essentially rules out a stroke, just as seeing an intrauterine pregnancy on ultrasound in a patient with first-trimester vaginal bleeding excludes an ectopic pregnancy (barring two simultaneous diagnoses in the first case and a heterotopic pregnancy in the second).

In ED patients with acute dizziness, it is the characteristics (not simply the presence or absence) of nystagmus that can be extremely helpful in making a confident diagnosis and yet studies show that emergency clinicians harbor misconceptions about how nystagmus informs the diagnostic process.[14,56–58] Collectively, these studies show that when nystagmus is documented by front-line providers, the descriptions of the nystagmus are often inconsistent with the recorded diagnoses, suggesting that either the clinician was misinterpreting the type of nystagmus or they were misinterpreting its diagnostic significance, for example, diagnosing BPPV in a patient with spontaneous nystagmus. Furthermore, a recently completed clinical trial (AVERT NCT02483429) found nystagmus descriptions by emergency clinicians frequently did not match eye movements recorded contemporaneously in the ED by portable VOG (D.E. Newman-Toker, unpublished data). The details of nystagmus can be extremely helpful both in making a specific diagnosis of peripheral vestibular causes and in distinguishing peripheral from central ones (Table 2 and Figure 2).

Thus, use of a flawed symptom quality paradigm, knowledge gaps related to bedside diagnostic and therapeutic maneuvers, limitations of brain imaging, and inconsistent availability of MRI all contribute to non–evidence-based management.[16] It is not surprising that emergency clinicians consistently select dizziness and vertigo as a high priority for a clinical decision rule for adult patients.[77,78] It also helps explain the high misdiagnosis rate in patients with acute dizziness, with one study showing that emergency clinicians missed over a third of strokes presenting with dizziness (16 of 46 validated stroke or TIA cases were missed in the ED).[26] Figure 3 illustrates some of the factors related to misdiagnosis.

The objective of this guideline is to provide an evidence-based framework intended to support patients, clinicians, and other health care professionals in their decisions about the evaluation and management of adult ED patients with acute dizziness who do not have an obvious central cause with frank neurological findings or an obvious general medical one.

The important eye findings related to dizziness are dynamic. Seeing video clips of the various bedside techniques for diagnosis and treatment of acute dizziness is key to understanding them. Therefore, to maximize the impact of this guideline, the GRACE-3 committee have created a multimedia educational smartphone app

TABLE 2 Common nystagmus patterns useful for diagnosis of acutely dizzy patients.

Nystagmus pattern	Nystagmus characteristics	Common causes
Peripheral vestibular		
Positional	• Transient (lasts <30 s) upbeat–torsional nystagmus triggered by the Dix–Hallpike test • Transient (lasts <90 s) horizontal nystagmus triggered by the supine roll test and beating toward the lowermost ear ("geotropic"). In hc-BPPV, the nystagmus is seen no matter which side the head is turned	• pc-BPPV • hc-BPPV
Persistent	• Horizontal spontaneous[a] (present on primary gaze) nystagmus that is unidirectional (never changes direction with different gaze positions or positional tests ["direction-fixed"])	• Vestibular neuritis (*but can be seen in stroke*[b])
Central vestibular		
Positional	• Persistent positional downbeating vertical nystagmus	• CPPV (*but can be seen in BPPV variants*[b])
	• Positional horizontal nystagmus beating away from the lowermost ear ["apogeotropic"]) that does not dampen over time	• Often due to apogeotropic hc-BPPV (*but is also seen in CPPV*)
Persistent	• Dominantly vertical (upbeating or downbeating) or dominantly/purely torsional spontaneous[a] nystagmus • Gaze-evoked (direction changing) horizontal nystagmus (persistent left-beating nystagmus on leftward gaze and persistent right-beating nystagmus on rightward gaze)[c]	• Stroke, Wernicke syndrome, multiple sclerosis (or other structural central lesions), and medication side effects (e.g., anticonvulsants) or acute intoxication (e.g., alcohol)

[a]"Spontaneous nystagmus" refers to nystagmus that is present on routine testing when the patient opens their eyes and looks straight ahead (also known as "primary gaze"). "Positional nystagmus" refers to nystagmus that is not present when the head is held still but is elicited on specific positional movements of the head (e.g., the Dix–Hallpike test).

[b]Some findings can be seen in both peripheral and central causes. Spontaneous horizontal unidirectional ("direction-fixed") nystagmus is typical of vestibular neuritis but can also be seen with strokes. Persistent positional nystagmus that is horizontal and beats away from the lowermost ear ("apogeotropic") can be seen both in CPPV and with a hc-BPPV variant (cupulolithiasis). The more persistent the nystagmus, the greater the concern for CPPV.

[c]Pathologic gaze-evoked nystagmus must be differentiated from physiologic end-gaze nystagmus (sometimes called "end-point nystagmus"). The physiologic (normal) form is (a) present only on extreme lateral gaze, (b) of low amplitude, and (c) nonsustained (i.e., lasts just a few beats). While an occasional "normal" individual will have more prominent physiologic end-gaze nystagmus, it must generally be assumed in a patient with acute dizziness that sustained gaze-evoked nystagmus is pathologic, rather than physiologic, until proven otherwise.

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