Noninvasive measurement of head dielectric properties as a novel method for monitoring intracranial volume variations

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Noninvasive measurement of head dielectric properties as a novel method for monitoring intracranial volume variations

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Introduction: Intracranial pressure (ICP) monitoring and craniospinal compliance (CC) assessment can provide information for the diagnosis and management of neurological disorders. However, these tests are invasive, requiring probe placement within the cranial or spinal space. We present a method that may allow for the noninvasive derivation of CC surrogate metrics. It is based on the concept that cardiac and respiratory action modulate the dielectric properties of the head by periodic displacement of blood and cerebrospinal fluid (CSF), and that such modulations can be assessed noninvasively using capacitive measurements[1]. Through acquisitions on healthy volunteers, we show that the time course of the signal obtained in this fashion has characteristics reminiscent of typical ICP waveforms. We further show by physiological testing that the measured signal is, in part, of intracranial origin.
Methods: A custom device for capacitive measurements was coupled to the volunteers' heads through electrically isolated electrodes and hyperventilation testing was performed. As intracranial arteries have markedly higher CO_2 reactivity than extracranial ones, changes in the measured signal produced by hypocapnia are expected to be primarily of intracranial origin. We focused on the peak-to-peak amplitude of signal oscillations produced by cardiac action (AMP) as the metric to compare normocapnic and hypocapnic conditions.
Results: The acquired signal showed cardiac and respiratory oscillations. Hyperventilation reduced the end-tidal CO_2 from 5.7% ± 0.2 to 4.2% ± 0.3 (P = 0.028). AMP was reduced in all investigated subjects during hyperventilation and returned to its baseline value during a control period.
Discussion: We have developed a method for probing changes in the dielectric properties of the head induced by natural oscillations of the composition of the intracranial space due to cardiac and respiratory action. Hyperventilation testing suggests that signals obtained are, in part, of intracranial origin. CC surrogates could potentially be derived using this noninvasive approach.

References

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Cumulative Roles for different Viruses in Driving an Inflammatory Cascade Underlying MS Pathogenesis

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Roles for viral infections and aberrant immune responses in driving localized neuroinflammation and neurodegeneration in multiple sclerosis (MS) are the focus of intense research. Epstein-Barr virus (EBV), as a persistent and frequently reactivating virus with major immunogenic influences and a near 100% epidemiological association with MS, is considered to play a leading role in MS pathogenesis, triggering localized inflammation near or within the central nervous system (CNS). This triggering may occur directly via viral products (RNA and protein) and/or indirectly via antigenic mimicry involving B-cells, T-cells and cytokine-activated astrocytes and microglia cells damaging the myelin sheath of neurons. The genetic MS-risk factor HLA-DR2b (DRB1*1501b, DRA1*0101a) may contribute to aberrant EBV antigen-presentation and anti-EBV reactivity but also to mimicry-induced autoimmune responses characteristic of MS. A central role is proposed for inflammatory EBER1, EBV-miRNA and LMP1 containing exosomes secreted by viable reactivating EBV+ B-cells and repetitive release of EBNA1-DNA complexes from apoptotic EBV+ B-cells, forming reactive immune complexes with EBNA1-IgG and complement. This may be accompanied by cytokine-or EBV-induced expression of human endogenous retrovirus-W/-K (HERV-W/-K) elements and possibly by activation of human herpesvirus-6A (HHV-6A) in early-stage CNS lesions, each contributing to an inflammatory cascade causing the relapsing-remitting neuro-inflammatory and/or progressive features characteristic of MS. Elimination of EBV-carrying B-cells by antibody-and EBV-specific T-cell therapy may hold the promise of reducing EBV activity in the CNS, thereby limiting CNS inflammation, MS symptoms and possibly reversing disease. Other approaches targeting HHV-6 and HERV-W and limiting inflammatory kinase-signaling to treat MS are also being tested with promising results. We will present an overview of the evidence that EBV, HHV-6, and HERV-W may have a pathogenic role in initiating and promoting MS and possible approaches to mitigate development of the disease.

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The microbe-heart-brain dialogue: Vagal activity is associated with gut-microbiome patterns in women

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Introduction: A functional reciprocity between the gut microbiome and vagal nerve activity has been suggested, however, human studies addressing this phenomenon are limited.
Methods: 24- hour cardiac vagal activity (CVA) was assessed from 73 female participants (aged 24.5±4.3 years). Additionally, stool samples were subjected to 16SrRNA gene analysis (V1–V2). Quantitative Insights Into Microbial Ecology (QIIME) was used to analyze microbiome data. Additionally, inflammatory parameters (such as CRP and IL-6) were derived from serum samples.
Results: Daytime CVA correlated significantly with gut microbiota diversity (r=0.254, p=0.030), CRP (r=-0.348, p= 0.003), and IL-6 (r=-0.320, p=0.006). When the group was divided at the median of 24 hour CVA (Mdn=1.322), the following features were more abundant in the high CVA group: *Clostridia* (Linear discriminant analysis effect size (LDA)= 4.195, p= 0.029), *Clostridiales* (LDA=4.195, p= 0.029), *Lachnospira* (LDA=3.489, p=0.004), *Ruminococcaceae* (LDA=4.073, p=0.010), *Faecalibacterium* (LDA=3.982, p= 0.042), *Lactobacillales* (LDA=3.317, p=0.029), *Bacilli* (LDA=3.294, p=0.0350), *Streptococcaceae* (LDA=3.353, p= 0.006), *Streptococcus* (LDA=3.332, p=0.011). Based on Dirichlet multinomial mixtures two enterotypes could be detected, which differed significantly in CVA, age, BMI, CRP, IL-6 and diversity.