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The vestibulocochlear bases for wartime posttraumatic stress disorder manifestations

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ABSTRACT

Preliminary findings based on earlier retrospective studies of 229 wartime head injuries managed by the Walter Reed Army Medical Center (WRAMC)/National Naval Medical Center (NNMC) Neurosurgery Service during the period 2003–08 detected a threefold rise in Posttraumatic Stress Disorder (PTSD) manifestations (10.45%) among Traumatic Brain Injuries (TBI) having concomitant vestibulocochlear injuries compared to 3% for the TBI group without vestibulo-cochlear damage (VCD), prompting the authors to undertake a more focused study of the vestibulo-auditory pathway in explaining the development of posttraumatic stress disorder manifestations among the mostly Blast-exposed head-injured. The subsequent historical review of PTSD pathophysiology studies, the evidence for an expanded vestibular system and of a dominant vestibular system, the vascular vulnerability of the vestibular nerves in stress states as well as the period of cortical imprinting has led to the formation of a coherent hypotheses utilizing the vestibulocochlear pathway in understanding the development of PTSD manifestations. Neuroimaging and neurophysiologic tests to further validate the vestibulocochlear concept on the development of PTSD manifestations are proposed.

Introduction/Background

The story of WWI trench warfare as the historic cauldron for what we now know as Posttraumatic Stress Disorder (PTSD), is also the untold story of the vestibular system. In the first published study on "shell shock", author Charles Myers, an English psychologist and Army volunteer, reported 3 cases with transient amblyopia (contracted vision), anosmia and amnesia whose presentation then eluded explanation for a unified pathophysiology [1]. In the light of the current knowledge establishing a functional connection between the semicircular canal- vestibular innervations and hippocampal "Place cells" and its limbic connections as part of an extended vestibular system, the reported clinical features are now better understood as afflictions affecting the hippocampal, parahippocampal (entorhinal cortex) and the visual posterior sylvian area behind the Parieto-insular vestibular cortex (PIVC) [2–7]. The archival post-WWI photographs of soldiers exhibiting bizarre tilted posturing actually depicted a higher order vestibular (PIVC) deficit known as Verticality [8,9]. Even the classic "startle reflex" of PTSD can be explained by a patient's accommodation to oscillopsia, a vestibular disturbance producing an unstable or oscillating visual field [10]. Although Myers used the popular yet ill-defined term,"shell shock" in 1915 to reflect an assumed link between observed symptoms of tinnitus, amnesia, headache, dizziness, tremor, and hypersensitivity to noise among soldiers exposed to trench warfare and the effects from artillery shell explosions, the treatment which he and his co-military psychologist Arthur Hurst developed based on psychotherapeutic guidelines prevail to this day in the treatment of PTSD [11–15]. A little known but revealing historical note documented Charles Myers' frustration and refusal to contribute further wartime posttraumatic stress studies with the War Committee Inquiry into 'Shell Shock'[16,17] which attributed the causes of shell shock to 'leadership' issues and concluded that regular units with high morale were immune from "Shell shock" [18]. The later decision by the British Military to ban further use of the term "shell shock" from medical literature caused subsequent authors to ascribe different terminologies in place of shell shock [19–22] explaining the seeming historical disconnect between shell shock and PTSD. Kardiner in 1941 was the first to systematically describe the five clinical features noted among afflicted soldiers with "Chronic War Neuroses" citing the startle

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response, and denoting the clinical entity now known as PTSD. The evolving nomenclature of Posttraumatic stress responses which varied with its causality; i.e., eg, "shell shock syndrome" [23] "war neurosis" [21], "rape trauma syndrome" [24], "survivor syndrome" [25] and "Post-Vietnam Syndrome" [26,27] led to the formation of a theoretical framework whereby the concepts of fear reduction could be eventually accommodated under PTSD [28–33] which reached its current recognition as a distinct disease entity, with the incorporation of Kardiner's criteria in the Diagnostic and Statistical Manual of Mental Disorders- as written in its 3rd edition (DSMMD-III) during the background of the Tet offensive in Vietnam [34,35]. The vestibular manifestations of "deafness, dizziness and disorientation", however, continued to be treated independent of its noted association with PTSD.

The hypothesis/theory

Vestibular innervations of the semicircular canal, extend to the hippocampal "Place cells" [2,3], the parahippocampal "Grid cells" [4–6] and the limbic "Head Direction" cells [36–39]. This links the superficial interoceptive vestibular organelle and the deeper exteroceptive hippocampus in a common functional tract within the vestibulo-auditory pathway [40–43]. Blast wave exposure compounds its destructive effect in fluid-filled structures such as the semicircular canals and its friable vestibular innervations so that a deafferentation neural injury results with the frayed vestibular nerve [44–47]. A deafferented vestibular nerve causes faulty signal transmission affecting not only the vestibular nuclei but also its deeper-seated hippocampal connection to the amygdala and in turn, its downstream stress hormonal firing sequences to the hypothalamus [48–53]. When the adjacent amygdala is damaged, the ability to generate fear responses is lost. When only the hippocampus is with indirect functional damage however, as with Blast-induced vestibular deafferentation injuries transmitted from the semicircular canals, the amygdala's ability to generate fear responses is still retained [54–56], but the contextual memory of fear to which the amygdala links to in the hippocampus in order to trigger the fear response appropriate to the situation, is dysfunctional or absent, in which case, the amygdala may fire at a lower threshold or maladaptively, causing the manifestations we see in PTSD [57–63]. These maladaptive reactions are fed back to the thalamic projections and cortically imprints to the sensorintegrative parieto-insular vestibular cortex (PIVC) [7,64–69] completing a functional neural circuit preserving the balance between environmental orientation, reflexive postural adaptation and cognitive resilience on one hand and disorientation, maladaptive postural responses and cognitive dysfunction on the other. Affliction of a full-blown PTSD symptomatology only happens when the dominant vestibular tract in the non-dominant cerebral hemisphere) is affected and compensatory reflexive corrections from the contralateral vestibular tract is overcome [70–73]. The period when a dysfunctional circuitry cortically imprints in the Parieto-Insular Vestibular Cortex (PIVC) is 2 months [74] – also the average observed period post-trauma when PTSD becomes clinically manifest. For non-Blast cases of wartime PTSD as in Military Sexual Trauma (MST) [75], ischemic vestibular injury can be induced in stress states with occurrence of the "steal" effect when hypertensive perfusion favors the higher–calibered parent artery to the detriment of the smaller-calibered labyrinthine artery which perfuses the vestibular nerve [76] resulting in ischemia-induced deafferentation injuries in the vestibular nerve and its subsequent faulty signal transmission to the hippocampal 'Place cells' with the resultant mismatch between hippocampal contextual memory and amygdalar emotional memory before the inadvertent amygdalar firing for release of downstream hypothalamic stress hormones. This same imbalance of disorientation, maladaptive postural responses and cognitive dysfunction can ensue leading to PTSD manifestations using the same functional tract described even if the initial cause is not Blast- but rather mental stress–induced, to the level that would cause hypertensive ischemic deafferentation injury to the vestibular nerve (Fig. 1a).

Evaluation of the hypothesis/idea

The pairing of the auditory (cochlear) with the vestibular nerve in humans has been established embryologically and persists functionally in adult life [77]. The cochlear component organizes sounds that filters through the ears as consonance versus dissonance using the tonotopic cochlear-auditory cortex association to create a coherent registry to the brain [78,79], while the vestibular part gives the body a sense of position relative to the inner and outer environment. The vestibular nerve has also been identified with its own share of motor efferents from the brainstem flowing adjacent to the periolivary cell groups of the superior olivary nucleus (cochlear) joining the auditory nerve [80]. There exists an exchange of nerve fibers between the cochlear nerve and the superior and inferior vestibular nerve within the internal auditory canal [81]. The medial and lateral efferent cochlear fibers run within the inferior vestibular nerve, only joining the cochlear nerve at the anastomosis of Oort, a bundle of 1300 fibers running from the saccular branch of the inferior vestibular nerve to the cochlear nerve [81,82]. Functionally, this is manifested as sensory compensation and is often observed when enhanced audition and vestibular function substitutes for loss of vision as in echo-localization seen in bats and traditional coping mechanisms of the blind [83–87]. More importantly, direct neural projections traced from the auditory somatosensory nuclei of the thalamus to the amygdala were found which ran in parallel with an indirect circuit running first to the auditory cortex before ending up in the amygdala [56,88–92] which formed the basis of long-term potentiation (LTP), an experience-dependent form of neural plasticity involving mechanisms that underlie memory formation extensively studied in the hippocampus [93].

Although sharing conjoined neuroanatomical space from its peripheral extent in the middle ear to its central representation within the Sylvian fissure where the Auditory cortex lies adjacent the Vestibular cortex (Parieto-Insular Vestibular Cortex [PIVC]), respective vestibulocochlear nerve pathways in between central and peripheral connections take independent turns within the brain and the spinal cord making both pathways one of the most extensive cranial nerve network in the body. The inner environment refers to the body's position relative to the head wherein the vestibular apparatus serves as an interoceptive organelle correcting body movements in reference to an upright head position. It apparently is not alone in its function of assessing space and position, which is essentially a multi-modal cognitive task. The hippocampus, with its position or "place" cells whose signals were first detected in its CA1 region combines inputs from several different sensory modalities in its integration with the limbic system and fires when the rat model or human arrives at a familiar location it has previously "marked" in exploring unknown territory [2,3], thus serving as the exteroceptive biological equivalent of a Global Positioning System (GPS) receiver sensing the body's position relative to the external environment. Further studies exploring the origins of the signaling pattern have demonstrated "grid cells" [4–6,94,95] in the adjacent medial entorhinal cortex which fired impulses to the hippocampus in equally- spaced coordinates subserving the higher-level hippocampal circuit. Hippocampal stroke cases in humans showing reduplicative paramnesia wherein memory directions to familiar places in one's past were preserved with