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Original article

Deformation of the corona radiata and internal capsule in normal pressure hydrocephalus

Déformation de la corona radiata et de la capsule interne dans l'hydrocéphalie à pression normale

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ABSTRACT

Background and purpose. – The pathophysiology of the clinical manifestations in normal pressure hydrocephalus (NPH) remains obscure. Ventricular dilatation could generate forces on the paracentral fibers of the corona radiata (CR), hence interfering with their function and producing the classical clinical triad. The analysis of the regional displacement and deformation of the white matter bundles, forming the corona radiata and internal capsule, may clarify the relationship between ventricular dilatation and clinical manifestations in NPH.
Method. – An experimental finite element (FE) analysis was used to simulate ventricular dilatation in 3 dimensions (3D) and to calculate the strain and deformation on the surrounding parenchyma. Magnetic resonance diffusion tensor imaging-based white matter tractography was then applied to retrieve the displacement and deformation exerted along various fiber bundles of the corona radiata and internal capsule. Anterior and posterior limb displacements and elongations were compared using a paired samples *t*-test.
Results. – The internal capsule, hence the corona radiata, of each cerebral hemisphere was segmented into anterior and posterior limbs. Mean displacements and elongations were calculated for each limb. Mean displacement was significantly larger in the anterior limb whereas mean deformation was larger in the posterior limb ($P < 0.01$).
Conclusion. – The present simulation demonstrates that ventricular dilatation does not have a homogeneous effect on the periventricular fibre tracts, with a particular load on the corticospinal tract. The affection of this tract remains thereby a potential factor in the generation of the NPH gait disorders.

Mots clés :
Hydrocéphalie à pression normale
Corona radiata
Analyse par éléments finis
Modèle du cerveau en maillage 3D
Tractographie de la substance blanche

RÉSUMÉ

Introduction. – La physiopathologie des manifestations cliniques dans l'hydrocéphalie à pression normale reste obscure. La dilatation ventriculaire pourrait générer des forces sur les fibres paracentrales de la corona radiata et interférer par-là avec leur fonction, ce qui pourrait expliquer la triade clinique classique. L'analyse du déplacement et de la déformation régionale des faisceaux de substance blanche constituant la corona radiata et la capsule interne pourrait aider à clarifier la relation entre la dilatation ventriculaire et les manifestations cliniques de la maladie.
Méthode. – Un modèle expérimental en éléments finis a été utilisé pour simuler la dilatation ventriculaire en trois dimensions et calculer la contrainte et la déformation subies par le parenchyme environnant. Une tractographie de ces faisceaux, basée sur une imagerie par tenseur de diffusion, fut ensuite fusionnée sur ce modèle afin de retrouver le déplacement et la déformation subis par les divers faisceaux de fibres de la corona radiata et la capsule interne. Les déplacements et les élongations des bras antérieurs et postérieurs ont été comparés en utilisant un test-*t* sur échantillons appariés.

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Résultats. – La capsule interne, et par là la corona radiata, de chaque hémisphère a été segmentée en bras antérieur et postérieur. Les déplacements et les déformations moyens ont été calculés pour chaque bras. Le déplacement moyen était significativement plus grand dans le bras antérieur alors que la déformation moyenne était significativement plus grande dans le bras postérieur ($p < 0{,}01$).
Conclusion. – La présente simulation démontre que la dilatation ventriculaire n'a pas un effet homogène sur les faisceaux périventriculaires de substance blanche, avec une déformation particulière du faisceau corticospinal. L'affection de ce faisceau reste donc un facteur potentiel dans la génération des troubles de la marche de l'hydrocéphalie à pression normale.

1. Introduction

The genesis of the clinical manifestations in normal pressure hydrocephalus (NPH) remains obscure [1,2]. Originally, Hakim et al. postulated that ventricular dilatation generates shearing forces on the fibers of the corona radiata (CR), hence interfering with their function and producing the clinical syndrome [3]. In hydrocephalus, in general, the enlarged lateral ventricles impinge on the periventricular white matter (WM) and cause a deformation of the myelinated fascicles of axons running within it. Yakovlev had already inferred that the longest corticospinal fibers, originating from the leg area of the primary motor cortex and descending closest around the dilated lateral ventricles, were the most stretched ones [4]. Thus, the deformation and ensuing dysfunction of the periventricular WM, and particularly of the paracentral fibers in the most medial region of the CR, where fibers from the cortical leg area run, has been hypothesised to cause one of the cardinal clinical manifestations of NPH, namely gait disturbance [5].

Finite element (FE) models of hydrocephalus have shown that ventricular dilatation results in particularly high levels of stress in the periventricular regions [6]. However, there are regional differences in the strain endured by the periventricular parenchyma. According to Pena et al., expansion forces predominate around the frontal and occipital horns whereas compressive forces have been observed along the body of the lateral ventricles [7,8]. These regional differences in mechanical strain could explain the preferential location of pathophysiological alterations found in hydrocephalus, such as CSF oedema around the ventricular horns [8] and the regional decrease of cerebral blood flow in the periventricular WM [9]. Since the ventricular system and the parenchyma demonstrate heterogeneities in dilatation and strain, it seems reasonable to presume that the periventricular WM tracts suffer differently from hydrocephalus depending on their location, orientation and spatial configuration in relation to the ventricles. Determining the pattern of displacement and deformation endured by the CR fibers during NPH constitution may enable us to clarify the unclear relationship between pathology and clinical manifestations. This analysis may in fact indicate which functional systems are significantly affected by the ventricular dilatation. The dysfunction of the specific WM bundles sustaining a particular mechanical load could then be reasonably considered as potentially responsible for at least a subset of the typical clinical picture. The absence of a differential mechanical impact, with homogeneous deformation and load among the WM bundles, might point to alternative pathogenic mechanisms, such as ischaemia or metabolic toxicity, to explain the clinical manifestations.

In this study, a new methodology was applied by combining finite element (FE) analysis and DTI-based white matter tractography. Ventricular dilatation and the resulting displacement and elongation on the brain parenchyma were simulated in 3 dimensions (3D) using a FE model. White matter tractography was then integrated in this model in order to determine and analyze the displacement and the deformation forced upon the white matter bundles forming the CR.

2. Materials and methods

2.1. Image acquisition

The MRI DTI of the brain of a 60-year-old healthy normal subject without significant medical history was acquired on a 1.5 T MRI scanner at the University Hospital of Geneva (70 slices, 128×128 matrix, voxel size $2 \times 2 \times 2$ mm^3, 30 diffusion directions). A T1-weigthted morphological volume was acquired in the same session and, apart a cavum septum pellucidum and a cavum vergae as variations of normality, proved normal (Fig. 1A). written and informed consent was obtained from our subject, one of the co-authors (DB). Fractional anisotropy (FA) and apparent diffusion coefficient (ADC) maps were calculated using standard methods [10,11].

2.2. Finite element model

The ADC volume was used to segment the brain surface and the ventricles. The anatomical information extracted from the MR images was used to construct a realistic 3D geometry that served as input for the simulation. The complex ventricular system configuration was thus accurately reproduced. The exact geometry of the basal brain was considered to be of little importance for the deformation of the entire brain, notably at the level of the lateral ventricles, and was therefore simplified. A 3D mesh was created semi-automatically using the COMSOL® software (Fig. 1B). The definition of the mesh was as accurate as possible near the ventricles in order to best represent the deformation of the parenchyma of this region and hence of the WM tracts running around the ventricles. Since the deformation closer to the brain surface was considered to be of less importance and more regular, a less precise mesh was tolerated in this region.

The material properties validated in a previous 2D FE model were implemented in the present 3D FE model. Briefly, the brain parenchyma was considered as a porous material with non-linear elastic and plastic behaviours. Homogeneous and isotropic initial material properties were assumed and the boundary conditions were the same as in the authors' previous 2D model [12]. As both hemispheres were taken into account, the falx cerebri (FC) was implemented in the FE model to avoid unrealistic ventricular and parenchymal displacements. The FC was modeled as a high ($E_{falx} = 100E_0$) elastic modulus fine slice above the ventricles in order to improve the quality of the model.

The boundaries constituted by the ventricular and brain surfaces define the domain in which the differential equations were solved to acquire the deformation (displacement and elongation) of the brain in hydrocephalic conditions. We used COMSOL® (Version 3.1) to solve the system of differential equations and Matlab® (R2007a) for pre- and post-treatments. The pre-treatment consisted essentially of image processing in order to acquire the brain and ventricular geometry. The post-treatment consisted of the calculation of the WM tracts deformation. Please refer to the appendix of the article describing the previous 2D model [12] for a detailed presentation of the poro-elastic equations involved in the present 3D model.