



Letter to the Editor

Appeal to Ignorance

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Appeal to ignorance is also known as argument from ignorance, in which ignorance represents “a lack of contrary evidence” and becomes “a fallacy in informal logic.” It asserts that a proposition is true because it has not yet been proven as false. This is the consequence of lack of knowledge and insufficient information to prove the proposition to be either true or false.

We can refer to Russell’s teapot analogy as an illustration. Russell, a philosopher, asks us to imagine a man claiming that there is a teapot orbiting the sun between Earth and Mars. The teapot is too small to be seen by the most powerful telescopes. Russell’s hypothetical statement says “since you can’t prove the teapot is not there, you must assume that it is there.” It is patently ridiculous to claim that we must believe in a teapot orbiting the sun simply because we have no means to prove that it is not there. It is common sense to reply to the person claiming the existence of the teapot to provide positive evidence for us to believe this claim.

Interestingly, it is not uncommon that people speak as though it were the business of others to disprove the received dogmatic assertions rather than to prove them. Therefore, to believe without evidence, it is so common that in science as well as medicine, such interferences are made to some degree of probability.

Vertigo disorders represent a conducive area to rely merely on the fact that the veracity of the unfalsifiable assertion is not disproved to reach to a definitive conclusion.

In spite of recent improvements in our knowledge and assessments in vertigo, VNG, VEMP, posturography, and radiology, many aspects in equilibrium disorders are still unclear, and their treatments are more often symptomatic or empirical than etiological.

Another reason for the dissemination of logic confusion in this field is that vertigo involves several specialties, such as ORL, vestibular testing, neurology, physiotherapy, psychology, and sciences, which offer equal opportunity for different opinions following their own patients, classification, and treatment. Each of them has their own societies with their own privilege to promote. Recently, I had the honor to be involved in an international working group for Meniere’s disease. For such a well-identified disease, I was amazed that its definition is still questionable. Suspicious, uncertain, probable, atypical, and pseudo-Meniere, what do they mean? So far, I do not know the diagnosis. We are reluctant to confess our ignorance and prefer to appeal to ignorance. Vestibular migraine and vestibular paroxysmia are the most typical examples.

Vestibular migraine

There are several subtitles including migraine-associated dizziness, migrainous vertigo, migraine-related vestibulopathy, basilar migraine, and lastly, migraine in Meniere’s disease. The scope of vestibular migraine is not restrictive.

The analogy between migrainous crisis and episode of vertigo is the main concept. Considering that 80% of the general population of women have some type of headache in a given year, this is consistent in women, but inconsistent in men.

In vestibular migraine, there is repetition of dizziness or vertigo in patients who have a history of migraine symptoms. However, in contrast to traditional migraines, headache is not a constant symptom. Therefore, is the leading and specific facial pain symptom, which individualizes and defines the disease, missing? Isn’t it funny to enlarge the disease to the last 20% of the population without headache, and how do we identify them?

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Assuming a patient with migraine is progressing to vestibular migraine if he experiences some equilibrium disorders, does this announcement modify the treatment of migraine? No.

Does the treatment of migraine resolve vertigo? No.

There is no specific medication for vestibular migraine because it is not an entity.

Feeling unsteady is a common symptom in migraine crisis. Till date, patients with migraine are so frequently encountered that they can be presented with associated vertigo but without evidence to be related to migraine.

Vestibular paroxysmia

Vestibular paroxysmia appears to be similar to pleonasm. All peripheral vestibulopathies are most commonly paroxysmal: Meniere and benign paroxysmal positional vertigo, to name the most well-known.

In vestibular paroxysmia symptoms, the paroxysms do not come in attack, evolve on a minor mode, and spontaneously resolve.

The leading symptoms are recurrent, spontaneous short attack of spinning or non-spinning vertigo that last a fraction of a second to a few minutes, and occur with or without ear symptoms (tinnitus and hypo or hyperacusis) with possibility of motion intolerance. Therefore, the clinical description that substitutes the disabling positional vertigo still encompasses a broad spectrum of typical or atypical vertigo.

The key point of vestibular paroxysmia is not its clinical presentation but on MRI assessment the image of a vascular loop (most commonly AICA) is in contact with the acousticofacial nerve bundle in the CPA or IAC. In contrast to most vertigo entities, in which symptoms are first noted, vestibular paroxysmia proceeds in reverse order from the image assessment toward “the heterogeneous collection of signs and symptoms, far from a reliably diagnosable disease entity” as vascular compression-induced vertigo.

Assuming the neurovascular conflict with the auditory nerve is the cause of vertigo disorders, what is the decision making?

It is not to treat the cause but to give an obsolete treatment, i.e., carbamazepine, which never provided any evidence of a positive result in the vascular compression of the auditory nerve as well as the facial nerve. Despite this, the response to medical treatment could be the criteria to confirm the diagnosis of cross-compression.

Vestibular paroxysmia was first described as disabling positional vertigo that was already considered as “a controversial illness with a controversial treatment.” The real entity providing a clear identification of what we are talking about, in place of a factitious terminology, is neurovascular compression of the auditory nerve. The frequency of a vascular loop in contact with the acousticofacial bundle and the numerous causes of vertigo disorders lead to a “thorny issue.” How do we prove that the artery is offending the nerve and the cause of the unilateral vestibulocochlear symptoms of the patient?

Vestibular migraine and vestibular paroxysmia are both “academic diseases.” The evidence of the clinical entity is weak. Even in the absence of symptom, the diagnosis can be evoked (absence of headache and absence of trouble with vascular loop). The evidence of pathophysiology and treatment is poorly understood and poorly understandable. It is a fallacy to draw a conclusion precisely based on ignorance “ignorance-based medicine” because this does not satisfactorily provide warrant for their proposals.

How do we disprove a disease that we are not able to prove? This is the main reason why they have been so easily accepted by the medical profession. Their repetition in different meetings and publications converts a simple hypothesis to a virtual disease.

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