but those who frequently evaluate dizzy patients are not surprised by the typical reply, "I don't know, Doc, I'm just *dizzy*." In the pages that follow, I review the physiology and phenomenology of dizziness symptoms in preparation for the scientific arguments that follow. By the end of this dissertation, I hope to have offered the reader solid evidence in support this anecdotal assertion.

How Does Dizziness Happen? (The Physiology of Dizziness)

The Physiology of Dizziness — The Vestibular System

The neurobiology of the *vestibular system* (balance system) is complex, reflecting a dynamic, distributed network of combined sensory, motor, and integrative neural elements that work cohesively to serve three crucial bodily functions: (1) prevent falls, especially during locomotion, (2) stabilize vision when the head is in motion, and (3) adjust autonomic tone, especially blood pressure, to prevailing gravitational conditions (e.g., upright vs. recumbent posture).

It is arbitrary to draw fixed anatomic boundaries around a system as distributed and integrative as the one serving balance. However, in practice, "the vestibular system" is generally defined as the inner ear balance organs (semicircular canals and otolith

organs, located within the bony "labyrinth" [a] inside the skull base), its central connections (located principally within the brainstem and cerebellum), and the vestibular portion of the 8th cranial nerve[b] that connects these "peripheral" (inner ear) to "central" (brain) vestibular structures. Central structures, in turn, project to motor nuclei in the spinal cord to stabilize the trunk during walking, eye movement nuclei in the pons and midbrain to stabilize vision during head motion, and autonomic centers in the medulla to adjust blood pressure and other visceral responses. These central vestibular structures are located principally in the lateral zones of the middle and lower brainstem (lower pons and upper medulla) and inferior portion of the cerebellum, and are housed within the infratentorial intracranial space known as the *posterior fossa*.[c]

The Physiology of Dizziness — The Genesis of Vestibular Symptoms

In considering the link between the balance *system* and balance *symptoms*, we must remember that most day-to-day vestibular "sensations" (inputs) never rise above a subconscious level in humans.[36, 37] Although vestibular structures in the brainstem (e.g.,

[a] The bony *labyrinth* is, literally, a "maze" of tunnels and chambers within the petrous portion of the temporal bone of the skull base. This maze of tunnels and chambers houses fluid-filled, soft tissue ("membranous") sensory end organs serving hearing (cochlea) and balance (vestibular labyrinth, comprising semicircular canals and otolith organs). Together, the cochlea and vestibular labyrinth are colloquially known as "the inner ear." Although, technically-speaking, the cochlea lies within the anatomic confines of the bony labyrinth, the unmodified term *labyrinth* is often used in medical parlance to refer specifically to the *vestibular* labyrinth.

[b] The vestibulo-cochlear (8th) nerve is technically a "peripheral" nervous system structure, although it can be damaged by diseases typically considered "central" (e.g., multiple sclerosis).[35] On occasion, "peripheral" diseases affecting the 8th nerve are lumped together with diseases affecting "central" auditory or vestibular pathways,[29] subsumed under the heading of "retro-cochlear" or "retro-labyrinthine" (behind/beyond the inner ear) pathology.

[c] The *tentorium cerebelli* is a folded-over double layer (meningeal "reflection") of the hard, fibrous membrane surrounding the brain (dura mater). It physically separates the cerebral from cerebellar hemispheres, and segregates the intracranial spaces known as anterior/middle cranial fossae (housing the cerebrum), from the intracranial space known as the posterior fossa (housing the brainstem and cerebellum). **Supratentorial** – above the *tentorium cerebelli*. The cerebral hemispheres lie within the anterior and middle fossae, above the tentorium. **Infratentorial** – beneath the *tentorium cerebelli*. The brainstem and cerebellum lie within the posterior fossa, below the tentorium.

vestibular nuclei in the lateral medulla/pons) and cerebellum (e.g., flocculus, nodulus in the vestibulocerebellum) *do* send significant projections to the cerebral hemispheres,[38] the surface area of cerebral cortex devoted *exclusively* to balance perception is relatively small. These projections synapse heavily on areas of the brain (chiefly insular and parietal cortex) primarily involved in higher-order sensory and visuospatial integration. These areas of *heteromodal association cortex* bring together inputs from surrounding areas devoted to single sensory inputs (visual, somatosensory, auditory).

From a naturalistic perspective, we might posit the small cortical area devoted *exclusively* to vestibular sensation as a possible explanation for why we typically only "feel" (or notice) balance sensations when presented with exaggerated stimuli outside the system's normal dynamic range (e.g., amusement park rides).[36, 39] Regardless of whether this provides a sensible explanation (as opposed to mere mnemonic) for the system's quiet operation under *normal* conditions, it is absolutely clear that a *broken* vestibular system rises quickly to the level of conscious awareness.[d] It is believed that when the balance system is not working properly, vestibular signals do not match other sensory inputs (e.g., those from neck proprioceptors or vision), leading to the abnormal sensation of balance or spatial perception commonly described by patients as "dizziness." [40, 41]

In the medical setting, a percept of dizziness usually reflects a *pathologic* mismatch between vestibular and other sensory inputs.[40, 41] The mismatch often results from direct damage to the vestibular system by focal structural disease (e.g., vestibular neuritis) or specific physiologic insult (e.g., alcohol intoxication). However, dizziness

[d] As described later, the vestibular system produces the most dramatic symptoms when it is damaged asymmetrically (generally unilaterally, i.e., right only or left only) and rapidly (such that there is little opportunity for the nervous system to adapt).

may also reflect mismatch linked to dysfunction of a *different* sensory system, especially vision[42] (e.g., new spectacle correction; uncorrected irregular astigmatism or diplopia.[43])

The precise mechanism for dizziness in other pathophysiologic contexts remains obscure. For example, the dizziness associated with cardiac insufficiency, anxiety, or hyperventilation might result from secondary dysfunction of the vestibular system; alternatively, it might somehow relate to dysfunction of other sensory (or motor?) systems that bear on balance or spatial awareness. The dysfunction in these pathologic contexts might be more one of spatial "uncertainty" rather than "rivalry" (mismatch), similar to what is presumed to occur in patients with age-related loss[e] of input from multiple, interrelated sensory systems,[19, 47] sometimes known as "multisensory dizziness." [1] Independent of precise mechanism or site of action, it is presumed that, somehow, all forms of perceived dizziness ultimately reflect information failure at the level of cerebral cortex with regard to spatial orientation.[42]

Damage to the vestibular system can occur with diseases that affect either peripheral or central vestibular structures, and such damage tends to produce a constellation of symptoms and signs that reflect disruption of the three principal vestibular domains described (walking, eye stability, autonomic).[48] During walking or standing, there is typically a tendency to fall, and often a sense or impulse of falling, tilting, or turning. Vision is usually disrupted, with inappropriate bobbing or motion of the visual world (oscillopsia) during head movement, with head motionless, or both; corresponding intrusive eye movements (nystagmus) or impaired eye movement responses (abnormal vestibulo-ocular reflex [VOR]) are frequently-associated signs.

[e] Age-related *vestibular* loss (one component of so-called "multisensory dizziness") is also known as "disequilibrium of aging," and occasionally called *presbyastasis*[44, 45] or *presbylibrium*.[46]

Autonomic instability is a common accompaniment, often with nausea and/or vomiting, hypertension, reflex (vasovagal) hypotension, or blood pressure lability.

Why is Dizziness So Complex and Varied? (The Phenomenology of Dizziness)

The Phenomenology of Dizziness — The Variety of Vestibular Dizziness

Vestibular dizziness is highly variable, with symptoms ranging from severe, world-spinning vertigo to vague sensations of spatial disorientation (e.g., a floating sensation,[23, 49] "peculiar sensation in the head," [50] or "muddled brain" [51]) Intermediate symptoms include those of rocking, bouncing, swaying, tilting, falling, and the like.[20, 29, 36, 50, 52, 53] Three key principles link balance-system physiology to variation in clinical symptom phenomenology.

The first, and most important, of these principles is that asymmetries (e.g., right vs. left) in neural activity of vestibular sensory inputs are perceived by the brain as head movement.[36] At rest, there is a tonic level of symmetric neural activity coming from each inner ear balance organ. With head motion (e.g., rightward head turn), the balance organs respond asymmetrically (e.g., increase firing on the right, decrease on the left). When asymmetric firing occurs during such a head movement with an *intact* vestibular system, *the asymmetric firing is perceived by the brain as a normal head motion*. However, in the *pathologic state*, when asymmetric firing occurs solely as a result of vestibular system disease, *it is perceived as false (illusory[f]) head motion*, typically called *vertigo*.[36] Here,

[f] Although "vertigo" is generally referred to as an ***illusion*** of motion,[13, 54-56] it is probably more appropriately considered a ***hallucination*** when it occurs spontaneously (i.e., in the absence of head motion), since a hallucination is defined as "a sensory perception without external stimulation of the relevant sensory organ." [57] When the appropriate asymmetric firing that occurs during a real head movement is superimposed on a damaged vestibular system, asymmetric at baseline, it is perceived as inappropriate or distorted (illusory) head motion. Although the behavioral response to the transient spatial disorientation provoked by head movement with a damaged vestibular system is sometimes known as

with the head completely still, the vestibular signal (right-left asymmetry in firing, as if the head were turning), does not match input from neck proprioceptors, vision, or other sensory systems (e.g., absence of cutaneous sensation from stretching of the skin or changes in airflow over the skin's surface, normally encountered during head rotation).

The second principle is that the vestibular system responds primarily to *dynamic* change (acceleration), rather than *continuous* motion (velocity).[36] From a teleological perspective, this is presumably because the system's main purpose is to adjust trunk posture, eye position, and autonomic tone to unanticipated (i.e., changing) balance circumstances to prevent falls. Accordingly, all changes in firing rate are transient under normal conditions, and adaptive, central neural mechanisms mute any prolonged, consistent asymmetries in firing.[63] When considering the pathologic state from a symptom perspective, this means that (1) most dizziness is transient; (2) even patients with devastating, acute, unilateral loss of peripheral vestibular function only remain profoundly dizzy or vertiginous for a few days, until central, adaptive mechanisms adjust to the new, consistently asymmetric firing frequencies; and (3) people who remain dizzy for prolonged periods generally either have episodic diseases that produce physiologic dysfunction that fluctuates (which is not entirely amenable to central adaptation), or conditions affecting central vestibular structures themselves, that, therefore, impair adaptive mechanisms (e.g., cerebellar degeneration or brainstem stroke).

The third principle is that the vestibular system is highly subdivided, with each component serving a slightly different balance-related function. Linear accelerations

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vestibular space and motion sensitivity or similar term,[58] both illusory and hallucinatory percepts of motion are generally subsumed under the heading of "vertigo." A detailed discussion of the distinction between hallucinations and illusions is beyond the scope of this work, but has been considered elsewhere in the context of visual system dysfunction.[59-62] For the purposes of this discussion, we will apply the more commonly-used term, referring to "vertigo" as an *illusion* (rather than *hallucination*) of motion.

(head translations, e.g., riding in a car or elevator; and head tilts relative to the continuous linear acceleration of gravity) are sensed by the otolith organs (utricle and saccule), while angular accelerations (head rotations, e.g., turning your head to the side or tipping it backwards) are sensed by the semicircular canals.[64] Angular accelerations in the horizontal plane (e.g., turning your head to the right) are sensed primarily by two *horizontal* semicircular canals (e.g., right "on," left "off"), while those in the sagittal plane (e.g., pitching your head forward, as in tucking chin to chest) are sensed primarily by four *vertical* semicircular canals (e.g., right and left anterior canals "on," right and left posterior canals "off"). Thus, when individual components of the system fail, they produce different symptoms. For example, dysfunction of the otolith organs (or their central connections) tends to produce a sensation of falling, tilting, or disturbed perception of gravity, sometimes severe enough to feel as if one is being pushed over or thrown to the floor by a powerful force,[65] or as if the world has flipped on its side (90 degrees) or even upside down (180 degrees).[66-69] By contrast, dysfunction of the semicircular canals (or their central connections) tends to produce illusions of rotation in the plane of the affected semicircular canal,[36] along with a corresponding eye movement abnormality (nystagmus[g] or VOR failure) in the same plane.[70-73]

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[g] Nystagmus describes a rhythmic oscillation of the eyes. When caused by vestibular disease, the oscillation typically has a "fast phase" (also called "quick" phase) and a "slow phase," This type of nystagmus is known as "jerk" nystagmus because of the characteristic "jerking" of the eyes seen with each quick-phase movement. The nystagmus direction is named for the quick movement, because it is more visually obvious to the examiner than the slow movement. However, from a physiologic perspective, it is the slow-phase drift that reflects bias or asymmetry within the vestibular system; the quick phase is merely a "position reset" process designed to prevent the eyes from being displaced away from the straight-ahead (center) or otherwise desired position within the orbit. The word nystagmus is derived from a Greek word related to 'dozing off' or 'falling asleep' (New Latin, from Greek *nystagmos* drowsiness, from *nystazein* to doze http://www.britannica.com/dictionary?book=Dictionary&va=nystagmus&query=nystagmus). The association to the eye movement disorder is a visual metaphor from the head-nodding motion seen in people dozing ('nodding') off, with a slow downward drift of the head (neck flexion), and a fast upward jerk of the head (neck extension).

Bearing these principles in mind, it is perhaps no great surprise that vestibular symptoms will vary, depending on whether the disease process is (a) unilateral or bilateral, (b) acute or chronic, and (c) partial or total (Table 1.1).

The Phenomenology of Dizziness — The Issue of "Non-Vestibular" Dizziness

Naturally, a discussion of "vestibular" dizziness[5] begs a discussion of "non-vestibular" dizziness.[74] This, in turn, begs the question, "What do we mean by *'non-vestibular dizziness'*?," which is, in some sense, an oxymoron, given that vestibular forms of dizziness are not restricted to one particular dizziness type, and all "dizzy" sensations are ultimately believed to reflect information failure (whether due to mismatch or insufficiency) in cortical spatial perception.

One interpretation would be that "non-vestibular dizziness" refers to *vestibular-dizziness-like* symptoms reported by patients suffering from primary, non-vestibular diseases affecting other body systems (e.g., cardiac arrhythmia, hypoglycemia, panic disorder, etc.), whether or not such symptoms arise from secondary dysfunction of the vestibular system, *per se* (e.g., cardiac arrhythmia perhaps causing vertigo via cerebellar or labyrinthine ischemia[75]). An alternative interpretation would be to restrict use of the term "non-vestibular dizziness" to refer to symptoms deriving from dysfunction of neural systems *interacting with*, but not strictly *part of*, the anatomic vestibular system, as defined above — for instance, visual dizziness resulting from distorted or doubled vision (e.g., caused by an isolated eye muscle pathology[43]).

Unfortunately, the latter tack, though more firmly rooted in hard neuroscience, is not likely to prove helpful, scientifically or clinically. There is enormous visual-

vestibular[76] and somato-vestibular[77] interaction and adaptive changes that take place within the vestibular system in response to feedback from these other sensory systems,[78] so deciding where one sense "ends" and the other "begins" is generally an unanswerable, philosophical question. If, instead, we focus on the anatomic locus of original pathology in making this distinction, we devolve to the first explanation above, leaning heavily on the inciting etiology, rather than the precise nature of its downstream consequences for the vestibular (or other) system(s) in the brain (which, in most cases, remain unknown[h]).

The details of "vestibular" symptoms in such "non-vestibular" patients have only rarely been studied with any scientific rigor.[83, 84] There are isolated case reports of unexpected dizziness types as the dominant manifestation in non-vestibular disorders (e.g., vertigo in cardiovascular disease[75, 85]), and occasional case-series data for selected conditions (*see Chapter 3, Table 3.5*), but, more often, various dizzy sensations are lumped together or not described in detail when non-vestibular diseases are studied.[86-89] Accordingly, little can be said other than that the spectrum of dizziness symptoms among patients suffering from non-vestibular disorders appears to be roughly as broad as the spectrum of dizzy symptoms resulting from primary vestibular disorders.

[h] Precise pathomechanisms for dizziness are generally unknown for cardiovascular (e.g., reflex syncope, orthostatic hypotension, aortic stenosis, blood loss) and metabolic (e.g., hypoglycemia, anemia) causes. Toxic causes are generally better, though still incompletely, understood. For example, a fair amount is known about the pathogenesis of dizziness following exposure to systemic (or locally-applied) toxins that directly poison the peripheral vestibular apparatus (e.g., aminoglycoside antibiotics),[79] which might be thought of either as a "vestibular" or "non-vestibular" cause, depending on philosophical leanings. But, with the exception of alcohol,[40] relatively little, if anything, is understood about the pathomechanism of dizziness when it occurs as a result of most other toxic exposures affecting the central nervous system (e.g., carbon monoxide poisoning,[80] antiepileptic medications,[81, 82] etc.).

Part II: The Impact of Dizziness

Why Should We Care About Dizziness? (The Toll of Dizziness)

The Toll of Dizziness — Dizziness is Common and Costly

Dizziness is the chief complaint in 5% of walk-in-clinic visits,[90] and the third most common major medical symptom reported in general medical clinics.[91] In the acute-care setting, a chief complaint of dizziness accounts for a similar fraction (~4%) of Emergency Department (ED) visits.[92] Our research findings corroborate this *chief* complaint prevalence estimate, but suggest dizziness is even more common if one considers those with a *secondary* complaint of dizziness — a staggering 26% of all ED patients with other chief complaints state that dizziness is part of the reason for their visit.[93] These higher prevalence estimates match those reported by other authors who have systematically inquired about dizziness in unselected ED patients.[94]

Although dizziness is common at all ages, its prevalence rises slowly with age. Prevalence estimates among the elderly range as high as 61%[95] and even conservative, population-based figures suggest dizziness affects at least 10%[96] of those over age 65, with more typical estimates ranging from 20-35%.[15] Women are disproportionately affected at all ages.[6, 15, 96-98]

Dizziness, as a symptom, exacts its toll on individual patients through falls, fear, and loss of function. Dizziness doubles the risk of falls among those over 65-70 years of age.[99, 100] In the older age group, fall-related injuries frequently culminate in disability or death.[101] Dizziness is independently associated with an increased risk of hip fracture,[102] and increases the relative risk of a second fracture nearly three-fold.[103] It produces subjective functional impairment in 54% of patients and engenders the fear of serious

medical illness in 46%.[104] The symptom decreases health-related quality of life[105, 106] and functional capacity,[96] and leads to a secondary depression or anxiety disorder in 32%.[107] For society, dizziness is associated with substantial healthcare resource utilization in both primary[108] and acute-care[109] settings.

The Toll of Dizziness — Dizziness is Associated with Cerebrovascular Disease

Cerebrovascular disorders affect nearly a million Americans annually[110, 111] and include ischemic and hemorrhagic strokes, as well as transient ischemic attacks (TIAs). Most cerebrovascular events are ischemic strokes or TIAs,[112, 113] and these two disorders are most often responsible for dizziness as a cerebrovascular symptom.[50] Dizziness typically occurs when strokes or TIAs affect the brainstem or cerebellum,[114] within which the major central vestibular structures are located, although infarction of the inner ear may also occur.[115] The blood supply to these regions is from the vertebral and basilar arteries, which, together with their downstream branches, are commonly known as the *vertebrobasilar system* or *posterior circulation*.[i]

The most devastating cause of dizziness is stroke.[j] Stroke, which affects 700,000 Americans every year, is the third leading cause of death in the US and a leading cause of serious, long-term disability.[110] It consumes $50-60 billion in direct and indirect annual healthcare costs.[117, 118] The majority of strokes (90%) are ischemic, and posterior

[i] The *posterior circulation* comprises five large, named vessels — the paired *vertebral* arteries, which join to form the single, unpaired *basilar*, which then splits into paired *posterior cerebral* arteries — plus all of their medium and smaller branches. The posterior circulation is distinguished from the *anterior circulation*, which comprises six large, named vessels — the paired *internal carotid* arteries, which split into paired *anterior cerebral* and paired *middle cerebral* arteries — plus all of their medium and smaller branches.

[j] The term "stroke" is often used inclusively to embrace both ischemic and hemorrhagic strokes. In some cases, it is used as a shorthand substitute for "cerebrovascular disease" and incorporates TIAs as well.[116] In this discussion, after its initial introduction, we use the term "stroke" in its narrower conception, referring only to completed, ischemic stroke (i.e., brain infarction).

circulation strokes account for 28% of the these.[113] Dizziness is the most common posterior circulation ischemic symptom,[119, 120] occurring in about half of all cases,[50] and 70% of those with cerebellar involvement.[121] The only hemorrhagic stroke commonly associated with dizziness is cerebellar hemorrhage,[50] representing about 10% of intracerebral hemorrhages.[50, 113] It produces similar symptoms[122] to those seen in patients with ischemic stroke of the cerebellum,[50] but is more often (and more rapidly) lethal.[123]

TIAs are harbingers of ischemic stroke. Roughly 240,000 Americans suffer a transient ischemic attack (TIA) annually.[111] In the "brain attack" parlance of ischemic stroke, a TIA is the conceptual analog of angina pectoris for patients with incipient myocardial infarction.[124, 125] This "warning shot" carries with it a high risk of subsequent stroke, greatest in the days immediately following the TIA.[126, 127] The early risk of stroke may be highest after vertebrobasilar TIAs.[128] Roughly one in four cerebellar strokes is preceded by a TIA,[129, 130] and although the early risk for subsequent stroke is high, it has also been shown that *isolated* episodes of dizziness occurring repetitively for up to *two years* may be ischemic in etiology and portend eventual stroke.[114]

The Toll of Dizziness — Dizziness is an Under-recognized Manifestation of Stroke

Although there has been some improvement during "the decade of the brain," [131] public awareness of stroke risk factors[132] and warning symptoms that should prompt urgent medical attention[133] remains poor. Awareness is particularly lacking with respect to symptoms that resolve spontaneously. Knowledge about TIAs is inadequate in the general population, with fewer than 10% of Americans able to define what a TIA represents or identify a single TIA symptom.[134] Fewer than half of those with TIA

symptoms seek medical attention, and, even when they do, more than a third wait more than a day to do so.[134]

This lack of knowledge is unevenly distributed across symptoms, and worst for dizziness. Sudden-onset, focal neurologic symptoms that are highly specific for stroke,[135] such as unilateral motor weakness and speech disturbance,[117, 136] are more likely to be recognized by both patients[133, 137] and physicians[138] as manifestations of cerebrovascular disease. By contrast, non-specific symptoms such as dizziness, that may result from stroke,[116, 139] but are frequently caused by benign disorders,[135] are less likely to be correctly identified by patients[133, 137] or physicians.[116]

Unlike more "obvious" stroke symptoms such as motor and speech problems, dizziness does not seem to provoke a sense of urgency for patients. It is rarely cited as a reason for contacting Emergency Medical Services among those experiencing stroke-like symptoms.[140] While motor weakness and speech difficulties spur patients on to reach the hospital in a median time of 3 hours or less, dizzy patients take nearly twice as long to reach the hospital.[137] And, when patients do arrive in the ED, physicians are no less susceptible to the bias, misdiagnosing only about 4% of cerebrovascular patients with motor symptoms[138] compared to 35% of those with dizziness.[116]

Although there is nothing inherently wrong with focusing attention on symptoms more specific to stroke, it is crucial to recognize the potential loss of sensitivity for identifying cerebrovascular events in doing so. Only about 3-6% of dizziness is caused by cerebrovascular disease,[116, 141, 142] compared to about 80-90% for acute, hemi-motor symptoms.[135] However, because dizziness is 15-fold more common than motor

weakness,[143] it still represents a major manifestation of cerebrovascular disease in the general population.

What is the Link between Dizziness and Stroke? (Cerebrovascular Dizziness)

Cerebrovascular Dizziness — Vascular Supply to the Vestibular System

As mentioned previously, dizziness in cerebrovascular disease generally results from ischemia in the *posterior circulation* (vertebro-basilar territory),[50, 114] which supplies blood to both central and peripheral vestibular structures.[144] The vascular supply to the brainstem,[145] cerebellum,[146] and inner ear[147] is complex, but well characterized (Figures 1.2–1.4). The vertebral and basilar arteries are large trunks that deliver blood to the region, and give rise to two pairs of medium-sized arteries known as the PICAs (posterior inferior cerebellar arteries) and AICAs (anterior inferior cerebellar arteries), which nourish the central vestibular structures in the lateral brainstem and inferior cerebellum directly.[k] On either side, a smaller vessel, typically arising from the distal AICA,[149] feeds the inner ear and is known as the IAA (internal auditory artery). This vessel, in turn, splits into two small, end arteries supplying blood to the cochlea (cochlear artery)[150] and vestibular labyrinth (labyrinthine artery),[147] serving hearing and balance, respectively. Dizziness may therefore result from ischemia due to obstruction of flow in any of these posterior circulation vessels (vertebral, PICA, basilar, AICA, IAA, or

[k] The cerebellum also receives blood from a third pair of medium-sized vessels known as the SCAs (superior cerebellar arteries). The SCAs do not directly nourish the major central vestibular structures in most individuals (*see Figure 1.4*), but do supply blood to parts of the cerebellum that control balance and coordination during walking, reaching, and speaking. Thus, ischemia in this vascular territory tends to produce gait ataxia, limb ataxia/dysmetria, and dysarthria out of proportion to dizziness or vertigo.[148] Because of the typically prominent "neurologic" signs, SCA strokes usually present less of a diagnostic challenge to providers. Perhaps for the same reason, they are also less likely to be associated with major morbidity or mortality.[148] When obvious neurologic signs are absent, patients with SCA strokes or TIAs can look similar to patients with PICA-territory infarcts or ischemia. However, the diagnostic assessment is no different for SCA than PICA vascular events. Consequently, we will not dwell on SCA strokes further.

labyrinthine arteries), a state sometimes known generically as *vertebro-basilar insufficiency* (VBI).[115, 151]

Dizziness resulting from *anterior circulation* ischemia (i.e., internal carotid artery distribution) is thought to be much less common, and has been said to occur as an important or convincing vascular manifestation in only about 2% of cases.[50] Although anterior circulation stroke affecting supratentorial vestibular projections in the cerebral hemispheres (vestibular thalamus; insular, temporal, parietal cortex)[152] can, in theory, lead to dizziness, this appears to occur only rarely. Many patients with cerebrovascular lesions affecting the relevant hemispheric regions have subtle evidence of vestibular system disruption demonstrable in the laboratory,[153, 154] but few convincing cases have been reported in which dizziness or vertigo was a major clinical manifestation of stroke affecting these cerebral structures.[155, 156]

Nevertheless, anterior circulation disorders do cause dizziness, primarily through remote effects on the posterior circulation. In instances of certain rare congenital vascular variants, the posterior circulation actually derives its supply from the anterior circulation,[157, 158] and, in such cases, posterior circulation ischemia can *directly* result from anterior circulation disease.[159] Perhaps more importantly, however, vertebrobasilar ischemic symptoms can *indirectly* result from severe stenosis or occlusion of one or more large vessels in the anterior circulation (i.e., internal or common carotid artery),[160-162] apparently by "stealing" blood from the posterior circulation.[160, 163] The frequency with which this phenomenon, known as "steal VBI," occurs remains uncertain. Some authors have suggested vertebrobasilar symptoms may occur in roughly 10% of isolated carotid stenoses[164, 165] and surgical correction of the carotid lesion is "curative" more than 80%

of the time,[165] but these figures may be overestimates, given that large, randomized trials have shown no reduction in vertebrobasilar stroke rates following carotid endarterectomy, despite demonstrating reductions in *contralateral* hemispheric stroke.[166] Nevertheless, since research guidelines[120] and most studies of carotid artery stenosis do not consider dizziness a "symptomatic" manifestation of carotid disease,[167] nor vertebrobasilar strokes an outcome of interest,[168] the question of frequency remains unanswered.

Cerebrovascular Dizziness — Relation between Vascular Territory and Stroke Symptoms

The constellation of neurologic symptoms or signs that accompany dizziness during a stroke or TIA is, naturally, a function of which brain or inner ear regions are ischemic, and these, in turn, a function of the vascular territory[1] supplied by the affected vessel (Table 1.2). The ***brainstem*** is tightly packed with numerous "eloquent" structures serving major neural functions (e.g., eye/facial movements, limb strength, sensation), so even small brainstem strokes usually produce obvious (or, at least, easily demonstrable) clinical symptoms or signs. However, the ***cerebellum*** serves many functions that are more distributed and redundant (e.g., motor learning), so damage to large regions of the cerebellum may be associated with only unimpressive clinical findings.[170] With uncomplicated, unilateral, inferior cerebellar strokes (typical of those seen in distal PICA or AICA occlusions), the only clinical symptoms reliably present are vestibular in nature (dizziness, nausea, vomiting, and gait unsteadiness), and, importantly, classic cerebellar signs (e.g., limb ataxia) are frequently absent.[139] The ***inner ear*** serves both balance

[1] Standardized maps of the arterial territories of the brain are available elsewhere.[145, 169] It is important to note, however, that such maps represent average vascular distributions, and there is substantial inter-individual variation in actual blood supply, particularly in the posterior circulation (*see main text*).

(vestibular labyrinth) and hearing (cochlea) functions, and unilateral strokes here produce identical vestibular symptoms to uncomplicated, unilateral, inferior cerebellar strokes, except that labyrinthine[m] ischemia is typically accompanied by auditory symptoms.[149, 171]

The single **basilar artery** supplies blood to the upper two thirds of the brainstem, most of the thalamus, part of the medial temporal lobes, and most of the occipital lobes (the latter two via the posterior cerebral arteries). Accordingly, when basilar artery flow is significantly obstructed, ischemic symptoms are often profound, and may include nearly any combination of visual, cranial nerve, motor, sensory, autonomic, and cognitive symptoms.[172] Nevertheless, *isolated* dizziness is the initial complaint in roughly 20% of cases of basilar artery occlusion.[50] Both **AICAs** usually arise directly from the mid-basilar, and each typically sends a small, proximal branch to the lateral pons before feeding a large region of the ipsilateral inferior and middle cerebellum, before giving rise to the internal auditory artery on that side. Thus, both vestibular and cochlear symptoms (including bilateral ones[173, 174]) may result from basilar ischemia.[50, 149, 175] The paired **vertebral arteries** join together to form the unpaired basilar. Before this merger, however, each vertebral artery generally gives rise to a single, medium-sized cerebellar vessel, the **PICA**, which sends a small, proximal branch to the lateral medulla before feeding the bulk of the inferior cerebellum. The PICA territory is the only posterior circulation vascular distribution fed by only a single vertebral (making it uniquely susceptible to ischemia when one vertebral artery is occluded). So, when either the right

[m] Although, technically-speaking, the cochlea lies within the anatomic confines of the bony labyrinth, the unmodified term "labyrinth" is often used in medical parlance to refer specifically to the vestibular labyrinth. We use the terms "labyrinthine ischemia" and "labyrinthine infarction" to refer to TIAs and strokes involving the vestibular labyrinth, *whether or not* there is associated cochlear ischemia or infarction (i.e., *whether or not* there are associated auditory symptoms such as sudden hearing loss).

or left vertebral artery is occluded, non-vestibular neurologic symptoms are usually[n] far less dramatic than with a basilar occlusion. Dysfunction is usually restricted to the lateral medulla and inferior cerebellum on the affected side — since a single, normal vertebral artery on the unaffected side is generally sufficient to maintain blood flow to the basilar artery territory.

When ischemia occurs "downstream" in one of the medium-sized (distal AICA, distal PICA) or smaller (IAA, labyrinthine artery) vessels, symptoms are often deceptively "non-neurologic" in nature, mimicking those seen in patients with benign diseases of the peripheral vestibular system. The **distal PICA** (after the take-off of the branch to the lateral medulla) supplies *only* the inferior cerebellum. Thus, distal PICA ischemic symptoms (as seen with embolic or local occlusion) mimic those seen in patients with benign vestibular neuritis (dizziness, nausea, vomiting, and gait unsteadiness), resulting in a clinical syndrome now known as "vestibular pseudo-neuritis." [176] The **distal AICA** (after the take-off of the branch to the lateral pons) supplies *only* the inferior/middle cerebellum and inner ear. Thus, distal AICA or internal auditory artery ischemic symptoms mimic those seen in patients with benign labyrinthitis, differing only from vestibular neuritis and PICA ischemia (pseudo-neuritis) in the co-morbid presence of auditory symptoms (pseudo-labyrinthitis). The **labyrinthine artery** supplies *only* the vestibular labyrinth, and may mimic vestibular neuritis or distal PICA infarction (pseudo-neuritis) precisely.

[n] Vertebral artery occlusions usually produce dominantly (or exclusively) vestibular symptoms, without impressive co-morbid neurologic features. The exception to this rule occurs when contralateral vertebral artery flow is limited (e.g., prior occlusion, congenitally-small contralateral vertebral), or when an embolus from vertebral to basilar artery causes secondary basilar-territory ischemia.

In this discussion of vascular supply to the vestibular system, it should be noted that there is considerable inter-individual variability in the vascular anatomy of the posterior cerebral circulation. This anatomic variation is described in detail elsewhere.[177-186] Aside from those unusual cases in which posterior circulation ischemia results directly from anterior circulation disease (superimposed on rare congenital vascular variants),[159] this anatomic variability is most clinically relevant with respect to the vascular supply of the cochlea, and its relationship to the localizing value of auditory symptoms in patients with a primary complaint of dizziness.

Auditory symptoms (e.g., hearing loss, tinnitus) do not result from isolated cerebellar strokes, since the auditory pathways do not traverse the cerebellum.[187] Such symptoms only rarely result from brainstem or cerebral ischemia, due to redundancy of both vascular supply and innervation of central auditory stuctures.[187] As a result, their presence points to a peripheral disease localization (cochlea or cochlear division of the 8th nerve) and generally implies either IAA ischemia *or a non-ischemic etiology*. Auditory symptoms may, therefore, be (mistakenly) thought of as an indicator of less serious underlying pathology, on the grounds that ischemia in a small, distal vessel such as the IAA represents a "mild" form of stroke, and non-ischemic causes such as viral labyrinthitis are generally benign and self-limited. However, since the IAA can arise directly from the basilar trunk in 15-20% of individuals,[187] mixed auditory and vestibular symptoms may be a harbinger of basilar artery occlusion.[175, 188] Furthermore, the IAA may arise from the PICA in about 2-4% of individuals,[149, 187] which could explain the occasional association between mixed audio-vestibular symptoms and unilateral vertebral occlusion,[189, 190] which, more typically, causes isolated vestibular symptoms.[191] Since

large-vessel posterior circulation occlusions are often associated with significant morbidity or mortality,[192] considerable care should be taken in assigning a benign prognosis to those acutely dizzy patients with co-morbid auditory symptoms.

Part III: Mis-Diagnosing the Dizzy Patient

Is Diagnosing the Dizzy Patient Hard? (The Difficulty Diagnosing Dizziness)

Difficulty Diagnosing Dizziness — Dizziness Presents a Diagnostic Challenge

Some authorities consider dizziness the most difficult symptom to diagnose.[15] It may be caused by many diseases, some of which, if not diagnosed rapidly and treated emergently, can be disabling or fatal. For example, ischemic stroke of the cerebellum carries a significant risk of mortality due to secondary brainstem compression,[193] and cerebellar hemorrhage is often rapidly fatal without urgent neurosurgical decompression of the cranial vault.[123]

Although some authors have downplayed the association between dizziness and stroke, claiming the majority of cases of cerebrovascular dizziness are accompanied by other, more obvious, neurologic symptoms or signs,[12, 28] it has been shown that approximately 20% of basilar occlusion patients[50] and 10% of cerebellar strokes[139] initially manifest ***only*** dizziness or vertigo.

While it is true that the most common causes of dizziness in a general medical population are benign in nature and relate to conditions of the inner ear,[142] dangerous diseases such as cerebellar TIA or stroke,[114, 139, 194] cardiac dysrhythmia,[75, 195] or acute hypoglycemia[196, 197] can produce similar (or even identical) symptoms. In the outpatient setting, fewer than one in ten cases is attributed to a serious cause such as cerebrovascular event (6%) or cardiac dysrhythmia (1.5%).[142] However, the risk is probably much higher

in acute-care settings such as the ED. Serious causes are identified in about 34% of unselected ED dizzy patients.[141] It is in this clinical setting, therefore, that accurate medical diagnosis is essential.

Despite the overall high risk of dangerous diseases in acutely dizzy patients,[141] it is important to note that some causes probably remain a relative "needle in a haystack," even in the ED (e.g., stroke/TIA, accounting for about 3-6%[116, 141]). An extensive battery of laboratory and imaging tests might suffice to exclude dangerous diseases in most cases, but this approach is neither practical nor efficient, given that dizziness affects nearly one of every three ED patients.[93]

Neither blood tests (e.g., cell counts, electrolytes, glucose), nor imaging studies (e.g., CT head, MRI brain[o]), are cost-effective when applied indiscriminately to the evaluation of unselected dizzy patients.[198-200] Bedside evaluation emphasizing detailed history-taking and specialized physical exam techniques is thought to be the best means to identify those in urgent need of additional testing.[7, 198, 201] However, no prospective studies exist to document the success of this strategy.[15, 198, 202] Although the traditional bedside approach to evaluating dizzy patients, relying heavily on dizziness "type" (determined by symptom quality) to inform subsequent diagnostic inquiry,[1] was described more than 30 years ago,[19] it has never been rigorously validated.

Difficulty Diagnosing Dizziness — Dizziness is Frequently Misdiagnosed

Despite its high prevalence (or perhaps *because* of it), dizziness appears to be frequently misdiagnosed. Although data on overall misdiagnosis rates in unselected dizzy patients are lacking, disease-specific studies indicate diagnostic performance is poor. It is

[o] CT (computed tomography); MRI (magnetic resonance imaging)

estimated that 9% of elderly adults in the community have undiagnosed benign paroxysmal positioning vertigo (BPPV),[95] and that such patients frequently go undiagnosed, untreated, and un-referred for more than a year after first contact with their primary providers, despite a typical clinical presentation in most cases.[203] Although adverse outcomes (e.g., falls, hip fractures, etc.) may result from failure to promptly diagnose and treat even "benign" diseases such as BPPV, the major clinical impact of misdiagnosis occurs when life-threatening causes of dizziness are mistaken for benign disorders ("critical" misdiagnoses), and this appears to happen most often with cerebrovascular events (stroke and TIA).

Dizziness is the symptom most often associated with a missed diagnosis of ischemic stroke in the ED,[204] and it is estimated that 35% of cerebrovascular events in patients with dizziness go undiagnosed by ED physicians.[116] However, even this large figure may be a conservative estimate, since few patients in the cited study underwent MRI, most were never seen by a neurologist, and patients with isolated dizziness, discharged from the ED with a benign (non-stroke) diagnosis, were never actively followed up for the possibility of stroke or TIA. Such mis-*triaged* (i.e., inappropriately discharged) strokes among dizzy patients are known to occur, if only through press coverage when prominent public figures have been recipients of inadequate care[205] or when large jury settlements have been awarded for resulting adverse outcomes.[206] Although not yet systematically studied, it is likely that mis-triaged strokes among ED dizzy patients are fairly common, given the high frequency of missed strokes among *admitted* patients,[116] and the overall high risk of inappropriate discharge from the ED.[207]

Why is Dizziness Misdiagnosed? (The Pitfalls of Dizziness)

There are numerous possible explanations for the poor diagnostic performance seen in diagnosing dizzy patients, particularly when considering diagnosis in an acute-care setting. First, there are human factors that result in frequent misdiagnoses across healthcare settings. Second, there are healthcare-delivery system factors that place ED physicians at especially high risk for misdiagnosis. Third, there are symptom factors unique to dizzy patients that increase their risk being misdiagnosed, especially in the ED.

Pitfalls of Dizziness — Dizziness-Independent (Human & Delivery-System) Factors

Diagnostic errors are rampant. Conservative estimates suggest that at least 40,000-80,000 deaths result from misdiagnosis annually in the US,[208] but this figure is probably on the low side. Diagnostic errors often go unrecognized, or are recognized but not reported.[209-213] The ED is a hot spot for misdiagnosis. More than half of all hospital-associated adverse events deemed negligent occur in the ED.[214] The majority of these adverse events relate to inappropriate discharge, with half of those released having, in retrospect, met criteria for admission.[207] Although, elsewhere in the hospital, treatment errors are more prevalent, in the ED, errors in diagnosis probably represent the majority of errors[215, 216] with many cases involving serious injury or death.[217]

Research on safety from high-stakes industries (e.g., aviation, nuclear power plants) indicates that most errors ultimately derive from flaws in systems operations.[218] This is likely to hold true in medicine,[218] ***even when it comes to misdiagnosis***, where an individual physician seems inherently to blame.[210] Whether they result from limitations and biases in human decision-making capacity, failure to communicate on clinical teams, or shortcomings in medical education or dissemination of medical evidence, most

diagnostic errors ultimately relate to the human cognitive process.[210, 211, 219] In the ED, systems factors contributing to cognitive errors include, among others, the broad spectrum of complaints managed by ED physicians, lack of a pre-existing doctor-patient relationship with most patients, enormous variability in illness severity from patient to patient, huge fluctuation in patient volume from hour to hour, understaffing, intense time pressures, and an often chaotic, "interrupt-driven" work environment.[220-222]

Pitfalls of Dizziness — Dizziness-Dependent (Symptom-Specific) Factors

Important dizziness-independent factors notwithstanding, the problem cannot exclusively be a function of human cognitive limitations and the hectic ED environment, since frontline providers (both inpatient and outpatient) are more likely to misdiagnose *neurologic* problems than *general medical* ones. While only 2% of myocardial infarction patients[223] are misdiagnosed at first contact, 20% of awake subarachnoid hemorrhage patients[224] and 24% of stroke and TIA patients[204] are misdiagnosed at first contact. In a study examining the causes of 49 preventable deaths in 12 hospitals, most due to myocardial infarction reflected errors in management, while most due to cerebrovascular events reflected errors in diagnosis.[225]

In one study, only 26% of tentative neurologic diagnoses by ED physicians were considered correct and complete on review by a neurologist, and the neurologist completely changed the diagnosis in 52%.[204] However, in practice, neurologists are seldom involved in acute cerebrovascular care in the ED, with only 10% of stroke patients and 4% of TIA patients receiving a neurologic consultation.[226] Whether this occurs because neurologists are unavailable for consultation or because self-confident ED

physicians elect not to solicit their input remains an open question. However, it is clear that confidence alone affords no protection against medical misdiagnosis.[227]

However, evidence suggests there is more to misdiagnosis of dizzy patients than general shortfalls in neurologic diagnosis. Dizziness appears to be the neurologic symptom most likely to generate diagnostic confusion, at least with respect to misdiagnosis of cerebrovascular disease. When compared to active, on-site diagnosis by a neurologist, 24% of all cerebrovascular events are missed by ED physicians, and 22% of these misdiagnoses (representing the plurality) occur in dizzy patients.[204] Population-based estimates drawn from a single geographic region suggest that physicians misdiagnose only about 4% of cerebrovascular events in patients with motor manifestations[138] compared to 35% of those with dizziness.[116]

There are a number of symptom-specific factors that may increase the risk of misdiagnosis in dizzy patients. These include (1) high symptom prevalence coupled with the benign nature of underlying causes in most, (2) breadth and complexity of the etiologic differential diagnosis, (3) dearth of information about the prevalence of various *uncommon* causes in frontline healthcare settings, (4) inability of patients to clearly describe their dizziness symptoms, (5) high rate of misconceptions among providers about bedside assessment, (6) under-appreciated subtleties of clinical history and physical examination techniques, and (7) lack of sensitivity and specificity of commonly-applied laboratory and imaging tests for most causes of dizziness.

(1) **Common Symptom, Commonly Benign**: The fact that dizziness is part of the reason for 30% of all ED visits,[93] yet most cases are likely benign in etiology,[3, 141, 228] presents a signal-to-noise detection problem for acute-care providers.

(2) **Breadth of Differential Diagnosis**: Possible etiologies for dizziness in the ED are perhaps even more numerous than in general medical care settings — in one study, 46 different diagnoses were given to 106 dizzy patients.[3] This wide spectrum of causes makes bedside assessment of dizziness one of the most challenging tasks a frontline provider must face.[15]

(3) **Lack of Prevalence Data**: Robust estimates of the spectrum of likely diagnoses among ED dizzy patients are lacking, with only three previous English-language studies[142] describing unselected ED dizzy patients (total n=352).[3, 141, 228] Furthermore, providers may be confused by available prevalence estimates for critical diagnoses that vary widely — for cerebrovascular disease, the range is <1%[3] to 25%,[194] depending largely on study inclusion criteria. A recent population-based study of stroke provides the best current estimate, attributing 3.2% of ED dizziness presentations (n=1666) to a cerebrovascular cause over a 3-year period in an isolated, rural community.[116] No population-based data have been published for other causes of dizziness in the ED, but preliminary results from analyses we have conducted on the CDC's National Hospital Ambulatory Medical Care Survey (NHAMCS) data set suggest that the spectrum of dizziness causes is broad, and more heavily weighted towards acute general medical conditions than previously imagined (*Appendix 1.1*).

(4) **Trouble Describing Dizzy Symptoms**: When offered standard options to describe dizziness, general practice patients are unable to characterize their symptoms 7% of the time,[30] and older patients (among whom dizziness is most prevalent) report symptoms in two or more of the four dizziness categories more than half the time.[33, 229] This latter problem has forced some clinical investigators to develop a hierarchy for

classifying the dizziness complaint in an effort to reduce category overlap. Unfortunately, different investigators have assigned the highest priority to different dizziness types (e.g., vertigo[30] takes priority vs. disequilibrium[116] takes priority). As we shall see in Chapter 3, the difficulty describing dizziness symptom quality is not restricted to ambulatory-care settings or elderly patients, and represents one of the core problems in using symptom quality to inform subsequent diagnostic inquiry.

(5) **Misconceptions about Assessment**: Dangerous diseases can present with dizzy symptoms difficult to distinguish from more common, benign causes. Patients with dizziness as a (sole or dominant) manifestation of TIA or stroke may be especially prone to misdiagnosis due to the absence of lateralizing weakness, a finding often viewed as the hallmark of cerebrovascular events, with its absence mistakenly thought to exclude the diagnosis.[138] In support of this contention is a finding from a study comparing ED referring diagnoses to neurology consultant diagnoses. Among patients confirmed on consultation to have stroke, a cerebrovascular diagnosis was <u>not</u> entertained by the referring ED physician in 29% of those with *posterior circulation* stroke (who frequently have dizziness, but often do not have hemiparesis) compared to 12% of those with *anterior circulation* stroke (who rarely have dizziness, but typically do have hemiparesis).[230] In general, misconceptions about the assessment of dizzy patients among frontline providers appear to be frequent,[231] and, in part, may reflect misinformation presented in textbooks and other medical literature (*Appendix 1.2*).

(6) **Subtleties of Physical Diagnosis**: Making matters worse, even if entertained as a diagnostic possibility, acute strokes may mimic medically-benign vestibular neuritis or labyrinthitis, in all clinical aspects down to nuances of the bedside neuro-otological

examination.[139, 176, 201, 232, 233] The techniques used by specialists to distinguish stroke from vestibular neuritis or labyrinthitis require skill in detailed bedside assessment of eye movements (analyzing nystagmus,[176, 201] demonstrating a head thrust sign,[176, 234] and identifying skew deviation[176]) — skills unfamiliar to many frontline providers.[231]

(7) **Lab and Imaging Studies Ineffective**: Neither blood tests (e.g., cell counts, electrolytes, glucose), nor imaging studies (e.g., CT head or MRI brain), are cost-effective when applied indiscriminately to the evaluation of dizzy patients.[198-200] CT scans of the head, which are readily available in most EDs (and frequently used[200]), are generally insensitive for identifying acute ischemic strokes relative to MRI (61% vs. 91%).[235] CT sensitivity for brainstem and cerebellar infarcts, in particular, is even lower (≤40%[p]).[236, 237, 239] Posterior fossa imaging of the brain by CT is hindered by radiographic artifacts created by the dense bone of the skull base.[240] This phenomenon is worst for the inferior portion of the cerebellum[239] — the region where strokes are most likely to cause dizziness. The false sense of reassurance provided by a normal head CT may have deadly consequences for dizzy patients.[241] However, even modern MRI with diffusion-weighted imaging (DWI) can miss acute strokes,[235] and this appears to occur more frequently in patients with brainstem strokes,[242, 243] including those with strokes in the lateral medulla,[234, 244] who generally present to the ED with acute dizziness, nausea, and vomiting. Furthermore, transient ischemic attacks are associated with radiographic

[p] 40% percent (derived from the three cited studies, total n=22/55) is probably an overestimate for the sensitivity of CT in the assessment of the acutely dizzy patient. First, most of the CT scans in these studies were obtained days or even weeks after the initial symptoms.[236, 237] By contrast, most ED CT scans likely occur within hours of symptom onset, and the sensitivity of CT is known to be lower for detecting strokes in the first 48 hours than later.[238] Furthermore, the sensitivity of CT is lowest for the inferior portion of the cerebellum[239] (i.e., the vestibular portion, where strokes produce dominantly or exclusively dizzy symptoms). The cited studies included superior cerebellar and even posterior circulation cerebral[236] infarcts, which are more easily recognized by CT. Therefore, for acutely dizzy patients imaged within the first 48 hours, 40% sensitivity is likely to be a substantial overestimate.

stroke-like changes less than half the time.[245] Thus, no imaging study alone provides absolute protection against a missed diagnosis of a posterior circulation cerebrovascular event in the assessment of an acutely dizzy patient.

Although each of these possible explanations probably plays a role in the genesis of diagnostic errors among acutely dizzy patients, I theorize that the most important explanation may stem, instead, from the pervasive use of an oversimplified, inaccurate (or inappropriately applied) clinical heuristic for diagnosis. The traditional approach to diagnostic assessment of the dizzy patient relies heavily on dizziness symptom quality to inform diagnostic inquiry by associating dizziness types with specific underlying causes. I have hypothesized that this approach is in widespread clinical use, predisposes to misconceptions, is fundamentally flawed, and could, therefore, be linked to misdiagnosis. The Chapters that follow describe the work we have done to test these hypotheses.

Conclusion

Dizziness is an important, common symptom, and critical misdiagnoses are probably frequent. Although dizziness-independent factors likely contribute to misdiagnosis of dizzy patients, dizziness-dependent factors clearly play a role, and must be addressed in pursuit of accurate diagnosis for these patients. As part of a long-range program to improve diagnosis of acutely dizzy patients, we have recently conducted two pivotal studies focused on diagnosing dizziness in the ED:

Diagnosing Dizziness in the Emergency Department — Do Physicians Rely Too Heavily on Symptom Quality? ***Results of a Multicenter, Quantitative Survey*****.** Chapter 2 describes a multi-institutional survey of roughly 400 emergency physicians

regarding diagnosis of dizziness in the ED. This study reports the heavy emphasis providers place on symptom quality in diagnosing dizzy patients, to the relative exclusion of other dizziness symptom dimensions (e.g., timing, triggers) and associated symptoms (e.g., pain). It highlights related misconceptions in diagnostic reasoning, and the potential for resulting misdiagnosis.

Rethinking the Approach to the Dizzy Patient — Patient Reports of Symptom Quality are Imprecise. ***A Cross-Sectional Study Conducted in an Acute-Care Setting*****.** Chapter 3 presents a cross-sectional study conducted in a consecutive sample of ED dizzy patients, with data gathered through systematic screening and assessment of all ED patients over a one-month period at each of two university hospitals. This study reports detailed symptom descriptions in over 300 ED dizzy patients. It emphasizes measurement precision of patient-reported symptom dimensions for different historical features of dizziness (quality, timing, and triggers). It demonstrates the lack of clarity, consistency, and reliability of dizziness symptom quality, relative to timing and triggers. These findings indicate that the current "quality-of-symptoms" approach to dizziness diagnosis is misguided at its core, and a new approach is needed.

Taken together, these studies suggest that over-reliance on a flawed heuristic in the ED might predispose to dangerous misdiagnosis. They provide a solid foundation for subsequent research intended to develop and validate new strategies for accurately diagnosing acutely dizzy patients — most importantly, for reducing critical misdiagnoses.

In the final Chapter (**Towards a New Approach to Diagnosing Dizziness in Frontline Healthcare Settings.** ***Insights from Past to Present***), I describe the historical context in which the traditional paradigm arose, highlighting critical pitfalls to be avoided

in the development of a new diagnostic model. I conclude by outlining a new approach to diagnosis, and possible strategies by which this approach might be developed, validated, and implemented in the ED.

Table 1.1 Dizziness and balance symptoms vary by disease symmetry and onset

The table below illustrates some examples of how dizziness and balance symptoms vary with different clinical conditions, based on differences in disease asymmetry and rapidity of onset. The more *asymmetric* the pathology, the more likely there is to be severe dizziness that has a rotational or spinning component (at least in the acute phase of the illness). The more *acute* the pathology, the more likely there is to be disruption of gait, vision, and autonomic function.

Not shown are differences based on partial vs. total involvement of vestibular structures by disease. For example, both vertigo (symptom) and nystagmus (corresponding sign) are predominantly horizontal (axial) in orientation if the entire labyrinth is damaged on one side, as in labyrinthitis. However, both symptom and sign are mixed vertical (sagittal) and torsional (coronal) in orientation if a single posterior semicircular canal is involved, as in BPPV.

Table 1.1 Abbreviations and footnotes

VOR – vestibulo-ocular reflex (as tested at the bedside by the "head impulse test" [246])

* If rapid-onset vestibular dysfunction is transient, the disruption may produce only a partial clinical picture. For instance, with BPPV, the diseased vestibular stimulus is typically so brief (<40 seconds) that vomiting is rare (unlike acute vestibular neuritis, which lasts for days, where vomiting is the rule, rather than the exception).

† The issue from the brain's perspective is generally one of vestibular "asymmetry" rather than "unilaterality" or "bilaterality," *per se*. Unilateral disease generally results in asymmetry, but asymmetry could result from disease that is bilateral, but *unequal* (right vs. left). Alternatively, asymmetry could be produced by bilateral disease that created a front-to-back or top-to-bottom asymmetry (rather than right-to-left asymmetry), since the vestibular system operates on "balance" in three-dimensional space. An example of this is alcohol intoxication, which creates top-to-bottom (rather than right-to-left) asymmetry through gravity-dependent differential effects on the density of endolymph.[40]

Table 1.1 Dizziness and balance symptoms vary by disease symmetry and onset

	Acute/Rapid-Onset*	**Chronic/Insidious Onset**
Unilateral (asymmetric†)	*Examples*: - vestibular neuritis - acute medullary/cerebellar stroke *Typical Symptoms*: - continuous, severe dizziness (often *with* sense of motion or spinning), exacerbated by head movement - severe, spontaneous oscillopsia, worse horizontally than vertically - severe nausea *Typical Signs*: - severe gait unsteadiness - spontaneous nystagmus - abnormal VOR, unilateral - vomiting, blood pressure lability	*Examples*: - recovery post vestibular neuritis - vestibular schwannoma *Typical Symptoms*: - mild dizziness (usu. *without* rotary motion or spinning), brought on or exacerbated by head movement - mild oscillopsia with head motion, worse horizontally than vertically - +/- mild nausea *Typical Signs*: - mild gait unsteadiness - inducible nystagmus - abnormal VOR, unilateral - no vomiting or autonomic instability
Bilateral (symmetric†)	*Examples*: - acquired vestibular failure (aminoglycoside toxicity) *Typical Symptoms*: - moderate dizziness (often *without* sense of motion or spinning), *not* exacerbated by head movement - severe oscillopsia with head motion, worse vertically than horizontally - +/- nausea *Typical Signs*: - mild to moderate gait unsteadiness - no nystagmus - abnormal VOR, bilateral - +/- vomiting or autonomic instability	*Examples*: - hereditary bilateral vestibular loss - age-related vestibular loss *Typical Symptoms*: - mild dizziness (often *without* sense of motion or spinning), *not* exacerbated by head movement - severe oscillopsia with head motion, worse vertically than horizontally - no nausea *Typical Signs*: - gait unsteadiness only in darkness - no nystagmus - abnormal VOR, bilateral - no vomiting or autonomic instability

Table 1.2 Relationship between brain region, vascular territory, and neurologic symptoms and signs that may accompany dizziness during a stroke or TIA

Brain regions are "stacked" in order, with caudal (inferior) regions at the bottom of the table and rostral (superior) ones at the top (lower brainstem/cerebellum; middle brainstem/cerebellum; upper brainstem/cerebellum and inferior cerebrum).

Blue shading indicates stroke symptoms or signs that are typically obvious (e.g., hemiplegia) and, thus, represent cases where stroke diagnosis is usually self-evident. Yellow shading indicates stroke symptoms or signs that are usually clear (e.g., visual field cut, confusion, facial palsy, hoarseness) but could be mistakenly attributed to benign illness (e.g., migraine, intoxication, viral ear infection, viral laryngitis); note, some of the "yellow" signs are asymptomatic findings that are usually identified only if specifically sought (e.g., Horner syndrome). Pink shading indicates stroke symptoms or signs that are subtle and closely mimic those seen with benign conditions of the inner ear (e.g., vestibular neuritis, viral labyrinthitis); here, the risk of misdiagnosis is greatest.

It should also be noted that when symptoms are caused by a TIA rather than completed stroke, many of the telltale neurologic symptoms that accompany dizziness caused by disease in a particular vascular distribution may be absent. For example, basilar TIAs may produce isolated transient dizziness (vertiginous or not)[50] or transient dizziness accompanied only by auditory symptoms.[174] Thus, with transient symptoms, these rules cannot be consistently relied upon.

Table 1.2 Abbreviations and footnotes

AICA – anterior inferior cerebellar artery; PICA – posterior inferior cerebellar artery; SCA – superior cerebellar artery; V/N/V – vertigo/nausea/vomiting; VOR – vestibulo-ocular reflex

***** The AICA supplies the lateral pons, "middle" cerebellum (including part of the antero-inferior cerebellum), and the inner ear. When auditory symptoms occur with proximal AICA occlusion, they may reflect involvement of the cochlear nucleus in the lateral pons, or, instead, involvement of the ipsilateral inner ear (cochlea). When they occur with distal AICA occlusion, they reflect involvement of the ipsilateral inner ear (cochlea).

Table 1.2 Relationship between brain region, vascular territory, and neurologic symptoms and signs that may accompany dizziness during a stroke or TIA

	Brain/Ear Region (*Vascular Territory*)	**Typical Neurologic *Symptoms***	**Typical Neurologic *Signs***
UPPER	Occipital lobe *(distal posterior cerebral)*	Blurred or dim vision	Visual field cut
	Thalamus *(thalamic perforators)* Infero-medial temporal lobe *(proximal posterior cerebral)*	Confusion, amnesia, sleepiness	Short term memory deficit, impaired arousal/attention
	Midbrain *(upper basilar perforators, proximal SCA)*	Diplopia, weakness	Vertical gaze palsy, ptosis, 3rd nerve palsy, hemiplegia
	Superior cerebellum *(distal SCA)*	Clumsy, "drunk," +/- slurred speech	Severe limb & gait ataxia, +/- dysarthria
MIDDLE	Medial pons *(mid-basilar perforators)*	Dizziness, diplopia, weakness, numbness	Horizontal gaze palsy, 6th nerve palsy, hemiplegia, hemisensory loss
	Lateral pons* *(proximal AICA)*	V/N/V, oscillopsia, slurred speech, facial numbness, tinnitus or hearing loss, trouble walking/standing	VOR loss (8th), nystagmus, facial palsy/dysarthria (7th), facial sense loss (5th), unilateral deafness (8th), moderate gait +/- limb ataxia, Horner syndrome
	Middle cerebellum, labyrinth* *(distal AICA)*	V/N/V, oscillopsia, tinnitus or hearing loss, trouble walking	VOR loss (8th), nystagmus, unilat. hearing loss (8th), unsteady gait +/- limb ataxia
LOWER	Medial medulla *(anterior spinal artery, vertebral perforators)*	Dizziness, dysarthria, weakness, numbness	Nystagmus, tongue palsy (12th), hemiplegia, hemisensory loss
	Lateral medulla *(proximal PICA)*	V/N/V, oscillopsia, dysphagia/hoarseness, trouble walking/standing	Nystagmus, palatal (9th) or vocal cord palsy (10th), moderate gait +/- limb ataxia, Horner syndrome, crossed hemianalgesia face/body
	Inferior cerebellum *(distal PICA)*	V/N/V, oscillopsia, trouble walking	Nystagmus, unsteady gait

obvious stroke clear stroke subtle stroke

Figure 1.1 Traditional "quality-of-symptoms" approach to the dizzy patient

This figure illustrates the commonly-applied bedside rule that dizziness symptom quality, when grouped into one of four dizziness "types" (vertigo, presyncope, disequilibrium, or non-specific [ill-defined] dizziness), predicts the underlying cause. Although the description of these heuristics may be slightly oversimplified in this formulation, the rules presented here are basically those endorsed by proponents of the traditional approach, as articulated in the medical literature and, as we shall see, articulated by healthcare providers in describing their own practice (*Chapter 2*).

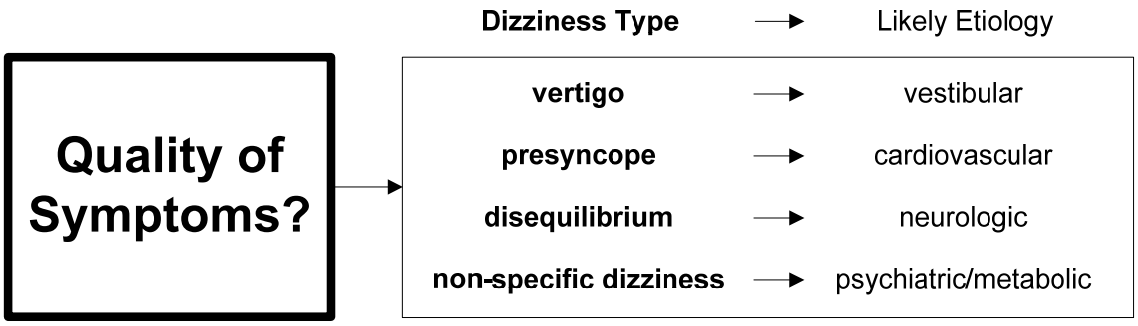


Figure 1.2 Posterior circulation vascular tree illustrating the classical vascular pattern and a common anatomic variant (reproduced from Oas & Baloh, 1992,[144] with permission)

The central vestibular structures are located principally in the lateral zones of the middle and lower brainstem (lower pons and upper medulla) and inferior portion of the cerebellum. The blood supply to these regions derives mainly from two pairs of medium-sized arteries known as the PICAs (posterior inferior cerebellar arteries) and AICAs (anterior inferior cerebellar arteries). Although there is inter-individual variability in blood supply patterns, each PICA most commonly arises from one vertebral artery (right or left), just before the two vertebrals join one another to become the basilar artery. Each PICA generally supplies blood to the lateral medulla and inferior-most portion of the cerebellum on either side. Both AICAs typically arise from the single basilar artery, and supply blood to the lateral pons, the anterior portion of the inferior and middle cerebellum, and the inner ear. The intermediate-sized branch that supplies the inner ear is known as the internal auditory artery (IAA), which itself splits into smaller branches supplying different *parts* of the inner ear.

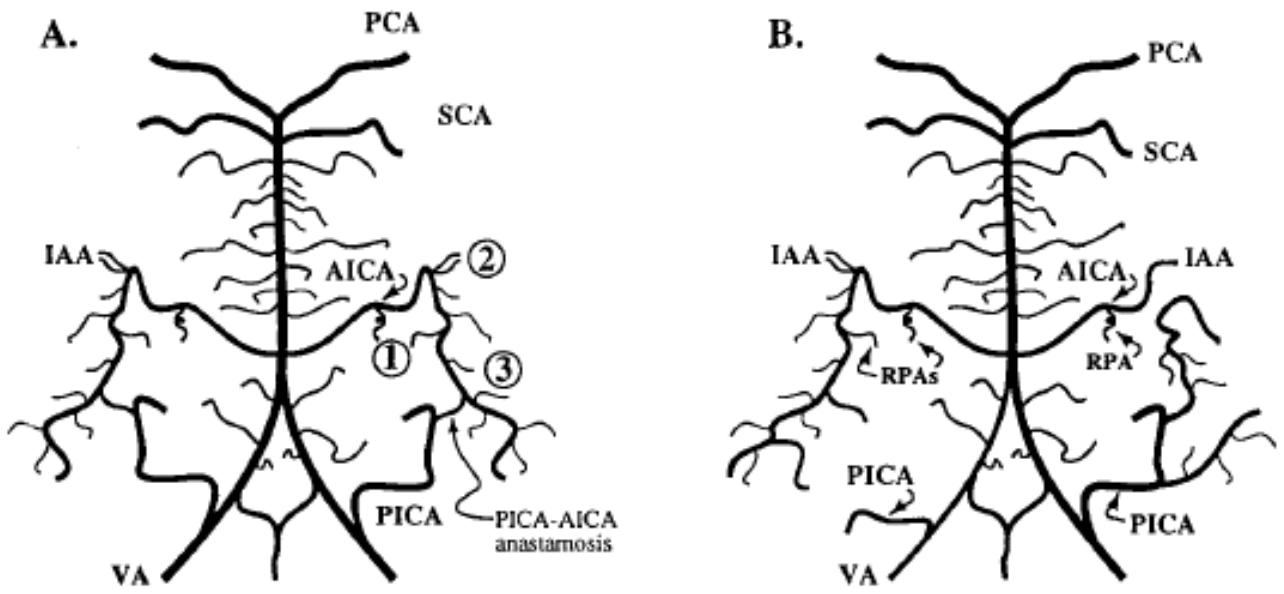
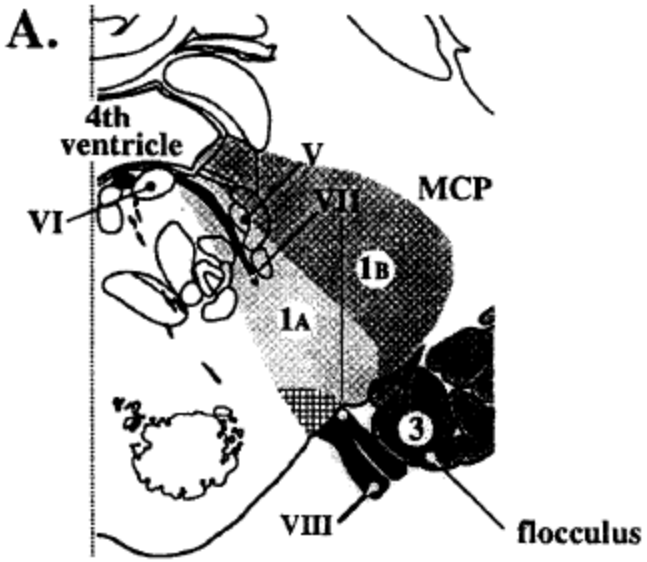


Figure 2. Common variations of AICA anatomy (modified from Atkinson,[6] with permission). Numbers refer to the zones of AICA shown in figure 3. Recurrent penetrating arteries (RPA) are branches off AICA. (A) Classical AICA anatomy with AICA and PICA of equal dominance. (B) Common variant with AICA dominance on the left and PICA dominance on the right. IAA = internal auditory artery; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; RPA = recurrent penetrating artery; SCA = superior cerebellar artery; VA = vertebral artery.

Figure 1.3 Blood supply to central and peripheral vestibular structures from the anterior inferior cerebellar artery (AICA) (reproduced from Oas & Baloh, 1992,[144] with permission)

Figure 1.3 – Central Vestibular Structures Supplied by AICA (see following page for description of numerical notations in figure)

Schematic diagram showing the distribution of blood supplied to the vestibular system by various branches of the AICA. Proximal branches supply blood to a small portion of the lateral pons (A). Next to receive blood is a large area of the anterior-inferior and middle cerebellum (C). Not shown is the PICA supply to the lateral medulla and postero-inferior portion of the cerebellum (*see Figure 1.4*).



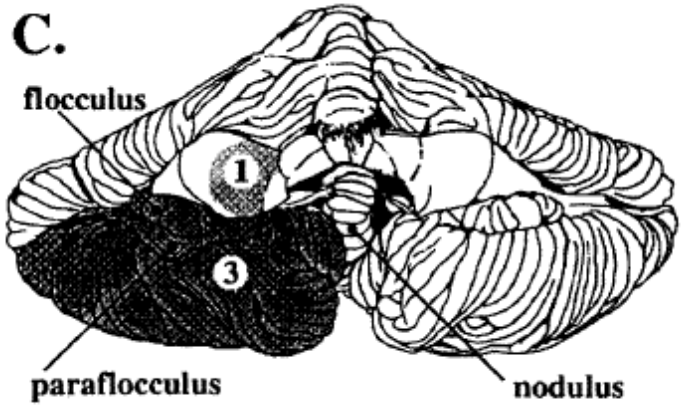


Figure 1.3 – Peripheral Vestibular Structures Supplied by AICA

Finally, the inner ear is nourished by the internal auditory artery (IAA) (B), which itself splits into smaller branches, one supplying predominantly the balance organs (anterior vestibular artery [AVA]) and the other supplying predominantly the hearing organ (common cochlear artery [CCA]).

The labyrinth is supplied by the IAA via two separate "paths." [147] The anterior vestibular artery (also known as the superior vestibular artery) arises directly from the IAA to supply blood to the anterior and lateral semicircular canals, as well as the utricle. The posterior vestibular artery (also known as the inferior vestibular artery) arises from the common cochlear artery (also known as the vestibulo-cochlear artery) to supply blood to the posterior semicircular canal, as well as the saccule.

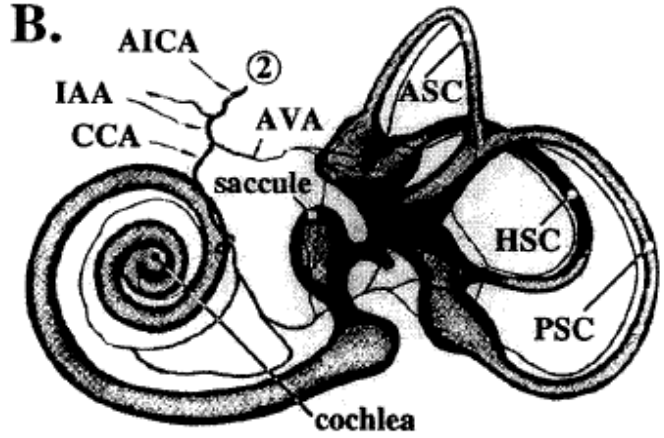


Figure 3. Three zones of AICA supply. Zone 1 is supplied by the recurrent penetrating arteries (RPA) off AICA, zone 2 by the internal auditory artery, and zone 3 by the terminal cerebellar branches of AICA. (A) Rostral pons at the level of the facial and abducens nuclei (vertical dotted line represents the mid-sagittal line). Zone 1A and zone 1B represent the arterial supply to areas supplied by a pre-meatal and post-meatal RPA (see figure 4A). Often a single RPA (originating from the pre-meatal or post-meatal AICA) supplies all of zone 1 (see figure 4B). The cross-hatched area represents the root entry zone of the facial and vestibulocochlear nerves. (B) Zone 2 represents the arterial supply to the inner ear (modified from Schuknecht, Pathology of the Ear,[28] *reprinted by permission of Harvard University Press). (C) Cerebellum, anterior view. Zone 3 represents the arterial supply from the terminal cerebellar branches of AICA. ASC = anterior semicircular canal; AVA = anterior vestibular artery; CCA = common cochlear artery; HSC = horizontal semicircular canal; IAA = internal auditory artery; MCP = middle cerebellar peduncle; PSC = posterior semicircular canal; V = spinal trigeminal tract and nucleus; VI = abducens nucleus; VII = facial nerve; VIII = vestibulocochlear nerve.*

Figure 1.4 Arterial territories of the inferior brainstem and cerebellum, in axial sections at the level of the major central vestibular structures (reproduced from Tatu, et al., 1996,[145] with permission)

Average arterial territories of the human brainstem and cerebellum from Tatu, et al., 1996.[145] The sections shown are representative of the blood supply from the level of the mid medulla (A) to the mid pons (D). Most of the central vestibular structures are located in the span between these planes of section.

PICA supply to the inferior cerebellum is shown in two shades of blue (medial and lateral branches). Note that the PICA supplies almost the entirety of the inferior-most portion of the cerebellum, so proximal PICA occlusions tend to produce very large infarctions. Despite their large size, "neurologic" symptoms are sparse in the early stages, and these patients often present with a pure mimic of benign viral vestibular neuritis.[139] Such patients are at high risk for death secondary to necrotic swelling of the infarct, either due to direct compression of the brainstem, or brainstem herniation secondary to raised intracranial pressure from cerebrospinal fluid outflow obstruction.

AICA supply to the anterior portions of the inferior and middle cerebellum is shown in red. The total cerebellar volume supplied by the AICA is less than that of the PICA. Therefore, even proximal AICA occlusions tend to produce slightly smaller cerebellar infarcts, at somewhat lower risk for brainstem compression or herniation from obstructive hydrocephalus. However, these patients often have co-morbid auditory symptoms (most likely from ischemia of the cochlea via the AICA branch known as the internal auditory artery). As a consequence, they may be mistakenly thought to have a "peripheral" (inner ear) pathology, assumed to be benign, and later go on to develop devastating brainstem infarction[247] which might otherwise have been prevented.

Figure 1.4A Arterial territories, level of the mid-medulla and inferior cerebellum

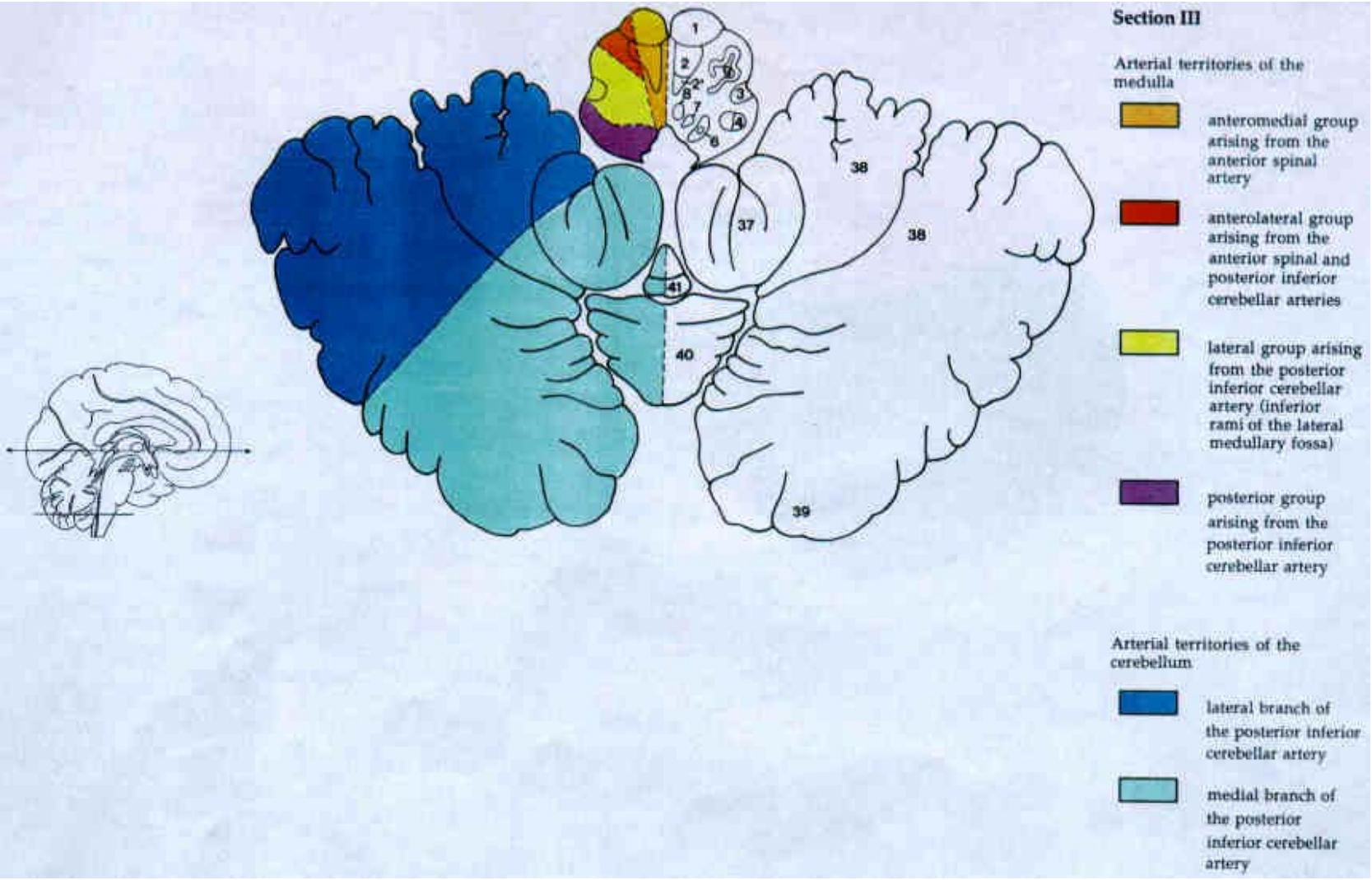


Figure 1.4B Arterial territories, level of the upper-medulla and inferior cerebellum

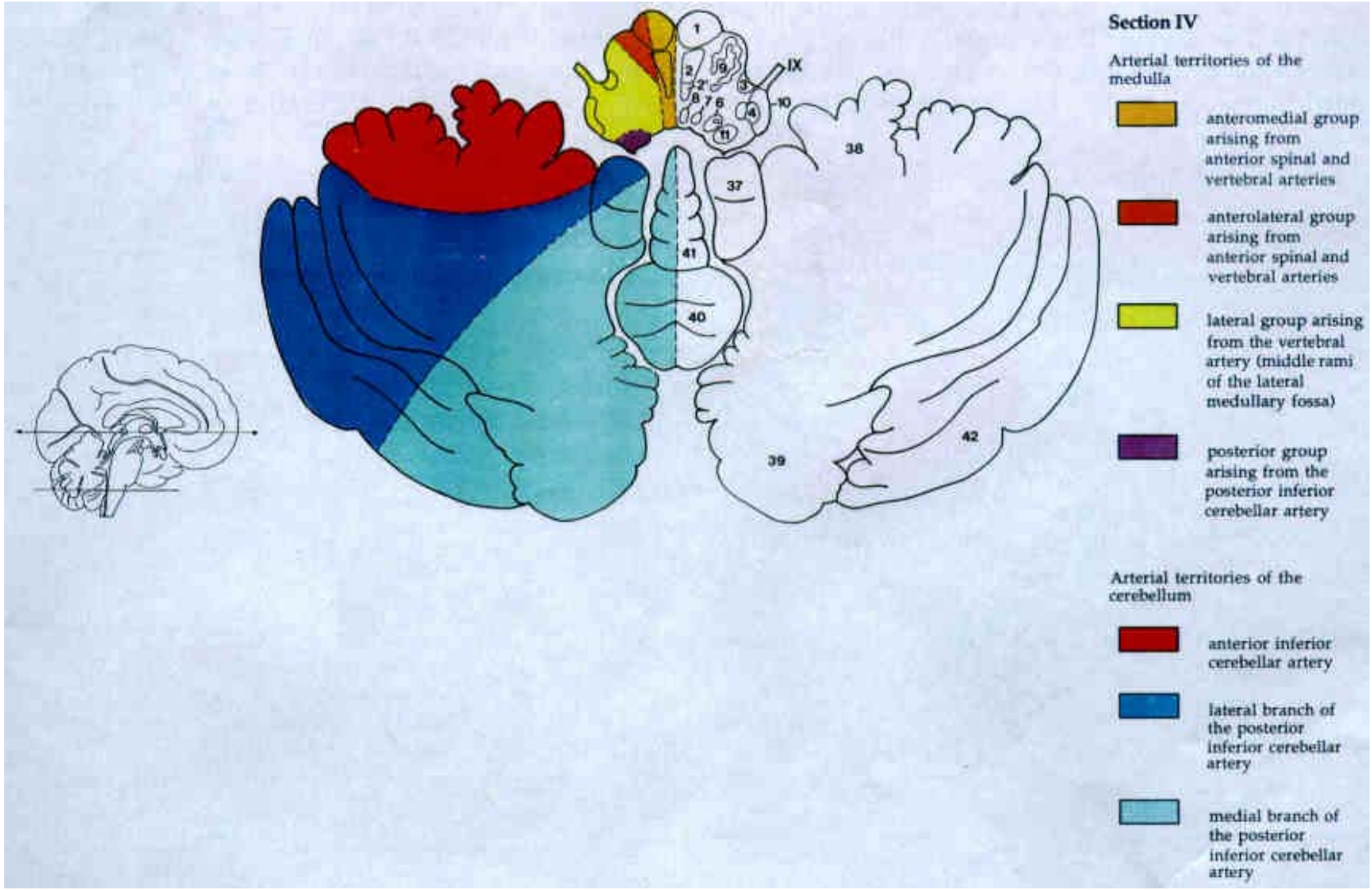


Figure 1.4C Arterial territories, level of the ponto-medullary junction and middle cerebellum

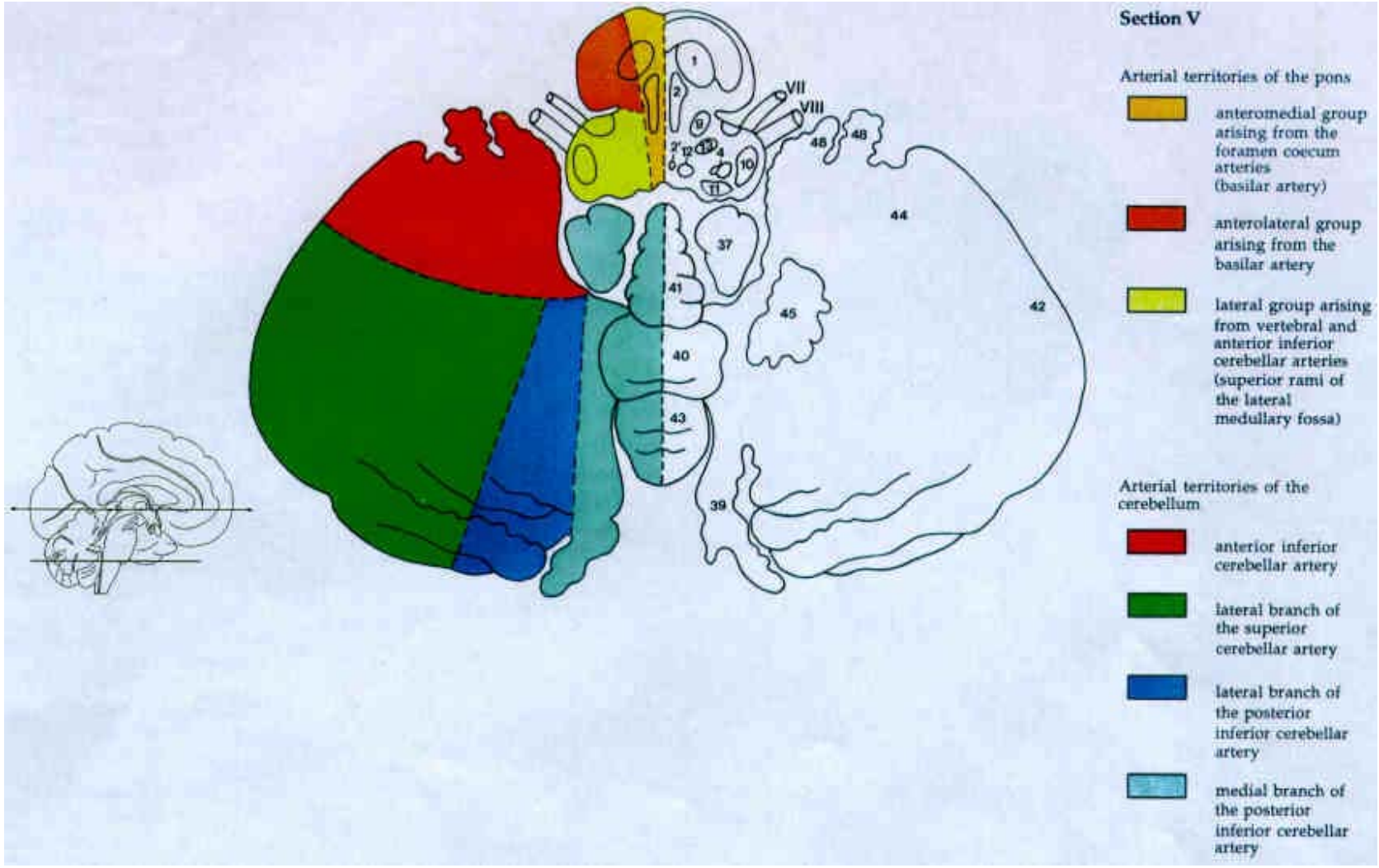
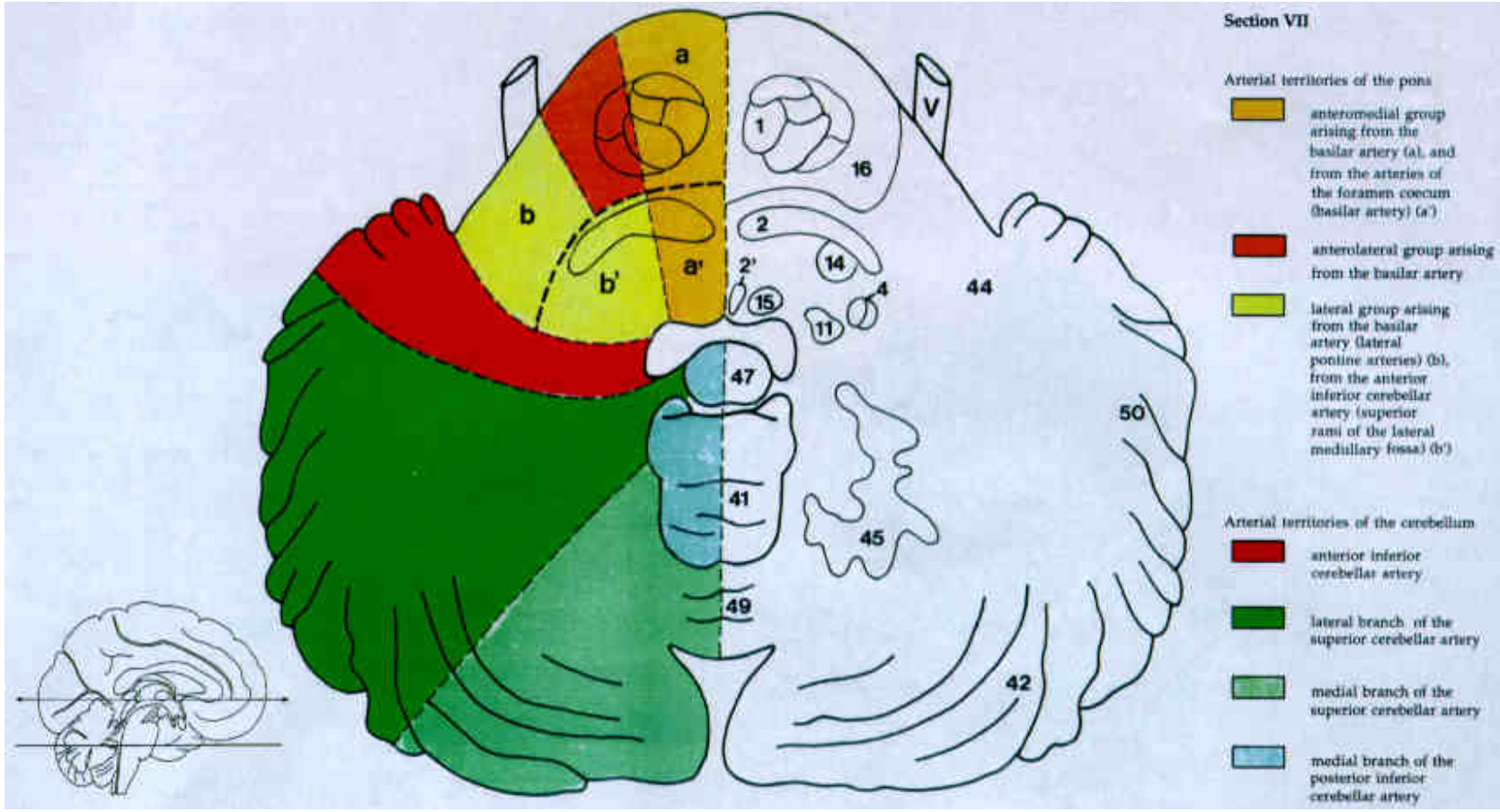


Figure 1.4D Arterial territories, level of the mid-pons and middle cerebellum



Chapter 2

Diagnosing Dizziness in the Emergency Department — Do Physicians Rely Too Heavily on Symptom Quality?

Results of a Multicenter, Quantitative Survey

Abstract

BACKGROUND: The textbook approach to diagnosing dizziness relies heavily on initially classifying the patient's qualitative complaint as vertigo, presyncope, disequilibrium, or ill-defined dizziness, with each "type" indicating a narrow spectrum of possible causes. It is unknown whether physicians use this approach in clinical practice, nor to what extent it might influence their subsequent diagnostic reasoning.

OBJECTIVE: Our goal was to quantify physicians' self-described practice in the diagnostic assessment of dizziness. We hypothesized that most would endorse the "quality-of-symptoms" approach, and that doing so might be associated with "risky" diagnostic reasoning.

DESIGN: Anonymous, internet-based survey.

SETTING: 17 academic-affiliated EDs.

SUBJECTS: Attending and resident physicians.

MEASUREMENTS: Ranked relative importance of symptom quality, timing, triggers, and associated symptoms. Level of agreement (Likert scale) with each of 20 statements about the diagnostic assessment of dizziness in clinical practice. Logistic regression for impact of "quality ranked first" on responses to clinical practice questions.

RESULTS: Response rate 82% (n=415/505). 93% (95%CI 83-100%) agreed that determining dizziness type is very important, and 64% (95%CI 54-74%) ranked "quality" the most important diagnostic feature. In a multivariate model, those ranking symptom quality most important more often reported risky clinical reasoning that might predispose to misdiagnosis (e.g., in a patient with persistent, continuous dizziness — who could have a cerebellar stroke — these physicians reported feeling reassured that a normal head CT indicates the patient is safe to go home: OR 2.43, 95%CI 1.23-4.77).

LIMITATIONS: Non-representative sampling method and reliance on self-reported clinical practice.

CONCLUSIONS: Physicians report taking a quality-of-symptoms approach to diagnosis of dizzy patients in the ED. Those who rely heavily on this approach may be predisposed to high-risk downstream diagnostic reasoning. Other clinical features (timing, triggers, and associated symptoms) appear relatively undervalued. Educational initiatives merit consideration.

Introduction

Dizziness is a complex neurologic symptom reflecting a disturbance of balance perception. It is the chief complaint in 5% of walk-in-clinic visits,[90] and third most common major medical symptom reported in general medical clinics.[91] A primary complaint of dizziness accounts for 4% of Emergency Department (ED) visits,[92] but another 24% of ED patients cite dizziness as part of the reason for their ED visit (*Chapter 3*). Although most cases are attributed to benign inner ear or cardiovascular disorders in either clinical setting,[141, 142] some result from dangerous cerebrovascular[142, 194] or cardiovascular[75, 85, 142] diseases requiring urgent attention. Dizziness is the ED

symptom most commonly associated with a missed diagnosis of stroke,[204] with population-based estimates suggesting a 35% misdiagnosis rate for cerebrovascular events.[116] The consequences of such misses can be profound, with one study indicating a 40% mortality rate for dizzy patients not initially recognized to have cerebellar stroke as the cause.[241]

Dizziness, like other symptoms (e.g., chest pain), may be thought of as having multiple symptom attributes such as quality, severity, duration, and provocative factors. Dizziness quality may be described by patients using words such as spinning, swaying, unsteady, lightheaded, foggy, disoriented, etc.[29, 50] It has traditionally been taught that the patient's description of dizziness quality should be classified as one of four "types" (vertigo, presyncope, disequilibrium, or ill-defined dizziness) in order to direct subsequent diagnostic inquiry.[19] In abbreviated form, this "quality-of-symptoms" approach states that vertigo indicates a vestibular cause, presyncope indicates a cardiovascular cause, disequilibrium indicates a neurologic cause, and ill-defined dizziness indicates a psychiatric or metabolic cause.[1] This approach can be traced back to Drachman and Hart's landmark 1972 article, "An Approach to the Dizzy Patient," which described detailed diagnostic assessments in a series of 104 outpatients attending a university dizziness clinic.[19] The quality-of-symptoms approach has been frequently endorsed in the medical literature across disciplines,[1, 7, 12, 15, 26, 28, 30-32, 141, 198, 248] but to what extent this approach is relied upon in clinical practice remains unknown, with one study of dizziness in primary care suggesting decision-making may be primarily driven by other factors (e.g., diagnostic uncertainty).[51]

It is also unclear whether this diagnostic model (Figure 2.1) will work well to guide *frontline* diagnosis, particularly in the acute-care setting. Disease-based data from recent studies suggest the quality-of-symptoms approach may not help identify key dangerous disorders in the ED, with the odds of cerebrovascular disease equal in patients with either "vertigo" or non-vertiginous "dizziness," [116] and myocardial infarction as likely to present with "vertigo" (8%) as "faintness" (5%).[249] Furthermore, new studies have shown that ED patients have trouble reliably reporting dizziness symptom quality (*Chapter 3*). These findings question whether the quality-of-symptoms approach can be relied upon to accurately inform diagnosis and work-up in an acute-care setting.

This concern is heightened by the fact that the quality-of-symptoms model derives from a study conducted in a subspecialty clinic more than three decades ago, prior to the advent of modern neuroimaging (both CT and MRI), during which each subject underwent a four-half-day battery of tests. In the ED, the spectrum of causes is broad,[3] the chances of acute, life-threatening pathology are high,[141, 194] and evaluations are time-pressured and oriented towards risk-stratification in pursuit of disposition decisions, rather than final diagnoses.[215] In this setting, the quality-of-symptoms approach may not be the most appropriate.

Data regarding diagnostic reasoning of emergency physicians (EPs) in the assessment of dizzy patients are scant. It is presumed that the EP approach reflects what is written in emergency medicine (EM) literature[31] and textbooks,[250-252] which generally endorse the quality-of-symptoms approach. One small study has shown that EPs preferentially document symptom quality and suggested the possibility that over-reliance

on symptom quality, to the relative exclusion of other clinical parameters (e.g., timing, triggers, and associated symptoms), might increase the risk of misdiagnosis.[253]

Given the importance of accurately diagnosing acutely dizzy patients, and the paucity of data on clinical reasoning in this domain, we sought to assess EPs' diagnostic approach to the dizzy patient, using a multicenter, quantitative survey. We hypothesized that EPs would (a) endorse the quality-of-symptoms approach in theory, (b) describe clinical decision-making that reflects reliance on symptom quality in practice, and (c) demonstrate risky diagnostic reasoning about bedside evaluation of dizzy patients that could relate to an over-reliance on symptom quality. We also sought to characterize EP use of dizziness terminology, comfort level with bedside dizziness diagnosis, and desire for decision tools that might assist in diagnosis.

Methods

Study Design, Setting, and Subjects

Multicenter, anonymous, web-based survey of EPs conducted in September-October 2006. The study was developed and implemented at Johns Hopkins University, in collaboration with the Emergency Medicine Network (www.emnet-usa.org). The survey was approved by the human subjects committees at all participating institutions.

All EM resident and attending-level EPs (n=505) at 17 hospitals affiliated with five academic centers (NewYork-Presbyterian — The University Hospital of Columbia and Cornell, Harvard Medical School, the Johns Hopkins University School of Medicine, University of California at San Francisco School of Medicine, University of Pennsylvania School of Medicine) were eligible to participate. Potential participants were excluded if they did not have a functioning email address (<1%).

Site leaders were recruited to identify possible subjects from their affiliated hospitals. Email invitations to participate in the study were distributed by the leader at each site. No incentive was offered for survey completion. An initial invitation was followed by one to two follow-up email reminders, at approximately one-week intervals, depending on response rate (target >80%). Potential subjects were informed that they could opt out of further study notification by emailing that they did not wish to be contacted further.

Study Procedures

To minimize respondent burden, while still gathering data on the full spectrum of questions of interest, the survey was disseminated as two partially overlapping versions (A and B). Email invitations from a given site leader contained a hyperlink to a site-specific portal webpage that automatically re-directed the participant to one of the two survey versions. Effectively-random allocation was achieved by computing the difference in milliseconds between an arbitrary, fixed start time and the time of the participant's computer's clock when the portal webpage was accessed. If this number was even, version "A" was loaded; otherwise, version "B" was loaded.

Anonymity was maintained by segregating physician identifiers (gathered by site leaders) from physician responses (gathered by the coordinating center). Site leaders removed respondent identifiers (names, emails, rank) prior to sending recruitment logs to the coordinating center. Data cleaning was performed at the coordinating center, and only aggregate data were sent back to site leaders.

The survey was prepared using standard methods for web-based survey development,[254] including a pilot testing phase. Several site leaders and seven attendings

at two institutions participated in pilot testing. These subjects were not excluded from final survey participation.

The survey was delivered using a commercial online survey vendor (Surveymonkey.com LLC, Portland, OR). Questions were presented two per page, and participants were required to answer both questions before continuing. They could not return to a previous page after moving on to the next. Internet cookies prevented subjects from taking the survey more than once at the same computer.

The two survey versions had nine overlapping (both A and B) and six discrete (either A or B) questions about the provider's beliefs and practices regarding the bedside evaluation of dizziness. Respondents graded level of agreement with 14 statements on a 7-point Likert scale (strongly agree to strongly disagree). They then ranked four clinical attributes of dizziness, based on their relative importance to diagnosis (attributes were presented in a randomly-determined order for each respondent).

Six demographic questions and one about participation in the pilot phase were followed by an opportunity to provide feedback. Three of six demographic questions were free-response (year of graduation; total years of clinical experience; percentage time clinical work in previous 2 years), while the others were categorical (academic rank; EM board eligibility/certification; prior exposure to the study hypothesis).

Data Analysis

Response rates were calculated using data on the number of invitations sent by site leaders. Survey duration was calculated from meta-data provided by the online survey vendor (difference from start to submit time). Results by site could not be segregated for sites E/F, due to a technical problem with one survey hyperlink.

Means and standard deviations for Likert-scale questions (20 across the two survey versions) were calculated using a linear conversion of responses to a numerical scale (+3 to -3), assuming equidistance between response options. Percent agreement with survey statements was calculated by combining all affirmative (+1 to +3) responses. We report percent agreement, number of respondents, and 95% confidence intervals (CI). We framed four statements such that agreement (Q10, 18) or disagreement (Q11, 19) would indicate a high-risk clinical decision with regard to misdiagnosis of a dangerous underlying disorder (e.g., cerebrovascular). For these questions, we identified the percentage of "risky" responses. For false statements, agreement or neutrality (scaled answers 0 to +3) was considered "risky"; for true statements, disagreement or neutrality (scaled answers 0 to -3) was considered "risky."

For continuous demographic variables we calculated mean and standard deviations. All residents graduating medical school in 2006 were assigned 0.25 years of experience. For categorical demographic variables we calculated the proportion of respondents in each category. Due to the relatively small number of higher-ranking (associate/full) professors, academic rank was dichotomized into "residents" and "attendings" (i.e., fellows, instructors, and professors at all ranks) for subgroup analyses. For an analogous reason, board eligibility/certification was dichotomized into "EM" (i.e., EM, with or without additional board eligibility/certification) and "not EM" (i.e., only internal medicine, surgery, or other). Prior exposure to research or teaching related to the study hypothesis was categorized by respondents as "Yes, a lot," "Yes, a little," or "No, not at all."

Multivariate logistic regression was used to calculate the impact of endorsing the quality-of-symptoms approach on responses to questions about clinical behaviors. We report odds ratios and 95% CI for ranking symptom quality most important (Q21) after adjusting for academic rank, EM board eligibility/certification, and percent clinical effort. Generalized Estimating Equation (GEE) regression techniques were used to control for within-site correlations.

Data were handled in Microsoft Excel 2003 (Redmond, WA). For statistical analyses, data were exported into SAS v9.1 (Cary, NC). Percentages and proportions are reported with accompanying 95% CI. All p-values were 2-sided, with $p<0.05$ considered statistically significant.

Role of the Funding Source

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Results

Overall, 82% (n=415/505) of those surveyed responded, and 94% of responders (n=389/415) completed all survey questions. Of the 415 responders, 200 were randomly assigned to survey version A, and 215 to version B. Completion rates for versions A and B were not significantly different (91% vs. 96%, respectively; p=0.36). The median survey duration, including demographic questions, was 5.3 minutes (interquartile range 4.1–7.3 min). The breakdown of demographic variables by site is shown in Table 2.1.

Survey questions and responses are provided in Appendix 2.1, and question responses by site are provided in Appendix 2.2.

Overall Experience With and Attitude Towards Dizziness

EPs agreed that dizziness was one of the "top 10" non-trauma chief complaints they encountered in clinical practice (Q1: 77%, 95%CI 73–81%). Reports of dizziness as a common symptom in the ED varied by site, but at all sites, the majority agreed it was a "top 10" complaint.

Respondents expressed overall confidence in assessing dizzy patients without specialist consultation (Q2: 76%, 95%CI 71–80%). They expressed less confidence in identifying a common physical diagnostic sign — the typical upbeat-torsional nystagmus seen among patients with benign paroxysmal positioning vertigo (BPPV) [255] (Q17: 57%, 95%CI 50–64%).

Regarding dizzy patients, the vast majority of EPs were open to the possibility of using a clinical decision rule to help guide diagnostic testing (e.g., neuroimaging) (Q13: 94%, 95%CI 90–97%), and the majority expressed willingness to use recommendations produced by a computer-based decision support kiosk that interviewed patients in the waiting area (Q20: 66%, 95%CI 59–72%).

Endorsing the Quality-of-Symptoms Approach in Theory

There was broad consensus that the quality-of-symptoms approach to dizziness is the dominant diagnostic paradigm presented in the medical literature and teaching, and providers personally endorsed a belief in this approach (Table 2.2).

When asked to rank the relative importance of several attributes of dizziness to diagnostic assessment of an ED dizzy patient, the majority ranked symptom quality first

(Table 2.3). The strongest predictor of ranking symptom quality first was site (Q21: Site A 49%, Site B 52%, Site C 63%, Site D 71%, Site E/F 75%; $p<0.001$). As hypothesized, there was a dose-response relationship between ranking symptom quality first and prior exposure to research or teaching that the quality-of-symptoms approach might be flawed: 69% (104/151) if not exposed, 64% (129/202) if exposed a little, and 53% (19/36) if exposed a lot. However, the number of subjects exposed "a lot" was sufficiently small that statistical power was limited, and the trend was of borderline statistical significance ($p=0.08$ by Cochran-Armitage Trend Test).

Interpretation of Quality-of-Symptoms Terminology

The majority of respondents endorsed thinking of "lightheadedness" as a mild form of "presyncope" and pursuing cardiovascular causes in such patients (Q7: 68%, 95%CI 61–74%). This is in contrast with the original quality-of-symptoms approach advocated by Drachman and Hart (Figure 2.1 legend), where "lightheadedness" was clearly segregated from "presyncope" and classified as "ill-defined 'lightheadedness' other than vertigo, syncope, or disequilibrium." [19] To sidestep confusion on this point, "Type 4" dizziness is now often referred to as "vague," [1] "non-specific," [25] or simply "other" [24] dizziness.

The majority of respondents also endorsed restricting use of the term "vertigo" to describe an unmistakable spinning sensation (Q15: 68%, 95%CI 62–74%), which represents a more stringent interpretation than that advocated by Drachman[1] and many neuro-otologists.[9]

Endorsing the Quality-of-Symptoms Approach in Practice

Providers were mixed about whether "vague" dizzy symptoms in the ED were usually associated with metabolic disorders (Q14: 40%, 95%CI 34–47%). In the original Drachman and Hart study, conducted in a specialty clinic, the majority of these "vague" cases were deemed to have psychiatric disorders.[19]

Many providers agreed that they make clinical decisions based on the quality-of-symptoms model, including when *not* to pursue certain diagnoses. The majority acknowledged they typically do not pursue cardiovascular causes when the patient reports "vertigo" nor vestibular causes when the patient reports "presyncope" (Q6: 69%, 95%CI 64–73%). Responses varied by site, but at all but one, the majority agreed. Those ranking symptom quality the most important dizziness attribute (Q21) were more likely to report *not* pursuing cardiovascular causes in patients with "vertigo" nor vestibular causes in patients with "presyncope" (adjusted OR 1.76, 95%CI 1.30, 2.37).

Roughly half agreed they do not pursue cerebrovascular causes when the patient reports "vague" dizziness symptoms unassociated with obvious neurologic symptoms or signs (Q8: 48%, 95%CI 41–55%). Responses varied by site. Again, those endorsing symptom quality as the most important attribute were more likely to report *not* pursuing neurologic causes in patients with "vague" dizziness (adjusted OR 1.94, 95%CI 1.14, 3.28).

Relation Between Quality-of-Symptoms Approach & Other Dizziness Attributes

Although the frequency of "risky" responses was non-trivial (Table 2.4), all but one (Q10) of four questions was answered "safely" by the majority of ED physicians. Eighty percent of providers mistakenly endorsed the idea that, in patients with persistent

dizziness, head motion triggering an exacerbation of symptoms is an indicator of benign pathology (Q10) (*see Discussion and Box 2.1 for clarification*). For all four of these questions, those who chose symptom quality as the most important attribute were at increased odds of a risky response, significantly so for two questions (Table 2.4).

Discussion

Our survey results demonstrate that the quality-of-symptoms approach to dizziness (i) is the dominant diagnostic paradigm in the ED, (ii) drives physician thinking and self-reported behaviors at multiple levels, and (iii) could be contributing to risky clinical reasoning in the diagnostic assessment of dizziness. These findings are significant because recent evidence indicates that the quality-of-symptoms approach appears flawed (*Chapter 3*) and critical misdiagnosis of ED dizzy patients may be frequent.[116, 204]

EPs uniformly agreed that the quality-of-symptoms approach is the most common approach to the dizzy patient described in EM literature and teaching. However, it is noteworthy that two-thirds of respondents defined the dizziness symptom categories (which purportedly indicate etiology) differently than the original paradigm suggests. Almost all respondents personally endorsed a key role for symptom quality in helping to determine dizziness etiology, and the majority ranked symptom quality the most important attribute for diagnosis (even that small minority previously exposed to "a lot" of research or teaching to the contrary). They generally endorsed decision-making behaviors that reflected these stated beliefs.

The majority of providers harbored one important misconception about the diagnostic assessment of dizzy patients — that exacerbation of dizziness by head motion is a sign of a peripheral vestibular disorder in patients with persistent, continuous

dizziness (*see Box 2.1 for clarification*) — a potentially lethal misconception that has been described previously[231] (*see Appendix 1.2 for additional details*). Among the minority harboring other misconceptions, providers who endorsed the quality-of-symptoms approach were less likely to assign diagnostic importance to the presence of head or neck pain in a dizzy patient, despite the well-recognized association between dizziness and vertebral artery dissection[191, 256, 257] and the potential risks associated with missing that diagnosis.[241] They were also more likely to take a normal head CT scan as excluding a diagnosis of cerebellar stroke, despite the known low sensitivity of CT scans for identifying posterior fossa infarcts.[237, 239, 241, 258]

Whether these associations are causal or not remains speculative, but they raise the possibility that adopting a quality-of-symptoms approach might constrain diagnostic reasoning in a way that predisposes to errors. For example, diagnostic emphasis placed on symptom quality might be to the relative exclusion of details such as episode duration.[253] Failure to distinguish between patients with brief, episodic dizziness and those with a single, protracted episode could then lead to confusion about the diagnostic meaning of key historical (e.g., head motion triggers) and physical examination (e.g., nystagmus) findings (Box 2.1).

Limitations

This study has a few potential limitations. Threats to internal validity include (1) an imperfect survey design, (2) the potential disconnect between self-reported behavior and actual behavior, and (3) the possibility of unmeasured confounders explaining the relationship between a symptom quality-focused view and dangerous misconceptions.

The principal threat to external validity is the relatively narrow group of EPs (n=415) drawn from only 17 hospitals.

In the interest of brevity (in pursuit of a high response rate), we were forced to make compromises in survey design — (i) asking fewer questions, (ii) not including "realistic" clinical vignettes, and (iii) framing many questions as two-part statements. The first of these means we still have unanswered questions (e.g., "Do the 60% of EPs who disagree that patients with vague dizziness have metabolic disorders think they have psychiatric disorders, or something else?"). The second means it is possible that other clinical factors (e.g., age or co-morbid medical conditions), not assessed, mitigate the importance of quality-of-symptoms reasoning to EPs. The third, however, poses the greatest threat to validity. Two-part ("double-barreled") questions are generally frowned upon in survey design, because their results can be difficult to interpret.[259]

In this study, we wished to draw out the link between a conceptual endorsement of a diagnostic principle and the corresponding clinical behavior (e.g., *Q7. When a patient reports "lightheadedness," I think of this as a mild form of "presyncope" (about to faint) even if they don't expressly describe a feeling of impending faint. Therefore, I focus on cardiovascular causes in such patients.*). Without employing multi-part questions, this would have required at least three separate questions, leading to a prohibitively long survey. When respondents "disagree" with two-part statements, nothing can be known about which part(s) of the statement are disagreed with (i.e., part one, part two, or both). However, we contend that when respondents "agree" with two-part statements, it is reasonable to infer that they agree with both parts of the statement. Accordingly, we framed all of the two-part questions with the intent that they would be

agreed with by the majority. In all but one case in which we employed this strategy (Q18), the majority agreed with the two-part statement.

Self-reported behaviors do not always match observed real-world performance.[260] Because this survey relies solely on physician self-report, it is impossible to assess how accurately the responses portray actual clinical practice. However, our results do match previous findings from a small study examining EP charting habits in which 70% documented dizziness quality, 50% documented associated pain, 30% documented triggers, and only 13% documented episode timing,[253] strengthening the link between belief and action.

Unmeasured confounders represent a threat to validity in any non-randomized study suggesting a causal association.[261] We do not contend that our study provides conclusive evidence of a causal link between the quality-of-symptoms approach and dangerous misconceptions in the evaluation of dizzy patients. However, we believe that (a) the "biologic plausibility" of the association (Box 2.1), (b) its statistically-significant relationship to dangerous misconceptions (Q18, 19), and (c) the fact that the association was prospectively hypothesized, all argue in favor of a real link that could be causal in nature.

With any survey there is concern that respondents differ from the population as a whole. Our high response rate (82%) makes significant sampling bias *within* our sampling frame unlikely. However, the issue of generalizability *from* our sampling frame (EPs affiliated with five academic institutions) to the larger physician population could be viewed as a limitation. Survey responses varied by site, and contrary to our *a priori*

hypothesis, the majority of the inter-site variability was not explained by prior exposure to research and teaching on flaws in the quality-of-symptoms approach.

Geographic differences in medical practice are known to be common, and there is no reason to believe that the diagnostic approach to dizzy patients would be an exception. So-called "small area variations" in clinical practice have been recognized for decades,[262] and are presumed to reflect complex local social and healthcare delivery system factors.[263] It might be that community physicians take a different approach to dizziness than academic physicians. However, this seems unlikely, given that there was such a broad consensus that the quality-of-symptoms approach is the dominant diagnostic paradigm presented in the medical literature and teaching. Furthermore, although responses to individual survey questions varied by site, in almost all instances, these differences were in magnitude only, rather than in direction (i.e., majority agree vs. majority disagree). This general agreement across sites, despite inter-site demographic differences, argues in favor of generalizability of the results. Finally, although the study was coordinated through five academic centers, 35% (n=6/17) of the affiliated hospitals from which EPs were recruited are community-based hospitals, further bolstering the contention that the results are likely generalizable to other frontline healthcare settings.

Conclusions

Despite its potential limitations, our study presents a strong case that the quality-of-symptoms approach is the dominant paradigm for diagnosing the acutely dizzy patient in the ED. Furthermore, it suggests this approach may be displacing alternative diagnostic models, such as those emphasizing other symptom dimensions (e.g., timing, triggers, and associated symptoms) to guide diagnostic reasoning.

Given recent evidence questioning the standard approach to diagnosis and suggesting a potential link to misdiagnosis, future studies should clarify whether a physician's focus on symptom quality is associated with real-world misdiagnosis of ED dizzy patients. Related research should seek to rigorously determine the accuracy and utility of alternative diagnostic models. As shown in our study, EPs are open to new approaches, whether in the form of well-validated clinical decision rules, or workflow-sensitive forms of computer-based decision support. In the meantime, strong consideration should be given to training frontline healthcare providers to approach dizziness with a different diagnostic emphasis.

Table 2.1 Demographic description of physician survey respondents by site (academic institution)

The table shows response rate, academic rank, board eligibility/certification in emergency medicine, prior exposure to the underlying study hypothesis or pilot testing of the survey instrument, mean years since medical degree, and mean percent clinical effort, in aggregate, and by site. Results suggest there was some demographic variability in respondents by site.

Note that totals by site (column) do not always sum to aggregate number of respondents (total 'n') due to missing data on a particular variable.

Table 2.1 Abbreviations and footnotes

EM – emergency medicine

* Site E/F represents mixed results from two survey sites. We intended six "sites" among five institutions, with the largest split into two separate sites. However, a technical problem occurred during survey dissemination. Participants at two of the six sites (affiliated with different institutions) were sent the same survey hyperlink in error. Accordingly, data from those two sites were analyzed together (as site E/F), and represent an amalgam of results across the two institutions.

† Prior exposure to research or teaching suggesting that the quality-of-symptoms approach may be flawed.

‡ Only individuals at sites A and C were offered an opportunity to be pilot test subjects. Therefore, the other positive responses (n=4, Sites D, E/F) represent mis-clicks or misunderstanding of the question.

Table 2.1 Demographic description of physician survey respondents by site (academic institution)

Demographic		All Sites	Site A	Site B	Site C	Site D	Site E/F*	p-value
Response Rate (completed entire survey)		77% (389/505)	64% (59/92)	78% (66/85)	73% (43/59)	94% (97/103)	75% (124/166)	<0.001
Academic Rank	*Resident*	44% (171)	56% (33)	47% (31)	0% (0)	35% (34)	59% (73)	<0.001
	Fellow/ Instructor/ Asst. Prof.	43% (169)	37% (22)	48% (32)	58% (25)	54% (52)	31% (38)	
	Assoc./Full Professor	10% (38)	5% (3)	3% (2)	42% (18)	2% (2)	10% (13)	
Board Eligible or Certified EM		72% (280)	61% (36)	71% (47)	100% (43)	78% (76)	63% (78)	<0.001
Prior Exposure to Study Hypothesis†	*A lot*	9% (36)	10% (6)	18% (12)	7% (3)	8% (8)	6% (7)	0.04
	A little	52% (202)	54% (32)	52% (34)	67% (29)	48% (47)	48% (60)	
	Not at all	39% (151)	36% (21)	30% (20)	26% (11)	43% (42)	46% (57)	
Pilot Testing‡		2% (9)	5% (3)	0% (0)	5% (2)	2% (2)	2% (2)	0.30
Mean Yrs Since Degree (SD)		8.7 (8.9)	7.4 (8.7)	8.5 (8.5)	14.2 (8.8)	9.7 (9.6)	6.6 (7.8)	<0.001
Mean % Effort Clinical (SD)		70% (27)	67% (30)	75% (26)	70% (25)	73% (24)	66% (28)	0.14

Table 2.2 Emergency physicians endorse the quality-of-symptoms approach

The table shows percent agreement (with 95% confidence interval) and mean level of agreement (7-point Likert scale from +3 to -3) with three statements about the quality-of-symptoms approach to diagnosing dizzy patients. The first (Q3) demonstrates the pervasiveness of the quality-of-symptoms paradigm in the medical literature and teaching. The second (Q4) confirms the common understanding of this textbook approach. The third (Q5) establishes the respondent's personal belief in or endorsement of the approach in their clinical practice setting. Together, these results identify the quality-of-symptoms approach as the dominant diagnostic paradigm for assessment of dizzy patients in frontline care settings.

Note that the total number of respondents (total 'n') is lower for Q5 than Q3 and Q4 because of attrition of respondents during the survey.

Table 2.2 Emergency physicians endorse the quality-of-symptoms approach

Survey Statements Endorsing The Quality-of-Symptoms Approach	**% Agree (n)**	**95% CI**	**Mean (SD) (+3 to -3)**
Q3. Most articles, textbooks, and lectures about dizziness say that asking a dizzy patient to describe the quality of the dizzy symptoms is the first (and most important) step.	90% (366/407)	87–92%	1.8 (1.0)
Q4. These sources of information also generally say that the patient's response should be categorized as one of four "types" of dizziness, each implying a likely etiology: i. VERTIGO (spinning/motion), implying a vestibular (peripheral or central) cause ii. PRESYNCOPE (about to faint), implying a cardiovascular cause iii. DISEQUILIBRIUM (unsteady walking), implying a neurologic (motor/sensory) cause iv. NON-SPECIFIC (other vague dizzy feelings), implying a psychiatric or metabolic cause	87% (354/407)	83–90%	1.6 (1.1)
Q5. I believe that the "type" of dizzy symptoms is very important in determining the underlying etiology when evaluating an ED dizzy patient.	93% (371/400)	90–95%	1.8 (1.1)

Table 2.3 Ranked importance of four dizziness attributes in the assessment of a typical ED patient

Respondents were presented with a single ranking question about the importance of four different dizziness attributes. The table shows the proportion of respondents ranking an attribute "most important" (i.e., 1) and the mean rank (possible range, 1.0–4.0) for each of the four attributes. Symptom quality was ranked most important five-fold more often than the next nearest attribute, and mean rank for symptom quality was significantly higher than each of the three other dizziness attributes.

Table 2.3 Abbreviations and footnotes

ED – emergency department

* The precise instructions were, "Please rank the overall relative importance to you of these historical features in assessing a typical ED dizzy patient. (We recognize that various elements of clinical history may be of different value under different clinical circumstances.)" The four response options were presented to each respondent in random order to eliminate any potential biasing effect of presentation order. Each rank position could be used only once, and the respondent could not continue the survey without ranking all four dizziness attributes.

† $p<0.001$ for each comparison (quality vs. timing; quality vs. triggers; quality vs. associated symptoms) by paired t-test

Table 2.3 Ranked importance of four dizziness attributes in the assessment of a typical ED patient

Dizziness Attribute	Proportion Ranked* Most Important (n)	95% CI	Mean Rank* (SD)
Quality	64% (254/394)	60–69%	1.7† (1.0)
Timing	11% (44/394)	8–15%	2.8 (1.0)
Triggers	12% (46/394)	9–15%	2.6 (1.0)
Associated Symptoms	13% (50/394)	10–16%	2.9 (1.0)

Table 2.4 Association between reliance on symptom quality and increased risk of dangerous reasoning

Respondents were presented with four statements framed such that agreement (Q10, 18) or disagreement (Q11, 19) would indicate a high-risk clinical decision with regard to possible misdiagnosis of a dangerous underlying disorder (e.g., cerebrovascular). The table compares the percentage of "risky" responses among those ranking symptom quality most important (#1) to the percentage among those who did not. The majority of emergency physicians chose a less risky response for three of the four statements. However, risky response rates were higher among those ranking symptom quality first, and, for two of the four statements, those ranking symptom quality most important had more than double the odds of endorsing a risky decision.

Table 2.4 Abbreviations and footnotes

ED – emergency department; TIA – transient ischemic attack

* Denominators (and respondents) for questions 10 and 11 (survey version A) differ from those for questions 18 and 19 (survey version B) due to different numbers of respondents taking each survey version, and differ slightly from the total number of respondents described in results (n=200 for version A, n=215 for version B) due to attrition during the survey.

† Odds ratios for answering question with "risky" response based on ranking symptom quality #1 vs. anything else (#2, #3, or #4) are adjusted for academic rank, EM board eligibility/certification, and percent clinical effort, after controlling inter-subject correlations within site.

‡ Statistically significant difference between groups ranking symptom quality first and those who did not.

Table 2.4 Association between reliance on symptom quality and increased risk of dangerous reasoning

Survey Statements Regarding Diagnostic Reasoning	Risky Response (Risk)	% (n*) Endorsing Risky Response by Quality Rank		Odds Ratio† (95% CI)
		Quality Ranked #1	Quality Not Ranked #1	
Q10. In those still "sick and dizzy" at the time of assessment, exacerbation of symptoms with any head motion gives me confidence the patient has a peripheral vestibular disorder.	Agreement (missed stroke)	80% (96/120)	70% (45/64)	1.48 (0.69, 3.19)
Q11. In patients with brief, unprovoked, episodic dizziness (lasting seconds to minutes) and negative Dix-Hallpike (Nylen-Bárány) positional testing, I focus on ruling out cardiac arrhythmias and TIAs.	Disagreement (missed TIA, arrhythmia)	43% (52/120)	41% (26/64)	1.20 (0.62, 2.33)
Q18. In patients with a single, continuous bout of dizziness (lasting hours to days) and spontaneous nystagmus, the diagnosis is likely to be vestibular neuritis or cerebellar stroke. In these cases, a normal CT reassures me that these patients are safe to go home.	Agreement (missed stroke)	21% (106/134)	13% (66/76)	2.43‡ (1.23, 4.77)
Q19. In ED dizzy patients with head or neck pain and a normal ear exam (including otoscopy), I aggressively pursue aneurysm or vascular dissection, unless I am confident the patient has migraine-associated dizziness (a.k.a. vestibular migraine, basilar migraine).	Disagreement (missed TIA or stroke)	43% (77/134)	30% (53/76)	2.26‡ (1.82, 2.80)

Figure 2.1 Textbook "quality-of-symptoms" approach to the dizzy patient, as might be applied in the ED setting

This figure illustrates the textbook approach to the dizzy patient, as it might be carried out in the ED setting. The emphasis is placed on identifying symptom quality for classification as one of four dizziness "types" (vertigo, presyncope, disequilibrium, or non-specific [ill-defined] dizziness), in order to predict the underlying cause. The original definitions of these categories are provided below. The precision of this approach relies, in part, on a shared understanding among physicians of the precise meaning of these qualitative symptom descriptors. However, results presented in this study suggest that variations in terminology are commonplace (*see Results, Interpretation of Quality-of-Symptoms Terminology*).

Drachman & Hart's Original Definitions for the Four Types of Dizziness[1, 19]

Type 1: Vertigo = "a definite rotational sensation"

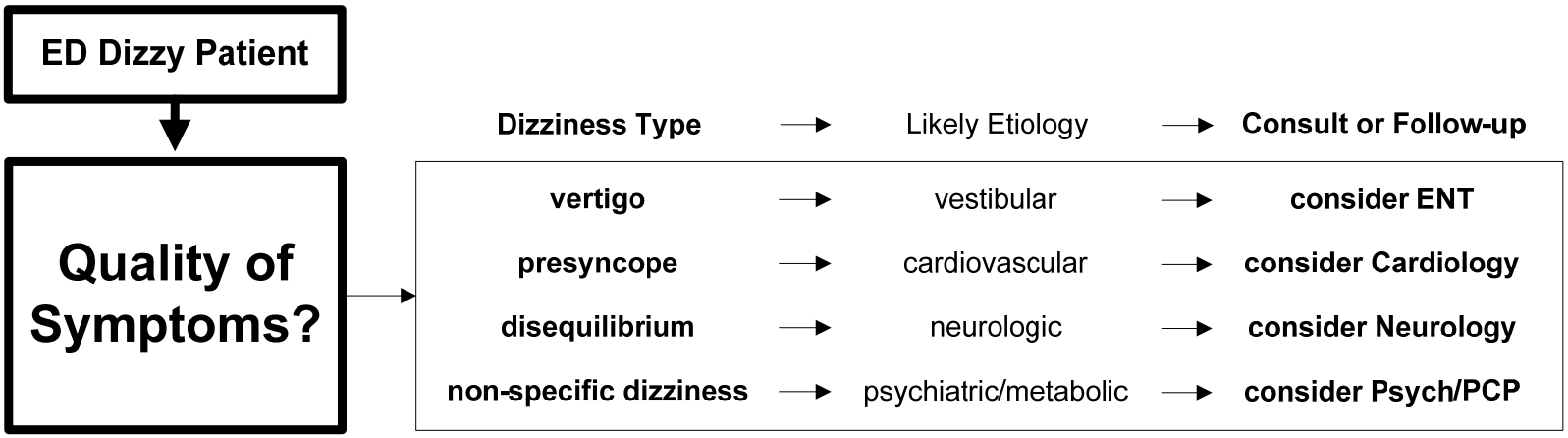
Type 2: Presyncope = "a sensation of impending faint or loss of consciousness"

Type 3: Disequilibrium = "dysequilibrium [*sic*] or loss of balance without head sensation"

Type 4: Vague light-headedness = "ill-defined 'lightheadedness' other than vertigo, syncope, or dysequilibrium [*sic*]"

Figure 2.1 Abbreviations

ED – emergency department; ENT – ear, nose, and throat physician (otolaryngologist); PCP – primary care provider; Psych – psychiatrist



Box 2.1 Bedside rules to distinguish central from peripheral causes of dizziness are contingent on symptom timing

Commonly-applied bedside rules that use dizziness attributes or findings to distinguish central (often dangerous) from peripheral (usually benign) causes may be true or false, depending on symptom timing (episode duration and frequency). Two such rules, as cited in current emergency medicine textbooks are listed below in the box. The rationale for why the truth or falsity of each is contingent on symptom timing is provided.

Bedside Rule #1: Head motion or position triggers usually indicate a peripheral (benign) etiology[250, 251]

- This rule is **TRUE** if and only if the patient has brief, episodic dizzy spells. BPPV is almost invariably incited by head-back triggers,[255] while the chief dangerous causes of recurrent, episodic dizzy spells [TIA and cardiac arrhythmia] are generally spontaneous, rather than triggered.[75]

- This rule is **FALSE** when the patient has a single, protracted bout of dizziness lasting hours to days. Cerebellar strokes produce persistent dizziness symptoms that are exacerbated by head motion[264] or position,[265] just like vestibular neuritis.[264] So this clinical finding will fail to provide evidence of a peripheral cause in a patient with a single, prolonged episode of dizziness.

Bedside Rule #2: Vertical/torsional nystagmus usually indicates a central (dangerous) pathology[251, 252]

- This rule is **TRUE** if and only if the patient has a single, protracted bout of dizziness lasting hours to days. Brainstem or cerebellar strokes sometimes cause dominantly vertical[148] and/or torsional nystagmus,[266] while vestibular neuritis causes nystagmus with a dominantly horizontal vector,[201] with or without a small degree of rotation[267] or trivial vertical vector[268] superimposed.

- This rule is **FALSE** when the patient has brief, episodic dizzy spells. Posterior-canal BPPV (constituting ~90% of cases[269, 270]) produces a characteristic canal-specific nystagmus[70, 71] that is reproduced at the bedside by the Dix-Hallpike maneuver.[255] The nystagmus is mixed vertical-torsional in appearance with the more dominant vector being vertical in one eye and torsional in the other.[271] So this clinical finding will fail to provide evidence of a central cause in a patient with repetitive, brief spells of dizziness lasting less than a minute.

Chapter 3

Rethinking the Approach to the Dizzy Patient — Patient Reports of Symptom Quality are Imprecise

A Cross-Sectional Study Conducted in an Acute-Care Setting

Abstract

BACKGROUND: Traditional teaching instructs clinicians to classify dizziness as vestibular if the patient reports vertigo, cardiovascular if the patient reports presyncope, neurologic if the patient reports disequilibrium, and psychiatric or metabolic if the patient reports ill-defined dizzy symptoms.

OBJECTIVE: To determine whether dizzy patients clearly, consistently, or reliably report symptom quality, and, secondarily, symptom duration or triggers.

DESIGN: Cross-sectional study (2005)

SETTING: Two urban, academic Emergency Departments (EDs)

PATIENTS: Adult ED patients (24x7 recruitment). ***Exclusions***: Too sick to be interviewed, risk to research assistant. ***Inclusions***: "Dizzy, lightheaded, or off balance" ≤7 days, or "bothered" by same previously. 5415 ED patients, 1674 screened, 872 met inclusions, 316 completed interview.

MEASUREMENTS: Description of dizzy quality elicited by four questions in different formats (open-ended, multi-response, single-choice, directed questions). Clarity assessed qualitatively (vague, circular) and quantitatively (dizzy "type" overlaps). Consistency measured by frequency of mismatched responses across question formats. Reliability determined by test-retest.

RESULTS: ***Clarity****:* Open-ended descriptions were frequently vague or circular. 62% selected >1 dizzy type on the multi-response question. ***Consistency****:* On the same question, 54% did *not* pick 1 or more types endorsed previously in open description. Of 218 subjects *not* identifying vertigo, spinning, or motion on first 3 questions, 70% endorsed "spinning or motion" on directed questioning. ***Reliability****:* Asked to choose the single best descriptor, 52% picked different response on "retest" ~6 minutes later. Relative to dizziness quality, reports of dizziness duration and triggers were non-overlapping, internally consistent, and reliable.

LIMITATIONS: Lack of clinical diagnosis data.

CONCLUSIONS: Descriptions of dizzy quality are often vague and overlapping, internally inconsistent, and unreliable, casting doubt on the validity of the traditional approach to the dizzy patient. Alternative approaches, emphasizing "timing and triggers" over "type," should be investigated.

Introduction

Dizziness accounts for 5% of walk-in-clinic[90] and 4% of Emergency Department (ED)[92] visits, making it a "top 10" chief complaint across ambulatory care settings. Among key symptoms reported in general medical clinics, it is third most common.[91] Some consider dizziness the most difficult symptom to diagnose,[15] in part due to myriad possible causes — in one study, 46 different diagnoses were given to 106 patients.[3] Failure to recognize dangerous causes (e.g., arrhythmia, stroke) can have life-threatening consequences[75, 232] and this risk is greater in the ED[141, 194] than the outpatient setting.[142] Dizziness is the symptom most often associated with a *missed* ED diagnosis of stroke.[204]

Even among those with non-urgent causes such as benign paroxysmal positioning vertigo (BPPV), misdiagnosis can lead to unnecessary diagnostic testing and treatment delays.[203]

Extensive laboratory testing and imaging studies might suffice to exclude dangerous causes in most cases, but this approach is not practical. Neither blood tests (e.g., blood count, electrolytes, glucose), nor imaging studies (e.g., MRI brain), are cost-effective when applied indiscriminately to the evaluation of dizzy patients.[198-200] Bedside assessment emphasizing careful history-taking and specialized examination techniques has been touted as the best means to identify those in urgent need of additional testing,[7, 55, 198, 201], but prospective studies are lacking.[15, 198, 202]

In 1972, Drachman and Hart's landmark paper, "An Approach to the Dizzy Patient" [19] defined four "types" of dizziness: vertigo, presyncope, disequilibrium and "ill-defined lightheadedness" (also called "vague" or "non-specific" dizziness). Since that time, the patient's qualitative description of dizziness has been thought to reflect the underlying cause.[1] This "quality-of-symptoms" approach (Figure 3.1) is used widely in clinical practice (*Chapter 2*) and cited frequently in the medical literature across disciplines[1, 7, 12, 15, 26, 28, 30-32, 141, 198, 248] with occasional modifications (*see Figure 3.1, legend*). Although commonly used, the quality-of-symptoms model has not been adequately validated, particularly in frontline healthcare settings.

Advances in vestibular science over the past several decades have cast doubt on whether an approach relying heavily on symptom *quality* (as opposed to other symptom dimensions such as episode duration, provocative factors, etc.) will yield accurate diagnosis. In specialty clinics, symptom quality does not differentiate vestibular from psychiatric causes.[58, 272] In the ED, stroke has equal odds of being associated with

"vertigo" as the more non-specific complaint of "dizziness." [116] When offered standard options to describe dizziness, 7% of ambulatory patients cannot classify their symptoms at all,[30] and the majority of older patients report symptoms in two or more categories.[33, 51, 229] In general, these findings have been viewed as lamentable, yet tolerable, shortcomings of the quality-of-symptoms approach[15] or a marker of complexity inherent to evaluation of geriatric patients.[33, 51] However, recent reports have called into question fundamental tenets of the traditional approach, including the notion that true, spinning vertigo never results from primary cardiac disease.[75]

Brief, recurrent episodes of dizziness are believed to imply a starkly different differential diagnosis (e.g., BPPV, transient ischemic attack [TIA]) than a single, acute, prolonged bout (e.g., vestibular neuritis, cerebellar stroke).[55] Once a limited differential based on duration is defined, dizziness triggers are thought to differentiate key disorders (e.g., BPPV vs. TIA).[75] An alternate approach to diagnosing the dizzy patient emphasizing "timing and triggers" over "type" has been proposed,[253, 273] but not validated.

As part of a broader effort to develop a comprehensive, evidence-based approach to bedside diagnosis of "the dizzy patient," we began by focusing on the history. We sought to clarify the *potential* diagnostic value of different symptom dimensions by asking whether unselected dizzy patients could clearly, consistently, and reliably report their symptom quality, duration, or triggers. Our primary hypothesis was that reports of dizzy *quality* would be unclear, inconsistent, and unreliable. Our secondary hypothesis was that reports of dizzy *duration* and *triggers* would be clear, consistent, and reliable.

Methods

Study Design, Setting, and Subjects

We conducted a cross-sectional study at two urban, academic EDs, each with ~50,000 patient visits per year (one serving a predominantly black population, the other a predominantly white population). Recruitment was "24x7" over an 8-week period in summer, 2005 (4 weeks at each ED, in series). All adult, non-Level-1 trauma patients in an ED bed were eligible for pre-screening. Patients leaving without treatment were ineligible. The study protocol, which included a HIPAA waiver for screening and oral consent procedure, was approved by the Johns Hopkins Medical Institutions IRB.

Derivation of the study population is outlined in Figure 3.2. Potential subjects were logged from "the board" and pre-screened. Patients were not eligible if <18 years old or recently enrolled in our study (<14 days). Principal exclusions were (a) frankly altered mental status (e.g., coma), (b) too sick to participate per caregivers, or (c) risk of violence or infection to research assistants (e.g., police custody, respiratory isolation, exposed blood).

Those not excluded during pre-screening were offered a structured screening interview (~5min) to determine their chief complaint, whether they were dizzy, and whether dizziness was germane to the visit. We defined dizziness broadly in order to study the diagnostic approach to the *undifferentiated* dizzy patient. Patients with any complaint of "dizziness" in the previous seven days, or "bothered" by dizziness in the past met inclusion criteria. For a recent complaint we asked, "Have you been dizzy, lightheaded, or off balance in the past seven days?" Those endorsing dizziness indicated if this was "part of the reason" or "the main reason" for the visit. Those denying recent

dizziness were asked, “Have you ever been *bothered* by dizziness, lightheadedness, or being off balance?” (an entry criterion adapted from a previous study[274]).

To guard against bias in symptom reporting, patients were told the study was about “symptoms in the Emergency Room” and were masked to the primary study focus (dizziness) using six other medical symptoms as distracters. Questions were formatted similarly for all seven symptoms (dizziness, dyspnea, chest pain, abdominal pain, back pain, neck pain, and headache) and randomly ordered. For example, for chest pain, we asked, “Have you had pain, pressure, or tightness in the chest in the past seven days?”

After screening, additional patients were excluded (Figure 3.2) for impaired mental state, visual impairment, illiteracy, non-fluent English, or other reasons that precluded full and active participation in the detailed interview (~30–45min) that followed.

Sample size was determined by outcomes related to a longitudinal study of long-term morbidity and mortality in these patients. The sample achieved (n=316) was adequately powered on the primary hypothesis for this study (i.e., that reports of symptom quality are unreliable). Assuming a point estimate of 50% test-retest reliability, a sample of 300 subjects provides 93% power to detect an upper 95% bound of 60% and >99% power to detect an upper bound of 75%.

Study Procedures

The initial interview segment was a detailed, dizziness-specific module (~15min) conducted by one of 13 research assistants using a tablet PC-based, adaptive questionnaire running on a modified version of a commercially-available software package — Digivey Survey Suite CSR™ v2.3 (Phoenix, AZ). A portion of the interview

was self-administered by the patient under the assistant's supervision. Data collection was paperless, and mis-click rates were calculated using responses to a binary question with a verifiable answer (male vs. female).

The complaint-specific history about dizziness included open-ended, multiple-choice, and directed (yes-no type) question formats. Multiple choice questions either allowed multiple responses (multi-response), or limited patients to a single response (single-choice). Descriptions of dizzy quality were elicited by questions in all four formats: open-ended (verbal description of dizziness), multi-response (six descriptors, pick all that apply), single-choice (six descriptors, pick the best), and directed questions about vertigo. Clarity was assessed qualitatively (vague, circular) and quantitatively (dizzy "type" overlaps). Consistency was measured by frequency of inconsistent responses across different question formats. Reliability was based on test-retest comparison.

Patients were first asked, "People use words like 'dizzy' to describe a lot of different things — what do *you* mean when you say you've been dizzy, lightheaded, or off balance?" Patients giving "off target" responses were pursued with structured follow-up. A digital audio recording of responses was made using the tablet PC, and transcribed immediately post-interview by the research assistant. Responses were later coded into categories by one of the authors (LMG) for quantitative analysis (*see Appendix 3.1 for sample responses and coding*).

We gave patients six options to describe their dizziness: (1) "spinning or vertigo," (2) "about to faint or 'fall out'," (3) "unsteady on my feet," (4) "dizzy," (5) "lightheaded," and (6) "disoriented or confused." We chose six categories instead of the

traditional four types in order to conduct analyses with the subtypes of "non-specific" dizziness (i.e., options 4, 5, and 6). Descriptors were presented in random order on a single screen. Subjects were offered the option to choose more than one (multi-response), then were asked to pick the "best" (single-choice) if they selected more than one ("Test"). After additional questions, response options were randomly re-ordered and subjects were again asked to choose the "best" ("Retest").

Because some authors[54, 275, 276] point to the presence of "true vertigo" as the most important qualitative distinction, those *not* choosing "spinning or vertigo" as the "best" were asked, "When you are feeling 'xxx,' do you have a sense of motion or spinning?" (where 'xxx' was their "best" choice, e.g., "lightheaded"). Response options were "Yes, definitely," "Yes, sort of," or "No, definitely not." Any patient endorsing spinning or vertigo on either the first "best" or on the "spinning or motion" question was asked to clarify what was moving, using a multi-response question for which one option was "the room is spinning" (*Appendix 3.2*). Again, because definitions of "vertigo" are controversial,[9] we used two different standards for "vertigo" — one sensitive (any sense of spinning or motion with their dizziness), and one specific (["yes, definitely" spinning or motion ***OR*** prior best choice "spinning or vertigo"] ***AND*** "the room is spinning").

Multiple parameters were recorded about dizziness, including details of timing (e.g., newness, episode duration and frequency), triggers (e.g., standing quickly, head motion), severity, and associated symptoms. Demographic variables included race and ethnicity, selected by subjects from a list of options according to NIH guidelines.

Specific timing and trigger questions were asked in ways that paralleled questions about symptom quality, for subsequent comparison regarding clarity, consistency, and

reliability. For example, for episode duration clarity (overlap), we asked a six-item, multi-response question, "How long does ONE complete 'xxx' spell last? You may choose more than one:" (where, again, 'xxx' was replaced by their "best" descriptor, e.g., "lightheaded"). Response options were "less than 10 minutes," "10 minutes – 1 hour," "1 hour – 1 day," "1 day – 2 weeks," "2 weeks – 6 months," and "longer than 6 months." Categories were chosen to approximate clinical episode duration groupings thought to distinguish different underlying etiologies for dizziness.[54, 75, 277]

Data Analysis

Verbal responses were mapped onto our six dizziness categories to mirror the multi-response quality-of-symptoms question. For part of the analysis (Figure 3.3), these six categories were collapsed into the traditional four-type schema, applying "dizzy," "lightheaded," and "disoriented or confused" to the "non-specific dizziness" group (Type 4), in keeping with the original criteria. Because classification of the term "lightheaded" remains controversial, additional analyses were conducted applying "lightheadedness" to the "presyncope" group.

For "disequilibrium," we did not exclude patients who endorsed other symptom types. Drachman & Hart originally used "disequilibrium" only if patients had balance problems in the absence of "other head sensations [of dizziness]." In their model, *a priori*, anyone with "overlap" symptoms was not classified as having disequilibrium. For this analysis, however, we felt it important to demonstrate the overlap as other investigators have previously.[21, 24]

Subgroup analyses were conducted to test for significant demographic heterogeneity in our primary hypothesis results. NIH race categories were mapped to

mutually-exclusive groups (black only, white only, other). We analyzed whether relevance of dizziness to the visit influenced the primary results. Because the dizziness "main reason" group was small, we further analyzed this variable dichotomized as "part of" or "not part of" the reason for the ED visit.

Analyses of other dizzy symptom dimensions were conducted to test our secondary hypothesis. Because of length and redundancy limitations in interview instrument design, data gathered for timing and triggers were less extensive than for quality, and comparisons were partial. We compared (a) *Clarity*: proportion of patients reporting >1 category in a six-option, multi-response format (quality vs. duration); (b) *Consistency*: proportion of patients failing to select a category (multi-response) they had previously reported in an open-ended format, and proportion of patients endorsing a response on directed inquiry they had not mentioned on prior questions (quality vs. triggers); and (c) *Reliability*: proportion of patients giving different answers to a repeated question (quality vs. duration). All secondary hypothesis comparisons were conducted using the same "n" (i.e., the subjects who completed both halves of the comparison).

Data were handled in Microsoft Excel 2003 (Redmond, WA). Venn diagrams were drawn by hand using Microsoft Visio 2003 (Redmond, WA). For statistical analyses, data were exported into SAS v9.1 (Cary, NC). All p-values were 2-sided, with $p<0.05$ considered statistically significant (*see Appendix 3.3 for details*).

Role of the Funding Source

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design, but was uninvolved in the collection, analysis, and interpretation of data; in writing of the report; and in the decision to submit the paper for publication.

Results

Of 1342 patients screened, 65% (n=872) were "dizzy, lightheaded, or off balance" in the past 7 days (n=677) or "bothered" by dizziness before (n=195). Among these 872 dizzy patients, 44% considered dizziness "the main reason" or "part of the reason" for the ED visit. Enrollment and completion rates were equivalent across groups (Table 3.1), and demographic characteristics were comparable (Table 3.2). Mis-click rates were low (<1%).

Open-ended descriptions of dizziness were often vague, circular, or hard to understand (*Appendix 3.1*). For example, "*Um; I think the general meaning would be the point where that woozy feeling; now I don't know how you want to describe the adjective for that; I guess woozy at that point.*" Or, "*Yes, like your head is becoming empty.*" Analogies to common experience (e.g., drunkenness) were sparse.

Responses were overlapping both within question types (unclear) (Figure 3.3, Panels A, B) and between question types (inconsistent) (Figure 3.3, Panels C, D; Table 3.3, pink panels). Test-retest responses were unreliable (Table 3.3, red panels).

We performed several subgroup analyses and found few differences by age, sex, race, education, or hospital site. Those unreliable on test-retest were slightly older (45.3 vs. 41.5 years; p=0.02), but there were no significant differences by age in category overlap (open-ended or multi-response). The only clear association between demographic variables and dizzy symptom reporting was use of the phrase "fall out" to describe fainting, used only by a subset of African Americans (27%, n=44/161).

When analyzed by relevance of dizziness to the visit (four categories or dichotomized), results were either equivalent across groups, or *worse* among patients towards the "chief complaint" end of the reason-for-visit spectrum. For example, the proportion of patients endorsing more than one type of dizziness (multi-response) increased with greater relevance of dizziness to the visit: "not dizzy" (49%), "not part of reason" (63%), "part of reason" (66%), "main reason" (74%) (p=0.01, Cochran-Armitage trend test).

By comparison to symptom quality, reports of dizziness duration and triggers were clear, consistent, and reliable (Table 3.4).

Discussion

Our data show that patients (a) lack clarity when describing dizzy symptoms and endorse more than one type of dizziness, (b) are internally inconsistent in their choices, and (c) are unreliable in their responses when forced to pick a single dizzy type. These findings question the validity of relying on the traditional quality-of-symptoms approach to diagnose dizziness in the ED.

Why is dizziness so hard to describe? Unfamiliarity of disease-related symptoms may contribute — if you have never fainted, it may be hard to know whether it feels like you are "about to faint." The brief, intermittent nature of most dizziness may not afford sufficient opportunity to focus on symptom details. Associated symptoms such as nausea, vomiting, or fear may distract from reflective consideration of one's dizziness. It may also be worth considering that, dating back to the time of Aristotle, humans were thought to have five senses.[278] The balance system ("sixth sense") went undiscovered for centuries, at least in part, because it operates below conscious perception most of the

time.[37] As a result, we may never develop the rich lexicon to describe vestibular experiences that we develop for our other senses (e.g., vision). This may make using the quality of dizzy symptoms to aid bedside diagnosis uniquely difficult.

Why is this important? The quality-of-symptoms approach is being used in clinical practice (*Chapter 2*), and may be failing physicians… and patients. Although Drachman and Hart never suggested that diagnostic investigation of dizzy patients should *end* with symptom quality, the meticulous details of their original approach have not been carried forward in the abridged rule-of-thumb — "dizziness type predicts etiology." Symptom quality has become the main focus for directing diagnostic inquiry in dizzy patients. For example, a recent academic review (drawing heavily on quality-of-symptoms principles) states, "The sensation of motion effectively removes [*sic*] the differential diagnosis from the cardiovascular into the realm of a specific neurological disturbance." [31] This statement is probably not accurate,[75] but does reflect current clinical thinking about dizziness (*Chapter 2*). Furthermore, there is some evidence that overemphasis placed on symptom quality correlates with under-emphasis on other symptom features, such as episode duration and triggers.[253] In a time-pressured environment such as the ED or busy primary-care clinic, where exhaustive diagnostic testing is not a practical option, an abridged quality-of-symptoms approach could guide physicians down the wrong path with dangerous consequences.[75] We believe our results should prompt frontline clinicians to rethink their basic understanding of dizziness, and to de-emphasize a strict reliance on symptom quality to direct their diagnostic reasoning.

What *should* be the first line of inquiry for clinicians then, when evaluating a dizzy patient? We speculate that timing (e.g., episode duration) and triggers (e.g.,

provocation by particular head movements), which have long been described as important secondary features in the diagnostic assessment of dizzy patients,[55, 198] will prove more helpful than symptom quality. Although we do not yet know the relative clinical importance of these historical elements for predicting ultimate diagnosis, at the very least, our data support the contention that these parameters are more reliably reported than quality in the ED. This empiric fact is not surprising, since symptom duration and triggers are inherently more "objective" in some sense than symptom quality. For instance, there is a certain universality and common understanding of time in the modern world — independent of language, culture, race, education level, etc… a second is still a second, a minute is still a minute, and an hour is still an hour. Whether or not this difference between quality and timing/triggers will hold true for symptoms *other* than dizziness is an open question, but studies have begun to cast doubt on the utility of other well-worn quality-based clinical heuristics, such as "burning chest pain implies a gastrointestinal cause." [279]

Limitations

We identified several potential limitations to our study, including three threats to internal validity and three threats to external validity.

Threats to Internal Validity

First, the study did not focus exclusively on patients with a *chief* complaint of dizziness. Some may contend that by admixing chief complaints of dizziness with those whose dizziness was a secondary complaint, minor associated symptom, or remote occurrence, we tainted our subject pool, invalidating our contentions about "the dizzy patient." Our data, however, do not bear this out. When analyzed by relevance of

dizziness to the ED visit, category overlap was progressively *worse* the *more* germane the symptom was to the visit (49%, 63%, 66%, 74%; p=0.01).

Second, the study focused on answers of patients, not conclusions of physicians. Perhaps doctors can better discern which dizziness type the patient is experiencing. However, this would seem unrealistic. Patients changed answers frequently, and the deeper we probed, the less clearly defined their symptoms appeared. Furthermore, evidence indicates considerable confusion among physicians about terminology and diagnostic implications of qualitative categories. Some cast a wide net around dizziness, including everyone with generalized weakness or fatigue[3] or all patients with syncope and falls,[4] while most do not. Precise definitions, which determine quality-based categorizations, vary amongst physicians. Although "lightheaded" is taken by some authors[7, 15-17, 24, 26-29] and many clinicians (*Chapter 2*) to indicate a mild version of "presyncope," others adopt Drachman and Hart's original stance that "lightheadedness" is distinctly separate from "near faint," [12, 30, 31] while others choose to deliberately avoid the term.[32, 33] Otologists and neuro-otologists (trained almost exclusively to evaluate dizzy patients) cannot agree to a precise meaning for "vertigo" — they are evenly divided whether it should describe any illusory sense of motion, or only a frank "spinning" or "turning" sensation.[9] Even among those restricting vertigo to a spinning sensation, there is disagreement. Some say it refers only to an external sense of the world spinning,[6] while others include as a subset those with spinning "inside the head." [10] This nuanced distinction is further muddied by use of the qualified terms "objective vertigo" and "subjective vertigo" to describe world-referenced and self-referenced motion, respectively.[11-14]

Third, this study does not include clinical outcomes. This should not matter, since an *unreliable* measure cannot be a *reliable* predictor of anything (unless it is sampled repetitively and averaged over numerous trials). However, in theory, it is possible that despite considerable vagueness, overlap, and self-contradiction, the initial description given by patients correlates with the actual diagnosis.[34] Studies describing dizziness symptoms in well-defined disease populations make this nearly impossible, because individual diseases produce poly-quality symptoms (Table 3.5). Furthermore, studies in humans conducted under controlled conditions suggest that (oversimplified) etiologic inferences drawn from dizziness quality are fundamentally misguided. If it were true that cardiovascular disease produced exclusively "lightheadedness" or "presyncope," but not "vertigo," we should expect this to be so when patients with orthostatic intolerance undergo tilt table testing, but this is not the case.[83] Likewise, if it were true that vestibular disease produced exclusively "vertigo" but not "lightheadedness," "presyncope," or other "non-specific" dizzy sensations, we should expect this to be so when patients undergo caloric irrigation, but this is not the case either.[280-282] Put simply, a *specific* etiology does not predict a *specific* symptom quality. Consequently, if any meaningful inferences could be drawn from symptom quality *back* to etiology, simple bedside rules would certainly not suffice — one would need a complex mathematical prediction model.

Threats to External Validity

First, the study was conducted in two busy, urban EDs. One might argue that patients in this clinical setting are too beleaguered to respond reliably to *any* questions at all, and that the results should not be extrapolated to "calmer" healthcare sites (e.g.,

primary-care office). But relative consistency in reports of duration and triggers make this an implausible contention.

Second, the study was conducted in only one city. However unlikely, it is conceivable that our results are not generalizable to other geographic regions. Against this argument, we found no significant differences in results between hospital sites, despite the fact that they serve demographically-distinct populations.

Third, the study focused on English-speaking Americans. It is theoretically possible that those speaking other languages might be better equipped to describe their dizziness. However, the current schema relies on a link between particular *American-English* words (or concepts) and underlying medical causes. But these words do not necessarily have comparable synonyms in other languages[283] or, for that matter, international English. For example, the use of particular "dizzy" words or phrases such as "giddiness" or "funny turns" is largely restricted to the British Commonwealth,[21, 284-286] and these terms lack clear parallels in the four-type approach.[287]

Conclusions

Thus, despite its limitations, we believe our study presents a compelling case that emphasizing the quality of dizziness symptoms to inform diagnosis is likely flawed, at least when applied in abbreviated form in a frontline healthcare setting. Future research should assess whether a revised approach to the dizzy patient, emphasizing "timing and triggers" over "type," will yield accurate diagnosis. In the meantime, the quality of the patient's dizzy symptoms should be given less diagnostic weight than it presently enjoys.

Table 3.1 Triage characteristics, rates of enrollment, and rates of completion by relevance of dizziness to the ED visit

Characteristics of patients screened and enrolled, by the extent to which dizziness was part of the reason they came to the ED. As expected, patients reporting greater relevance of their dizziness to the ED visit were more likely to have been coded by triage nurses as "dizzy" ($p<0.001$), and more likely to be coded as "dizzy" now or in the past ($p<0.001$). In addition to these expected differences across groups, there was a trend towards lower mean triage acuity scores (i.e., higher acuity) towards the "dizzy, and the main reason" end of the ordinal groupings ($1/10^{th}$ of a triage acuity level per transition from one group to the next). This latter finding may well reflect true triage acuity coding patterns (i.e., "dizzy" as a reason for visit is given a higher triage acuity rating than average for the group).

Table 3.1 Abbreviations and footnotes

ED – emergency department

* Triage acuity was scored on a 4-point scale with Level 1 representing the sickest patients. Note that triage data were not available for all patients (~10% of triage sheets could not be located; ~2% of interviews were truncated prior to gathering triage data; and ~2% had missing or illegible acuity scores or complaints).

† There were 339 dizzy enrollees, but only 316 completed questions relevant to the primary hypothesis.

‡ Patients for whom dizziness was <u>not</u> part of the reason for ED visit were asked dizzy quality questions if they endorsed being "bothered" by dizziness in the past. There were also a small number of patients (n=7) who initially endorsed being dizzy, but were later considered "not dizzy" by Research Assistants. We believe these were likely patients who recanted their original statements, but it is also possible some were mis-click data entry errors.

§ One-way ANOVA for difference between groups; linear regression for linear trend: slope -0.10, 95% CI (-0.14, -0.06), $p<0.001$

‖ Chi-squared test

¶ Cochran-Armitage trend test

** Fisher's exact test

Table 3.1 Triage characteristics, rates of enrollment, and rates of completion by relevance of dizziness to the ED visit

	Is dizziness part of the reason for the ED visit? (n=1342)	Not dizzy in the past 7 days (n=665)	Dizzy, but not part of the reason (n=290)	Dizzy, and part of the reason (n=328)	Dizzy, and the main reason (n=59)	*P* Value
Patients Screened	**mean triage acuity* (n)**	2.5 (571)	2.4 (237)	2.3 (291)	2.2 (50)	**<0.001**§
	% coded "dizzy" at triage* (n/n)	1.0 (6/577)	3.0 (7/235)	12 (36/298)	47 (24/51)	**<0.001**‖¶
Enrollees	**% of those screened enrolling (n/n)**	42 (279/665)	38 (109/290)	43 (140/328)	42 (25/59)	**0.56**‖
	% "dizzy" now or in the past (n/n)	26 (72/279) ‡	94 (103/109) ‡	99 (139/140) ‡	100 (25/25)	**<0.001**‖
	% completing dizzy quality description† (n/n)	94 (68/72)	92 (95/103)	94 (130/139)	92 (23/25)	**0.90****

Table 3.2 Triage, demographic, and record characteristics by relevance of dizziness to the ED visit

Characteristics of patients completing dizziness interview, by the extent to which dizziness was part of the reason they came to the ED. In addition to the expected trend across groups (fraction coded as "dizzy" at triage), there were more audio files lost among "dizzy, and the main reason" patients than in the other groups (p=0.02); we could identify no explanation for this finding.

Table 3.2 Abbreviations and footnotes

ED – emergency department

* Triage acuity was scored on a 4-point scale with Level 1 representing the sickest patients. Note that triage data were not available for all patients (~10% of triage sheets could not be located; ~2% of interviews were truncated prior to gathering triage data; and ~2% had missing or illegible acuity scores or complaints).

† "Off target" responses to initial open-ended questions were those that required follow-up questions to elicit a more substantive response. For instance, if patients gave a very meager or limited response, such as "I'm just dizzy" they were probed further with additional questioning designed to draw them out.

‡ Kruskal-Wallis test

§ One-way ANOVA

‖ Chi-squared test

¶ Cochran-Armitage trend test. As expected, as patients reported an increasing relevance of their dizziness to the ED visit, an increasing proportion of them were coded by triage nurses as "dizzy" ($p<0.001$).

** Fisher's exact test

Table 3.2 Triage, demographic, and record characteristics by relevance of dizziness to the ED visit

	Is dizziness part of the reason for the ED visit? (n=316)	Not dizzy in past 7 days, but bothered by dizziness in the past (n=68)	Dizzy, but not part of the reason (n=95)	Dizzy, and part of the reason (n=130)	Dizzy, and the main reason (n=23)	*P* value
Triage Characteristics of Completers	**mean triage acuity* (n)**	2.4 (59)	2.3 (76)	2.2 (119)	2.2 (19)	**0.20**§
	% coded "dizzy" at triage* (n/n)	1.7 (1/59)	2.6 (2/76)	13 (16/119)	58 (11/19)	**<0.001**‖¶
Demographic Characteristics of Completers	**mean age (range)**	43 (19–78)	44 (18–86)	44 (18–88)	50 (20–87)	**0.33**‡
	% female (n)	54 (37)	65 (62)	56 (73)	61 (14)	**0.46**‖
	% white (n)	51 (35)	40 (38)	41 (53)	39 (9)	**0.33****
	% black (n)	46 (31)	49 (47)	52 (68)	61 (14)	
	% other race (n)	2.9 (2)	11 (10)	6.9 (9)	0.0 (0)	
	% Hispanic (n)	1.5 (1)	4.2 (4)	0.8 (1)	0.0 (0)	**0.35****
	% English as a second language (n)	2.9 (2)	4.2 (4)	3.1 (4)	13 (3)	**0.21****
	median educational level attained (range)	12th grade (8th grade – graduate degree)	12th grade (7th grade – multiple graduate degrees)	12th grade (9th grade – multiple graduate degrees)	12th grade (9th grade – multiple graduate degrees)	**0.64**‡
Record Characteristics of Completers	**% "off target" responses† (n)**	10 (7)	12 (11)	10 (13)	4.3 (1)	**0.88****
	% of audio files damaged (n)	5.9 (4)	16 (15)	11 (14)	30 (7)	**0.02****

Table 3.3 Lack of reliability or consistency in ED patient reports of dizziness symptom quality

RELIABILITY: The blue zone of the table represents the Test-Retest paradigm in which patients were asked to select the "best" descriptor for their dizziness. Green shading indicates matching (reliable) responses on Retest (145/304, 48%, 95% CI 42%–53%). Red shading indicates different responses on Retest, demonstrating unreliability. Although the small n's in any individual cell in the table do not allow for certainty in point estimates, the lack of test-retest reliability is broadly distributed across symptom descriptors, and not being driven by outlier cell values.

CONSISTENCY: The orange zone of the table represents the directed inquiry about spinning or motion. The presence of vertigo is shown by two different criterion standards for "vertigo" — one sensitive (any spinning or motion) and the other specific (definite room spinning *or* initial best choice of "spinning or vertigo"). Pink shading indicates category overlap generated by directed vertigo inquiry (i.e., vertigo present by directed inquiry, but not selected as the best descriptor during either Test or Retest). Note that in further analyses of consistency, only a subset of these pink-shaded responses were considered inconsistent, depending on whether vertigo was endorsed in open-ended and multi-response questions).

Table 3.3 Abbreviations and footnotes

ED – emergency department

* Because there was some attrition of participation during the interview, and the Retest question occurred outside the confines of the dizzy quality segment of the interview, only 304 of 316 patients described in this manuscript reached the Retest point in the Test-Retest paradigm.

Table 3.3 Lack of reliability or consistency in ED patient reports of dizziness symptom quality

		REPEAT TRIAL: "RETEST" (n=304*)						ENDORSED VERTIGO	
	QUALITY "BEST" CHOICE	V (n=15)	D (n=52)	L (n=95)	F (n=50)	U (n=58)	C (n=34)	SENSITIVE CRITERIA (n=236)	SPECIFIC CRITERIA (n=88)
FIRST TRIAL: "TEST" (n=304*)	Spinning or vertigo (V) (n=20)	30.0% (n=6)	20.0% (n=4)	20.0% (n=4)	15.0% (n=3)	10.0% (n=2)	5.0% (n=1)	100% (n=20)	75.0% (n=15)
	Dizzy (D) (n=69)	2.9% (n=2)	33.3% (n=23)	36.2% (n=25)	8.7% (n=6)	10.1% (n=7)	8.7% (n=6)	81.2% (n=56)	23.2% (n=16)
	Lightheaded (L) (n=85)	1.2% (n=1)	17.6% (n=15)	54.1% (n=46)	12.9% (n=11)	9.4% (n=8)	4.7% (n=4)	72.9% (n=62)	23.5% (n=20)
	About to faint (F) (n=39)	10.3% (n=4)	5.1% (n=2)	10.3% (n=4)	53.8% (n=21)	12.8% (n=5)	7.7% (n=3)	79.5% (n=31)	33.3% (n=13)
	Unsteady on my feet (U) (n=58)	3.4% (n=2)	6.9% (n=4)	15.5% (n=9)	10.3% (n=6)	56.9% (n=33)	6.9% (n=4)	72.4% (n=42)	24.1% (n=14)
	Disoriented or confused (C) (n=33)	0.0% (n=0)	12.1% (n=4)	21.2% (n=7)	9.1% (n=3)	9.1% (n=3)	48.5% (n=16)	75.6% (n=25)	30.3% (n=10)
ENDORSED VERTIGO	SENSITIVE CRITERIA (n=236)	100.0% (n=15)	67.3% (n=35)	77.9% (n=74)	76.0% (n=38)	79.3% (n=46)	82.4% (n=28)		
	SPECIFIC CRITERIA (n=88)	46.6% (n=7)	28.8% (n=15)	23.2% (n=22)	30.0% (n=15)	29.3% (n=17)	35.3% (n=12)		

Table 3.4 Relative imprecision of patient responses with respect to "type" vs. "timing" or "triggers" of dizziness

The table shows intra-subject comparisons with respect to precision of reporting different dizziness symptom attributes. Comparisons proportions of imprecision are shown only for participants who completed a given measure both for "type" *and* either "timing" or "triggers." Not all possible comparisons (e.g., "type" vs. "triggers" on measure of clarity) were tested due to length and redundancy constraints in interview instrument design. Timing (duration) and triggers (position/motion) were more clearly, consistently, and reliably reported by patients than type (quality).

Table 3.4 Abbreviations and footnotes

* Some n's are relatively small both because of attrition during the interview, and because not all downstream questions were asked of all patients (adaptive interview contingent on prior responses). The patients responding to each question pair for a given measure (e.g., one question related to clarity on *quality*, and the other to clarity on *duration*) are partially overlapping with those subjects answering question pairs related to other measures (i.e., consistency, reliability); thus, the total 'n' (denominator) across measures sums to greater than the number of enrollees.

† McNemar test

Table 3.4 Relative imprecision of patient responses with respect to "type" vs. "timing" or "triggers" of dizziness

Comparison Measure	Type % (n*)	Timing % (n)	Triggers % (n)	*P* Value
Unclear [proportion "overlapping" on multi-response question (chose 2 or more of 6 possible categories for quality or episode duration)]	83% (175/212)	16% (34/212)	–	<0.001†
Inconsistent [proportion failing to select a category previously reported in an open-ended format (overlap for quality vs. postural/positional, head motion, etc. triggers)]	41% (9/22)	–	0% (0/22)	0.002†
Inconsistent [proportion endorsing a category on directed inquiry not mentioned in prior answers ("vertigo" for quality, "rolling in bed" for triggers)]	76% (80/105)	–	8.5% (9/105)	<0.001†
Unreliable [proportion different response on re-asking the same (quality) or similar (episode duration) question within minutes of the initial question]	60% (46/77)	19% (15/77)	–	<0.001†

Table 3.5 Overlap in dizzy symptom types as reported in disease-based studies, arranged by etiologic class and risk to patient

The table summarizes findings regarding dizziness types (vertigo, presyncope, other dizziness, off balance) reported in the medical literature in disease-specific populations. Representative benign and dangerous (D) diseases are shown across the major etiologic classes of disorders known to cause dizziness (vestibular, neurologic, cardiovascular, psychiatric, metabolic). If the traditional, quality-of-symptoms approach to bedside diagnosis were correct, we would expect particular dizziness types to cluster around each etiologic class — but this is not the case.

Note the presence of "unexpected" symptom-quality reporting both within a particular disease or clinical syndrome, and across diseases within an etiologic class (e.g., within the "vestibular" category, vestibular neuritis is predominantly "vertigo," while bilateral vestibular loss is predominantly "dysequilibrium" [*sic*]). Note that percentages in a single row often exceed 100% because patients complained of more than one type of dizziness (category overlap).

Also note differences in symptom quality reported by patients with the same disease, but being evaluated in different clinical settings. In particular, note the frequency with which rotational vertigo was reported in patients with BPPV or migraine, depending on whether they were identified from an otology clinic (~80–90%) or elsewhere (~30–40%). This indicates that referral bias is a potential contributor to perpetuating dizzy quality stereotypes in specialty settings.

Table 3.5 Abbreviations and footnotes

BPPV – benign paroxysmal positioning vertigo; (D) – dangerous; NR – not reported

* For those studies where a distinction was made, lightheadedness was generally classified with presyncope, rather than as "other dizziness" (except the row for Panic Disorder, where the study explicitly recognized it as distinct from faintness[21]); thus, these results differ from the original Drachman and Hart criteria, though represent a commonplace modern adaptation, as evident from recent work (*Chapter 2*) and in the definitions from several of studies which distinguished "lightheadedness" from other generic, non-specific forms of "dizziness."

† Studies did not differentiate patients who were solely off balance, without a "[dizzy in the] head sensation," so, again, this category is not a precise facsimile of Drachman and Hart's original criteria.

‡ In some instances, published results permitted only minimum (≥) or maximum (≤) estimates (e.g., orthostatic intolerance — authors reported only that 37% experienced BOTH a sense of motion and an impulse of falling, but not how many reported one or the other).

Table 3.5 Overlap in dizzy symptom types as reported in disease-based studies, arranged by etiologic class and risk to patient

Etiologic Class	Disease or Syndrome	Vertigo	Presyncope, Lightheaded*	Other Dizziness	Unsteady or Off Balance†
Vestibular	**BPPV** (otolaryngology clinic) (n=28) [288]	89% (vertigo)	11% (non-vertiginous, including postural dizziness & falls)		
	BPPV (Falls & Syncope Unit) (n=31) [288]	40% (vertigo)	77% (non-vertiginous, including postural dizziness & falls)		
	BPPV (neuro-otology clinic) (n=59) [49]	80% (vertigo)	NR	47% (floating sensation)	NR
	Vestibular neuritis (n=43) [23]	81% (vertigo)	NR	19% (floating & unsteady or "unclear")	
	Unilateral vestibular loss (n=126) [289]	46% (vertigo)	54% (non-vertiginous dizziness)		NR
	Bilateral vestibular loss (n=35) [290]	17% (vertigo)	NR	NR	91% (dysequilibrium)
	Autoimmune inner ear disease (n=28) [291]	25% (vertigo)	29% (lightheadedness)	NR	4% (ataxia)
	Hereditary otovestibular dysfunction (COCH gene) (n=60) [292]	≥37%‡ (vertigo)	28% (light-headiness)	28% (drunken feeling)	≥62%‡ (instability in darkness; tendency fall sideways)
	Labyrinthine fistula (D) (n=25) [293]	40% (vertigo)	44% (light-headiness)	16% (dizziness)	NR

Etiologic Class	Disease or Syndrome	Vertigo	Presyncope, Lightheaded*	Other Dizziness	Unsteady or Off Balance†
Cardiovascular	**Orthostatic intolerance** (n=90) [83]	≥37%‡ (sense movement)	88% (lightheadedness; dizziness)		≥37%‡ (impulse of falling)
	Syncope (n=77) [24]	35% (vertigo = spinning)	68% (presyncopal lightheadedness = impending faint)	4% (other dizziness)	55% (disequilibrium = losing balance)
	Myocardial infarction (D) (n=1546) [249]	8% (vertigo)	5% (faintness)	NR	NR
Neurologic	**Migraine** (neuro-otology dizzy clinic) (n=90) [20]	78% (rotational vertigo)	7% (light-headedness)	38% (to-and-fro sensations)	93% (postural imbalance & unsteadiness)
	Migraine (neurology headache clinic) (n=200) [284]	27% (vertigo)	NR	28% (giddy sensations)	
	Cerebellar stroke (D) (n=66) [148]	59% (vertigo)	≤41%‡ (non-vertiginous)		71% (gait instability)
	Basilar occlusion (D) (n=86) [50]	22% (rotational vertigo)	78% (non-vertiginous dizziness)		NR
Psychiatric	**Panic disorder** (n=57) [21]	23% (vertigo = moving, spinning, rocking)	33% (fainting = about to faint or lose consciousness)	72% (giddiness = lightheadedness or wooziness)	61% (instability = unsteady or off balance, so that you might fall or veer)
Metabolic	**Hypoglycemia (D)** (n=132) [84]	NR	69% (lightheadedness)	40% (dizziness)	69% (unsteadiness)

Figure 3.1 Traditional "quality-of-symptoms" approach to the dizzy patient

This figure illustrates the commonly-applied bedside rule that dizziness symptom quality, when grouped into one of four dizziness "types" (vertigo, presyncope, disequilibrium, or non-specific [ill-defined] dizziness), predicts the underlying cause. Frontline healthcare providers endorse this approach to diagnosis as the standard of clinical practice (*Chapter 2*).

The emphasis in diagnosis is placed on identifying dizziness symptom quality for classification as one of the four possible types. From the type, etiologic inferences are drawn, and subsequent diagnostic inquiry is shaped. The original definitions of these four categories are provided below.

The precision of this approach relies, in part, on a shared understanding among physicians of the precise meaning of these qualitative symptom descriptors. However, variations in terminology that would influence classification are commonplace among practicing physicians (*Chapter 2*). Similar variations are found across the medical literature, even in subspecialty circles (*see below*).

Drachman & Hart's Original Definitions for the Four Types of Dizziness[1, 19]

Type 1: Vertigo = "a definite rotational sensation"

Type 2: Presyncope = "a sensation of impending faint or loss of consciousness"

Type 3: Disequilibrium = "dysequilibrium [*sic*] or loss of balance without head sensation"

Type 4: Vague light-headedness = "ill-defined 'lightheadedness' other than vertigo, syncope, or dysequilibrium [*sic*]"

Modern Adaptations of the Original Drachman & Hart Definitions

Type 1: Some redefine "vertigo" more generically to include to-and-fro motion,[20] rocking,[21] or even any sense of motion relative to the environment with an impulse of falling.[83]

Type 2: Some redefine "presyncope" to include lightheadedness[16, 17, 24] or even "vertigo," "unsteadiness," and "weak spells." [18]

Type 3: Some refer to "disequilibrium" as "imbalance," [20] "postural instability," [21] or (postural) "unsteadiness." [22, 23] The category often no longer expressly excludes the co-morbid presence of "[dizzy-in-the] head sensations." [21, 24]

Type 4: Some refer to "vague light-headedness" as "giddiness," [21] "non-specific dizziness," [25] or simply "other" dizziness.[24] Some remove lightheadedness entirely from this group and place it with "presyncope." [24] Others remove to-and-fro or rocking motions from this category and place them with "vertigo." [20, 21]

Figure 3.1 Abbreviations and footnotes

ED – emergency department; ENT – ear, nose, and throat physician (otolaryngologist); PCP – primary care provider; Psych – psychiatrist

Figure 3.1 Traditional "quality-of-symptoms" approach to the dizzy patient

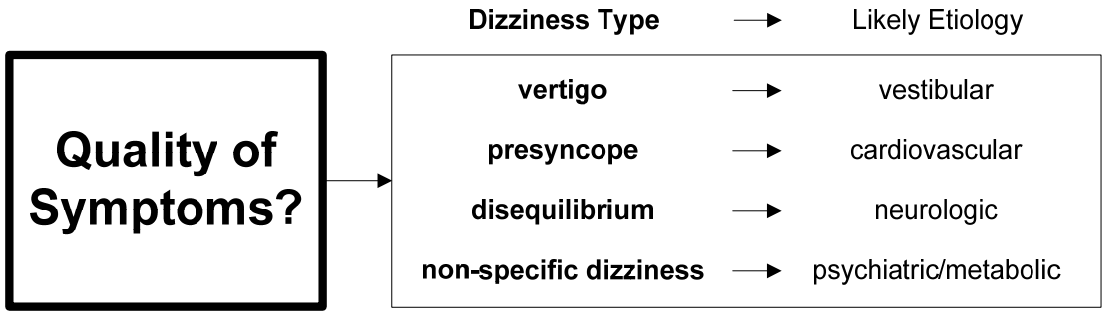


Figure 3.2 Derivation of study population for cross-sectional study of dizzy patients

The figure shows the derivation of the study population, denoting reasons for non-participation, as appropriate, during the case finding, pre-screening, screening, and enrollment processes.

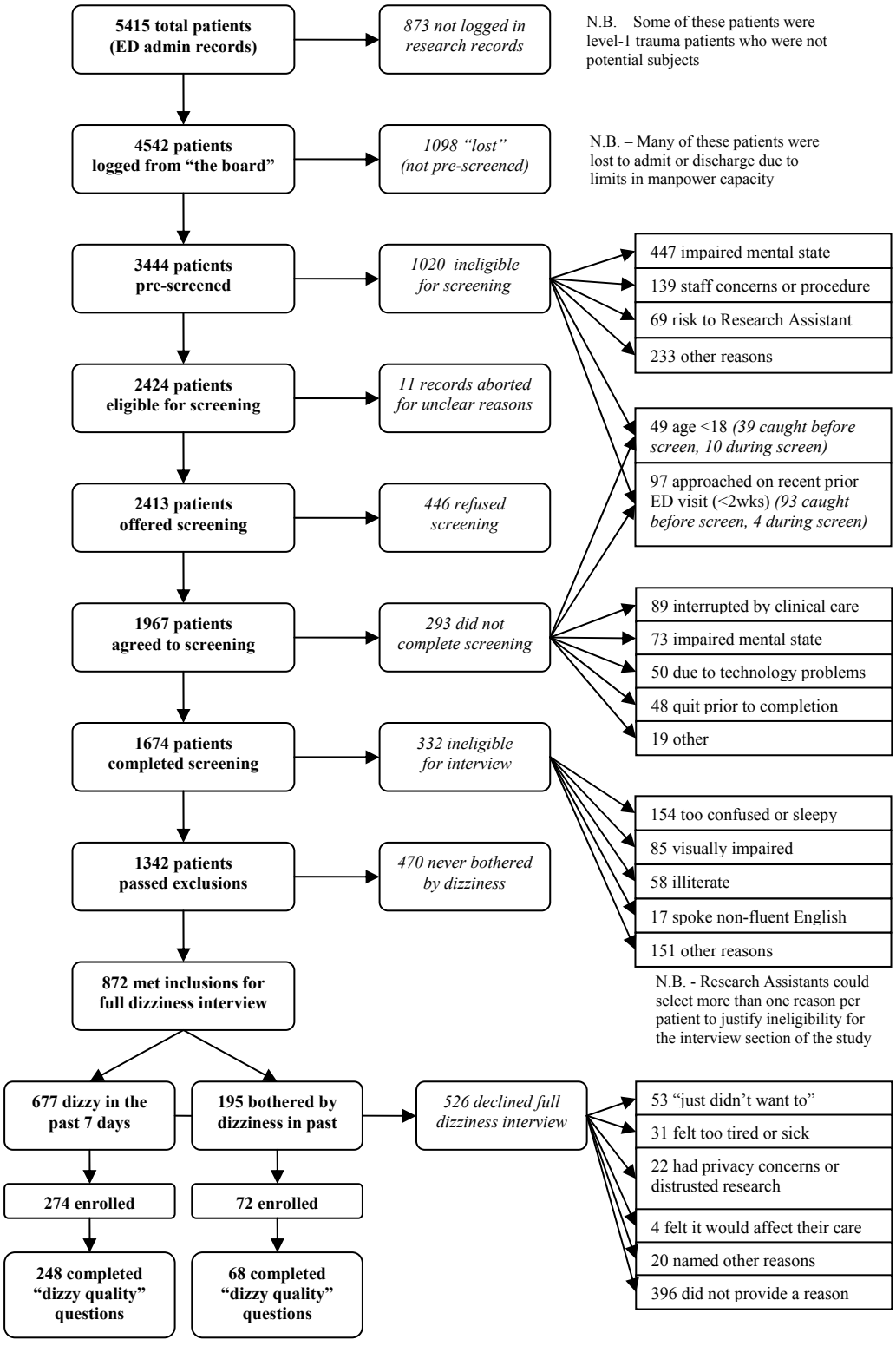


Figure 3.3 Overlap in reported "type" of dizziness by different methods of inquiry

The figure illustrates the spectrum of qualitative dizzy symptoms reported by patients, using a minor adaptation of the traditional four-type classification schema, as described in the Methods section. Shown are responses to an open ended question (Panel A), a multi-response question (Panel B), combined results from these two questions (Panel C), and these combined results, further combined with a directed inquiry about vertigo (Panel D). The left column demonstrates overlap in symptom quality reported by patients (Venn diagrams are drawn to approximate area proportionality); the right column displays population histograms for patients identifying 1, 2, 3, or 4 different types of dizziness.

Panels A and B reflect lack of "clarity" (overlap within a question), while Panels C and D reflect lack of "consistency" (overlap across questions). After adjusting for correlated data within subjects, both the proportion of overlap and mean number of types endorsed by any one individual significantly increased from Panel B to C and C to D, demonstrating a linear trend ($p<0.001$).

Figure 3.3 Abbreviations and footnotes

CI – confidence interval; SD – standard deviation

* The number of patients represented is fewer in Panel A than in subsequent panels because verbal responses from patients were recorded using a tablet PC computer, and 13% of audio files were lost for technical reasons (*see % audio files damaged in Table 3.2 for breakdown by visit reason*).

Figure 3.3 Overlap in reported "type" of dizziness by different methods of inquiry (Panel A)

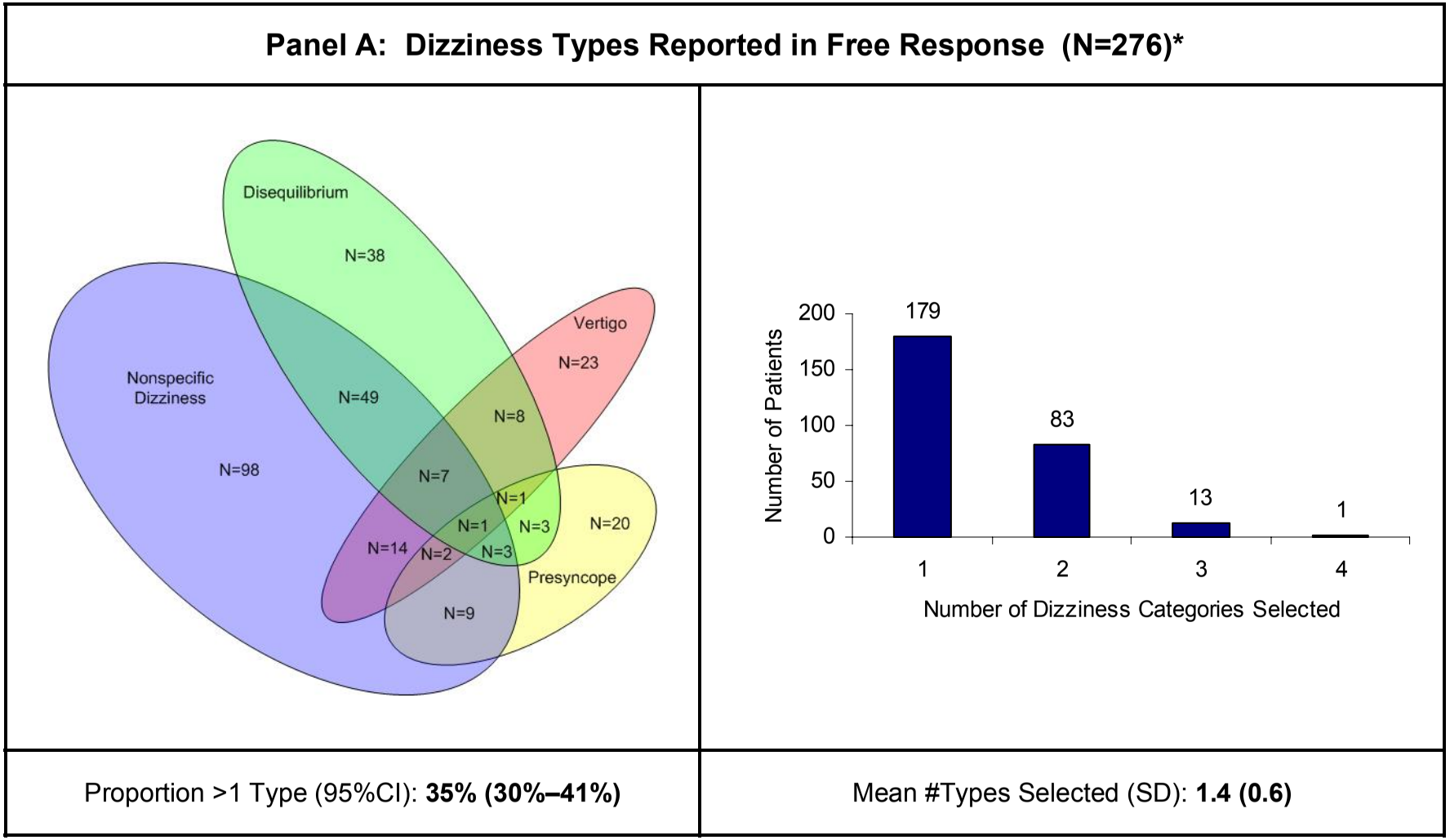
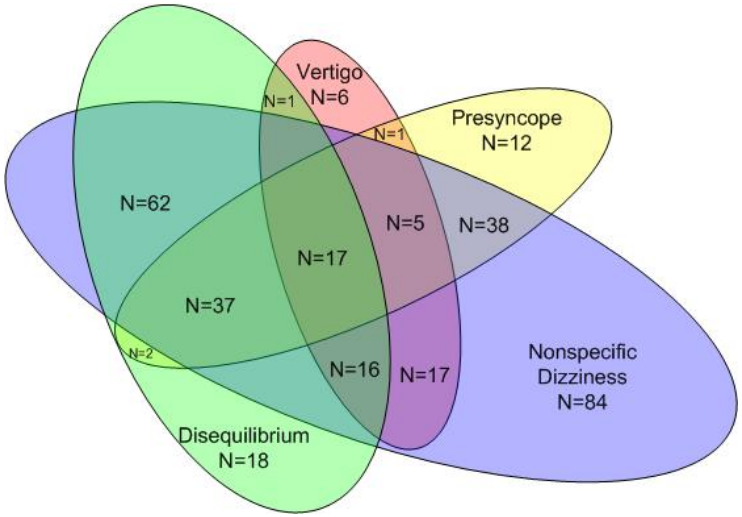
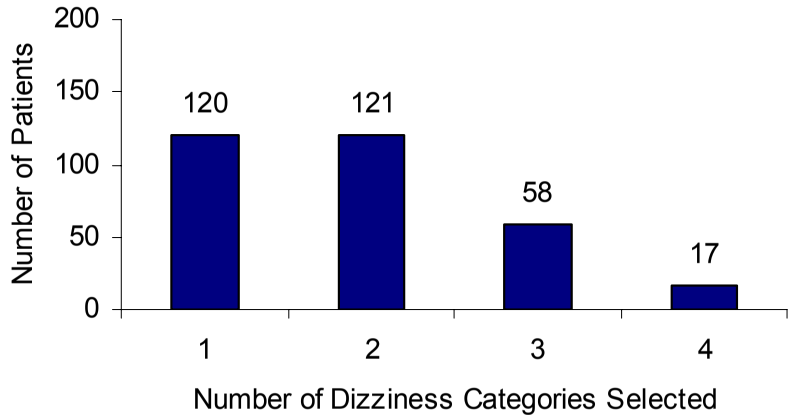


Figure 3.3 Overlap in reported "type" of dizziness by different methods of inquiry (Panel B)

Panel B: Dizziness Types Reported in Multi-Response (N=316)





Proportion >1 Type (95%CI): **62% (56%–67%)**

Mean #Types Selected (SD): **1.9 (0.9)**

Figure 3.3 Overlap in reported "type" of dizziness by different methods of inquiry (Panel C)

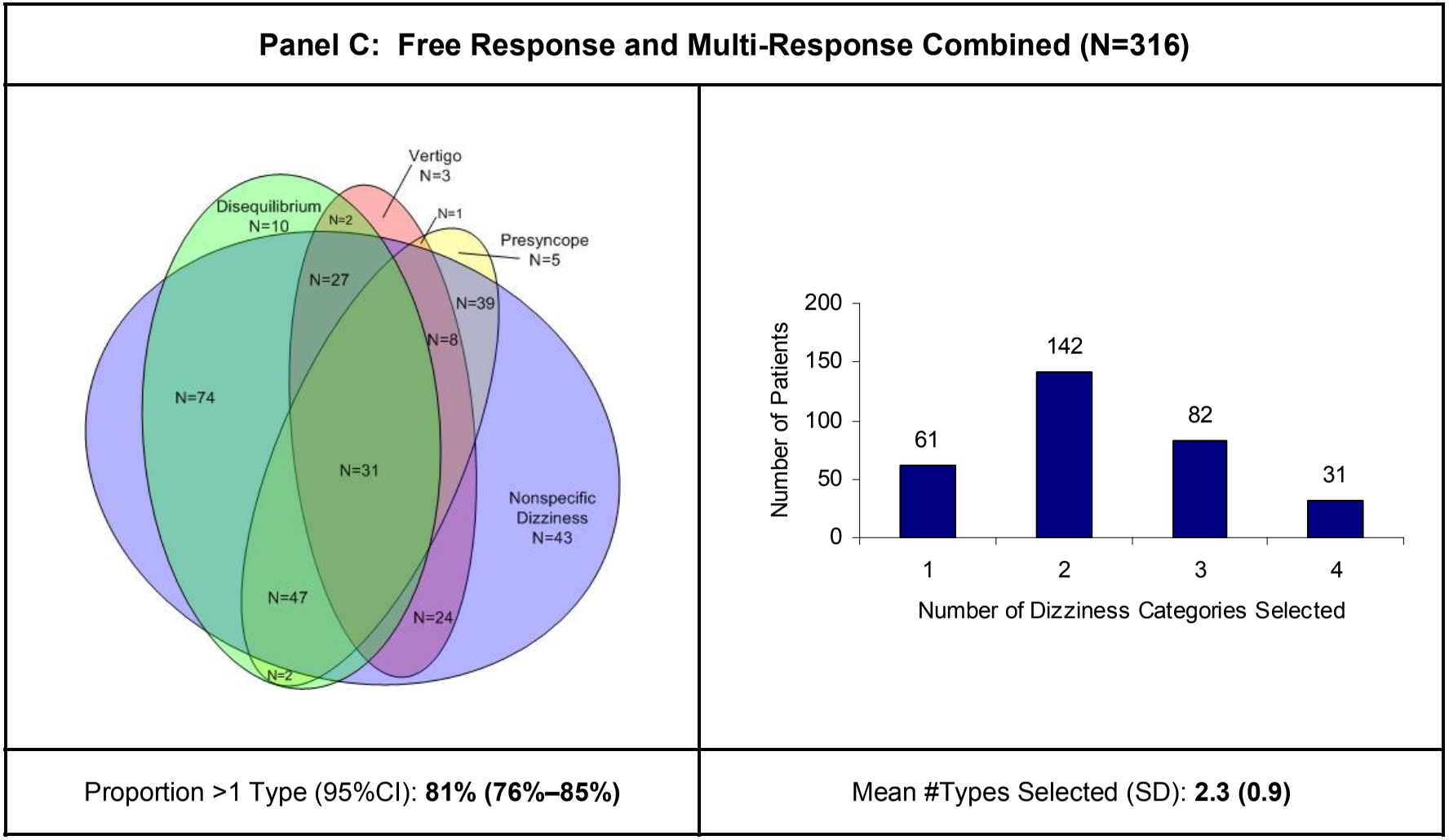
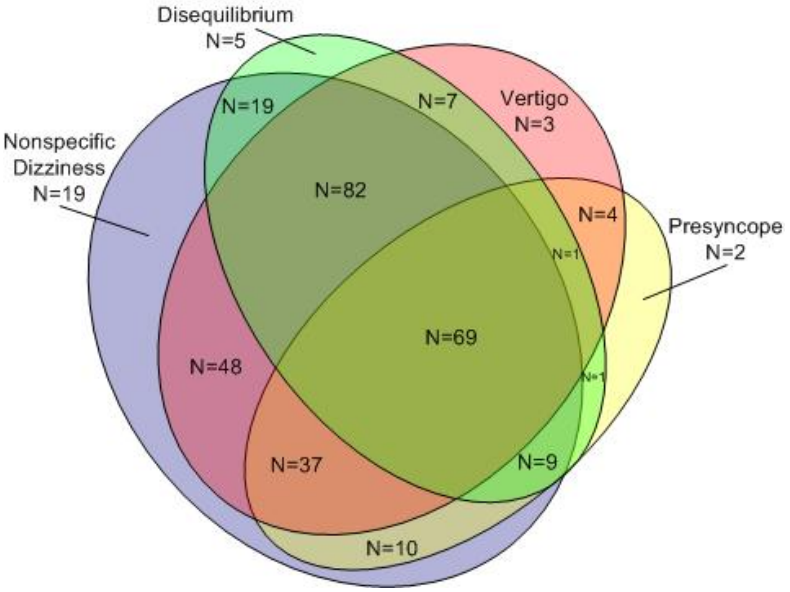
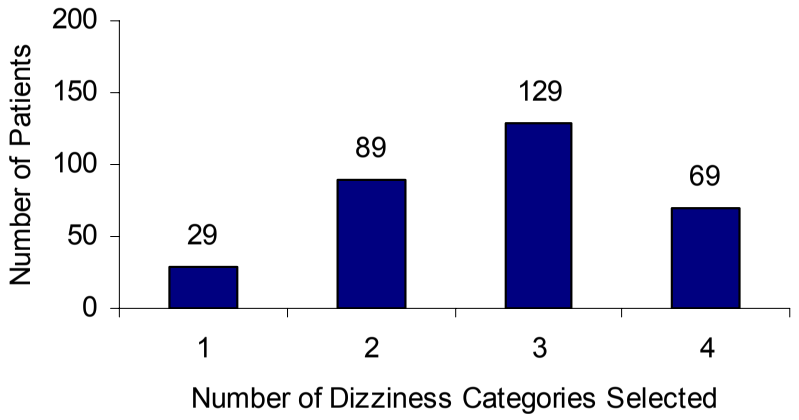


Figure 3.3 Overlap in reported "type" of dizziness by different methods of inquiry (Panel D)

Panel D: Free Response, Multi-Response, and Directed Vertigo Inquiry Combined (N=316)





Proportion >1 Type (95%CI): **91% (87%–94%)**

Mean #Types Selected (SD): **2.8 (0.9)**

Chapter 4

Towards a New Approach to Diagnosing Dizziness in Frontline Healthcare Settings

Insights from Past to Present

Abstract

Dizziness is an incredibly common symptom that appears to be misdiagnosed frequently. This is particularly an issue in the acute care setting, where the risk of dangerous underlying disorders is great, and the premium on accurate diagnosis is high.

The traditional approach for diagnosing the dizzy patient relies heavily on symptom quality to inform subsequent diagnostic reasoning, by classifying dizziness into one of four "types" (vertigo, presyncope, disequilibrium, non-specific), said to indicate the underlying cause. We have shown that this "quality-of-symptoms" approach to diagnosis is (a) in widespread clinical use, (b) predisposes to misconceptions, and (c) is fundamentally flawed.

Bedside diagnosis of "the dizzy patient" has been plagued by a dearth of strong scientific studies regarding diagnosis in unselected patient populations, particularly those derived from acute-care settings. The vast majority of dizziness research has been conducted in specialty or subspecialty clinics (including that which forms the basis of the traditional approach). Since there is evidence that referral bias may be an important source of misinformation about dizziness diagnosis, it may not be appropriate to extrapolate specialist diagnostic methods to generalist populations.

In this chapter, I discuss the historical context in which the traditional paradigm arose, and factors that may have influenced its persistence for over three decades. I also begin to explore the possibility of a new approach to the dizzy patient, emphasizing dizziness timing, triggers, and associated symptoms over dizziness type. I outline steps that might be taken to develop and validate such an approach, and offer a possible implementation strategy. I conclude with some general thoughts on the broader impact of this work on the field of symptom-oriented research.

Introduction

Dizziness is common, costly, confusing, and potentially catastrophic when misdiagnosed (*Chapter 1*). The accepted paradigm for diagnosing the dizzy patient relies heavily on symptom quality (e.g., is the world spinning vs. feeling faint, etc.), categorized into one of four dizziness "types" to direct subsequent diagnostic inquiry (*Chapter 2*). This "quality-of-symptoms" approach suggests dizziness is vestibular if the patient reports vertigo, cardiovascular if the patient reports presyncope, neurologic if the patient reports disequilibrium, and psychiatric or metabolic if the patient reports ill-defined symptoms.[1] In simpler terms, "symptom quality predicts underlying etiology." This approach is endorsed by emergency physicians both in theory and practice, and appears to drive their diagnostic reasoning to the relative exclusion of other dizziness parameters, such as timing, triggers, and associated symptoms (*Chapter 2*). However, this traditional approach rests on shaky ground in the emergency department (ED), since the assessment of dizziness quality at the bedside is plagued by imprecision, and, mathematically speaking, "nothing good can come of a bad measurement" (*Chapter 3*).

Although, in theory, measurement properties for dizziness type might differ in another clinical setting (e.g., a specialty dizziness clinic), it seems unlikely that the traditional approach could accurately inform diagnosis. Disease-based literature suggests that, even if symptom quality *could* be measured accurately and reliably, there would be no clear one-to-one relationship between symptom quality and underlying etiology. Thus, simple, rule-based diagnostic reasoning driven *largely* or *exclusively* by the quality-of-symptoms model is unlikely to yield accurate diagnosis, even under optimal conditions when dizziness "type" is more reliable. This raises an important question of whether all physicians rely equally heavily on the quality-of-symptoms approach, and whether its use presents the same potential pitfalls across clinical settings.

In Chapter 4, I will argue that the quality-of-symptoms approach to diagnosing dizziness is fundamentally misguided, but is probably used differently by generalists than specialists, and far more likely to end in adverse outcomes (i.e., serious misdiagnosis) in frontline healthcare settings, compared to specialty clinics. I go on to suggest an alternative model for diagnosis, and detail the steps that might be taken to develop and validate such an approach in the future.

Part I, "Diagnosing Dizziness Today — How did we get here?" begins with an historical consideration of the context in which the quality-of-symptoms model arose, and speculation on why, absent a strong scientific foundation, this approach might have gained such broad acceptance. I turn to the issue of the model's diagnostic role in generalist versus specialist settings, and the consequences its use might have in each. Finally, I touch on the broader impact this paradigm may have had in shaping the way we

frame clinical research questions and in setting the classification boundaries around diseases associated with dizziness.

Part II, "Diagnosing Dizziness Tomorrow — Where do we go?" begins with a discussion of the goals and expectations that attend any proposal for a new approach to dizziness diagnosis in frontline care settings. I then briefly describe the inferential and scientific foundation for an alternative approach, focused on timing, triggers, and associated symptoms. I provide examples of how such an approach might be instantiated. I go on to discuss possible strategies for validation, acknowledging potential pitfalls, and, finally, conclude with a possible strategy for implementation in the acute care setting.

Part I: Diagnosing Dizziness Today — How did we get here?

The study which informs our present approach to diagnosis represents one of only a dozen or so studies dedicated to diagnosis of "unselected" dizzy patients published in the English language literature.[142] It was conducted in a specialty referral clinic more than three decades ago,[19] prior to the advent of modern neuroimaging. Although elements of the model can be traced back even farther,[294] it was Drachman and Hart's landmark 1972 study[19] which ultimately consolidated the quality-of-symptoms approach and launched it to prominence. Since that time, until now, this paradigm has gone largely unquestioned, and the article has been cited more than 150 times (ISI Web of Knowledge Cited Reference Index, January, 2007).

Origins of the Quality-of-Symptoms Approach

To understand the foundations of this approach, it is instructive to review the historical context and some specific details related to Drachman and Hart's original

study. At the time, dizziness was very poorly understood, and, in many cases (except perhaps those complaining of spinning vertigo), was believed to defy diagnosis. Almost all extant literature had focused on specific dizziness subpopulations[19] (e.g., those with vestibular disorders[294] or cerebrovascular ones[50]). Drachman and Hart[19] framed this problem as follows (D&H p323-4):

> *Although dizziness is a nearly ubiquitous complaint, few physicians other than otologists have devoted more than passing interest in recent years to the evaluation of this symptom. The attention of otologists has been focused on the vestibular disorders... Considerably less effort has been directed to the evaluation of other medical problems that may present with a similar complaint of dizziness. These include neurological, cardiac, psychiatric, hematologic, vascular, ophthalmological, and other disorders, many of which are well understood but are not as closely identified with the complaint of dizziness.*
>
> *The need for a more systematic approach to the diagnosis of dizziness prompted the development of a model "dizziness clinic" designed to bring to bear the insights of several medical specialties, using an organized method of collecting data on each patient. The goals of the clinic, in addition to improving the accuracy of individual patient diagnosis, were to accumulate information regarding the frequency of the various disorders subsumed within this complaint; to learn the most useful distinguishing features (historical, physical, and laboratory), or "profile" of each of the disorders; and ultimately to derive a "least moves" strategy for future accurate diagnosis of the causes of dizziness. It was also hoped that the lessons of this "complaint-oriented" clinic approach might serve as a paradigm in other areas.*

This was a lofty and laudable goal, and one that Drachman and Hart, to a certain extent, achieved. They did create such a clinic, and did manage to "diagnose" the vast majority of patients they reported on in their manuscript. The fact that they were able, through a systematic, four-half-day battery of tests, come to *any* diagnosis at all, was, at that time, a remarkable feat. This point was not lost on the authors, whose first statement in their Comment was, "The most striking observation of this study was the unexpectedly high

proportion of patients in whom a reasonably secure diagnosis could be reached: 91% of those who completed the battery of tests."

Whether these diagnoses were accurate or not remains an open question. Although a major advance for its time, the study was constrained by limitations to scientific knowledge of that era. For example, certain diagnoses now known to be common causes of dizziness (e.g., vestibular migraine[295]) did not yet "exist," so were not considered possible diagnoses. How might this have impacted study results? In the absence of a plausible alternative, patients with migraine, a neurologic disorder, might have been misclassified as having a psychiatric disorder. This might have occurred because vestibular migraine patients frequently experience non-vertiginous dizziness symptoms,[20, 284] and not infrequently have co-morbid anxiety or depressive disorders.[296]

In addition to *diagnoses* that did not exist, there were diagnostic *tests* that did not exist (i.e., CT, which was first used clinically circa 1974, and MRI, which arrived nearly a decade later). In Drachman and Hart's study, the only form of neuroimaging was skull x-ray, so claims about the rarity of cerebrovascular events as a cause for dizziness in these patients must be viewed with some skepticism (D&H p330: "The small percentage of patients with cerebrovascular accidents probably reflects accurately the infrequency with which a primary complaint of dizziness is due to cerebrovascular disease."). In Drachman and Hart's case series, those diagnosed with cerebrovascular causes invariably had co-morbid symptoms such as "diplopia, facial weakness and numbness, unilateral hyperreflexia or weakness of the extremities, or mild impairment of cerebellar function." Of course, this is not surprising, since the presence of such co-morbid symptoms is the only way they might reasonably have made such a diagnosis antemortem, absent

neuroimaging techniques to identify strokes *in vivo*. Drachman and Hart were presumably aware of this issue, since they cited other work indicating that "vertigo due to ischemia rarely occurs as an isolated phenomenon in the absence of neurological deficits." Unfortunately, modern imaging techniques have shown that this classical dictum is far from true.[114, 139, 194]

Issues of diagnostic accuracy notwithstanding, this study was still an important milestone on the road towards a better understanding of dizziness, and the notion of complaint-specific clinics for patients with undifferentiated symptoms has proven prescient. However, Drachman and Hart's attempt to identify "the most useful distinguishing features… and… derive a 'least moves' strategy for… diagnosis," fell somewhat short. Although many subsequent publications, including ones written by the authors themselves,[1] would cite this original study as evidence supporting the quality-of-symptoms approach, the notion that "symptom quality predicts etiology" was neither tested nor validated in Drachman and Hart's original study.

Deriving the QOS Approach — Inferential Problems and the 'Missing Link'

In the original manuscript, the approach was never explicitly described, only hinted at. It was *assumed* (and stated as a Method), that quality somehow informed diagnosis (D&H p324 "An accurate description of the patient's subjective experience of dizziness was obtained by first separating and classifying all complaints of dizziness into 4 types… Once the type of complaint had been sorted out, secondary inquiries appropriate to each type of dizziness were sought..."). Although never formally stated as a conclusion, the relationship between etiology and symptom quality was pointed out repeatedly throughout the manuscript (Box 4.1).

The study was only formally re-framed as evidence for the quality-of-symptoms approach in retrospect.[1] Unfortunately, any post-hoc conclusions about the diagnostic implications of symptom quality are thoroughly tainted by (i) revisionist history-taking, (ii) diagnostic inclusion bias, and (iii) circular reasoning.

(i) *Revisionist History-Taking*: For many patients in Drachman and Hart's study, the original verbal description of dizziness quality was reformulated by examiners on the basis of subsequent testing (D&H p331 "The dizziness simulation battery proved to be of great value in identifying accurately the type of dizziness experienced by many patients when verbal descriptions did not suffice." [underline added for emphasis]). This revisionist approach creates problems for any predictions based on the *initial* assessment of symptom quality. Any clinical prediction rules derived from these data would require that Drachman and Hart's full 9-manuever "simulation battery" be applied to each patient before deciding on dizziness type for that individual.

(ii) *Diagnostic Inclusion Bias*:[297] In situations where one is trying to accurately assess the predictive properties of an unstudied test, it is essential that the test under study be compared to an *independent* standard. In Drachman and Hart's study, a test "battery" (i.e., history, physical exam, lab tests, electrophysiology, etc.) was used to arrive at final diagnoses. When a new "test" (e.g., classifying the symptom quality into one of four groups) is being studied for its predictive value (e.g., predicting underlying etiology) compared to the battery as the reference ("gold") standard, it is crucial that the test under study not be part of the reference diagnostic battery. If the test *is* part of the reference battery, the test's predictive power will almost universally be overestimated. Unfortunately, in this case, the test (i.e., classifying the type of dizziness) *was* part of the

battery (e.g., D&H p327, “The diagnosis of a peripheral vestibular disorder was, typically, applied to a patient who complained of unmistakable rotational vertigo, frequently with nausea and at times with vomiting.” [underline added for emphasis]; D&H p328, “positional vertigo was defined as a true rotational sensation occurring only on change of position and for a brief duration (minutes)” [underline added for emphasis]).

(iii) *Circular Reasoning*: In Drachman and Hart’s study, symptom quality was used to drive downstream inquiry and decision-making with respect to diagnosis (D&H p324, “Once the type of complaint had been sorted out, secondary inquiries appropriate to each type of dizziness were sought to identify related neurological, otological, cardiac, psychiatric, gastrointestinal, visual, or other symptoms.” [underline added for emphasis]). In the presence of conflicting or overlapping results, symptom quality appears to have been the final arbiter of ultimate diagnosis (D&H p327 “Thirty-nine patients had significant peripheral vestibular disorders as a major cause of dizziness; of these, 32 had vestibulogenic dizziness alone while 7 had an additional cause of dizziness (Table 4). The hyperventilation syndrome was responsible for the second type of dizziness in 5 of these patients. They complained of light-headedness as well as vertigo.” [underline added for emphasis]; D&H p330, “There were, as previously noted, 15 patients with the hyperventilation syndrome related to underlying psychiatric disturbances. These patients are not included in the psychogenic dizziness group, however, since the mechanism of production of the dizziness, and the type of complaints, differed.” [underline added for emphasis]).

We can see how this circular approach might lead to errors in diagnostic reasoning using the disorder benign paroxysmal positioning *vertigo* (BPPV) as an

example. BPPV is now known to result from accumulation of mobile crystalline debris within the semicircular canals; its pathophysiology is well understood, allowing for strong, symptom-independent confirmation of the diagnosis by detailed bedside eye movement analysis during positional testing.[255] Using Drachman and Hart's original definition for the disorder (*see last line of subsection 'ii' above*) and applying the logic they used for hyperventilation syndrome patients (*see previous paragraph*), if a patient with BPPV were to complain of episodic lightheaded or presyncopal dizziness, we might feel compelled, *a priori*, on the basis of the "unexpected" type of dizziness symptoms, to demand the presence of a second diagnosis. However, there may be little cause to do so, given the a high frequency of *non-vertiginous* dizzy complaints encountered in patients with *confirmed* BPPV who were referred to a "Falls and Syncope Unit," (77%) rather than an otolaryngologist's office (11%).[288] These findings raise the possibility that we may be unwittingly trapped in a linguistic web of self-fulfilling prophecy when diagnosing dizzy patients.

Unfortunately for the quality-of-symptoms approach, there is one additional problem (the 'missing link'). The data in Drachman and Hart's original study, under the most generous of interpretations, provide evidence only that "underlying etiology predicts symptom quality." For a clinical decision rule to be useful, it must flow in the opposite direction, as suggested by the simpler formulation of the quality-of-symptoms approach (i.e., "symptom quality predicts underlying etiology"). However, for such a transformation to be inferentially and logically correct, there must be a fairly tight correspondence between symptom quality and underlying etiology. More precisely, in mathematical jargon, the symptoms must be conditionally independent, given each

mutually-exclusive and jointly-exclusive relevant etiology, including "unknown" etiologies. Unfortunately, there is no such tight correspondence between dizziness symptom-type groupings and underlying causes, either at the disease-specific level, or the broader etiologic-class level (*Chapter 3, Table 3.5*).

Acceptance, Dissemination, and Entrenchment of the QOS Approach

In the absence of scientific evidence, how could this approach become established and so firmly entrenched in the medical consciousness (*Chapters 1 & 2*)? I speculate that at least six ingredients were necessary: (1) an important problem; (2) an associated knowledge void; (3) scientists and clinicians eager to fill the void; (4) a simple, uncontroversial solution; (5) a dearth of symptom-oriented science; and (6) a lack of clinical skepticism. I theorize that the last two of these had the greatest impact on the longevity and pervasiveness of the quality-of-symptoms approach.

Important Problem, Knowledge Void, Eager Physicians, Simple Solution

Dizziness as a symptom and medical complaint was, and remains, incredibly common. Yet, in 1972, there was no unified approach to the assessment of "the dizzy patient." Medicine abhors a vacuum. Physicians needed a solution to the problem of dizziness and Drachman and Hart's study, for the first time, seemed to offer one. Everyone was willing to defer skepticism in the interest of patient care. The new paradigm was appealing in its simplicity and coherence with a popular idea at the time — that true, room-spinning vertigo was only associated with vestibular disorders, and non-vertiginous dizziness was caused by something else.[52]

That the approach might have taken root is perhaps no great surprise. Many failed diagnostic[298] and therapeutic[299] technologies have been initially embraced with overzealous enthusiasm. It required only mild over-reaching on the part of the original authors, and moderate over-reaching on the part of readers to conclude the quality-of-symptoms approach was a sensible one. It has been popularized by some of the most prominent educators of our time.[34] But, if it were not true, surely the scientific community would have recognized it to be false long ago, before 35 subsequent years had passed? This is where I believe the scientific community went astray.

Dearth of Symptom-Oriented Science

A tremendous amount of vestibular research has been conducted over the past 35 years. From the time of Drachman and Hart's paper to the present, more than 12,000 research abstracts have been catalogued in PubMed (searching the part words "vestibul*" OR "labyrinth*" in the title). Yet there are fewer than 3,000 research abstracts with the part words "dizz*" or "vertig*" in the title, and only a dozen or so clinical research studies addressing diagnosis of the undifferentiated dizzy patient.[142] The vast majority of vestibular research, whether basic science or clinical, has been disease-oriented, rather than symptom-oriented.[15] In the process of conducting such disease-based research, investigators have sought to define tight, homogeneous populations of patients. Unfortunately, in so doing, vestibular science has produced few robust insights as to the utility of various historical, physical examination, or laboratory parameters in prospectively assigning diagnoses to undifferentiated dizzy patients.[15, 198]

The impact of such disease-based science has been worse than neutral, however, with respect to dizziness diagnosis. Unfortunately, such disease-specific research is never

truly “silent” on diagnosis for most readers (although in most cases it should be). When a disease-based study defines the clinical or physiologic phenotype of an illness, that information is generally inverted to help inform diagnostic reasoning (“if these are the characteristics of this disease, then when I see these characteristics, they will be indicators of this disease”). This transformation is identical to the ‘missing link’ described above, and almost always unjustified[300] in clinical medicine.

There are many examples of such erroneous inference in the literature on dizziness, but I will briefly describe one. There is a bedside test of vestibular function known as the “head impulse test” that was described in patients with clear peripheral vestibular loss,[246] and has since been characterized in great detail. Its measurement properties have been studied extensively in the oculomotor laboratory, and its physiologic correlates are highly reproducible.[301, 302] Because this test has been studied almost exclusively in patients with peripheral vestibular disorders, its presence is now conceptually associated with loss of peripheral vestibular function. In the absence of data to the contrary, it has been presumed that an abnormal head impulse is therefore a clinical sign of peripheral vestibular disease. Accordingly, some authors have suggested it be used as a single measure to distinguish peripheral (benign, e.g., vestibular neuritis) from central (dangerous, e.g., stroke) causes in patients with acute dizziness or vertigo.[56] Unfortunately, recent studies indicate the test does not neatly distinguish between the two disease populations, since nearly 50% of patients with central causes have an *abnormal* result,[176] and nearly 20% of patients with peripheral causes have a *normal* result.[303]

So, in the absence of rigorous, symptom-oriented diagnostic data to the contrary, erroneous inferences about the diagnostic value of dizziness types may have been easily perpetuated.

Lack of Clinical Skepticism

Bedside medicine is considered an "art" and much of what is practiced is learned through apprenticeship from experienced clinicians. Most of what is used and taught in clinical practice to inform bedside diagnosis has only longevity to commend it, since notions of "evidence-based medicine" have only infrequently crossed the divide from treatment to diagnosis.[210] Even when medical "evidence" has been brought to bear on diagnosis, it has generally been used to mathematically ascertain the value (or lack thereof) of specific laboratory or physical diagnostic techniques,[304] not the fundamentals of clinical history-taking.

In the absence of scientific "evidence" (and even in its presence) it is incumbent upon clinicians to remain skeptical and self aware, lest they be caught up in a vicious circle of self-fulfilling prophecy when they practice. Obtaining a diagnostic history from a patient is a complex process. The story may change, evolve, or be clarified during the course of an interview. More importantly, it may be revisited in light of subsequent information obtained from the physical examination (as in Drachman and Hart's "stimulation battery," described above), laboratory testing, or even longitudinal follow-up. During this process, most clinicians are entirely capable of "massaging" the patient's description of their symptoms into the "correct" category. I have seen physicians convince patients they experienced vertigo rather than lightheadedness because the physician knew the overall story sounded like vestibular disease. I have also seen

physicians convince patients (or themselves) that they did *not* experience vertigo because physicians knew the overall story sounded like a cardiovascular problem. There is nothing inherently wrong with taking this "artistic" approach at the bedside, and, in experienced hands, this strategy probably ends in a correct diagnosis most of the time. *However, it is problematic to take these "corrected" patient responses as evidence confirming the truth of a preconceived, oversimplified bedside rule*. Doing so may have contributed to the inordinately long lifespan of the quality-of-symptoms approach, and risks relegating clinical practice to remaining unscientific in the long run.

On the Differences in Diagnosis between Generalist and Specialist Settings

The quality-of-symptoms paradigm arose in a specialist setting. Even if Drachman and Hart's attempt was to create a clinic for evaluation of *undifferentiated* dizzy patients (i.e., not restricted to vertigo, etc.), there is no presumption that, in doing so, they studied an *unselected* population. Tertiary care, university referral clinics do not serve the same population as seen in primary ambulatory care clinics, nor the same as that seen in acute-care settings, such as urgent care clinics or the ED. In addition, even different specialty-based tertiary care referral clinics (e.g., otolaryngology vs. neurology vs. cardiology) serve different referral patient populations.

In specialist settings, pre-selection referral patterns generally insure (i) a narrower spectrum of causes, (ii) a narrower spectrum of symptoms, and (iii) a lower level of illness severity. When they occur in clinical research studies, these three patient selection phenomena form the basis of what is commonly known as "referral bias." As we shall see, such referral bias may adversely affect both internal validity (truth) and external validity (generalizability) of research results.

(i) A narrower spectrum of *causes* is seen in referral clinics. This phenomenon is the natural consequence of a healthcare system that uses generalist providers to help guide patients to see the correct organ-system-specific specialist for further diagnosis or treatment. For example, those dizzy patients suspected of ear disease (e.g., on the basis of co-morbid auditory symptoms and ear pain) are more likely to be sent to an otolaryngologist, while those suspected of cardiac disease (e.g., on the basis of co-morbid chest pain, palpitations, and dyspnea) are more likely to be sent to a cardiologist. In such cases where patients are *polysymptomatic*, and all the elements of the history or exam point to a single organ system diagnosis, this process is straightforward. It is self-evident that specialty clinics populated by such referrals will be enriched with a particular subset of possible etiologies (a fact which many specialists rely upon in diagnosis). Some specialists even pre-screen their referrals or accept only patients with confirmed diagnoses, in order to help insure that patients in their clinics have the "right" type of disease that they handle.

(ii) A narrower spectrum of *symptoms* is seen in referral clinics. This phenomenon is also the natural consequence of the referral triage system as described above. Here, the generalist tends to associate certain symptoms with certain organ systems (e.g., auditory = ear; chest pain = heart). These *monosymptomatic* referrals (which rest on shakier ground than the polysymptomatic cases from a diagnostic standpoint) usually only occur after the generalist has expended some energy insuring that the referral is "appropriate." This may mean additional testing, observation over time, or even assessment of response to empiric or symptomatic therapy. When a patient needs a referral for a single symptom that could be caused by many different disorders across different organ systems (e.g.,

fatigue, dizziness), the choice of referral specialist may be based upon a "best guess" strategy. For dizziness, this may mean taking the quality-of-symptoms approach — if the patient says "spinning," that means they have "vertigo," and they are sent to an otolaryngologist; if the patient says "lightheaded, like I'm about to pass out," that means they have "presyncope," and they are sent to a cardiologist. Specialty clinics populated by such referrals will be enriched with a relatively narrow subset of possible symptoms or subtypes of symptoms.

(iii) A lower level of illness severity is seen in referral clinics. This point is perhaps best illustrated by a brief personal anecdote. During elective time in my neurology residency, I worked with a world-renowned neuro-otologist in his dizziness clinic in Sydney, Australia. After about four weeks without a single case of cerebrovascular disease being diagnosed, I asked, "Where are all your stroke cases causing dizziness?" He replied, "I haven't made that diagnosis in about ten years." I briefly mused to myself that perhaps he had become jaded over the years and was no longer looking for them (or perhaps that limited access to MRI scans in the Australian healthcare system had prevented him from doing so). That afternoon, we were called to see a dizzy patient across the street in the ED, and the Professor diagnosed an acute cerebellar stroke as the cause.

This fortuitous event crystallized in my mind what is surely an obvious fact for those who think about it for even a moment — patients in the hospital setting (ED or inpatient) are sicker than those in the ambulatory outpatient setting. Initially, it was not obvious to me why this should be, but, on further reflection, the reasons became clear. First, there is the effect of illness severity on the patient's decision about how urgently to

seek care. Although patients may not always be aware of the urgency of their symptoms,[134] some symptoms either spark enough concern (e.g., chest pain; inability to speak) or are disruptive enough (e.g., trouble walking; vertigo with protracted vomiting) to force people to seek care emergently. So patients with certain dangerous illnesses that tend to produce dramatic symptoms, such as acute myocardial infarction (causing crushing chest pain) or acute stroke (causing hemiplegia), are rarely found in the ambulatory outpatient setting. Second, for those patients with symptoms that seem less urgent, there is the effect of appointment scheduling waiting times on disease selection. In the Professor's clinic, there was a 6-month wait to get an appointment. Transient ischemic attacks (TIAs) are harbingers of ischemic stroke. The greatest risk of subsequent stroke occurs within days of the initial event,[126, 127] and slowly returns to a baseline level of risk over months. So many of these patients, even if they made (or were referred for) an appointment to see the Professor about the initial symptoms, never made it to his clinic — they went to the hospital with a stroke… or directly to the morgue.

Linked to these differences in patient population across care settings are differences in goals and approach to diagnosis for physicians. In the ED, the spectrum of dizziness causes is broad,[3] the chances of acute, life-threatening pathology are high,[141, 194] and evaluations are time-pressured and oriented towards risk-stratification in pursuit of disposition decisions, rather than final diagnoses.[215] This is in stark contrast to Drachman and Hart's original study, where the spectrum of causes was limited, the risk of life-threatening pathology (e.g, cerebrovascular or dangerous cardiovascular causes) was low, and extended evaluations took four half-day clinic visits in pursuit of a final, definitive diagnosis. We shall next examine why these differences matter.

Application of the QOS Approach in Generalist vs. Specialist Settings

So what are the implications for dizziness diagnosis related to these differences in clinical setting? First, the specialist's job in diagnosis is easier and more secure. Second, if not inherently skeptical, the specialist is liable to be falsely reassured about the utility of their paradigms regarding diagnostic approach (and to pass these on to generalists as "useful" rules). Third, the generalist is at much greater risk for dangerous misdiagnoses if what the specialist tells them about diagnosis is taken too literally and applied directly to their clinical population.

(i) The specialist has the *luxury* of referral bias in assessing patient symptoms and making a diagnosis. Because much of the initial triage work to "weed out" other organ system causes has been done for them, specialists only infrequently concern themselves with diseases that "belong" to other specialists. They are able to focus on "their" diseases, and look for pattern matches according to well-defined *illness scripts*.[305] An otolaryngologist need generally not consider the possibility that a patient with spinning vertigo might harbor an underlying cardiac arrhythmia[75] or aortic dissection,[85] since it is highly unlikely that such patients would ever reach their clinic.

(ii) If specialists are not duly skeptical about their own bedside approach, they run the risk of drawing erroneous inferences about the accuracy and utility of the methods they employ. Since the spectrum of symptoms and causes is narrow in the referral setting, there will necessarily be a tighter correspondence between symptoms and disease, *purely as a function of referral bias*. In other words, patients with vertigo in an otolaryngology clinic are likely to have vestibular disease, and patients with vestibular disease in an otolaryngology clinic are likely to have vertigo. This fact is not, per se, a problem.

Patients get the diagnoses and care they deserve (as long as they were sent to the correct specialty clinic). However, when specialists write and teach, they tend, like other physicians, to draw from their own personal experience. When this occurs, specialists are liable to overvalue the diagnostic relevance of certain clinical parameters, such as symptom quality. This problem has become entirely apparent in the case of BPPV (briefly described above). It appears that the likelihood of a patient with BPPV reporting "vertigo" as their main symptom is determined not by the disease, but by the site where they have sought medical care,[288] suggesting that referral bias may be an important contributor to perpetuating dizzy-quality stereotypes.

(iii) In the acute-care setting, in pursuit of efficiency, the quality-of-symptoms approach may be abridged to a set of oversimplified clinical decision rules (*Chapter 2*). Using such *heuristics* is a time-saving cognitive strategy adopted by many physicians practicing in fast-paced clinical settings,[306] but taking this strategy comes at a price — increased risk of misdiagnosis based on biases and oversimplified reasoning.[306] In keeping with the notion of a need for heuristic simplicity, one of my neuro-otology colleagues told me the following about rules and parameters for dizziness diagnosis in frontline care settings: "If you can't fit it on a credit card, no one will ever use it." Even if specialists do not oversimplify for them, frontline providers may choose to simplify for themselves. This is presumably done in pursuit of a digestible distillate that can be used effectively in their clinical practice setting. For example, a recent academic review in the emergency medicine literature, drawing heavily on quality-of-symptoms principles, emphatically states, "The sensation of motion effectively removes [*sic*] the differential diagnosis from the cardiovascular into the realm of a specific neurological disturbance."

[31] This statement reflects current frontline thinking about diagnosing dizzy patients (*Chapter 2*) and makes for a simple, efficient, credit-card-sized rule… just one that is probably dangerously inaccurate.[75] Furthermore, when presented with unscientific rules to guide diagnostic reasoning, acute-care generalists, unlike specialists, do not have the comfortable safety net that comes from practicing in a clinical setting where nobody dies of their underlying illness after a misdiagnosis.[241] A sub-optimally tuned "first pass approximation" for diagnosis might lead a frontline provider down the wrong arm of a decision tree with potentially-lethal consequences.

Thus, although the quality-of-symptoms approach to dizziness diagnosis might be equally wrong *in theory* across healthcare venues, it is might only lead to wrong diagnoses *in practice* in generalist clinical settings, and *dangerous* misdiagnoses in acute-care locations.

The Impact of the Quality-of-Symptoms Approach

Aside from the obvious impact for individual dizzy patients who may have been misdiagnosed over the years as a result, what might the consequences of adopting a quality-of-symptoms approach have been? I believe there is evidence that this focus on symptom quality has hurt clinical science by drawing boundaries between diseases in places that do not entirely make sense (at least from the perspectives of carefully-constructed nosology and frontline diagnosis).

This issue is encapsulated by a brief exchange I had with a colleague in cardiology who sees many patients with cardiac arrhythmias, and has conducted important clinical research in this domain. I asked him, "How often do your patients with

arrhythmias complain of spinning vertigo?" His response was matter-of-fact: "I don't know. If they say 'vertigo,' I send them to *you*." In other words, his clinical approach to diagnosis is so heavily influenced by symptom quality that he has closed off his mind to even the *possibility* that vertiginous symptoms might have resulted from primary cardiac disease.

This perspective, however, is not unique to this individual. In a systematic review of the medical literature (including review of over 1300 abstracts), we were able to identify only 5 studies of patients with primary cardiovascular disorders that reported on the relative frequency of vertiginous vs. non-vertiginous dizzy symptoms (*Newman-Toker, unpublished data*). Why have so few asked this scientific question? In my opinion, it is because the quality-of-symptoms mindset is so firmly entrenched in the medical consciousness that the question does not occur to them as one that needs to be asked. This line of reasoning was articulated by Sloane,[307] when he analogized the clinical approach to dizziness to the story of the three blind men and the elephant:

> *For the practicing [clinician], making sense of the literature on dizziness is a lot like the story of the blind men and the elephant. In that story, three blind men each feel a different part of the elephant's body, and each observation provides accurate but biased information about what the elephant is like. The same is true about dizziness: no comprehensive clinical or epidemiological studies exist; instead, each published study evaluates a subpopulation of persons and suffers from certain diagnostic and inclusion biases.*

This issue of whether we have misplaced the "frame" around the problem has potential implications for disease classification, clinical research, medical education, and clinical practice. Although an extensive treatment is beyond the scope of this discussion, I will touch briefly on some of the possible ramifications.

First, from the nosologic (disease classification) perspective, we have become stubbornly narrow-minded about disease phenotypes. This is now apparent for BPPV (*described above*), but may be true for other vestibular disorders as well.[308] Misclassification of disease may contribute to misdiagnosis, leading to unnecessary diagnostic testing and delays in treatment, as has been shown for BPPV.[203]

Second, from the frontline diagnostic perspective, we have failed to cast a wide enough net around dizziness in clinical research studies. In focusing research on either vestibular workups in patients with vertigo, or cardiovascular ones in those with presyncope/syncope, we have missed the opportunity to discover the overlap. The segregation of neuro-otologic and cardiovascular research has hurt both fields, with persistent confusion about the most likely causes for common problems, such as unexplained falls in the elderly.[65, 309] In the process, patients with dangerous, acute illnesses causing unexpected symptoms may have slipped between the cracks.[75]

From either perspective, clinical science has probably been hurt by the inappropriate segregation of patients with one dizziness symptom quality from another.

Part II: Diagnosing Dizziness Tomorrow — Where do we go?

In frontline healthcare settings, the spectrum of causes for dizziness is broad and different to that seen in tertiary care referral centers (*Chapter 1*). Unfortunately, few studies of dizziness have been conducted in primary-care settings, and fewer still in acute-care settings.[15] We are aware of only three prior published English-language manuscripts that describe diagnostic studies of unselected dizzy patients in the ED (total n=352 patients),[3, 141, 228] only one of which was prospective (n=125 patients).[141] The

prospective study used the quality-of-symptoms approach to frame diagnostic inquiry, and was subject to the same issues of diagnostic inclusion bias and circular reasoning as seen in the original Drachman and Hart study (*see Part I, above*).

We have seen in Part I that, despite the well-worn status of the simple heuristics outlined in the quality-of-symptoms approach, they have never been adequately studied or validated, and their value has likely been overestimated and overstated in the medical literature. I have argued that, although the quality-of-symptoms approach has little scientific foundation to commend it in *any* setting, the consequences of adopting this diagnostic approach could be more deleterious for generalists than specialists, particularly for those generalists practicing in acute-care settings.

So what next? As one irate reviewer said when presented with the work described in Chapter 3, "It seems premature to discount current practice without having another demonstrably better method to replace it." In order to adequately answer this simple question, and address the reviewer's concern, we must first decide on a set of goals for such a "better method."

Diagnosing Dizziness in Frontline Healthcare Settings — What is the Goal?

In frontline healthcare settings, final diagnosis is an unrealistic goal for most dizzy patients.[15] In the ED, in particular, final diagnosis is almost never the goal, regardless of the symptom.[310] Instead, emergency physicians focus on what we might call "diagnostic triage." We will define *diagnostic triage* as the dynamic, iterative process of early branch-point decision making focused on practical clinical decisions such as "image or not," "observe or not," "admit or not." Such workflow-related decisions rely heavily on working-diagnostic classification, but not, per se, on final diagnosis.[311] Most

importantly, these disposition decisions revolve around risk stratification with respect to dangerous target diseases — those at moderate to high risk (mandatory testing and admission), those at very low risk (limited testing and discharge), and those in equivocal risk range (additional information required).

Given the breadth of causes and the difficulties inherent in diagnosing dizziness (*Chapter 1*), is accurate risk stratification a realistic goal for ED dizzy patients? Studies indicate that effective clinical decision rules to identify high-risk patients are possible. For example, although the prevalence of acute ischemic stroke in unselected dizzy patients is only about 3%,[116] it is known that 25% of patients over age 50 *with*... new, isolated, severe, persistent dizziness, *without*... auditory symptoms or obvious neurologic symptoms or signs …have ischemic stroke as a cause.[194] That is, these clinical findings multiply the risk by a factor of 8, to a point well above the threshold for clinical action. Another study indicates that dizzy patients whose dominant manifestation of dizziness is balance problems when walking are at 4-fold increased odds of stroke relative to dizzy patients without such a clinical presentation.[116] The presence of such risk indicators suggests that the goal of diagnostic triage is at least *theoretically* achievable. Whether it can *practically* be attained is a separate matter, and one I explore in greater detail below.

How Can We Achieve Simplicity and Efficiency… yet Maintain Accuracy?

In fairness to my colleague who believes in credit-card-sized rules, "simple" is always better than "complicated," *all other things (e.g., rule accuracy) being equal*. One can always make a rule simpler. However, the material question is, "Can you make it simpler *and still be right*?"

In the case of dizziness in the ED, this question remains unanswered. No one has yet gathered all the data that would be necessary to assess how simple (or complex) a set of rules, diagnostic algorithm(s), or mathematical/statistical model(s) is needed to adequately risk stratify ED dizzy patients. Answering this question requires detailed data on all (potentially) relevant *clinical parameters* and all (potentially) relevant *diagnoses* (or, at least, *diagnostic triage decisions*) in a representative sample of all ED patients with dizziness. It also requires a mathematical method to ascertain the relative importance of the various clinical parameters with respect to the outcomes of interest (diagnoses and/or diagnostic triage decisions), such that a limited, "simpler" set of data might be focused on by providers.

Somewhat ironically, this was essentially the solution that Drachman and Hart set out to derive in their original 1972 study. They wished "to learn the most useful distinguishing features (historical, physical, and laboratory), or 'profile' of each of the disorders; and ultimately to derive a 'least moves' strategy for future accurate diagnosis of the causes of dizziness." Other than focusing on *diagnostic triage*, rather than *diagnosis*, we wish to achieve the same end.

So how might we achieve this goal? Gathering the background clinical research data may well be laborious, expensive, and time consuming. Doing so will also require a fair amount of domain expertise on the part of observers, and rigorous methods to control for bias in data acquisition. But, although this process may be logistically cumbersome, it will not be conceptually complex. On the other hand, distilling these raw data into a "least moves" approach requires more thoughtful consideration. Fortunately, others have already done most of the work on the conceptually challenging issue of deriving

mathematically-sound decision rules from large data sets. A detailed treatment of this subject matter is beyond the scope of this dissertation, but I will briefly mention a few possible strategies.

(i) Pure rule-based approaches allow a list of simple rules to be chained together (e.g., "*if* the dizzy patient has chest pain, *then* the risk of a cardiac cause is high"; "*if* the risk of cardiac cause is high, *then* obtain an electrocardiogram"). The rules can be assigned a priority to determine the order in which they are applied. If a rule does not apply (e.g., the patient does *not* have chest pain), the next highest priority rule is tested, and so on. Rules may be determined by prior scientific knowledge, or derived from a large data set (as mentioned above) using mathematical techniques for discerning item-category relationships, such as principal components analysis.

(ii) Tree-based (algorithmic) approaches force specific decisions at each branch point (node) (e.g., "Does the dizzy patient have chest pain?"; "if yes, obtain an EKG"; "if no, ask whether the episodes were brought on by exercise"). The rules for decision making are not merely a function of the individual nodes, but are embodied in the structure of the tree (e.g., downstream questions about palpitations might only be asked if the patient had chest pain, but not otherwise). Mathematical modeling techniques, such as recursive partitioning (also called CART, Classification And Regression Trees), facilitate development and testing of tree structures from large data sets.

(iii) Network approaches define a more complex, interactive set of relationships between clinical variables. They are harder to understand, at face value, than simple rules or trees, but more closely approximate real-world decisions in their complexity. Unlike rules or simple trees, they are usually hard to draw or represent visually on a single piece

of paper, and often require a computer-based graphical interface of some sort to work with in clinical practice. Some networks (e.g., Bayesian belief networks) are built in "top-down" fashion, with known relationships between clinical variables structured as part of an influence diagram (a circuit diagram or blueprint, of sorts). Other networks (e.g., neural networks) are built in "bottom-up" fashion, driven solely by statistical relationships derived from raw data. These types of network approaches have generally been used as the framework for robust, computer-based, diagnostic decision support systems (whether generic or symptom-specific).[312]

Regardless of the precise strategy taken to derive simple decision rules or more complex, computer-based decision support systems, it is crucial to remember that results will be (a) limited in accuracy by the spectrum and quality of the data used for development, and (b) only firmly established when validated prospectively using clinical outcomes, *after initial development*.[313]

What Might a New Approach to Diagnosing the Dizzy Patient Look Like?

For it to merit serious consideration by physicians, any proposal for a new approach to the dizzy patient should be evidence-based and rigorously validated. A dearth of strong symptom-oriented research studies in unselected dizzy patients presents a significant challenge to building such an approach *de novo*. However, despite the caution that must be taken in doing so, there are still some important evidentiary insights about diagnosis that can be drawn from a growing body of disease-based studies. Furthermore, if we restrict ourselves to diagnostic triage decisions, and emphasize differentiating common, benign causes from uncommon, dangerous, emergent ones, we may narrow the problem sufficiently for it to be made tractable.

For example, now that BPPV is well understood, it is clear that symptom quality (vertigo vs. postural lightheadedness) varies from patient to patient.[288] However, it is also clear that patients have characteristic, reproducible episode triggers that spark short-lived symptoms, whose duration is easily measured, since they can be confirmed and reproduced at the bedside.[255, 288] BPPV is believed to be the second most common (vestibular) cause of dizziness in the ED.[228, 314] If this is true, then what are the dangerous mimics that also produce short-lived episodes of dizziness? When one considers this question, it conjures up a fairly short list, headed by malignant cardiac arrhythmias and brainstem/cerebellar TIAs.[75] As it so happens, these dangerous mimics, to the best of our knowledge, are almost never triggered by changes in head position.[75] Since timing and triggers are fairly reliably reported by ED dizzy patients (*Chapter 3*), we might reasonably derive a simple, duration-specific heuristic for dizziness that says "brief episodes of dizziness are likely to be benign, if-and-only-if they are triggered by characteristic shifts in head position" (*see Chapter 2, Box 2.1 for additional details*).

We can extrapolate from this example, building up additional comparisons between common, benign causes and dangerous mimics, and emphasizing disease-specific factors believed or known to distinguish the two groups. This might lead to a set of clinical decision rules that helped frame the bedside approach to diagnosis and identified patients considered "safe to go" (i.e., low risk) (*Appendix 4.1*). Alternatively, it might be framed in the form of a decision tree, building on the well-studied "level-of-sickness" paradigm for risk stratifying ED patients,[315] with downstream branch point decisions determined by critical clinical factors that distinguish between benign causes

and dangerous mimics (*Appendix 4.2*). Alternatively, the rules could be encoded as a Bayesian belief network (allowing for more complex decision-making strategies).

As a caveat, it is important to remember that the same problems of historical revisionism, bias, and circular reasoning, seen in the analysis of the implications of dizziness symptom quality, might be encountered with any other clinical parameter. This risk may be mitigated by the fact that dizziness timing and triggers are more reliably reported parameters than dizziness type (*Chapter 3*), but it is certainly not eliminated. Until prospectively validated (or invalidated) as predictors of risk or outcome, these disease-derived associations (be they rules, trees, or networks), focused on other symptom dimensions (timing, triggers, associated symptoms) deserve no special epistemic privilege not afforded to symptom quality. Instead, any such putative predictors deserve the same high level of scientific scrutiny and clinical skepticism.

How Could Diagnostic Decision Support be Implemented in the ED?

Regardless of its final form when presented to clinicians, the new diagnostic model would need to be easy to use in order to achieve acceptance. More specifically, it would need to fit cleanly into the clinical workflow.[316] This could mean a simple clinical decision rule with about 3-5 steps, or limited algorithm (fitting on a single page) that could be placed in a handy reference format (e.g., pocket card), affixed to the patient's medical record (e.g., sticker placed on the chart), or both. Alternatively, it could mean workflow-sensitive, computer-based diagnostic decision support. This might be in the form of complaint-driven disease checklists generated from very limited diagnostic information entered into the system by busy clinicians (e.g., ISABEL).[317] Alternatively, it might be in the form of simple diagnostic summaries (Box 4.2) generated by an

automated diagnostic triage agent (waiting-room kiosk) that conducted a complaint-specific, detailed medical interview prior to the physician encounter, as we have proposed (*Newman-Toker, NIH National Library of Medicine Application R01 LM009630-01*). In either case, emergency physicians have expressed a willingness to use such decision aids to assist with diagnostic triage decisions in dizzy patients, as long as they are well validated (*see Chapter 2 for additional details*).

Conclusions

In summary, as a case study of how misinformation becomes standard practice in clinical care, dizziness makes for a fascinating tale. New approaches to dizziness diagnosis are needed, and are currently under investigation. Perhaps more importantly, however, a new level of introspection about our general approach to diagnosis and diagnostic accuracy is required. Greater efforts to conduct systematic, symptom-oriented diagnostic research must be applied across the broad range of medical symptoms, if we can ever hope to bring science to the art of bedside diagnosis.

Box 4.1 Implicit claims regarding the relationship between dizziness cause and dizziness type in the original Drachman and Hart study[19]

These descriptions from the Drachman and Hart study provide examples of indirect, inferential claims of *characteristic* dizziness complaints falling into each "type" in association with a particular etiologic class of disorders (i.e., etiology predicts quality).

Note the extremely small numbers of patients in three of four etiologic classes: cardiovascular (n=4), neurological (n=4), psychogenic (n=9). In each of these three etiologic groups, high rates of *uncharacteristic* dizziness complaints (cardiovascular 25%; neurological 25%; psychogenic 33%) are brushed aside by authors with language implying these cases represent unimportant exceptions or inconsequential subgroups (e.g., psychotic patients), rather than findings that invalidate the putative association.

Peripheral vestibular disorders. Thirty-nine patients had significant peripheral vestibular disorders as a major cause of dizziness; of these, 32 had vestibulogenic dizziness alone while 7 had an additional cause of dizziness (Table 4). The hyperventilation syndrome was responsible for the second type of dizziness in 5 of these patients. They complained of light-headedness as well as vertigo.

Cardiovascular disorders. Four patients (4%) had dizziness due to impairment of total blood flow to the brain. Two patients had orthostatic hypotension and hypersensitivity of the carotid sinus, 1 with varying tachyarrhythmias as well. One patient had micturition syncope, while the fourth had marked anemia. All of these disorders except anemia produced sudden episodes of syncope-like sensations (type 2), sometimes quite brief but at other times resulting in actual loss of consciousness.

Neurological disturbances, other. Four patients with other neurological disorders leading to dizziness, or 4%, were seen in the present series. Two had Brun's apraxia of gait with impairment of ambulation on the basis of frontal lobe disease, most likely degenerative in origin (Alzheimer's disease). One patient had early parkinsonism and interpreted the motor impairment as dizziness. These 3 patients complained of dysequilibrium with difficulty in controlling the legs and balancing (type 3). The fourth had suffered from vague lightheadedness ever since a partial temporal lobectomy and the clipping of a ruptured cerebral aneurysm; she also had multiple sensory deficits, including impairment of sound localization, accounting for her symptoms.

Psychogenic dizziness. Dizziness caused by a psychiatric disturbance was diagnosed in 9 patients, or 9% of the study series (Table 7). Six of these patients were primarily depressed and anxious and characteristically complained of vague light-headedness (type 4)… Three patients were psychotic, 2 with chronic schizophrenia requiring institutional care in the past and 1 with a probable diagnosis of schizophrenia. Symptoms of dizziness were variable and numerous and did not fit any of the recognizable patterns, an observation consistent with other evidence of a thought disorder.

Box 4.2 Sample mockup of a computer-based, diagnostic decision support printout following an automated, symptom-focused diagnostic medical interview conducted at a waiting-room kiosk

Decision Support Output (printout affixed to chart)

- **Diagnostic triage recommendation(s)** (e.g., bedside Hallpike test to confirm BPPV — *if positive*, treat with Epley particle repositioning maneuver & discharge home with primary care or neuro-otology follow-up in 1-2 weeks; *if negative*, consider neurology consultation and/or admission for TIA workup)
- **Case Summary & Rationale** (e.g., Chief Complaint: Dizzy. This patient reports brief, episodic dizzy symptoms provoked by head position change, including rolling in bed, and unassociated with auditory symptoms, chest pain, or vomiting. This symptom pattern is consistent with the benign condition BPPV, but is occasionally mimicked by transient ischemic attacks [risk:*very low*] or cardiac arrhythmias [risk:*very low*].)
- **Patient Reliability Statistic** (false positive/negative responses on repeated questions)
- **Generic Evidence Summary** (includes description of BPPV, Hallpike/Epley maneuvers, with citations)
- **Interview Transcript** (i.e., questions asked and answered, in order asked, grouped by topic)

Appendices

Appendix 1.1 Preliminary analysis of NHAMCS ED visit data for dizziness

Draft abstract, figure, and tables from a cross-sectional analysis of dizziness in a nationally-representative sample of US hospital ED visits, with data derived from the CDC's National Hospital Ambulatory Medical Care Survey (NHAMCS). This study reports the spectrum of co-complaints, diagnostic tests, ED diagnoses, disposition, and treatments in the largest sample of ED dizzy patients ever described (nearly 9,000), representing over 32 million visits during a 12-year period. It demonstrates the impressive co-morbidity of dizziness with other symptoms, the high prevalence of medical diagnoses not traditionally thought of as common causes of dizziness, and the heavy resource utilization associated with this common complaint relative to others.

Appendix 1.2 Misconceptions about the bedside evaluation of dizzy patients

Draft abstract, figure, and table from a manuscript entitled, "Misconceptions about the Bedside Evaluation of Dizzy Patients — Are Textbooks Leading Frontline Providers Astray?" This appendix provides evidence of poor performance by generalists relative to specialists on a brief, paper-and-pencil assessment of dizziness knowledge. The figure displays poor performance by generalists — so poor as to indicate non-random misconceptions, rather than lack of information. The study was limited by a small, potentially biased sample, but effect sizes were large and comparable across disparate groups of frontline providers (both emergency physicians and primary care providers). The table that follows explores the possible relationship between misconceptions and misinformation presented in standard textbooks of emergency medicine.

Appendix 2.1 Emergency physician survey questions and results

Survey questions from the web-based survey of emergency physicians described in Chapter 2. Provided are question numbers (Q1–Q21), survey version(s) (A vs. B vs. A and B), question text, and aggregate responses across sites. We report means and standard deviations using a linear conversion of the 7-point Likert scale from strongly agree (+3) to strongly disagree (-3).

Appendix 2.2 Breakdown of survey responses by question and site

Survey results, by site, from the survey of emergency physicians described in Chapter 2. For questions 1-20 (Likert scale), we report means and standard deviations. For question 21 (rank response), we report proportion ranking quality first, with 95% confidence intervals.

Appendix 3.1 Sample responses to the open question 'What do *you* mean by dizzy?'

Sample responses (and associated dizzy "type" coding) from the open-ended question about dizziness symptom quality in the cross-sectional study of dizzy patients described in Chapter 3. We present sample free-text responses from patients, denoting the category or categories to which they were coded using a minor modification of the traditional Drachman & Hart coding schema.

Appendix 3.2 Directed vertigo inquiry details — '*What* is spinning or moving?'

Detailed analysis of the directed questions about vertigo from the cross-sectional study of dizzy patients described in Chapter 3. We present proportions of respondents endorsing the presence of "spinning or motion" with follow-up responses regarding the nature of the sensation. We also present samples of free-response dizziness descriptions that note a sense of spinning or motion.

Appendix 3.3 Statistical methods for cross-sectional analysis of dizziness attributes

Detailed description of statistical methods used to analyze data on various dizziness attributes derived from the cross-sectional study of dizzy patients described in Chapter 3.

Appendix 4.1 Proposed "safe-to-go steps" for bedside evaluation of acute dizziness

The first table in this appendix presents a duration-based differential diagnosis for acute dizziness, emphasizing comparison of common, benign causes and dangerous mimics.

The second table presents a possible new approach to bedside diagnosis of dizziness, using an episode duration-based classification schema, and a prioritized set of clinical rules to determine whether patients are "safe to go" (i.e., at very low risk of a dangerous underlying disorder). Such an approach might be printed on a pocket card for physicians, or, alternatively encoded as part of an assessment algorithm within a computer-based diagnostic decision support system.

Appendix 4.2 Proposed "triage" algorithm for bedside evaluation of acute dizziness

The appendix presents an algorithm (decision tree) describing a new, "diagnostic triage"-oriented approach to assessment of the acutely dizzy patient. The approach begins with an undifferentiated dizzy patient presenting for ED care, and capitalizes on the concept of level of illness severity for its first steps, mimicking the initial "triage" assessment that is standard practice in the ED. The algorithm relies on easily-ascertained clinical parameters (abnormal vital signs or mental state, pain) to identify those patients who are sickest, before focusing on a trigger and timing-based schema for those patients with relatively isolated acute dizziness symptoms, where common benign disorders must be segregated from duration-specific, dangerous mimics. The first page of the algorithm provides an overview of the diagnostic process. The second page of the algorithm describes, in detail, bedside techniques to distinguish benign disorders from dangerous mimics.

The Spectrum of Dizziness in United States Emergency Departments: Demographics, Workup, and Frequency of Pathologic Diagnoses

David E. Newman-Toker, MD; Carlos A. Camargo, Jr, MD, DrPH; Andrea J. Pelletier, MS; Jonathan A. Edlow, MD

Abstract (DRAFT)

Context: Dizziness is a common Emergency Department (ED) complaint that may result from a broad array of underlying medical conditions, both benign and dangerous. Traditional teaching about dizziness has generally focused on mono-symptomatic dizzy patients and vestibular causes, but small observational studies have suggested that ED dizzy patients do not conform to traditional notions of "the dizzy patient."

Objective: To describe the full spectrum of ED dizzy patients in the US, including their demographics, co-complaints, diagnostic tests, diagnoses, and disposition. Secondarily, to identify important clinical differences between "dizzy" patients and those with other presenting symptoms such as syncope.

Design: Cross-sectional study of national ED visits from the National Hospital Ambulatory Medical Care Survey (NHAMCS)

Setting: Weighted sample of US ED visits from the NHAMCS database (1993-2004)

Patients: *Inclusions*: "Dizzy" cases were defined as NHAMCS reason for visit code of vertigo/dizziness (1225.0), *or* final ICD-9 diagnosis of vertigo/dizziness (780.4) *or* a vestibular disorder (386.x). *Exclusions*: None (though patients under age 16 were excluded for subgroup analyses of co-complaints). Several prospectively-defined subgroup analyses were conducted, and multiple comparison populations were used.

Main Outcome Measures: Comparison of demographic and visit-related variables for "dizzy" vs. "not dizzy" subjects. Comparison of NHAMCS co-complaints and ICD-9 diagnoses between patients (>16yo) complaining of dizziness vs. those with five other presenting symptoms (ataxia, fatigue/malaise, syncope, chest pain, headache). Co-complaints grouped using an adaptation of the NHAMCS Reason for Visit coding schema. Diagnoses grouped using the HCUP Clinical Classification System for ICD-9 diagnoses.

Results: The total 12-year sample of dizzy patients was 8,987 (weighted estimate 32.1 million ED visits nationally over that same period), 92% of whom were coded with dizziness as a reason-for-visit complaint. Dizzy patients were more likely to be older (mean age 49 vs. 35), female (61% vs. 53%), in the ED longer (mean 3.9 vs. 3.1 hours), tested extensively (mean number of diagnostic tests 4.5 vs. 2.9), imaged by CT or MRI (17% vs. 5%), and admitted (19% vs. 13%) (all $p<0.001$).

Patients complaining of dizziness were more likely to be poly-symptomatic (80% vs. 52% for chest pain, $p<0.001$), with the most frequent co-complaint classes being nausea/vomiting (19%), cranio-cervical pain (13%), malaise/fatigue (12%), and neurological symptoms (9%). Major medical co-complaints (e.g., chest pain, dyspnea, URI symptoms, abdominal pain, syncope, bleeding, palpitations, fever/chills) were common, with one or more affecting 25%, while auditory/otologic (2.3%, most often tinnitus) and psychiatric (2.0%, most often anxiety) co-complaints were uncommon.

The most frequent diagnoses made were otologic/vestibular (27%), cardiovascular (21%), respiratory (12%), metabolic (11%), neurologic (11%, including 4% cerebrovascular), injury/poisoning (11%), psychiatric (7.5%), digestive (7.4%) and genitourinary (5%). Dangerous cardiovascular causes were diagnosed with comparable frequency among dizzy patients as among syncope patients (angina/myocardial infarction, 2.2% vs. 2.8%; arrhythmia 3.7% vs. 4.5%). Age was a significant predictor of a dangerous disease diagnosis, with more than 20% of those over 50 harboring a serious underlying cause. Age (OR 1.7, $p<0.001$) and co-morbid neurologic symptoms (OR 4.6, $p<0.001$) were independent predictors of a cerebrovascular diagnosis (transient ischemic attack), and co-morbid medical symptoms reduced the likelihood of the same (OR 0.3, $p=0.002$).

Conclusions: ED dizzy patients tend to be older and to use more medical resources than their non-dizzy counterparts, even when adjusted for age. ED patients experiencing dizziness do not conform to traditional notions of "the dizzy patient." Dizziness is rarely mono-symptomatic, not attributed to a vestibular disorder in most, and often associated with cardiovascular and medical causes. Associated symptoms generally predict final diagnoses, although exceptions are not uncommon, and prospective studies with independent diagnostic assessment are needed to confirm these associations.

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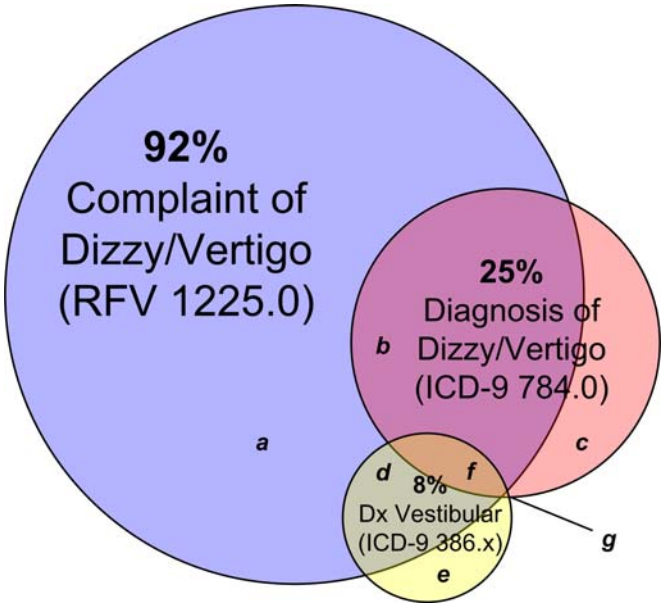
Figure 1 (DRAFT)

Area-proportional* Venn diagram illustrating the makeup of the study population.

All Dizzy Subjects Age 16 Years or Older (1993-2004, Total Weighted N = 30.6 million)	Group	Proportion (weighted N†)
	a. RFV Dizzy Only	68.2% (20.9 million)
	b. RFV Dizzy & Dx Dizzy	17.3% (5.5 million)
	c. Dx Dizzy Only	6.1% (1.9 million)
	d. RFV Dizzy & Dx Vestibular	5.0% (1.6 million)
	e. Dx Vestibular Only	1.3% (0.4 million)
	f. RFV Dizzy & Dx Dizzy/Vestib	1.1% (0.3 million)
	g. Dx Dizzy & Dx Vestibular	0.2% (0.1 million)

Dx – Diagnosis

ICD-9 – International Classification of Diseases, 9th Revision Diagnosis Code

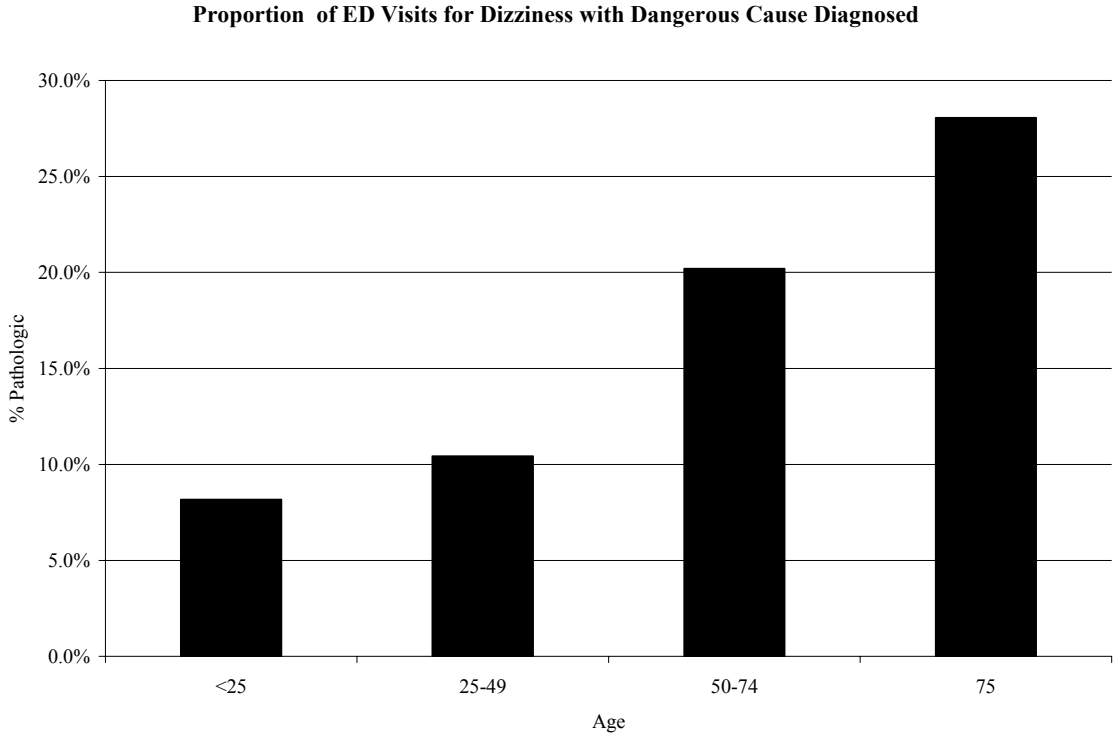
RFV – NHAMCS Reason for Visit Code

* Area-proportional diagram drawn free hand in Microsoft Visio 2003. Areas are approximate.

† Numbers do not sum exactly due to rounding artifacts.

The Spectrum of Dizziness in United States Emergency Departments: Demographics, Workup, and Frequency of Pathologic Diagnoses

Figure 2 (DRAFT)



Includes Dizzy Reason for Visit or Dizzy Diagnosis (ICD9 code 780.4 or 386)

The Spectrum of Dizziness in United States Emergency Departments: Demographics, Workup, and Frequency of Pathologic Diagnoses

Table 1 (DRAFT)

Demographic and Visit Differences Dizziness Cases v. Non-dizziness cases (1993-2004)

	Dizziness Cases					Non-dizziness Cases				p-value
Group	N	%	95% CI			N	%	95% CI		
female	5,451	60.8%	59.5%	62.1%		174,629	52.8%	52.5%	53.1%	p<0.001
admitted	1,696	18.9%	17.8%	20.1%		44,352	12.9%	12.5%	13.3%	p<0.001
arrived by ambulance*	1,082	22.8%	21.2%	24.4%		24,279	14.3%	13.8%	14.8%	p<0.001
coded as urgent	3,792	65.8%	63.4%	68.2%		127,147	57.2%	54.9%	59.4%	p<0.001
MRI/CT scan†	1,371	17.3%	16.1%	18.5%		15,331	5.3%	5.1%	5.6%	p<0.001
treated for vertigo/vomiting	1,690	18.5%	17.4%	19.6%		10,004	3.1%	2.7%	3.4%	p<0.001
		mean					mean			
mean age	8,987	49.1	48.4	49.7		332,299	34.5	34.1	34.9	p<0.001
mean wait time (min)*	4,713	46.6	43.5	46.9		144,404	44.8	42.8	46.9	p=0.16
mean length of visit (min)‡	4,321	235.7	224.6	246.7		165,238	187.2	181.6	192.8	p<0.001
mean # diagnostic tests§	6,730	4.5	4.3	4.7		235,104	2.9	2.8	2.9	p<0.001

* data available 1997-2000; 2003-2004

† data available 1995-2004

‡ data available from 2001-2004

§ data available from 1997-2004

Table 2 (DRAFT)

Co-Complaint Classes* by Visit Reason Group, Ranked Based on Frequency† for Dizziness; Ages >=16 years

	Dizziness (n=7,925)		Ataxia (n=930)		Fatigue/Malaise (n=7,024)		Syncope (n=2,756)		Chest Pain (n=20,811)		Headache (n=14,535)	
Complaint Class	N	%	N	%	N	%	N	%	N	%	N	%
Nausea	1461	19.0%	27	3.0%	904	13.5%	213	8.3%	885	4.5%	2039	14.5%
Craniofacial pain	1026	12.9%	34	3.4%	352	5.3%	135	5.0%	881	4.1%	n/a	n/a
Fatigue/Malaise	952	11.8%	98	11.2%	n/a	n/a	156	6.4%	359	1.6%	319	2.1%
Neurological	722	9.0%	n/a	n/a	597	8.5%	149	5.2%	612	2.9%	951	6.3%
Chest Pain	561	7.0%	6	0.3%	373	5.2%	131	5.1%	n/a	n/a	536	3.5%
Injury	459	5.5%	125	13.4%	158	2.2%	241	8.4%	521	2.4%	1052	6.8%
Dyspnea	447	5.4%	25	2.5%	612	8.9%	77	3.2%	3362	15.8%	291	2.0%
URI	314	3.8%	12	1.3%	549	8.4%	45	1.8%	1458	7.1%	1363	9.5%
Abdominal pain	300	3.8%	11	1.2%	256	3.6%	73	2.5%	580	2.8%	367	2.4%
Syncope	304	3.7%	7	1.0%	156	2.6%	n/a	n/a	127	0.6%	114	0.8%
Bleed	197	2.7%	3	0.3%	149	2.0%	37	1.4%	91	0.4%	124	0.8%
Palpitations	209	2.6%	2	0.5%	99	1.5%	33	1.3%	330	1.5%	54	0.4%
Fever/Chills	207	2.6%	8	0.8%	441	6.7%	28	0.8%	293	1.4%	766	5.3%
Auditory	178	2.3%	3	0.3%	38	0.5%	8	0.5%	54	0.2%	329	2.2%
Psychiatric	172	2.0%	33	2.8%	323	4.2%	66	1.7%	263	1.2%	251	1.5%
No co-complaints‡	1583	20.2%	106	11.6%	1426	19.6%	1132	40.5%	9846	48.2%	4742	33.7%

n/a = not applicable (complaint is within co-complaint class) Key: ≥10% 5-10% 0-5%

* Co-complaint classes based upon categories defined from NHAMCS visit reasons (described in Methods section)

† Top 15 co-complaint classes listed for dizziness (other co-complaints, not listed, could be in 'top 15' for other symptoms)

‡ No co-complaints: patients coded as monosymptomatic (coding schema lists up to 3 total complaints, without priority rank)

Misconceptions About the Bedside Evaluation of Dizzy Patients — Are Textbooks Leading Frontline Providers Astray?

David E. Newman-Toker, MD; Victoria Stanton, BA, MSII; Yu-Hsiang Hsieh, PhD; Richard E. Rothman, MD, PhD

Abstract (DRAFT)

Context: Dizziness is a common Emergency Department (ED) complaint that may result from a broad array of underlying medical conditions, both benign and dangerous. Bedside assessment is thought to offer the best possible opportunity for accurate diagnosis.

Objective: To assess emergency and primary-care physicians' understanding of bedside findings in the assessment of dizzy patients. Secondarily, to determine whether identified misconceptions correlated with misinformation in emergency medicine textbooks.

Design: Anonymous, true-false quiz administered as a pre-lecture assessment

Setting: Two university hospitals

Subjects: 28 emergency (n=14) and primary care (n=14) physicians; 10 vestibular specialists (trained in either neurology or otolaryngology) served as a comparison group

Main Outcome Measures: The percentage of correct responses was calculated for each individual and for each question. Content related to all quiz questions was qualitatively evaluated by reviewing relevant material in three leading emergency medicine textbooks. Quantitative responses were correlated with qualitative findings.

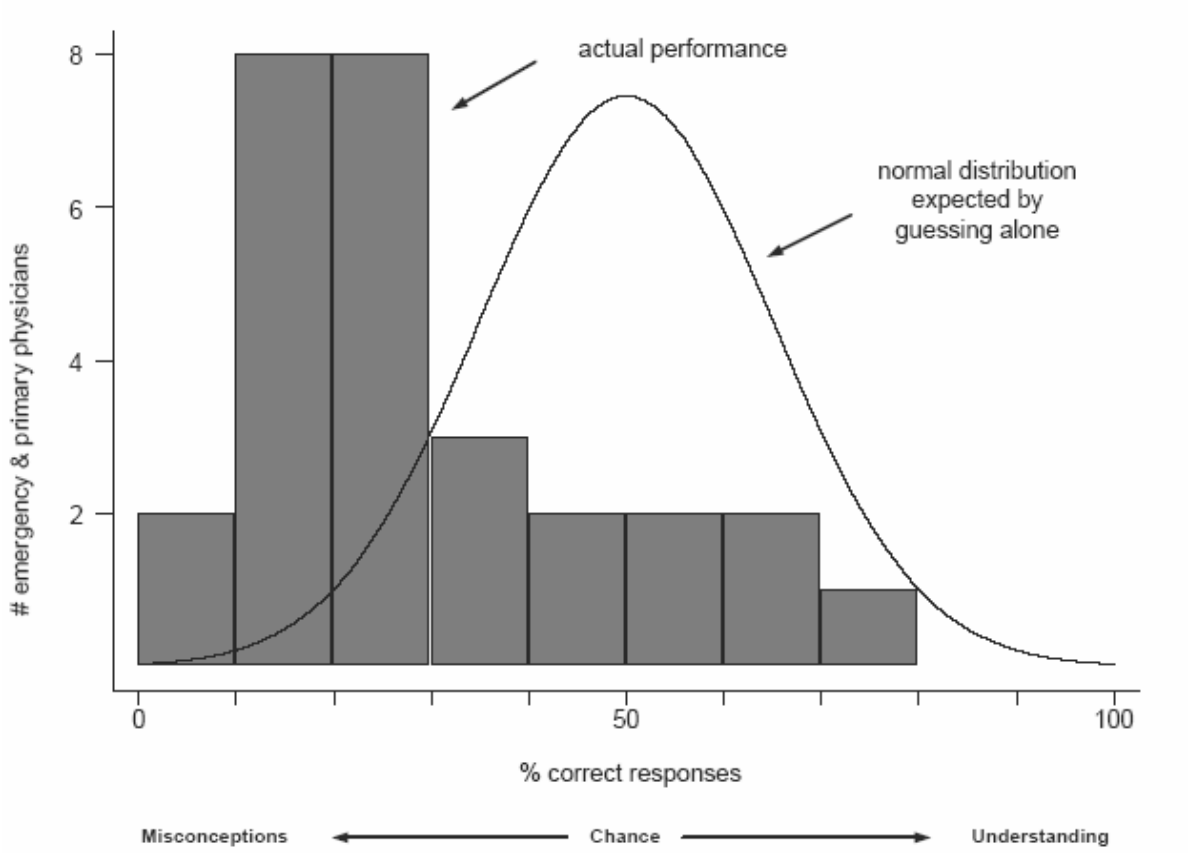
Results: The mean individual score for correct responses was 31% among emergency physicians and 29% among primary-care physicians. Combining the groups and analyzing by question, 6 of 10 questions were answered correctly at rates below those expected for guessing (8-26%, p=0.00002-0.02), implying misconceptions, rather than lack of knowledge. Vestibular specialists (control group) significantly outperformed generalists (mean total score 84% vs. 30%, $p < 0.0001$). Emergency medicine textbooks frequently presented misinformation, some of which correlated with erroneous responses. The most clinically relevant misconceptions were that (a) dizziness worsened by head movement is benign, (b) direction-changing nystagmus (rightward in right gaze and leftward in left gaze) is benign, and (c) episodic vertigo lasting 5-10 minutes is benign.

Conclusions: These findings suggest that misconceptions in the bedside approach to dizzy patients are probably common among frontline providers, and may, in part, reflect misinformation presented in textbooks. Such misconceptions could increase misdiagnosis and reduce patient safety. Educational initiatives should be considered.

Misconceptions About the Bedside Evaluation of Dizzy Patients — Are Textbooks Leading Frontline Providers Astray?

Figure 1 (DRAFT)

Histogram of scores for generalist physicians on a 10-question, true-false pre-lecture assessment about the bedside evaluation of dizzy patients.



Misconceptions About the Bedside Evaluation of Dizzy Patients — Are Textbooks Leading Frontline Providers Astray?

Table 1 (DRAFT)

Performance of generalists* vs. vestibular specialists* on a 10-question, true/false quiz, with comparison to textbook information

Question (Correct Answer)	Generalist % correct (95% CI) p-value†	Specialist % correct (95% CI) p-value‡	Correct information in textbook?		
			Rosen[1]	Harwood-Nuss[2]	Tintinalli[3]
Questions 1-5: "True or False: Likely to indicate a peripheral (as opposed to central) cause of PERSISTENT vertigo."					
1. Presence of new unilateral tinnitus. (True)	61% (41-78%) 0.34	90% (56-100%) 0.13	Correct	Correct	Correct§
2. Head motion or change in position exacerbates dizziness. (False)	25% (11-45%) 0.01	80% (44-97%) 0.006	Incorrect	Equivocal	Incorrect[a]
3. Horizontal nystagmus beating rightward in right gaze and leftward in left gaze. (False)	26% (11-46%) 0.02	100% (74-100%) 0.00005	Not addressed§	Not addressed§b	Correct§
4. Hallpike (Bárány) maneuver produces nystagmus which was absent in primary gaze. (False)	8% (1-26%) 0.00002	60% (26-88%) 0.0028	Equivocal§	Correct§	Correct
5. Nystagmus present in primary gaze increases with occlusive ophthalmoscopy. (True)	22% (7-44%) 0.01	90% (56-100%) 0.0004	Not addressed	Not addressed	Not addressed

Question (Correct Answer)	Generalist % correct (95% CI) p-value†	Specialist % correct (95% CI) p-value‡	Correct information in textbook?		
			Rosen[1]	Harwood-Nuss[2]	Tintinalli[3]
Questions 6-10: "True or False: General questions about vertigo/nystagmus."					
6. 'True vertigo' refers particularly to a spinning sensation in the horizontal plane. (False)	18% (6-37%) 0.0009	90% (56-100%) 0.0001	Correct	Correct	Correct
7. When elicited, classic benign paroxysmal positioning nystagmus is vertical and torsional. (True)	31% (14-52%) 0.08	90% (56-100%) 0.0023	Incorrect	Not addressed§	Correct
8. Nausea with vomiting is a frequent concomitant of benign paroxysmal positioning vertigo. (False)	46% (28-66%) 0.85	70% (35-93%) 0.28	Not addressed[c]	Equivocal	Correct[d]
9. Nystagmus in a unilateral vestibulopathy is always most evident looking away from the lesion. (True)	42% (22-63%) 0.54	90% (56-100%) 0.02	Incorrect	Not addressed	Not addressed[e]
10. A patient with several bouts of isolated vertigo lasting 5-10 minutes is most likely to have BPPV. (False)	23% (9-44%) 0.009	80% (44-97%) 0.0055	Correct	Equivocal	Correct

* Generalists: Johns Hopkins Hospital emergency physicians (n=14) and Massachusetts General Hospital primary care physicians (n=14); Specialists: Johns Hopkins Hospital vestibular specialists (n=10)

† p-values in the Generalist column are for comparison of mean proportion of correct responses among Generalists relative to that expected by chance guessing alone (i.e., 50% correct responses) (binomial exact)

‡ p-values in the Specialist column are for a comparison of mean proportion of correct responses among Generalists vs. Specialists (Fisher's Exact test)

§ Though not the express misconception tested, textbook does contain errors on the subject that, if relied upon, might contribute to misdiagnosis.

[a] A table in the 6th edition of Tintinalli[6] was updated to reflect that central vertigo may be aggravated by changes in position, but the text still reads that "the symptoms [of central vertigo] are not provoked by changes in position."

[b] Harwood-Nuss[5] is now updated to say that when a nystagmus is direction changing it is necessarily central.

[c] The most recent Rosen's[4] now lists nausea and vomiting as associated symptoms of BPPV.

[d] Tintinalli[6] has been updated from saying that BPPV is associated with nausea, vomiting and diaphoresis, to saying that "nausea is often present."

[e] This question is now correctly addressed in Tintinalli's latest edition.[6]

Misconceptions About the Bedside Evaluation of Dizzy Patients — Are Textbooks Leading Frontline Providers Astray?

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Appendix 2.1 Emergency physician survey questions and results

Shown below are question number, survey version, question text, and response average across sites. Means with standard deviations are reported for all 20 Likert-scale questions. These were calculated using a simple linear conversion of responses to a numerical scale (+3 to -3), assuming equidistance between response options. Positive means indicate overall agreement, while negative means indicate overall disagreement. No mean or standard deviation is reported for the rank-response question (Q21). A brief description of question "intent" or "focus" is provided in the following paragraph.

Between the two survey versions (A and B), there were 21 total dizziness-specific content questions. Five questions assessed physicians' experience and attitudes towards the evaluation of dizzy patients (Q1, 2, 13, 17, 20). Three questions assessed EPs' perceptions about the quality-of-symptoms approach to dizziness (Q3, 4, 5). One asked them to rank the importance of different symptom attributes (Q21). Two questions assessed EP interpretation of terminology linked to the quality-of-symptoms approach (Q7, 15). One question assessed whether the quality-of-symptoms model matched real world experience (Q14). Two questions assessed whether EPs reported not only *believing in* but *behaving in accordance with* the dictates of the quality-of-symptoms approach (Q6, 8). Seven questions (Q9–12, 16, 18, 19) focused on provider attitudes and behavior towards other dizziness attributes (timing, triggers, and associated symptoms). Four of these seven were framed in such a way that a "risky" response could be identified with respect to misdiagnosis of a dangerous underlying disorder.

Appendix 2.1 Emergency physician survey questions and results (continued)

Question (Version)	Survey Question Text	Mean (SD)
Q1 (A/B)	Excluding trauma and injury, dizziness is one of the most common medical complaints in the clinical setting where I work(ed), easily making the "top 10 symptoms" list.	1.12 (1.4)
Q2 (A/B)	I feel confident assessing, diagnosing, and triaging ED dizzy patients, and only infrequently feel the need to call for specialist consultation in these patients.	1.13 (1.3)
Q3 (A/B)	Most review articles, textbooks, and lectures about dizziness say that asking a dizzy patient to describe the quality of the dizzy symptoms is the first (and most important) step.	1.81 (1.0)
Q4 (A/B)	These sources of information also generally say that the patient's response should be categorized as one of four "types" of dizziness, each implying a likely etiology: i. VERTIGO (spinning/motion), implying a vestibular (peripheral or central) cause ii. PRESYNCOPE (about to faint), implying a cardiovascular cause iii. DISEQUILIBRIUM (unsteady walking), implying a neurologic (motor/sensory) cause iv. NON-SPECIFIC (other vague dizzy feelings), implying a psychiatric or metabolic cause	1.62 (1.1)
Q5 (A/B)	I believe that the "type" of dizzy symptoms is very important in determining the underlying etiology when evaluating an ED dizzy patient.	1.76 (1.1)
Q6 (A/B)	I generally don't pursue cardiovascular causes when I determine the patient has "vertigo." Likewise, I generally don't pursue vestibular problems when I determine the patient has "presyncope."	0.83 (1.5)
Q7 (A)	When a patient reports "lightheadedness," I think of this as a mild form of "presyncope" (about to faint) even if they don't expressly describe a feeling of impending faint. Therefore, I focus on cardiovascular causes in such patients.	0.70 (1.3)
Q8 (A)	I typically don't pursue stroke or TIA in patients with "vague" dizziness, unless they have obvious neurologic symptoms (e.g. double vision) or signs (e.g. hemiparesis).	0.14 (1.5)

Appendix 2.1 Emergency physician survey questions and results (continued)

Q9 (A)	I believe that patients with a single, continuous bout of dizziness (lasting hours to days), who are still "sick and dizzy" at the time of assessment, are much more likely to have suffered an acute cerebellar stroke than those with brief, episodic dizziness.	0.73 (1.4)
Q10 (A)	In those still "sick and dizzy" at the time of assessment, exacerbation of symptoms with any head motion gives me confidence the patient has a peripheral vestibular disorder.	0.42 (1.3)
Q11 (A)	In patients with brief, unprovoked, episodic dizziness (lasting seconds to minutes) and negative Dix-Hallpike (Nylen-Bárány) positional testing, I focus on ruling out cardiac arrhythmias and TIAs.	0.49 (1.1)
Q12 (A)	I generally don't pursue cardiovascular causes in dizzy patients with head motion triggers unless I suspect carotid sinus hypersensitivity associated with neck rotation.	0.86 (1.3)
Q13 (A)	If it were well designed and validated, I would use a clinical decision rule to help make decisions about diagnostic testing (e.g. imaging) in ED dizzy patients.	2.04 (0.9)
Q14 (B)	I find most ED patients with "vague" (non-vertiginous, non-presyncopal) dizziness have a metabolic disorder (medication side effect, hypoxemia, anemia, hypoglycemia, etc.).	0.13 (1.3)
Q15 (B)	I think "vertigo" is a term that should be reserved for patients with an unmistakable spinning sensation, and not be used for other "motion" symptoms (rocking, swaying, etc.).	0.93 (1.7)
Q16 (B)	I almost always use Dix-Hallpike (Nylen-Bárány) positional testing to try to reproduce clinical symptoms in patients with brief, episodic dizziness, assuming there is no obvious explanation (e.g. orthostatic hypotension).	0.86 (1.6)
Q17 (B)	I feel comfortable identifying the upbeat-torsional nystagmus characteristic of typical posterior-canal BPPV (benign paroxysmal positioning vertigo).	0.35 (1.5)
Q18 (B)	In patients with a single, continuous bout of dizziness (lasting hours to days) and spontaneous nystagmus, the diagnosis is likely to be vestibular neuritis or cerebellar stroke. In these cases, a normal CT reassures me that these patients are safe to go home.	-1.59 (1.3)

Appendix 2.1 Emergency physician survey questions and results (continued)

Q19 (B)	In ED dizzy patients with head or neck pain and a normal ear exam (including otoscopy), I aggressively pursue aneurysm or vascular dissection, unless I am confident the patient has migraine-associated dizziness (a.k.a. vestibular migraine, basilar migraine).	0.79 (1.3)
Q20 (B)	If a kiosk-based, automated interview in the waiting room could provide me with valid, evidence-based suggestions about diagnosis, I would consider using them to help make decisions about diagnostic testing (e.g. imaging) in ED dizzy patients.	0.74 (1.5)
Q21* (B)	Please rank the overall relative importance to you of these historical features in assessing a typical ED dizzy patient. (We recognize that various elements of clinical history may be of different value under different clinical circumstances.) 1 = most important, 4 = least important (CHOOSE EACH NUMBER ONLY ONCE) QUALITY (vertigo, presyncope, disequilibrium, other) TIMING (duration of illness, episode duration, etc.) TRIGGERS (head motion, change in posture, etc.) ASSOCIATED PAIN (head or neck pain, chest pain, etc.)	N/A

* Q21 was the only survey question that did not use a Likert response scale, and instead used a forced-choice rank response, in which respondents were required to rank all four dizziness attributes, and could not choose the same rank for more than one attribute. The four response options were presented in randomly-ordered sequence for each respondent, to prevent response bias related to order-of-presentation effects.

Appendix 2.2 Breakdown of survey responses by question and site

Means with standard deviations are reported for the 20 Likert-scale questions. These were calculated using a linear conversion of responses to a numerical scale (+3 to -3). For the rank-response question (Q21), we list proportions with 95% confidence intervals, by site. Totals for each question are identical to those in Appendix 2.1. Although there was heterogeneity across sites, most of the differences were in magnitude only, rather than direction of response (agree [positive means] vs. disagree [negative means]).

Question	Site	n	Mean	Standard Deviation
Q1	A	60	0.42	2.2
	B	67	1.94	1.4
	C	43	0.74	1.5
	D	108	1.28	1.4
	E/F	133	1.02	1.9
	Total	411	1.12	1.4
Q2	A	60	0.75	1.4
	B	67	1.24	1.3
	C	43	1.47	1.2
	D	108	1.37	1.4
	E/F	133	0.95	1.6
	Total	411	1.13	1.3
Q3	A	60	1.47	1.2
	B	66	1.65	1.3
	C	43	1.84	0.7
	D	107	1.97	1.3
	E/F	131	1.92	0.8
	Total	407	1.81	1.0
Q4	A	60	1.70	1.3
	B	66	1.68	1.1
	C	43	1.35	1.2
	D	107	1.81	1.8
	E/F	131	1.48	1.2
	Total	407	1.62	1.1
Q5	A	60	1.50	1.2
	B	66	1.70	1.2
	C	43	1.84	1.2
	D	101	1.96	1.4
	E/F	130	1.72	1.1
	Total	400	1.76	1.1

Appendix 2.2 Breakdown of survey responses by question and site (continued)

Q6	A	60	0.12	2.6
	B	66	1.00	1.5
	C	43	1.21	1.6
	D	101	1.23	1.3
	E/F	130	0.64	1.8
	Total	400	0.83	1.5
Q7	A	36	0.89	1.1
	B	23	0.87	0.7
	C	16	0.56	1.5
	D	49	0.67	1.3
	E/F	62	0.60	2.1
	Total	186	0.70	1.3
Q8	A	36	-0.53	1.8
	B	23	0.70	2.5
	C	16	0.19	1.6
	D	49	0.39	1.5
	E/F	62	0.11	2.6
	Total	186	0.14	1.5
Q9	A	35	0.43	1.5
	B	23	0.87	1.7
	C	16	0.69	1.6
	D	49	0.90	1.5
	E/F	62	0.73	1.8
	Total	185	0.73	1.4
Q10	A	35	0.51	1.2
	B	23	0.65	1.6
	C	16	0.19	1.3
	D	49	0.59	1.2
	E/F	62	0.21	2.0
	Total	185	0.42	1.3
Q11	A	35	0.66	1.1
	B	23	0.52	1.1
	C	16	0.38	1.3
	D	49	0.49	1.2
	E/F	61	0.41	1.4
	Total	184	0.49	1.1

Appendix 2.2 Breakdown of survey responses by question and site (continued)

Q12	A	35	0.40	2.0
	B	23	1.00	1.1
	C	16	0.75	1.6
	D	49	1.10	1.0
	E/F	61	0.92	1.4
	Total	184	0.86	1.3
Q13	A	35	1.97	1.1
	B	23	2.00	0.9
	C	16	1.75	0.9
	D	49	2.14	0.9
	E/F	61	2.10	0.9
	Total	184	2.04	0.9
Q14	A	24	-0.04	1.3
	B	43	0.21	1.9
	C	27	0.30	1.5
	D	50	0.28	1.4
	E/F	67	-0.04	1.2
	Total	211	0.13	1.3
Q15	A	24	1.21	1.8
	B	43	0.74	3.5
	C	27	0.81	1.7
	D	50	1.04	1.7
	E/F	67	0.91	2.7
	Total	211	0.93	1.7
Q16	A	24	-0.04	2.1
	B	43	0.95	2.1
	C	27	1.00	1.7
	D	50	0.98	2.0
	E/F	66	0.97	1.9
	Total	210	0.86	1.6
Q17	A	24	-0.08	1.9
	B	43	0.67	1.2
	C	27	0.22	1.4
	D	50	0.32	1.5
	E/F	66	0.38	2.6
	Total	210	0.35	1.5

Appendix 2.2 Breakdown of survey responses by question and site (continued)

Q18

A	24	-1.50	1.0
B	43	-1.42	2.1
C	27	-1.52	1.2
D	50	-1.66	1.7
E/F	66	-1.71	1.4
Total	210	-1.59	1.3

Q19

A	24	0.88	1.4
B	43	0.77	1.8
C	27	0.67	1.4
D	50	0.96	1.3
E/F	66	0.68	1.7
Total	210	0.79	1.3

Q20

A	24	1.38	1.6
B	43	0.47	2.8
C	27	0.74	1.2
D	50	0.58	1.7
E/F	66	0.82	1.8
Total	210	0.74	1.5

Q21

SITE	n	Proportion Quality #1	95% CI
A	59	49%	37–62%
B	66	52%	40–63%
C	43	63%	48–76%
D	99	72%	62–80%
E/F	127	73%	65–80%
Total	394	64%	60–69%

Appendix 3.1 Sample responses to the open question 'What do *you* mean by dizzy?'

Examples of free responses to open-ended questions about the quality of dizzy symptoms ("People use words like 'dizzy' to describe a lot of different things — what do *you* mean when you say you've been dizzy, lightheaded, or off balance?"). In parentheses are the Drachman and Hart dizziness types into which they were placed for quantitative analysis of the qualitative results (V = vertigo; P = presyncope; D = disequilibrium; N = non-specific dizziness).

Responses were occasionally…

1. STRAIGHTFORWARD

a. My head is spinning. (V)
b. Um, I mean it's like my head makes me feel like the room is spinning. (V)
c. Like I'm about to go out; like I'm about to faint. Or something like that. (P)
d. Feel like you getting to pass out. (P)
e. Off balance; when I can't steady myself. I can't control my balance. (D)
f. Not balanced; difficult walking; difficult, almost like you're about the fall. (D)

but often…

2. VAGUE OR CIRCULAR

a. Umm, I'm dizzy. (N)
b. What do I mean? When your head is dizzy and your eyes are dizzy and blurry. (N)
c. It means to lose yourself; I can't, I'm not coherent; I can't move around; I'm not mobile like I usually am; That's about it. (N)
d. I was just lightheaded. I would have to stop for a second like breathing; you know, my eyes would feel strange. (N)
e. Ah; Ah; I'd say like just the way your body feels; your legs kind of feel wobbly; lightheadedness, kind of like; that's what. (N)
f. Um; I think the general meaning would be the point where that woozy feeling; now I don't know how you want to describe the adjective for that; I guess woozy at that point. (N)

Even when relatively clear…

3. CROSSED CATEGORY BOUNDARIES

a. Room was spinning; felt lightheaded; like I was going to faint. (V,P,N)
b. Like close to falling, blacking out, like spinning, like the world's spinning. (V,P,D)
c. It feels like your knees are weak and that things are spinning around. It feels like I am going to faint. (V,P,N)
d. A combination of lightheaded; vertigo; to vertigo sometimes; the room spins around; sometimes just lightheaded; hard to concentrate; things like that. (V,N)
e. Dizzy, light head, off balance. I feel faintish, almost loss of consciousness. (P,D,N)
f. I sort of feel dizzy and lightheaded; I feel like I'm going to fall forward or backward. I just feel lightheaded like I might be tired. (D,N)

…and not infrequently…

4. DEFIED CONVENTIONAL MEDICAL TERMINOLOGY OR CLINICAL WISDOM

a. Lala land. (N)
b. Yes, like your head is becoming empty. (N)
c. My equilibrium's off; feeling like I can't walk straight; or even think very clearly. (D,N)
d. I see colors and spots; my balance is not good; I get this funny feeling in my head like this tightness. (N)
e. I can't focus; like when I'm trying to see, I just can't see right; everything's like a blur. (N)
f. Sometimes the room is spinning when I get up too fast from a lying position. (V)

Appendix 3.2 Directed vertigo inquiry details — '*What* is spinning or moving?'

The four panels demonstrate "best" dizziness descriptors and results of directed vertigo inquiry:

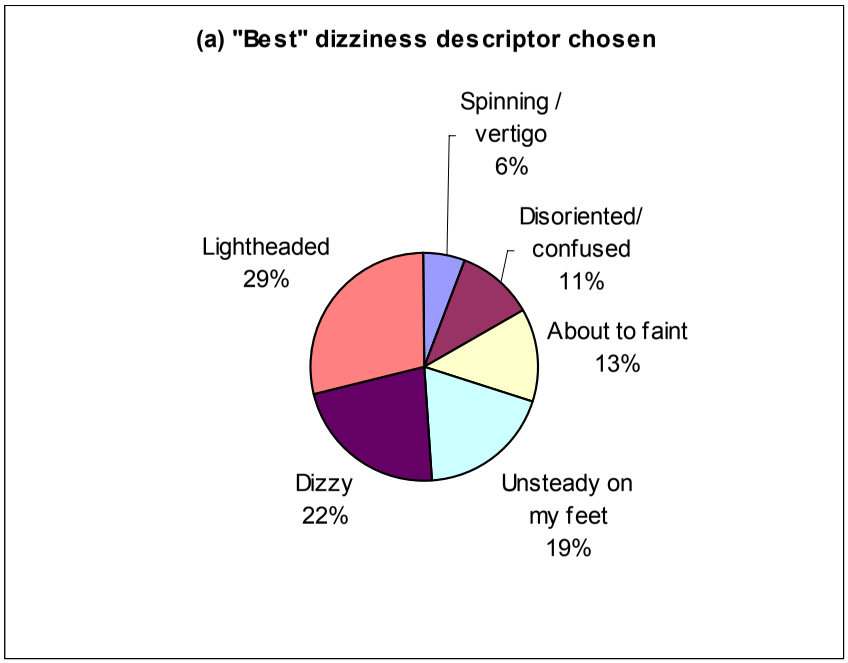
(a) single "best" response of 6 dizzy descriptors;

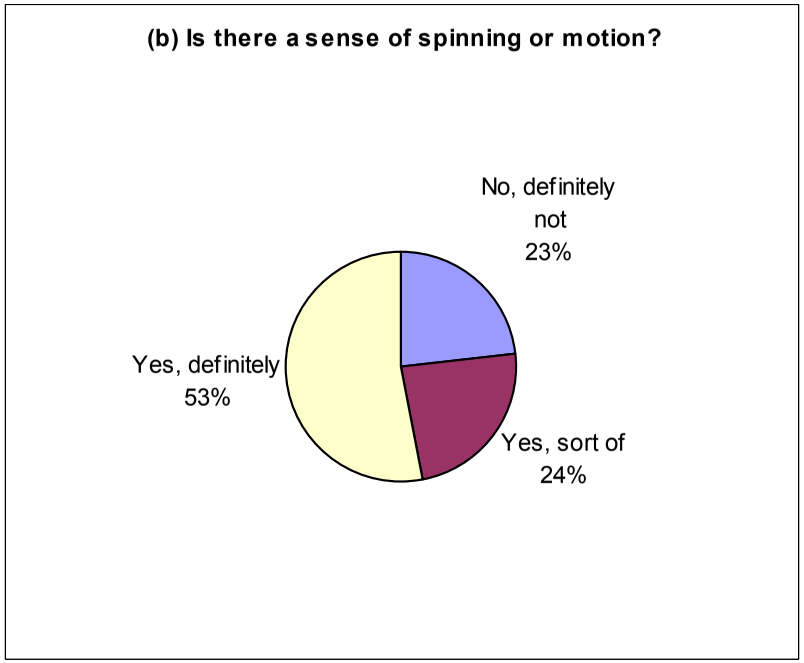
(b) presence of motion/spinning (for those **not** choosing "spinning or vertigo" as "best");

(c) 'what' is spinning or moving (for any endorsing spinning or motion) (multi-response format);

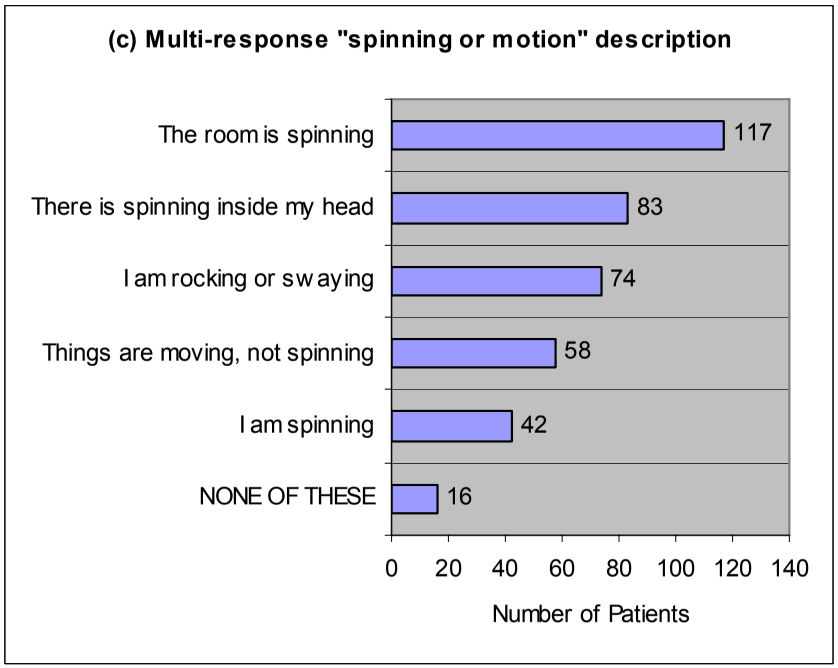
(d) nature of balance disturbance (for those *not* endorsing spinning or motion) (multi-response);

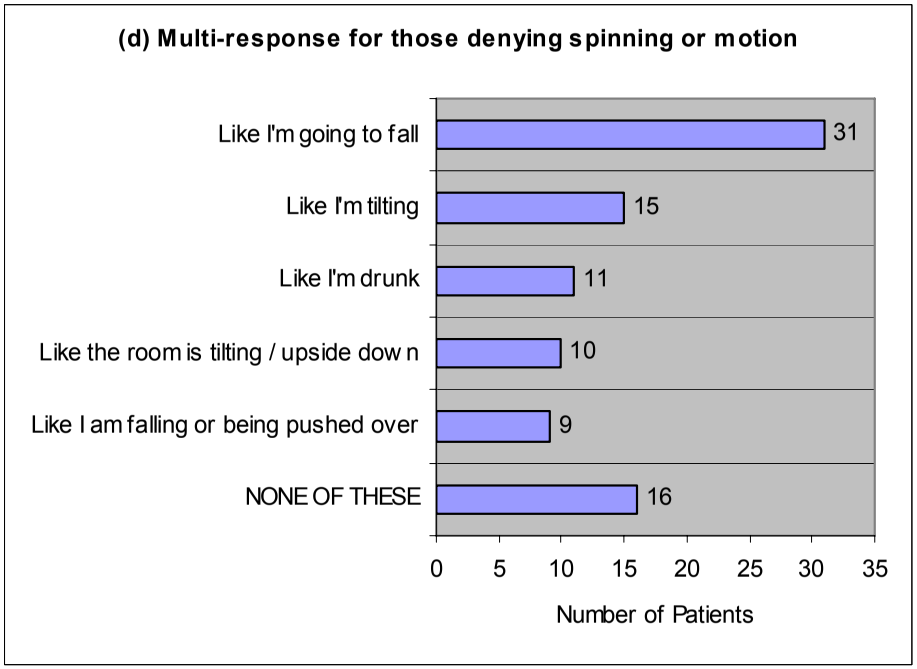
(e) examples of motion descriptions in free responses to open-ended question about dizzy quality





Appendix 3.2 Directed vertigo inquiry details — '*What* is spinning or moving?' (continued)





(e) Sample Open-Ended Responses Describing Motion

- *"It feels like the room is spinning and there is spinning in my head."*
- *"The room is spinning and uncoordinated."*
- *"I reach for something and it seems like it moves; the room moves."*
- *"My head feels like it's moving around in a different direction."*
- *"I feel unfocused, and everything's spinning around."*
- *"I go to stand and have to catch myself because everything be like waving around."*
- *"Feels like the whole world is turning upside down."*
- *"Dizziness [is when] things are going back and forth."*
- *"Feels like my eyes are spinning around; my head's spinning around in circles."*
- *"Your head go round and round."*

Appendix 3.3 Statistical methods for cross-sectional analysis of dizziness attributes

Statistical analyses were performed with SAS statistical software (v9.1, SAS Institute, Cary NC). All p-values provided were 2-sided and $p<0.05$ was considered statistically significant.

Table 3.1 – Characteristics of patients screened and enrolled with respect to the extent of dizziness as part of ED visit were compared by one-way ANOVA for continuous data, chi-square test or Fisher's exact test for category data, and Cochran-Armitage trend test for trend analysis.

Table 3.2 – Similarly, demographics and record characteristics by the extent of dizziness as part of ED visit were compared by nonparametric Kruskal-Wallis test for continuous data without normal distribution, one-way ANOVA for continuous data with normal distribution, chi-square test or Fisher's exact test for category data, and Cochran-Armitage trend test for trend analysis.

Table 3.3 – Percentage of agreement in reporting symptom quality of dizzy symptoms between test and retest was calculated with its 95% CI.

Table 3.4 – McNemar test was performed to assess the difference between the quality questions and timing or triggers questions in clarity, consistency, and reliability in subgroups of patients who completed both sets of questions.

Figure 3.3 – Proportions of overlap (>1 dizziness types selected) of qualitative dizzy symptoms reported by patients by different methods were calculated with their 95% confidence intervals. A generalized estimating equation (GEE) approach in this correlated data from repeated measurements was used to assess a linear trend of the proportions of overlap by different reported types completed by all participants: multi-response (Figure 3.3 panel B), free response and multi-response combined (Figure 3.3 panel C), and free response, multi-response, and directed vertigo inquiry combined (Figure 3.3 panel D).

Text (Results) – Subgroup analyses of demographics (age, sex, race, and years of education), hospital site, or whether dizziness as part of the reason for ED visit with respect to (1) overlap from free response question, (2) overlap from multi-response question, (3) unreliability from test-retest were performed by chi-square test, t test, or Cochran-Armitage trend test.

Appendix 4.1 Proposed "safe-to-go steps" for bedside evaluation of acute dizziness

Table 1. Common causes of dizziness and dangerous mimics, by duration

Duration*	Common, Benign† Causes	Dangerous Mimics
Seconds to Hours *(EPISODIC: transient or intermittent)*	• BPPV *(sec)* • orthostatic dizziness *(sec-min)* • reflex syncope *(sec-min)* • panic attack *(min-hrs)* • Meniere syndrome *(min-hrs)* • vestibular migraine *(sec-hrs‡)*	• transient ischemic attack *(sec-hrs)*[1] • cardiac arrhythmia *(sec-hrs)*[2] • other cardiovascular disorders (myocardial ischemia,[3] aortic dissection,[4] valvular heart disease, atrial myxoma, pulmonary embolus, etc.) • neuro-humoral neoplasm (insulinoma, pheochromocytoma, carcinoid, mastocytosis, etc.)
Days to Weeks *(NON-EPISODIC: persistent or continuous)*	• vestibular neuritis • viral labyrinthitis • drug toxicity (e.g., alcohol or anticonvulsants)	• brainstem,[5,6] cerebellar,[7,8] or labyrinthine stroke[9] • bacterial labyrinthitis/mastoiditis or herpes zoster oticus • brainstem encephalitis (e.g., listeria, herpes) • drug toxicity (e.g., lithium), drug withdrawal (e.g., alcohol), or toxic exposure (e.g., carbon monoxide)

* Patients with conditions producing vertigo lasting seconds to hours are often no longer symptomatic at the time of Emergency Department (ED) assessment. If they are still symptomatic, it is generally with intermittent symptoms triggered by certain actions (e.g., head movement, standing up quickly, etc.). By contrast, patients with conditions producing vertigo that lasts for days to weeks are generally symptomatic at the time of initial ED assessment. This clinical distinction is crucial, since the bedside examination findings one expects differ dramatically between the two groups. In the former group, with transient or intermittent symptoms, the physician should seek physical exam findings that provoke symptoms, but should not be surprised to find a completely normal exam — here, only the history offers the hope to differentiate between common, benign causes and their dangerous mimics. In the latter group, with persistent and continuous symptoms, the physician should expect that the exam findings will readily distinguish between benign causes and dangerous causes, and be surprised if they do not.

†Any disease causing vertigo can be considered a 'dangerous' medical problem, if the symptoms tend to occur in dangerous circumstances (e.g., highway driving or free-rock climbing). Furthermore, the high vagal tone that accompanies some vestibular disorders can provoke bradyarrhythmias in susceptible individuals. Nevertheless, although they may be quite disabling during the acute illness phase, diseases classified here as 'Common, Benign Causes' rarely produce severe, irreversible morbidity or mortality (unlike their 'Dangerous Mimics' counterparts).

‡Vestibular migraine episodes may last longer than a day in about 25% of cases.[10]

Table 2. 'Safe-to-Go' steps for bedside evaluation of acutely dizzy patients – History, Review of Systems, Physical Exam

I. HISTORY: **Plenty of Protective P's**	**II. REVIEW OF SYSTEMS:** **Dearth of Deadly D's***	**III. PHYSICAL EXAMINATION:** Choose either '**WAS DIZZY**' *or* '**STILL DIZZY**' Exam
If symptoms are old & recurrent… 1. **PERIODIC & PROLONGED**: recurrent, stereotyped episodes or bouts over a protracted period (longer than ~2-4 years); current episode is typical in all respects *Or, if symptoms are more recent…* 1. **PAINLESS**: head or neck pain, if present, should sound like migraine or tension-type headaches, and ***must not be any of the following***... • **SEVERE** • **SUDDEN** (peak intensity <30min) • **SUSTAINED** (duration >72hrs) *Plus, if there is vomiting…* 2. **PROPORTIONAL PUKING**: vertigo worse than vomiting might be o.k.; vomiting worse than vertigo is bad *Plus, if there is loss of consciousness…* 3. **PROTOTYPICAL PASSING OUT**: classic vasovagal syncope (with typical provocation & prodrome) is o.k.; anything else is probably bad	***No*** *Vascular Brainstem Symptoms…* 1. **Diplopia** (double vision) 2. **Dysarthria** (trouble speaking) 3. **Dysphagia** (trouble swallowing) 4. **Dysphonia** (hoarseness/hiccups) 5. **Dysmetria** (clumsiness) 6. **Dysesthesia** (facial numbness) 7. **Drop Attacks** (sudden falls without loss of consciousness) [i] 8. **Down-is-up Distortions** (room tilt & room inverted illusions) [ii] ***No*** *Vascular Inner Ear Symptoms…* 9. **Deafness** (any transient or bilateral hearing loss is bad; abrupt-onset unilateral loss [esp. if severe] may also be bad, but could be benign) ***No*** *Cardiovascular Symptoms…* 10. **Dyspnea** (any cardiorespiratory symptoms, unless clearly related to vasovagal or panic attack are bad)	'**WAS DIZZY**': *If symptoms are intermittent or gone, look for BPPV, orthostasis, or normal exam and classic history* **"P-Power to send Patient Packing"** 1. **Position-Provoked with Positive 'Pike'** (upbeat-torsional nystagmus on Dix-Hallpike), *or*... 2. **Postural with Predictable Pressure Plunge**, (*symptomatic* orthostatic BP drop on arising), *or*... 3. **Pristine exam & Paradigmatic Presentation** (BPPV,‡ vasovagal, migraine, Meniere, or panic) '**STILL DIZZY**': *If symptoms persist, confirm APV by excluding brainstem, cerebellar, & middle ear signs* **"IF SAFE & CLEAR THEN I'LL SEND HIM ON HOME"**† 1. **Intact Fields** (no visual field cut) 2. **Stands Alone** (able to stand unassisted) 3. **Face Even** (no weakness or droop, nor ptosis) 4. **CLear Enunciation** (no slurred or hoarse speech) 5. **Accurate Reaching** (no drift; normal reaching, RAM) 6. **THErmal Normal** (equal thermal or sharp sense) 7. **Isocoria in Low Light** (equal pupils in *dim* light) [iii] 8. **Straight Eyes** (normal ocular alignment, esp. vertical) 9. **Not Deaf** (no moderate to severe hearing loss) 10. **Head Impulse Misses** (abnormal head thrust VOR) [iv] 11. **One-way Nystagmus** (unidirectional, horizontal) [v] 12. **Healthy Otic & Mastoid Exam** (pearly; no pimples, pus, perforation, or pain on palpation of mastoid)

ABBREVIATIONS: APV – acute peripheral vestibulopathy; RAM – rapid alternating movements; VOR – vestibulo-ocular reflex

TABLE SYMBOLS & FOOTNOTES

* – Deadly D's should be excluded both by review of systems and corresponding examination (e.g., diplopia *or* ocular misalignment).

† – This acronym can be abridged to "SEND HIM ON HOME" by mandating that a thorough general neurologic exam be normal. These "Still Dizzy" criteria can also be met by a normal MRI with DWI that takes place after 24-48 hours of continuous vertigo (in the first ~24 hours, false negative DWIs have occurred).

‡ – Since BPPV is a relapsing and remitting disorder in which otoliths intermittently get stuck in the semicircular canals, it is possible for the patient to present clinically having experienced classic ("paradigmatic") historical symptoms with head-back triggers and short-lived vertigo episodes, yet not have the confirmatory eye findings on Dix-Hallpike maneuver. Thus, although it is comforting to identify the classic nystagmus on exam, it is not essential for diagnosis.

i – Drop attacks are bad. In association with vertigo, they imply brainstem ischemia until proven otherwise. Drop attacks are common in patients with Meniere disease[11] and occasional in patients with other inner ear diseases.[12-14] However, in the absence of a clear Meniere disease history (repeated episodes over years, and associated fluctuating tinnitus and/or hearing loss during the attacks), they should spark concern for an underlying cerebrovascular[15,16] or cardiovascular[17] etiology for vertigo. Although drop attacks are also seen in epilepsy and narcolepsy-cataplexy, in such cases, they are generally not associated with vertigo.

ii – Room tilt illusions are bad. These rare symptoms point to brainstem ischemia until proven otherwise. Tilt illusions can be seen in patients with Meniere and other inner ear diseases,[18] but, in the absence of an unequivocal history confirming otic involvement (i.e., longstanding attacks with fluctuating tinnitus, symptoms provoked by noise [Tullio phenomenon], etc.), a cerebrovascular cause should be surmised.[19,20]

iii – In a patient with vertigo lasting days, anisocoria is bad. Searching for anisocoria in *bright* light is insufficient. Pupils must be examined in *dim* light, since the anisocoria of Horner syndrome (oculosympathetic palsy, suggesting brainstem pathology) is usually only clinically apparent in dim light or darkness. Note that even in low light, the amount of anisocoria is typically less than 2mm. The associated ptosis of Horner syndrome is also generally subtle (1-2mm).

iv – In a patient with vertigo lasting days, a *normal* head impulse test is bad. An abnormal "head thrust sign" during the head impulse test[21] is seen in the vast majority of patients with APV.[22,23] Unfortunately, the same sign is routinely also seen in patients with labyrinthine infarction (e.g., from AICA occlusion), and sometimes seen in patients with brainstem or cerebellar stroke.[24] So, in the context of an acute vestibular syndrome with persistent vertigo, the finding's *absence* (suggesting brainstem or cerebellar stroke) is probably more informative than its *presence* (suggesting *either* benign APV or not-so-benign labyrinthine or brainstem/cerebellar infarct). An abnormal head thrust sign can therefore be thought of as *necessary* but *not sufficient* for a "safe-to-go" departure.

v – In a patient with vertigo lasting days, direction-changing nystagmus is bad. It generally indicates a central cause, often a stroke. Nystagmus associated with APV (a.k.a. labyrinthitis, vestibular neuritis) does not usually change direction when the patient looks in different directions (i.e., it remains unidirectional).[25] Since the nystagmus of APV is predominantly horizontal in vector, this means that APV nystagmus should *not* beat right-ward in right gaze, and left-ward in left gaze (so called 'gaze-evoked' nystagmus, which instead, is highly suggestive of brainstem or cerebellar stroke in this clinical context). Unfortunately, the converse is not true, since the majority of brainstem and cerebellar stroke patients, particularly those who present with acute vertigo, have unidirectional nystagmus that mimics APV.[8,24,26-28] Some such strokes mimic APV so closely as to be indistinguishable from APV even when eye movements are recorded and assessed using specialized electro-oculographic measurement devices.[29,30] The presence of unidirectional nystagmus can therefore be thought of as *necessary* but *not sufficient* for a "safe-to-go" departure.

Appendix 4.1 Proposed "safe-to-go steps" for bedside evaluation of acute dizziness (References)

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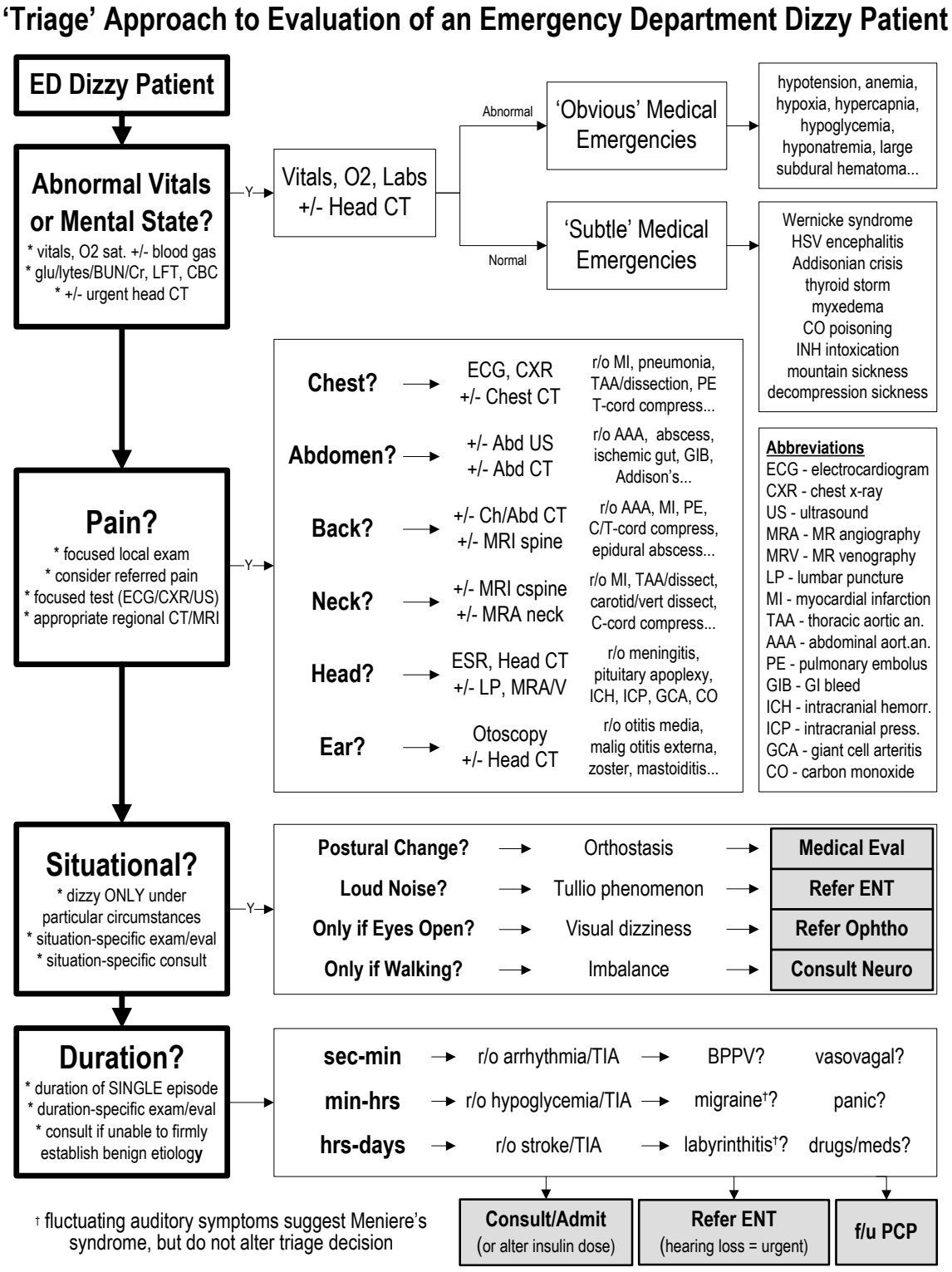
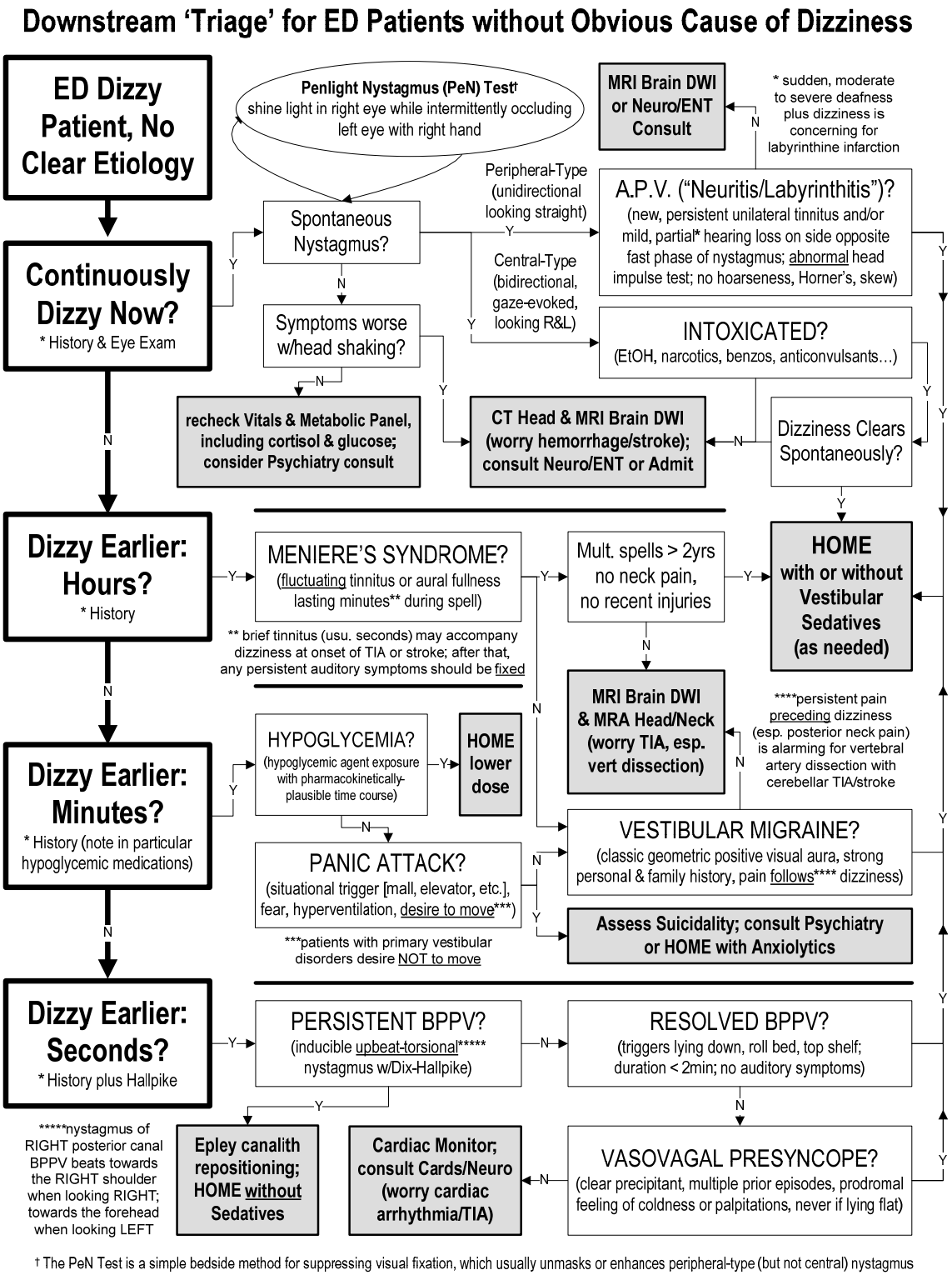
Appendix 4.2 Proposed "triage" algorithm for bedside evaluation of acute dizziness



Appendix 4.2 Proposed "triage" algorithm (continued)



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David E. Newman-Toker, M.D.

Curriculum Vitae, Johns Hopkins University School of Medicine

Last updated March 30, 2007

DEMOGRAPHIC INFORMATION

Current Appointments:

University Appointments (Johns Hopkins University)

Primary Appointment:

Assistant Professor of Neurology, full-time (7/1/02-present)

Secondary Appointments (School of Medicine):

Assistant Professor of Otolaryngology (7/1/02)
Joint Appointment in Ophthalmology (7/1/02)
Joint Appointment in Health Sciences Informatics (7/1/02)
Joint Appointment in Emergency Medicine (anticipated 5/07)

Secondary Appointments (Bloomberg School of Public Health):

Assistant Professor of Epidemiology (4/1/03)
Joint Appointment in Health Policy and Management (7/1/03)

Hospital Appointments

Active Staff, Neurology, The Johns Hopkins Hospital, Baltimore, MD
Active Staff, Neurology, The Johns Hopkins Bayview Medical Center, Baltimore, MD

Personal Data:

Date of Birth: 10/14/1969

Place of Birth: New York, NY

Mailing address:
Johns Hopkins Hospital
Pathology Building, 2-210
Baltimore, MD 21287

Contact Information:
410-614-1576 (phone)
410-614-1746 (fax)
toker@jhu.edu

Education and Training (in chronological order):

B.S., 1991, Yale University, Molecular Biophysics & Biochemistry
M.D., 1995, University of Pennsylvania School of Medicine
Intern, 1996, Harvard University/Massachusetts General Hospital, Internal Medicine
Resident, 1999, Harvard University/Massachusetts General Hospital, Neurology
Fellow, 2000, Harvard Univ./Massachusetts Eye & Ear Infirmary, Neuro-ophthalmology
Fellow, 2002, Johns Hopkins University/Johns Hopkins Hospital, Neuro-otology
Ph.D., 2007 (anticipated), Bloomberg School of Public Health, Clinical Investigation

Professional Experience (in chronological order):
Clinical Clerk in Neurology, Institute of Neurology, Queen Square, London, UK 3-5/95
Clinical Clerk in Neuro-Otology, Royal Prince Alfred Hospital, Sydney, AU 10-12/97
Clinical Associate in Neurology, Massachusetts General Hospital, Boston, MA 7/99-6/00
Senior Clinical Fellow in Neurology, Johns Hopkins Hospital, Baltimore, MD 3/01-6/02
Assistant Professor of Neurology, Johns Hopkins Hospital, Baltimore, MD 7/02-present

RESEARCH ACTIVITIES

Publications:

Selected Peer-Reviewed Scientific Articles (full-length articles)

Newman-Toker, DE. Charted records of dizzy patients suggest ED physicians emphasize symptom quality in diagnostic assessment. Annals of Emergency Medicine 2007 (research letter, in press).

Cheong R, Wilson RK, Cortese ICM, **Newman-Toker DE**. Mothball Withdrawal Encephalopathy – Case Report and Review of Paradichlorobenzene Neurotoxicity. Substance Abuse, 2007 Mar; 27(4):63-67.

Castle J, Sakonju A, Dalmau J, **Newman-Toker DE**. Anti-Ma2-associated encephalitis with normal FDG-PET: a case of pseudo-Whipple's disease. Nature Clinical Practice Neurology 2006 Oct; 2(10):566-572.

Newman-Toker DE, Camargo CA Jr. 'Cardiogenic Vertigo' – True vertigo as the presenting manifestation of primary cardiac disease. Nature Clinical Practice Neurology 2006 Mar; 2(3):167-172.

Newman-Toker DE, Horton JC, Lessell S. Recurrent visual loss in Leber hereditary optic neuropathy. Archives of Ophthalmology 2003 Feb; 121(2):288-291.

Kelly PJ, **Toker DE**, et al. Granulomatous compressive thoracic myelopathy as the initial manifestation of Wegener's granulomatosis. Neurology 1998; 51(6): 1769-1770.

Selected Peer-Reviewed Scientific Articles (abstracts & posters)

Cnyrim CD, **Newman-Toker DE**, Karch C, Brandt T, Strupp M. How to clinically differentiate between vestibular neuritis and "vestibular pseudoneuritis." Poster presentation at the European Neurological Society 16th Meeting, May 2006.

Newman-Toker DE, Guardabascio LM, Zee DS, Rothman RE. Taking the history from a dizzy patient – why "What do you mean by dizzy?" should not be the first question you ask. Poster presentation at the Society for Academic Emergency Medicine, May, 2006. Acad Emerg Med 2006 13(5 Supplement 1): S79.

Guardabascio, LM, Rothman RE, Zee DS, **Newman-Toker DE**. Chief complaint screening – a new method for symptom-oriented research in the Emergency Department. Poster presentation at the Society for Academic Emergency Medicine, May, 2006. Acad Emerg Med 2006 13(5 Supplement 1): S146.

Newman-Toker DE. Charted records of Emergency Department dizzy patients suggest overemphasis on symptom quality may be associated with diagnostic errors. American College of Emergency Physicians Research Forum. October 12-13, 2003, Boston, Massachusetts, USA. Abstracts. Annals of Emergency Medicine 2003 October; 42(4 Supplement):S80 (#295).

Newman-Toker DE. Common misconceptions in the evaluation of ED dizzy patients parallel those found in Emergency Medicine texts. Poster presentation at the Society for Academic Emergency Medicine, May, 2003. Academic Emergency Medicine 2003 May;10(5):491-2.

Newman-Toker DE, Rizzo JR III. Maddox Rod vs. Alternate Cover Testing in Neuro-ophthalmic Practice. Platform presentation at the North American Neuro-Ophthalmology Society Meeting, February, 2003.

Newman-Toker DE, Newman-Toker JR, Lehmann HP, Zee DS. Proposal for a multi-layer ontology to aid in classification of vestibular disorders. Abstracts of the XXII Barany Society Meeting. Seattle, Washington, USA. 26-29 September 2002. J Vestib Res. 2001-2002;11(3-5):281.

Newman-Toker DE, Zee DS. Building a new model for diagnosis of dizzy patients in the Emergency Department. Abstracts of the XXII Barany Society Meeting. Seattle, Washington, USA. 26-29 September 2002. J Vestib Res. 2001-2002;11(3-5):281-2.

Newman-Toker DE, Rizzo JR III. Intra-arterial thrombolysis of acute central retinal artery occlusion – preliminary data and methodologic approach. Poster presentation at the North American Neuro-Ophthalmology Society Meeting, March, 2000. Abs#38 p56.

Book Chapters & Monographs

Zee DS, **Newman-Toker DE**. Supranuclear and Internuclear Ocular Motor Disorders. In In Walsh and Hoyt's Clinical Neuro-Ophthalmology, 6th edition. Editors, Miller NR, Newman NJ, Biousse V, Kerrison JB. Baltimore, Lippincott-Williams & Wilkins, 2005.

Other Media (films, videos, CD-ROMs, slide sets, etc.)

Skew Deviation and the Ocular Tilt Reaction (slides, published on the web-based, open access Neuro-ophthalmology Virtual Education Library [NOVEL], Univ. Utah, 12/05).

Extramural Sponsorship:

Grants (Current)

Title:	Building a New Model for Diagnosis of ED Dizzy Patients
Dates:	12/1/02-11/30/07
Sponsor:	NIH (NCRR, K23)
Identification Number:	1K23RR17324-01
Role:	Principal investigator

Grants (Previous)

Title:	Building a New Model for Diagnosis of ED Dizzy Patients
Dates:	7/1/02-6/30/03
Sponsor:	FERNE
Role:	Principal investigator

Title:	BME Training Grant for Vestibular Research
Dates:	7/1/01-6/30/02
Sponsor:	NIH
Identification Number:	NRSA 5 T32 DC00023
Role:	Trainee
Principal Investigator:	Murray Sachs, PhD

Editorial Activities:

Journal peer review activities

Peer Reviewer, Annals of Neurology (Fall, 2001-present)

EDUCATIONAL ACTIVITIES

Teaching:

Course Directorships

JHU SOM 1st-Year Genes to Society Course, Mind-Brain-Behavior Block
Course Length: 10-12 weeks, 1 cycle per year
Role: Block Co-Director (with Jay Baraban, Dean MacKinnon)
Dates in Role: planning 10/05-present, to first be offered Spring 2010

JHU SOM 2nd-Year Pathophysiology Course, Neuro Block
Course Length: 2 weeks, 1 cycle per year
Role: Block Director
Dates in Role: 1/03-present

JHU SOM 2nd-Year Transition to the Wards Course
Course Length: 4 weeks, 1 cycle per year
Role: Course Director
Dates in Role: planning 10/05-present, to first be offered Spring 2011

JHU SOM 3rd-Year Neurology Clinical Clerkship
Course Length: 4 weeks, 10 cycles per year
Role: Clerkship Director
Dates in Role: 9/02-present

JHU SOM 4th-Year Neurology Sub-Internship
Course Length: 4 weeks, 12 cycles per year
Role: Sub-Internship Director
Dates in Role: 9/02-present

Classroom instruction

Current Didactic, Classroom Teaching Per Year: ~110-120 contact hours per year
(~30-35 hours 1st & 2nd year medical students, ~70-75 hours 3rd & 4th year students, ~4-6 hours Neurology residents, ~2-5 hours local community attending physicians)

INSTRUCTION AT RESIDENT LEVEL:

Johns Hopkins Hospital Neurology Resident Lecture Series
"Neurology of Eye Movements V: Ocular Tilt & Skew Deviation" (11/05)
"Neurology of Eye Movements III: Smooth Pursuit & VOR" (10/05)
"Neurology of Eye Movements II: Saccades & Vergence" (10/05)
"Neurology of Eye Movements I: The Oculomotor Plant" (10/05)
"Dangerous Headaches – What's NOT Migraine?" (8/04)
"Oculomotor Anatomy III – Torsional Eye Movements" (8/04)
"Oculomotor Anatomy II – Vertical Eye Movements" (8/04)
"Oculomotor Anatomy I – Horizontal Eye Movements" (8/04)
"Brainstem Anatomy" (8/04)
"Diplopia" (5/04)
"Diplopia" (8/03)
"Transient Neurologic Deficits" (8/03)
"Oculomotor Anatomy II – Horizontal Eye Movements (Advanced)" (8/02)
"Oculomotor Anatomy I – Horizontal Eye Movements (Basic)" (8/02)
"Bedside Evaluation of Ocular Motility Disorders" (8/02)
"Brainstem Neuroanatomy Made Ridiculously Simple" (8/02)
"The Neuroanatomy of Adaptive Supranuclear Oculomotor Control Mechanisms" (5/02)
"The Neuroanatomy of Primary Supranuclear Oculomotor Control Mechanisms" (4/02)
"Nystagmus and Related Oscillatory Eye Movement Disorders" (10/01)

Johns Hopkins Hospital Emergency Medicine Resident Lecture Series
"A New Approach to Evaluation of the Dizzy Patient" (3/04)
"Transient Neurologic Dysfunction: When to Worry" (1/01)
"Evaluation of Dizziness in the Emergency Ward" (12/00)

Massachusetts Eye & Ear Infirmary Ophthalmology Resident Lecture Series
"Migraine: Current Concepts & Clinical Approach" (6/00)
"Hysteria & Functional Visual Loss" (8/99)

Massachusetts General Hospital Neurology Resident Lecture Series
"Double Vision – A Practical Approach to Bedside Diagnosis" (8/01)
"Evaluation of Acute Dizziness in the Emergency Ward" (10/98)

INSTRUCTION AT MEDICAL STUDENT LEVEL:

JHU SOM 1st-Year Neuroscience Course (Lecturer, Small Group Leader)

Feb.-March, 2006: Discussion-group leader (clinical case correlations, 1 session)
Feb.-March, 2005: Discussion-group leader (clinical case correlations, 5 sessions)
Feb.-March, 2004: Discussion-group leader (clinical case correlations, 3 sessions)
Feb.-March, 2003: Guest lecturer & laboratory instructor (Brainstem & Vestibular Labs)

JHU SOM 2nd-Year Pathophysiology Course, Neuro Block (Lecturer, Lab Leader)

Feb.-Mar., 2007: Course Director, lab instructor (9 sessions), lecturer (9 lectures)
"Course Introduction & Overview of Functional Neuroanatomy"
"Functional Neuroanatomy of the Brainstem & Cranial Nerves"
"Localization in Neurology: Functional Pathoanatomy" (I & II)
"Patho-Anatomy of Neuro-op Signs in Cerebrovascular Disease"
"Headaches Syndromes and the Pathophysiology of Migraine Pain"
"Dizziness, Vertigo, and the Pathophysiology of Nystagmus"
"Episodic Neurologic Symptoms - Channelopathies & Beyond"
"Neuropathology-Pathophysiology Structured Review"

Jan.-Feb., 2006: Course Director, lab instructor (8 sessions), lecturer (9 lectures)
"Course Introduction & Overview of Functional Neuroanatomy"
"Functional Neuroanatomy of the Brainstem & Cranial Nerves"
"Localization in Neurology: Functional Pathoanatomy" (I & II)
"Dizziness, Vertigo, and the Pathophysiology of Nystagmus"
"Patho-Anatomy of Neuro-op Signs in Cerebrovascular Disease"
"Headaches Syndromes and the Pathophysiology of Migraine Pain"
"Episodic Neurologic Symptoms - Channelopathies & Beyond"
"Neuropathology-Pathophysiology Structured Review"

Jan.-Feb., 2005: Course co-director, lab instructor (10 sessions), lecturer (5 lectures)
"Functional Neuroanatomy of the Brainstem & Cranial Nerves"
"Localization in Neurology: Functional Pathoanatomy"
"Migraine, Tension, and Other Headaches"
"Vestibular Pathophysiology: Understanding Nystagmus"
"Neuropathology-Pathophysiology Structured Review"

Jan.-Feb., 2004: Course co-director, lab instructor (9 sessions), lecturer (4 lectures)
"Functional Neuroanatomy of the Brainstem & Cranial Nerves"
"Localization in Neurology: Functional Pathoanatomy"
"Migraine and Other Headache Syndromes"
"Neuropathology-Pathophysiology Structured Review"
Proctor, Student Written Examination

Jan.-Feb., 2003: Course co-director, lab instructor (10 sessions), lecturer (3 lectures)
"Localization in Neurology: Functional Pathoanatomy"
"Migraine and Other Headache Syndromes"
"Neuropathology-Pathophysiology Structured Review"

February, 2002: Laboratory instructor (10 sessions)

JHU SOM 2nd-Year Pathophysiology Course, Pain Block (Small Group Leader)

March, 2007: Small-group case session on headache (1hr)

JHU SOM Clinical Skills Course (1 cycle/year) (Lecturer, Small Group Leader)

Oct., 2002-05: "Introduction to the Neurologic History & Physical Examination"
February, 2002: Neurology Clinical Skills Instructor, Small Group (3 sessions)

JHU SOM 3rd-Year Neurology Clerkship (10 cycles/year) (Lecturer, S.G. Leader)

Sept., 2003-pres: Lecturer (4 lectures/mo)
"Neurologic Emergencies"
"History-Taking in Neurology"
"Dizziness"
"Headaches"

April, 2003-pres: Small group leader, patient case presentations (3-4 sessions/mo)
Jan.-Aug., 2004: Small group leader, 'Searching the Medical Literature' (1 session/mo)

JHU SOM 3rd-Year Ophthalmology Clerkship (5 cycles/year) (Lecturer)

Sept., 2005-pres: Lecturer "Optic Disc Edema & Optic Neuropathies"

Harvard SOM 2nd-Year Nervous System & Behavior (Lecturer, Lab Leader)

October, 1999: Guest lecturer (1 lecture)
"Basic Anatomy of the Afferent Visual System"

Sept.-Nov., 1997: Laboratory instructor (8 sessions), lecturer (3 lectures)
"Basic Anatomy of the Afferent Visual System"
"Basic Anatomy of the Brainstem: The Oculomotor System"
"Basic Anatomy of the Brainstem: The Lower Cranial Nerves"

Harvard SOM 3rd-Year Neurology Clerkship, Mass. General Hospital (Lecturer)

Summer, 1997: Course organizer and lecturer (12 lectures)

University of Pennsylvania SOM Clinical Skills Course (Small Group Leader)

Spring, 1994: Clinical Skills Instructor, Small Group (6 sessions)

INSTRUCTION AT ATTENDING LEVEL:

CME instruction

University of Maryland Health Center Annual CME Course 2007:
"Triage and Initial Management of the Acutely Dizzy Patient"
(University of Maryland-Sponsored CME Course, 1/07)

Neurology for the Neurologist 2006:
"Triage and Initial Management of the Acutely Dizzy Patient"
(Johns Hopkins University-Sponsored CME Course, 12/06)

Neurology for the Primary Care Provider 2006:
"Triage and Initial Management of the Acutely Dizzy Patient"
(Johns Hopkins University-Sponsored CME Course, 12/06)

Current Concepts in Ophthalmology 2006:
"'The World is Shaking' – Differential Diagnosis of Oscillopsia"
(Wilmer Eye Institute, Department of Ophthalmology CME Course, 12/06)

Hot Topics 2006 – Emergency Neurology:
"Emergency Evaluation of the Acutely Dizzy Patient"
(Medical University of Ohio-Sponsored CME Course, 5/06)

Topics in Clinical Medicine 2006:
"Meet the Professor" Roundtable Discussion – Neurology/Dizziness
(Johns Hopkins University-Sponsored CME Course, 5/06)

Neurology for the Neurologist 2005:
"Triage and Initial Management of the Acutely Dizzy Patient"
(Johns Hopkins University-Sponsored CME Course, 12/05)

Neurology for the Primary Care Provider 2005:
"Triage and Initial Management of the Acutely Dizzy Patient"
(Johns Hopkins University-Sponsored CME Course, 12/05)

Current Concepts in Ophthalmology 2005:
"Office Differentiation of Skew Deviation from 4th Nerve Palsy"
(Wilmer Eye Institute, Department of Ophthalmology CME Course, 12/05)

Topics in Clinical Medicine 2005:
"Meet the Professor" Roundtable Discussion – Neurology/Dizziness
(Johns Hopkins University-Sponsored CME Course, 5/05)

Neurology for the Primary Care Provider 2004:
"A New Approach to Evaluation of the Dizzy Patient"
"CNS Neurodiagnostics"
(Johns Hopkins University-Sponsored CME Course, 12/04)

Current Concepts in Ophthalmology 2004:
"Maddox Rod Testing in Patients with Diplopia – Does it Help?"
(Wilmer Eye Institute, Department of Ophthalmology CME Course, 12/04)

Neurology for the Primary Care Provider 2003:
"A New Approach to Evaluation of the Dizzy Patient"
"CNS Neurodiagnostics"
(Johns Hopkins University-Sponsored CME Course, 12/03)

Neurology for the Primary Care Provider 2002:
"A New Approach to Evaluation of the Dizzy Patient"
"CNS Neurodiagnostics"
(Johns Hopkins University-Sponsored CME Course, 12/02)

Current Concepts in Ophthalmology 2001:
"Neuro-ophthalmic Diseases Masquerading as Benign Strabismus"
(Wilmer Eye Institute, Department of Ophthalmology, 12/01)

CLINICAL ACTIVITIES

Certification:

Medical, other state/government licensure

Massachusetts State Medical License (5/12/99-10/14/01)
Massachusetts MCSR (5/24/99-7/1/00)
Maryland State Medical License (2/20/01-present)
Maryland CDS (2/22/01-present)
Federal DEA License (6/3/99-present)

Boards, other specialty certification

American Board of Psychiatry and Neurology Diplomate (April, 2000)

Service Responsibilities:

Clinic (Neuro-otology & Neuro-ophthalmology): 0.5 clinic days per week, 3/01-2/05
Ward Attending (General Neurology Service or Consults): 2-6 weeks per year, 8/02-pres.

ORGANIZATIONAL ACTIVITIES

Institutional Administrative Appointments:

- Educational Policy Committee
    a. EPC Member (SOM, 9/02-present)
    b. EPC Clerkship Directors Subcommittee Member (SOM, 9/02-present)
    c. Student Promotions Committee Member (SOM, 9/02-9/04)
- Student Assessment & Program Evaluation (SAPE) Committee
    a. SAPE Committee Member (SOM, 8/05-7/06)
- Simulation Center Steering Committee
    a. Simulation Center Steering Committee Member (SOM, 10/05-present)
- Curriculum Reform Committee
    a. Genes to Society Integration Committee Member (SOM, 12/06-present)
    b. CRC Steering Committee Member (SOM, 9/05-1/06)
    c. Educational Methods Subcommittee Member (SOM, 10/05-10/06)
    d. Measurement Subcommittee Member (SOM, 3/04-1/05)
    e. Basic Science Subcommittee Member (SOM, 2/04-1/05)
    f. Clinical Sciences Subcommittee Member (SOM, 11/03-10/06)
    g. Technology in Education Subcommittee Member (SOM, 11/03-1/05)
- 1st Year Genes to Society Course (new curriculum 2008-9)
    a. GTS Steering Committee Member (11/05-9/06)
    b. Mind-Brain-Behavior Block Co-Director (11/05-present)
    c. Mind-Brain-Behavior Curriculum Subcommittee Member (SOM, 1/05-11/05)
- 2nd Year Neurology/Neuropathology Course
    a. Neurology/Neuropathology Course Block Co-Director (SOM, 1/03-present)
    b. Neurology/Neuropathology Block Committee Member (SOM, 3/02-present)
    c. 2nd Year Pathophysiology Focus Group Member (SOM, 2/04-present)
- 2nd Year 'Transition to the Wards' Course (new curriculum Spring 2010)
    a. Planning Committee Chairman (10/05-present)
    b. Course Director (10/05-present)
- 3rd & 4th Year Neurology Clerkship
    a. Neurology Basic Clerkship Director (Neurology, 9/02-present)
    b. Neurology Advanced Clerkship Co-Director (Neurology, 9/02-present)
    c. Neurology Education Committee Member (Neurology, 9/02-present)

Professional Societies:

- American Academy of Neurology [AAN]
- Society for Academic Emergency Medicine [SAEM]
- North American Neuro-Ophthalmology Society [NANOS]
- American Federation for Medical Research [AFMR]

Conference Organizer, Session Chair:

- Faculty & Resident Development
    a. Organizer/Moderator for "Effective and Efficient Outpatient Clinical Teaching - a Primer and Panel Discussion" (12/21/06)

Advisory Committees, Review Groups:

- Search Committees
    a. Clinical Skills Director Search Committee Member (SOM, 11/04-2/05)

- Internal Review Committees
    a. Residency Training Program Review Committee Member (SOM, 11/03)

- Development Committees
    a. Simulation Center Development Committee Member (SOM, 2/03)
    b. ACGME Medical Residency Curriculum Workshop (Medicine, 6/02)
    c. Career Development Working Group Participant (Neurology, 4/02)

- Applicant Selection Committees
    a. Neurology Residency Applicant Interviewer (12/02-pres)
    b. Undergraduate Applicant Alumni Interviewer (Yale Univ., 11/01-present)

RECOGNITION

Awards, Honors:

Teaching Awards & Recognition

- American Neurological Association's Teaching Scholar Program, Fellowship Recipient (Rochester, NY 8/07-10/07). The purpose of this program is to train neurologists to become master teachers and administrators for integrated neuroscience courses.

- Nominee, Teacher of the Year for the Basic Sciences, Class of 2009 (JHU SOM, 3/07). This prize is awarded by JHU medical students to the best teacher in the basic science year 2.

- Runner Up, Teacher of the Year for the Clinical Sciences, Class of 2006 (JHU SOM, 5/06). This prize is awarded by JHU medical students to the best teacher in the clinical years.

- 2nd Runner Up, Teacher of the Year for the Basic Sciences, Class of 2007 (JHU SOM, 5/05). This prize is awarded by JHU medical students to the best teacher in the basic science year 2.

- JHU SOM LCME School-Wide Self-Study Survey Report Citations (4/05):

    a. **"Pathophysiology** Generally well received with no group lower than 50% satisfaction and 5 of 7 above 65% satisfaction. In particular, Renal and Neurology received excellent reviews. The quality of teaching in these sections was consistently complemented [*sic*] as outstanding. Michael Choi and David Newman-Toker were named again and again."

    b. **"Neurology** followed with a satisfaction level of 74.2% (*behind Emergency Medicine 80.2% and Internal Medicine 75.0%, and ahead of Pediatrics 67.6%, Psychiatry 63.5%, Surgery 62.1%, Ambulatory Medicine 59.1%, Obstetrics & Gynecology 53.8%, and Ophthalmology 44.9%*). It is currently approximately a 4 week rotation with 3 weeks of inpatient service or consult service and a week of outpatient Neurology. Students enjoyed the variety of inpatient and outpatient care seen — a week of outpatient medicine is built into the course. Students felt "very welcome" by the attendings and residents. There was much "personal attention." The course director, David Newman-Toker, was highlighted for his "great advances" and teaching ability."

- 2nd Runner Up, Teacher of the Year for the Basic Sciences, Class of 2006 (JHU SOM, 5/04). This prize is awarded by JHU medical students to the best teacher in the basic science year 2.

- Runner Up, Fellow of the Year Teaching Award (Massachusetts Eye & Ear Infirmary, 5/00). This prize is awarded by the MEEI Ophthalmology residents to the best teaching fellow.

Other Awards & Recognition

- Office of Behavioral & Social Sciences Research Scholarship for the NIH Summer Training Institute on the Design and Conduct of Randomized Clinical Trials Involving Behavioral Interventions (National Institutes of Health, Awarded 5/03)

- William T. Fitts, Jr. Memorial Prize (University of Pennsylvania School of Medicine, 5/95). "This prize is awarded to a graduating student for excellence in the surgery of trauma."

- *Cum Laude*, Molecular Biophysics & Biochemistry, Yale University, 1991.

Invited Talks, Panels:

Invited Extramural – Grand Rounds

"Diagnosing the Dizzy Patient: Why "What do you mean by 'dizzy'?" should NOT be the first question you ask" (Massachusetts General Hospital, Dept. Neurology, Grand Rounds, 5/07)

"Emergency Evaluation of the Acutely Dizzy Patient"
(University of Utah, Salt Lake City, UT, Department of Neurology, Grand Rounds, 7/06)

"Emergency Evaluation of the Acutely Dizzy Patient"
(York Hospital, York, PA, Department of Internal Medicine, Grand Rounds, 5/06)

"Triage and Initial Management of the Acutely Dizzy Patient"
(Presbyterian Hospital, New York, NY, Department of Emergency Medicine, Grand Rounds, 12/05)

"Skew Deviation & the OTR"
(Moran Eye Institute, University of Utah, Salt Lake City, UT, Dept. Ophthalmology, Grand Rounds, 11/05)

"21st Century Neuro-Otology: Towards Automated Triage of the E.D. Dizzy Patient"
(Hospital University of Pennsylvania, Philadelphia, PA, Department of Neurology, Grand Rounds, 5/05)

"A New Approach to Evaluation of the Dizzy Patient"
(Saint Agnes Hospital, Baltimore, MD, Department of Emergency Medicine, Grand Rounds, 3/04)

"Building a New Model for Diagnosis of ED Dizzy Patients"
(Massachusetts General Hospital, Boston, MA, Department of Neurology, Grand Rounds, 1/02)

"Preventing Misdiagnosis of Dizzy Patients in the Emergency Department - Designing a Systematic Approach to Bedside Diagnosis"
(Univ. Florida Health Science Center, Jacksonville, FL, Dept. Emergency Medicine, Grand Rounds, 1/02)

"Evaluation of Dizziness in the Urgent Care Setting"
(Massachusetts General Hospital, Chelsea Internal Medicine Group, Chelsea, MA, 11/99 [I], 4/00 [II])

Invited Extramural – National Meetings & Societies

Panelist, Trainee Education Luncheon, "Keeping the Balance" (tips on balancing career and home life for the junior investigator), AFMR National Meeting, 3/03

Invited Intramural Talks & Panels

"Diagnosing the Dizzy Patient: why 'What do you mean by dizzy?" should NOT be the first question you ask" (Johns Hopkins Hospital, Department of Neurosurgery, Grand Rounds, 1/07)

"Diagnosing the Dizzy Patient: why 'What do you mean by dizzy?" should NOT be the first question you ask" (Johns Hopkins Hospital, Department of Neurology, Grand Rounds, 11/06)

"Why 'What do you Mean By Dizziness?' Shouldn't Be the First Question You Ask"(Johns Hopkins Hospital, Department of Emergency Medicine, Research Day, 5/06)

"Why 'What do you Mean By Dizziness?' Shouldn't Be the First Question You Ask"(Johns Hopkins Hospital, Division of Health Science Informatics, Informatics Conference, 9/05)

"Identifying 'The Dizzy Patient' in the Emergency Department"
(Bloomberg School of Pub. Health, Dept. Health Pol. & Manage., Qualitative Res. Methods Group, 5/05)

"The Spiral Curriculum, A New Approach to Medical Education"
(Johns Hopkins University, School of Medicine, Curriculum Reform Committee Meeting, 5/04)

"Health-Related Quality of Life in Patients with Dizziness"
(Johns Hopkins Hospital, Departments of Neurology & Otolaryngology, Neuro-otology Conference, 11/02)

"Improving Patient Safety & Diagnostic Errors in Dizziness: How Rules Have Failed Us"
(Johns Hopkins Hospital, Multidisciplinary Health Sciences/Pathology Informatics Seminar, 2/02)

"Neuro-ophthalmic Diseases Masquerading as Benign Strabismus"
(Massachusetts Eye & Ear Infirmary, Department of Ophthalmology, Annual Fellows' Course, 5/00)

"Posterior Circulation Ischemia"
(Massachusetts General Hospital, Department of Internal Medicine, 8/98)

Invited Reviews, Editorials:

Newman-Toker DE. Time management top 10 list. J Investig Med 2004 May; 52(4):262-4.

Newman-Toker DE, Rizzo J. Neuro-ophthalmic diseases masquerading as 'benign' strabismus. International ophthalmology clinics 2001 Fall; 41(4):115-27.

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DR DAVID E NEWMAN TOKER
DEPT OF NEUROLOGY
PATHOLOGY 2-210
600 N WOLFE ST
BALTIMORE, MD 21287-0005

Invoice # B49800636 Customer # 000150562407 FEE: 0.00
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DOCTORAL DISSERTATION

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