A Causal Mediator for the Post-Concussion Syndrome

by

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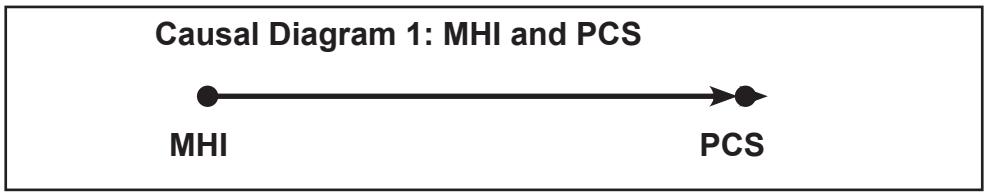
The post-concussion syndrome: The post-concussion syndrome diagnosis has proven controversial since it was first introduced about 80 years ago. It was described by Lewis (1942) as a "common, dubious, psychopathic condition," dismissed as an "accident neurosis" by Miller (1961) and legitimized as a form of mild traumatic brain injury by Binder (1986). Lishman (1988) points out the "striking disparity" in the perspectives of "those who argue for physiological or for psychological causation." These opposing and often irreconciliable perspectives continue to complicate clinical diagnostic work in mild head injury cases (McCrea, 2008).

There is little dispute about the symptoms that comprise the post-concussion syndrome: headache, dizziness, fatigue, light and sound sensitivity, visual blurring, impaired concentration and memory, and emotional distress. There is also general agreement in making a post-concussion syndrome diagnosis whenever this constellation of symptoms arises following a mild head injury.

The persistent symptoms that arise following mild head injuries have been designated as the post-concussion syndrome because of their presumed causal connection to concussive brain injury. Many physicians remain uncomfortable making a physical diagnosis of brain injury based on subjectively reported symptoms alone. The presence of physical damage to the brain cannot be proven in most cases, but the frequent occurrence of this syndrome following mild head injuries is undeniable. The post-concussion syndrome diagnosis has gradually become accepted by physicians, and it is now commonly used in head injury cases.

The name given to this diagnosis, the post-concussion syndrome, suggests that the syndrome is mediated by a concussive injury to the brain. Since the existence of a brain injury is questionable in most cases, Causal Diagram 1 illustrates only the accepted causal relationship between mild head injury (MHI) and the post-concussion syndrome (PCS) without a designated causal mediator.



Although there is no longer resistance to the clinical use of the post-concussion syndrome diagnosis, the presumed causal mediator is generally thought to involve some form of mild traumatic brain injury even though the existence of such an injury is not easily demonstrated in the majority of cases. An underlying brain injury is presumed to have occurred from signs of a concussion following the head injury. Only a transient alteration of consciousness is necessary to justify the post-concussion syndrome diagnosis, and this typically provides the only basis for the presumed mild traumatic brain injury (Kay, et. al., 1993).

The clinical data only reflects a positive correlation between mild head injuries and the post-concussion syndromes that follow. Although a correlation does not establish causality, the head injury is accepted as the cause of the resulting clinical syndrome. There is much less ground for agreement, however, regarding mild traumatic brain injury as the proposed causal mediator between the injury and the resulting syndrome.

McCrea's book, *Mild Traumatic Brain Injury and Post-Concussion Syndrome* (2008), summarizes the clinical research available at that time and attempts a synthesis based on statistical data alone. It represents the state of the art of clinical analysis of statistical data prior to what Judea Pearl (2018) refers to as the "causal revolution." It is instructive to

carefully reconsider McCrea's review of this data before turning to the causal diagrams that are the basis for this article.

The most immediate and predictable consequence of a pre-causal data analysis involves the emergence of a variety of plausible candidates for the role of causal mediator. The existence of a mild traumatic brain injury is difficult and often impossible to prove, and other possible mediators have been proposed. Since depression is common in patients who struggle with persistent symptoms, it has frequently been proposed as the cause of the post-concussion syndrome (Iverson, 2006). The correlation data alone do not enable us to know whether depression is the cause of the syndrome or one of its component symptoms. Iverson and his colleagues have proposed that depression accounts for the otherwise inexplicable persistence of the syndrome in very mild head injury cases.

When viewed from the perspective of sequellae of a brain injury, post-concussion symptoms have a generalized, nonspecific quality that provides a basis for questions about their legitimacy. This is especially true when minimal injuries result in persistently disabling symptoms. Leas-Haley and colleagues (2001) found that head injury disability claims shared similar sets of symptoms with other types of disability and that the symptoms reported lacked diagnostic specificity.

Many post-concussion syndrome patients develop significant anxiety that impacts their other symptoms and complicates their disability. Anxiety lacks specificity by definition, and it can be easily seen as the cause rather than a secondary development of a disability syndrome. Although dizziness can provoke anxiety, anxious individuals often generate dizziness symptoms. Ruff, Crouch and Troester (1994) suggest that a minority of fifteen percent of mild head injury patients remain "miserable" with persistent symptoms, but it remains an open question whether their misery is an effect or the cause of their syndrome.

The legitimacy of the post-concussion syndrome diagnosis is frequently challenged within the setting of disability claims and litigation. Charges of exaggeration, poor effort or malingering arise when substantial disability is claimed within the context of little or no evidence of concussion (Hayes, Hilsabeck and Gouvier, 1999). Neuropsychologists often rely on poorly validated malingering tests for questioning the validity of both the symptoms and the cognitive test results in post-concussion syndrome cases.

Attempts are often made to discredit the claims of mild head injury patients whose persistent symptoms appear to defy explanation in terms of the degree of concussive injury. When there is little or no alteration of consciousness, the brain injury involved must be so minor that it could not possibly cause the degree of disability that has been claimed (Larrabee, 1999).

The symptoms associated with the post-concussion syndrome constitute a disparate array of complaints that seem unrelated to any specific brain dysfunction. Psychological tests that identify personality traits like the MMPI interpret response patterns that report many seemingly unrelated types of symptoms as indicative of a Somatoform Disorder in which emotional distress is expressed in terms of physical symptoms. Glenn Larrabee (1997) has proposed that the persistent forms of the post-concussion syndrome represent Somatoform Disorders.

The clinical data from multiple research studies appear to indicate that the post-concussion syndrome is impossible to explain in terms of the claimed brain injury (McCrea, 2008). McCrea attempts to reconcile this conclusion with a synthesis of the viewpoint of those who understand the post-concussion syndrome as a legitimate response to mild head injury and those who take a more skeptical position. He concludes that "the neuropathophysiological

of MTBI start this process in motion, the development and maintenance of persistent PCS are more directly the result of psychological, psychosocial, and other non-MTBI-specific factors." This is an understandable conclusion from a review of the available data prior to the causal revolution.

The causal revolution: Judea Pearl (2018) defines the causal revolution as "a scientific shakeup that embraces rather than denies our innate cognitive gift of understanding cause and effect." He suggests that an analysis that confines itself to the data alone is susceptable to errors that are otherwise easily avoided, and he provides numerous examples. He demonstrates the inevitable confusion that follows when the confounding nature of proposed mediators between cause and effect is not recognized. He shows the power of counterfactual possibilities in developing our understanding of causal relationships. Most importantly, he establishes the critical role of causal diagrams in guiding our thinking about complex relationships.

Consider, for example, the confounded variable of depression in the causal sequence from mild head injury to post-concussion syndrome. The data that show a higher incidence of depression in those who develop persistent symptoms following mild head injury cannot be interpreted as establishing the role of depression as the mediator for the post-concussion syndrome. The depression variable is confounded by the fact that any set of persistently disabling symptoms leads to a high incidence of depression. The data alone do not allow us to determine the direction of the causal sequence.

A similar reasoning can be applied to anxiety, effort and even disability claims. Patients who struggle with persistent, disabling symptoms become anxious, they lose confidence in themselves and they often file disability claims. Each of these factors that have been proposed as mediators of the post-concussion syndrome is confounded so that the causal direction involved cannot be determined.

A striking instance of confounding arises in relationship to the psychological diagnosis of Somatoform Disorder. An array of disparate symptoms that are not obviously connected is the basis for the somatoform diagnosis. The post-concussion syndrome includes a variety of symptoms like dizziness, visual blurring and fatigue that appear to involve unrelated brain systems. Having the post-concussion syndrome, however, is the cause of the diagnosis rather than a diagnostic condition that causes the syndrome.

The all-too-common instances in which a persistent post-concussion syndrome arises following a minimal concussive injury or no concussion are counterfactual (Pearl, 2018) in the sense that they should not occur if a concussive injury to the brain is the causal mediator. Causal diagram 2 shows the causal sequence from mild head injury (MHI) to post-concussion syndrome (PCS) with mild traumatic brain injury (MTBI) as the causal mediator.

Causal Diagram 2: MHI to MTBI to PCS

In accodance with causal diagram 2, it should not be possible for a mild head injury to cause a post-concussion syndrome without a concussion that establishes the basis for a mild traumatic brain injury. The absence of the presumed causal mediator, mild traumatic brain injury as evidenced by the lack of signs of a concussive injury, has led many clincians to

question the legitimacy of their patients' symptoms.

These counterfactual examples of the post-concussion syndrome without concussion challenge the role of mild traumatic brain injury as the primary mediator of the syndrome. They lend support to the skeptics and detractors of the post-concussion syndrome as the product of other factors. McCrea's synthesis, however, retains mild traumatic brain injury as the initial cause even if it relies on psychological and psychosocial factors to explain the persistence of this syndrome over time.

When individual counterfactual cases are considered more closely, however, the plausibility of McCrea's synthese fades. There is no relationship between the degree of concussion and the persistence or severity of the post-concussion syndrome that follows. Although the concussive injury may not be irrelevant, it cannot be the primary causal mediator for the syndrome. It is time to question the specific components of what has been too often viewed solely as a syndrome and only through the lens of a brain injury.

Consider two of the more common features of the post-concussion syndrome, dizziness and visual blurring. The dizziness is often characterized as "episodic" as if it comes and goes in a way that brain dysfunction symptoms do not and might well be triggered by anxiety. A slightly closer look at this symptom reveals that the "episodes" are not random but are specifically provoked by head movements. Similarly, the visual blurring that patients report, usually in the context of normal visual acuity, also seems to come and go as if it were a stress response. It, however, is also a specific response to any tasks that require sustained visual focusing.

These symptoms are neither nonspecific as some critics of the post-concussion syndrome have claimed nor episodically. They are highly specific, predictable responses to certains activities of daily living. Although they are not typical brain injury symptoms, they cannot be explained away in terms of psychosocial issues. They constitute a well-established syndrome lacking an adequate causal mediator to explain them.

The peripheral vestibular trauma syndrome: It is possible to acknowledge the causal relationship between mild head injury and the post-concussion syndrome while challenging the causal role played by the presumed mediator, mild traumatic brain injury. The brain is not the only organ in the head that is subjected to trauma in mild head injury. The peripheral organs of the vestibular system, the otoliths and the semicircular canals located in the inner ear, are often functionally impaired by head trauma. The vestibular trauma syndrome that results solely from injury to structures that reside outside of the skull is virtually identical to what we presently understand as the post-concussion syndrome.

The peripheral vestibular trauma syndrome consists of primary symptoms that include movement-sensitive dizziness, imbalance, nausea, visual blurring and problems with sustained focusing as well as secondary symptoms of headaches, fatigue and anxiety. Not only does vestibular system trauma result in all the elements of the post-concussion syndrome, it provides a basis for explaining these symptoms entirely in terms of a disorder of the peripheral vestibular apparatus in the inner ear. This condition, known somewhat misleadingly as benign paroxysmal positional vertigo or BPPV, arises when calcium carbonate crystals from the otoliths, fragmented by trauma, float freely within the fluid filled cavities and drift into one or more of the semicircular canals. This crystalline debris interferes with the smooth movement of fluid in the affected canals and causes them to send aberrant movement signals to the brain.

The letter "V" in BPPV stands for vertigo, but this symptom is usually absent. The crystalline debris often provokes vertigo acutely, but in mild head injuries, the vertigo gradually resolves into movement-sensitive dizziness or lightheadedness. The preoccupation in the early

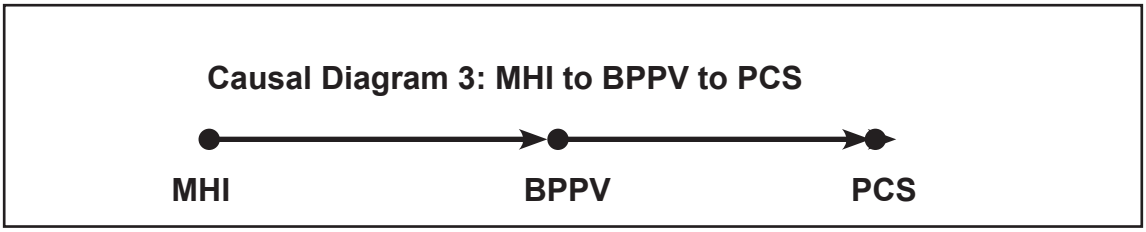
neurological literature with “true vertigo” appears to have been unwarranted (Jacob and Furman, 2001). The initial letter “P” in BPPV suggests that the symptoms are “paroxysmal” in that they arise spontaneously and unexpectedly. Although these symptoms often appear to arise “out of the blue,” they are entirely predictable responses to movement. Smooth, coordinated movement depends upon accurate vestibular signals. Patients fail to realize the connection between their own movements and their symptoms. They spontaneously adjust to the constraints imposed by this condition and learn to move more carefully without realizing how much they have accommodated their new limitations.

The second letter “P” in BPPV stands for positional, indicating that the vertigo is position sensitive. Although position-sensitive vertigo is possible, most patients with BPPV are disabled by movement sensitivity secondary to their aberrant canal signals. These aberrant signals not only interfere with movement, balance and equilibrium, but they also undermine the smooth movements of the eyes essential for living in a visual world. Because of this, the disability that results is anything but benign, as the letter “B” suggests.

The misleading label, benign paroxysmal postional vertigo, creates far more than a semantic difference because it misrepresents the vestibular trauma syndrome and interferes with our ability to evaluate, diagnose and treat a commonly encountered syndrome following mild head injury. Neurologists continue to dismiss patients who don’t present what they believe is “true” vertigo, and ophthalmologists falsely reassure patients who have dynamic visual focusing problems when they find normal static visual acuity using a Snellen chart.

Crystalline debris in the semicircular canals interferes with the vestibulo-balance reflex and causes dizziness, nausea and imbalance as primary symptoms and an understandably increased level of anxiety as a secondary symptom. It also interferes with the vestibulo-ocular reflex resulting in problems with dynamic visual focusing and undermining the ability to sustain a focus of concentration, especially on visual scanning tasks like reading. This peripheral vestibular syndrome is not necessarily associated with brain injury even though it is a common sequela of mild head injury.

Causal Diagram 3 illustrates the causaal sequence from mild head injury (MHI) to post-concussion syndrome (PCS) with benign paroxysmal positional vertigo (BPPV) as the primary causal mediator.



There is no difference between Causal Diagram 2 and Causal Diagram 3 other than the change from mild traumatic brain injury to benign paroxysmal positional vertigo as the causal mediator. This change, however, makes a world of difference by providing a mediator that doesn’t require a brain injury and by offering specific explanations for what otherwise appear to be nonspecific symptoms.

The presumed presence of a mild traumatic brain injury based on only the most minimal evidence of concussion seems unjustified and feels unacceptable to many clinicians. Moreover, the connection between the component symptoms of the post-concussion syndrome and brain injury has never been convincingly demonstrated. Benign paroxysmal positional vertigo, however, is an independently diagnosable condition syndrome even

though it cannot be easily assessed in an office examination. It is difficult to reliably provoke the torsional nystagmus that is the basis for the diagnosis without blocking fixation or to observe the subtle eye movements provoked while conducting a Hallpike-Dix maneuver. It is necessary to block fixation by using infrared goggles with cameras that create a video record of the eye movements that can be closely reviewed for signs of nystagmus.

Benign paroxysmal positional vertigo is specific to those semicircular canals that have been impacted by crystalline debris from the traumatic head injury. It is readily diagnosed by the characteristic nystagmus provoked by certain movements of the head and upper body. It is also treatable through repositioning maneuvers that thrust the crystalline particles out of the affected canals. Although a videonystagmogram can often satisfactorily complete the evaluation, a skilled physical therapist who can carry out the hands-on treatment remains essential.

Vestibular rehabilitation specialists who understand how to evaluate each of the six semicircular canals independently and apply the correct repositioning maneuver to treat the affected canals are in short supply. Too often, potentially treatable vestibular syndromes are never considered, and when they are considered not fully evaluated, and when they are evaluated inadequately treated. The most commonly available treatments involve retraining and habituation exercises that are conducted on patients who still have affected canals generating aberrant movement signals. Habituation exercises cannot succeed unless the semicircular canals have first been cleared of crystalline debris so that the brain is again receiving accurate movement signals.

It is useful to consider the vestibular trauma syndrome caused by crystalline debris in one or more of the semicircular canals in greater detail. Causal diagrams make it possible to represent the symptom specificity associated with these details The symptoms associated with balance can be differentiated from those that are related to dynamic visual focusing, and it becomes possible to define two symptom subgroups within the constellation of the post-concussion syndrome.

Causal Diagram 4 can be divided into two causal sequences representing the two distinct reflex pathways from the peripheral vestibular system to the brainstem. Both reflex pathways are impacted by the aberrant signals that cause benign paroxysmal positional vertigo (BPPV). They involve aspects of the post-concussion syndrome associated with balance through the vestibulo-balance reflex (VBR), Causal Diagram 4a, and with visual focusing through the vestibulo-ocular reflex (VOR), Causal Diagram 4b.

Causal Diagram 4a: MHI to BPPV to the VBR component of PCS

Causal Diagram 4b: MHI to BPPV to the VOR component of PCS

The balance reflex symptoms include vertigo, dizziness, lightheadedness, nausea, imbalance, nausea and secondary anxiety. The ocular reflex symptoms include visual

blurring, difficulty with sustained visual focusing, problems reading and related problems with concentration and short term memory, mental tracking, multitasking as well as secondarily induced headaches.

The post-concussion syndrome, differentiated into its two primary symptom groups, no longer seems nonspecific or easily explained in terms of psychosocial factors. The balance syndrome associated with the vestibulo-balance reflex and the visual syndrome associated with the vestibulo-ocular reflex make sense in accordance with the two causal diagrams. When the smooth movement of fluid in one or more of the semicircular canals is obstructed by crystalline debris, the affected canals send aberrant signals along both reflex pathways.

The two distinct symptom subgroups of the post-concussion syndrome are movement sensitive because they are vestibular in origin. They are the product of aberrant movement signals transmitted via the eighth cranial nerve to brainstem pathways. The symptoms make sense in terms of aberrant movement signals that result in dizziness and visual focusing problems respectively. Both sets of symptoms are provoked by a common etiology, crystalline debris in the fluid-filled semicircular canals generating aberrant movement signals.

The shift in causal mediator from brain concussion to vestibular system trauma changes our understanding of the post-concussion syndrome. The specific etiology of benign paroxismal position vertigo provides a causal substrate that can be evaluated and treated. Physical therapists with specialty training in vestibular rehabilitation have led the development of both evaluation techniques and treatment methods for the canal problems that are the basis for the vestibular trauma syndrome.

Physicians cannot reliably evaluate canal problems in an office evaluation. This may be easily accomplished in the most obvious cases, but it is extraordinarily difficult in more subtle instances. The skilled vestibular rehabilitation specialist who uses infrared goggles equipped with eye movement cameras to record and later observe subtle manifestations of nystagmus can localize the cause of the nystagmus to the specific canal involved.

Too often an adequate evaluation and treatment sequence is not achieved.

1. The vestibular origin of the persistent symptoms is not recognized because the symptoms are incorporated into a post-concussion syndrome diagnosis.

2. The vestibular origin of the persistent symptoms is recognized, but the physician’s office evaluation fails to demonstrate the pathology.

3. The vestibular origin of the persistent symptoms is recognized, but the patient is referred for retraining and habituation exercises without either an adequate evaluation or an attempt at clearing the canals. The exercises, carried out in the presence of aberrant signals from the semicircular canals, fail to alleviate the symptoms.

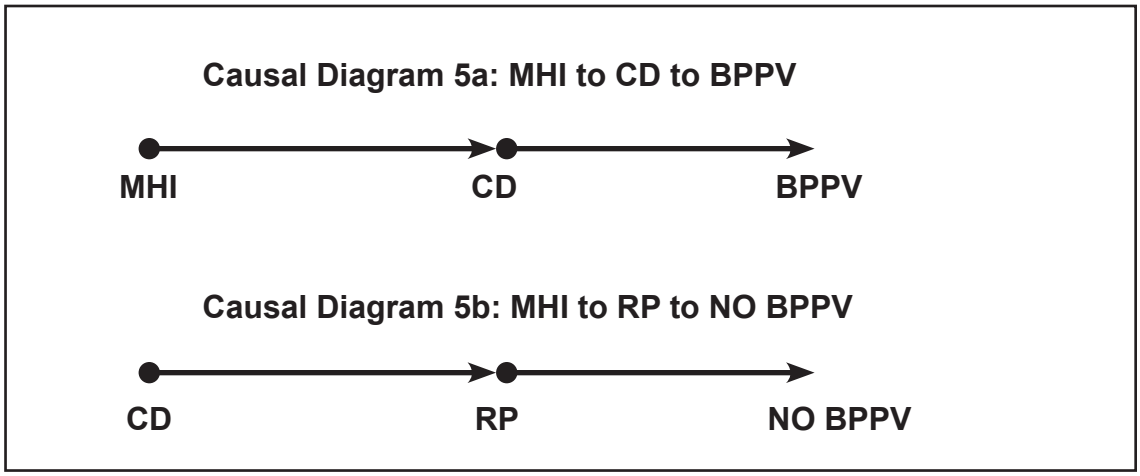
4. The vestibular origin of the persistent symptoms is recognized and the canals are evaluated with a video-nystagmogram. Positive findings are identified but no one is available to carry out the specific repositioning maneuvers necessary for clearing each of the canals involved. Retraining and habituation exercises, carried out in the presence of aberrant signals from the semicircular canals, fail to alleviate the symptoms.

A skilled physical therapist, trained to evaluate each of the six semicircular canals by blocking visual fixation while noting the eye deviations under varying conditions of head and upper body movement can then carry out repositioning maneuvers specific to each canal involved. This process of clearing the canals provides a new source of clinical information that is essential to validating the vestibular system as the primary causal mediator for the post-concussion syndrome.

When an abnormal eye finding like torsional nystagmus is observed in response to a particular repositioning maneuver, a skilled therapist can identify the specific canal or canals that have been affected. Each canal involved contributes one aberrant component to the overall vestibular system signal transmitted to the brainstem. These aberrant signals interfere with the normal functioning of both the vestibulo-balance reflex and the vestibulo-ocular reflex. Each of the six semicircular canals can become independently affected by crystalline debris and provoke aberrant eye movements on evaluation. Although the label benign paroxysmal positional vertigo appears to imply the presence of vertigo, the diagnosis depends solely on the eye movement findings.

The aberrant movement signals that cause symptoms related to eye and body movement can be corrected through repositioning maneuvers. The therapist can then reevaluate the treated canal or canals and check once again for eye movement findings. Once the crystalline debris has been successfully cleared through repositioning, the therapist can reevaluate the canal to demonstrate that the eye movement findings have resolved. The target symptom for the evaluation is not the post-concussion syndrome but its presumed mediator, the crystalline debris causing the nystagmus that establishes the diagnosis of benign paroxysmal positional vertigo.

Causal Diagrams 5a andd 5b represent the causal sequences from mild head injury (MHI) to crystalline debris (CD) to benign paroxysmal positional vertigo (BPPV) both before and after successful repositioning (RP) procedures.

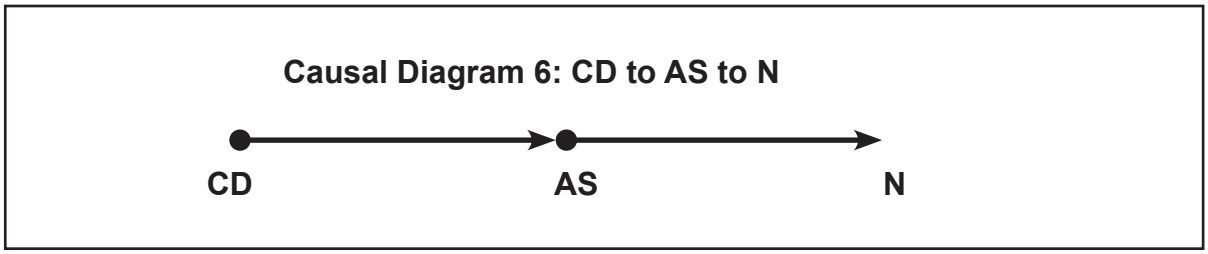


Causal Diagram 5a represents mild head injury as the cause of the crystalline debris in the right horizontal semicircular canal. The crystals in that specific canal serve as the causal mediator between the injury and the observed nystagmus that is the basis for the diagnosis of benign paroxysmal positional vertigo. Causal Diagram 5b represents the crystalline debris as the cause of the need for treatment with a repositioning maneuver to clear the debris resulting in an absence of eye findings and resolution of the condition. On reevaluation, the evaluation maneuver remains the same, but the crystals have been removed from the canal and the movements no longer generate nystagmus.

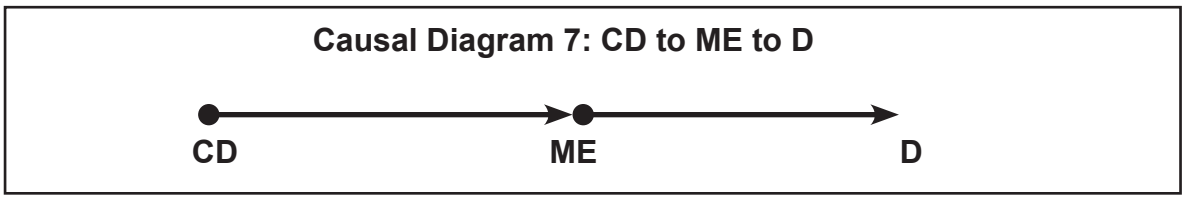
Causal diagrams 5a and 5b do not represent the mild head injury as the causal event because it is not the proximal cause of the post-concussion syndrome. The head can be hit in a variety of ways, at different angles, in diverse impact locations and with unspecified force. The proximal cause of the syndrome involves the crystalline debris and the aberrant vestibular signals that provoke benign paroxysmal positional vertigo. The resulting symptoms, including dizziness and visual blurring, are provoked by these aberrant signals.

The unspecified head injury need only to have had sufficient force to cause crystalline fragments to break off from the otoliths and migrate through the fluid-filled cavities into one or more of the semicircular canals.

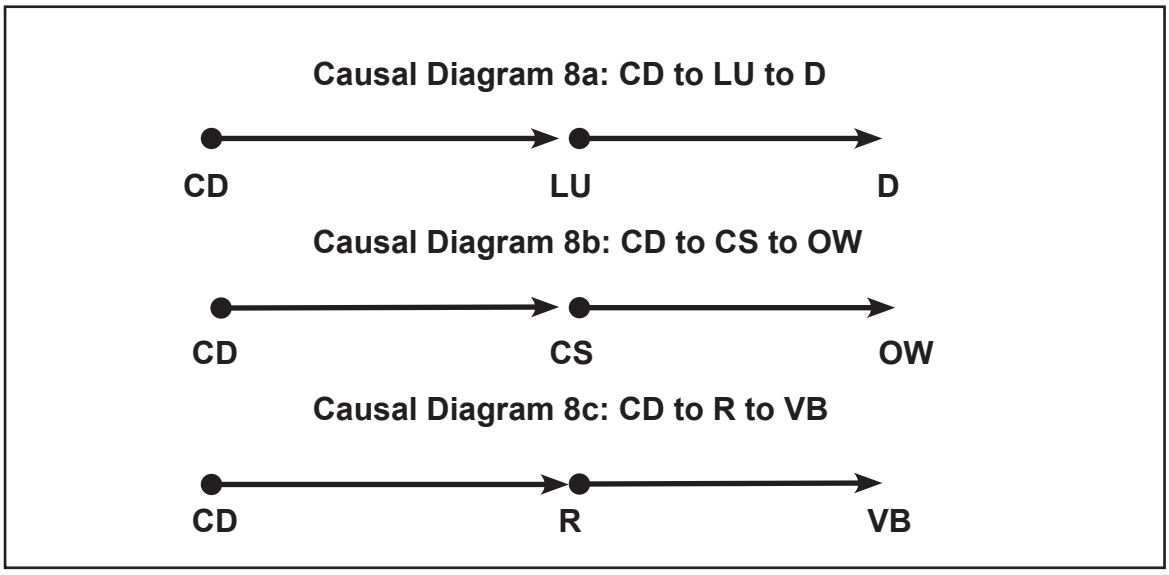
Causal Diagram 6 represents the causal sequence that takes place beginning with the traumatically induced crystalline debris (CD) that drifts into one or more of the semicircular canals and disrupts the movement of fluid in the affected canals resulting in aberrant signals (AS) that cause nystagmus (N) in the eye movement responses.



Since both the balance and visual symptoms are movement sensitive, it is possible to create a causal diagram that shows the movement event as the mediator between the cause, crystalline debris and the symptom effect. Causal Diagram Number 7 represents the crystalline debris (CD) as the cause, a movement event (ME) as the mediator and the resulting symptoms of dizziness (D) as the effect.



Causal Diagrams 8a, 8b and 8c differentiate the above causal sequence into three kinds of movements known to provoke symptoms, looking up (LU), walking through a crowded store (CS) and reading (R). Each of these movements results in a specific kind of symptom, dizziness (D), feeling overwhelmed (OW) and visual blurring (VB).

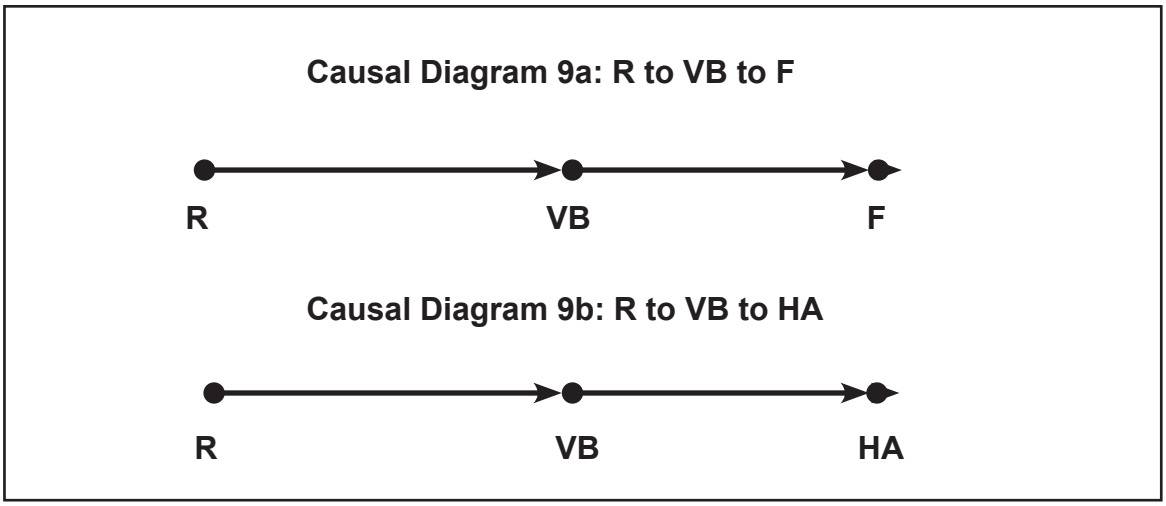


In the three causal diagrams given above, Causal Diagrams 8a, 8b and 8c, the initial causal agent is represented as crystalline debris in one or more of the semicircular canals. Aberrant signals, however, are not generated until they are provoked by specific movements. In each diagram, a particular movement event is specified as the mediator provoking the particular symptoms that arise. In Causal Diagram 8a, the movement event mediator involves "looking up," and the symptom effect is lightheadedness or dizziness. In Causal Diagram 8b, the movement event involves walking through a crowded store, and the symptom effect is feeling overwhelmed by too much stimulation. In Causal Diagram 8c, the movement event involves reading and the primary symptom effect is visual blurring.

These specific causal diagrams dissect the otherwise impenetrable post-concussion syndrome into its individual symptom components. In each case, the symptom effect can be understood as a direct product of a specific movement event. Symptoms that were previously characterized as "paroxysmal" and "nonspecific" are revealed as predictably provoked consequences of certain types of movement events.

The symptom complex of the post-concussion syndrome can be dissected further in relationship to the secondary symptoms that develop from attempts to sustain visual focusing in the presence of nystagmus and visual blurring. Reading is the most common example of a sustained visual focusing and scanning task, and it provokes two secondary symptoms of the post-concussion syndrome, headaches and fatigue.

Causal Diagrams 9a and 9b represent reading (R) as the causal event with the visual blurring (VB) caused by nystagmus as the mediator and the secondary symptoms of fatigue (F) and headache (HA) as the secondary symptoms provoked.



The above causal diagrams make it possible to differentiate the multiple causal steps that are involved in the connection between mild head injury and the post-concussion syndrome. The primary mediator involves peripheral vestibular system trauma and the resulting crystalline debris that drifts into the semicircular canals. The debris in the canals results in the aberrant vestibular signals that are the proximal cause of both benign paroxysmal positional vertigo and the symptom components of the post-concussion syndrome.

The relationship between post-concussion syndrome to its essential cause, vestibular system trauma and its array of primary and secondary symptoms sheds new light on a syndrome that has defied definition. It provides treating physicians a much needed specificity that has been previously lacking. In medical practice, increased specificity leads to improved evaluation and treatment, and this has been my experience with post-concussion syndrome

patients. I have had the good fortune to work with an experienced physical therapist skilled in evaluating the six semicircular canals and clearing any affected of crystalline debris following the methods developed by Susan Herdman (2007).

Patients experience the movement events that provoke their symptoms as the source of their problem and quickly constrain their movements and activities to accommodate these limitations. They may experience improvement over time that is solely due to the adjustments they have made in how they move. They learn to move more carefully, avoid crowds and take frequent breaks when reading. They control their symptoms by avoiding many former activities They often adjust their movements without realizing the degree to which they have constrained their lives.

It is difficult for patients to identify the vestibular origin of their symptoms. The aberrant signals provoked by crystalline debris in the semicircular canals that cause dizziness, imbalance and visual blurring is not experienced perceptually. The movement-event mediators are experienced as the cause of the symptoms they provoke while their vestibular origin remains hidden from awareness. The anxiety that is caused by disequilibrium and the headaches and fatigue from the strain of staying focused visually in the presence of ongoing nystagmus are often regarded as the source rather than the product of the disability.

Counterfactuals: Judea Pearl, in *The Book of Why?* (Pearl, 2015), describes counterfactual causal sequences as the "top rung" on what he calls the ladder of causality, above the data-based level of scientific analysis. Counterfactuals play a critical role in shaping our understanding of the causal relationships involved in the events we are considering. They pose questions about "What if...?" a possible event were to happen, and they ask us to consider "What would we expect to be the result." Counterfactual examples force us to think about the boundary conditions that surround the events we are considering.

The post-concussion syndrome response to mild head injury lends itself to the use of counterfactual questions like, "Could the post-concussion arise following a mild head injury when there only a minor concussion?" "Does the likelihood of a post-concussion syndrome decrease as the degree of concussive injury lessens?" "If there was nothing to indicate that a patient sustained a concussive injury, could he or she still develop a post-concussion syndrome?" All clinicians who evaluate patients following mild head injuries observe cases in which these supposedly counterfactural situations are realized in actual practice.

In Causal Diagram 2, the mediator between the mild head injury cause and the post-concussion syndrome effect is represented as mild traumatic brain injury. The documentation of the presumed brain injury is established through the degree of loss or alteration in consciousness. In a counterfactual instance, the post-concussion syndrome would occur in the context of a mild head injury but without a loss or even an obvious alteration in consciousness. When the post-concussion syndrome occurs without its presumed mediator, clinicians often blame the patient by suggesting a psychological weakness, psychosomatic tendencies, inadequate effort or even deliberate malingering.

My profession of neuropsychology has gone through decades of confusion and debate fueled largely by post-concussion syndrome cases. During the first twenty years of my work as a neuropsychologist, there was general agreement that the otherwise inexplicable symptoms and cognitive impairment demonstrated by these patients was best explained in terms of psychosocial factors. The MMPI often documents a high number of seemingly unrelated symptoms that have been interpreted as due to emotional factors (Cripe, 1999). The well-known profile with clinical scale elevations on hysteria and hypochondriasis commonly found in these patients was interpreted as diagnostic of a psychosomatic

condition in which patients “converted” emotional distress into physical symptoms.

During the past twenty years of my career, this way of explaining the post-concussion syndrome has been gradually replaced by accusations of exaggerated symptoms and malingered test results. These patients are no longer seen as having a psychological condition or emotional disorder but to be deliberately distorting their symptoms and test performances. So-called “effort” tests have been used to identify those who have too many symptoms or who perform too poorly on “easy” cognitive tests. Neuropsychologists have become locked in a polarizing debate over how to interpret impaired cognitive test results that appear to document disability associated with a post-concussion syndrome in the absence of a significant concussive injury.

While physicians generally accept the post-concussion syndrome diagnosis based on symptoms alone, even in the absence of well-documented changes in consciousness, neuropsychologists are often inclined to doubt the legitimacy of patients who perform poorly on effort testing. Neither group is inclined to confront the implications of the frequently encountered counterfactual examples of mild head injury patients with subjective symptoms that clearly constitute a post-conscussion syndrome and yet have little or no evidence of a concussive injury. These counterfactual cases arise repeatedly in clinical practice, but they only appear to be counter to the facts in a world described by Causal Diagram 2.

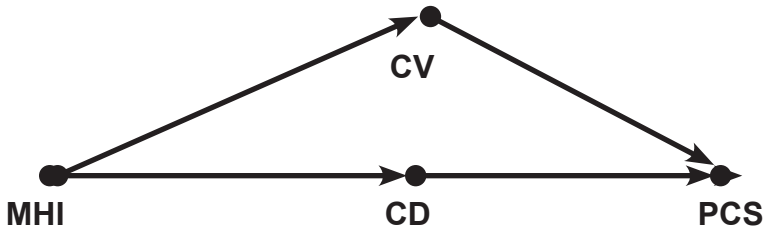
A misunderstanding of the primary causal mediator for the post-concussion syndrome continues to dominate the diagnostic thinking of most physicians and neuropsychologists. They remain unable to consider, evaluate or adequately treat mild head injuries. The incidence of peripheral vestibular system involvement following mild head injury is very high in patients who demonstrate persistent post-concussion syndromes. Research studies cannot accurately estimate this percentage because of the pervasive failure to recognize and evaluate vestibular syndromes, but Shumway-Cook (2007) estimates between 30 to 65 percent. Based on personal experience, however, it is reasonable to assume that patients with persistent post-concussion syndromes have peripheral vestibular involvement until it has been thoroughly evaluated and ruled out.

Brainstem trauma: The existence of crystalling debris in the semicircular canals as a causal mediator for the post-concussion syndrome does not preclude the possibility of mild traumatic brain injury. The alterations in or loses of consciousness following a mild head injury indicate at least minimal injury to the brain. Although alterations in consciousness cannot provide a general index of brain injury, they do specifically indicate brainstem trauma (Ropper and Gorson, 2007). The rotational movement of brain structures inside the skull following a head injury exerts specific trauma on brainstem systems essential for consciousness. The mild traumatic brain injury sustained in a concussion is thus brainstem trauma.

A recent clinical review highlights the incidence of vestibular system dysfunction in mild traumatic injury (Skora et. al., 2018), and Gottshall and Whitney (2019) describe the treatment strategies available. A thorough vestibular evaluation often reveals both peripheral and central vestibular system involvement. Central vestibular involvement can result from trauma to the brainstem nuclei that receive inputs from the semicircular canals via the eighth cranial nerve. Since both concussion and central vestibular dysfunction involve brainstem systems, a second causal pathway exists between mild head injury and the post-concussion syndrome mediated by the vestibular system. The symptoms of the post-concussion syndrome are still explained in terms of vestibular system dysfunction.

Causal Diagram 10 represents both causal sequences between a mild head injury (MHI) and the resulting post-concussion syndrome (PCS). The peripheral involvement is mediated by crystalline debris (CD) in one or more of the semicircular canals, and the central involvement is mediated by brainstem nuclei in the central vestibular system (CV).

Causal Diagram 10: MHI to both CD and CV to PCS



In addition to the direct effect of mild head injury on the peripheral vestibular apparatus in the inner ear through the trauma-induced crystalline debris in one or more of the semicircular canals, a secondary pathway is possible mediated by the brainstem trauma. The brain injury in concussion is brainstem trauma, and this is a major source of central vestibular dysfunction. It is important to note that both pathways explain the post-concussion syndrome in terms of vestibular system dysfunction. It is not unusual in a thorough vestibular evaluation following mild head injury for patients to show evidence of both central and peripheral vestibular dysfunction.

A thorough evaluation of all six semicircular canals is essential whenever there are symptoms of dizziness, imbalance or visual blurring following mild head injury or in the presence of persistent post-concussion symptoms of any kind. Any affected canals must be treated and reevaluated to demonstrate clearing of the crystals and alleviation of the BPPV. Only after the canals have been cleared can vestibular rehabilitation begin. This involves balance habituation exercises for the vestibulo-balance reflex and eye-movement retraining for the vestibulo-ocular reflex.

A case example: The causal mediator that is identified for the post-concussion syndrome has critical implications for clinical evaluation and treatment. Consider the case of Billy G., a high school student I evaluated a few years ago seven months after a motor vehicle accident in which he had lost control of his car and swerved into an embankment. He had no recall of the impact, and he was given an initial Glasgow Coma Scale rating of 14 for confusion. At the hospital emergency department, he was found to have a scalp contusion to the back of his head on physical examination, but an immediate CT brain scan and a later MRI brain scan were negative for signs of intracranial injury. Over the weeks that followed, he reported headaches, blurred vision, dizziness and difficulty with memory, and he was diagnosed with a post-concussion syndrome.

Billy was two months into his senior year at the time of the accident, and he had done well academically with all A's and B's. Although he was more than seven months from his accident when I first saw him, he had made minimal improvement over time, and his plans for college were in jepardy. I administered a battery of cognitive tests that yielded generally lower than expected scores consistent with the persistent problems he described in sustaining his concentration. I completed a brief vestibular survey survey with him that

indicated movement-sensitive dizziness, imbalance, nausea, feeling overwhelmed by crowds and having difficulty sustaining his focus while reading. I referred him to a highly skilled physical therapist with specialty training in vestibular rehabilitation for a vestibular evaluation. She found BPPV in both posterior semicircular canals as well evidence of central vestibular involvement.

Within five weeks of my initial assessment, she had completed three treatment sessions that resulted in both canals free of BPPV and the movement sensitive dizziness almost completely resolved. When he returned for a follow-up evaluation with me three month after our initial session, he felt improved in both his physical and cognitive functioning. His test scores were uniformly improved as well even though he had not yet returned to his likely preaccident baseline. He felt well enough to enroll in college at a local university and able to handle the academic demands.

This case illustrates many of the points made in the causal diagrams above.

1. Billy's post-concussion syndrome was not improving over time on its own.
2. Billy's future plans were at risk.
3. The vestibular aspects of his syndrome had not been recognized.
4. A brief survey was sufficient to reveal his vestibular syndrome.
5. A vestibular revealed treatable BPPV in two semicircular canals.
6. The canals were easily treated by a rehabilitation specialist.
7. Billy's cognitive and physical symptoms improved with treatment.
8. He was able to pursue his college plans.

Summary: The post-concussion syndrome, always contraversial, has been mistakenly attributed to concussive injury to the brain as determined by alterations in consciousness. It is, however, primarily a vestibular syndrome whether is solely caused by BPPV associated with the peripheral vestibular apparatus in the inner ear or also includes central vestibular involvement. When causal diagrams are used to represent various aspects of the post-concussion syndrome in terms of vestibular system mediators, the mystery of the post-concussion syndrome is resolved and its associated diagnostic contraversy vanishes. Moreover, a clear line of evaluation and treatment strategies emerges that not only makes sense theoretically but leads to effective treatment and recovery.

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