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Alteration of brain viscoelasticity after shunt treatment in normal pressure hydrocephalus

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Abstract
Introduction Normal pressure hydrocephalus (NPH) represents a chronic neurological disorder with increasing incidence. The symptoms of NPH may be relieved by surgically implanting a ventriculoperitoneal shunt to drain excess cerebrospinal fluid. However, the pathogenesis of NPH is not yet fully elucidated, and the clinical response of shunt treatment is hard to predict. According to current theories of NPH, altered mechanical properties of brain tissue seem to play an important role. Magnetic resonance elastography (MRE) is a unique method for measuring in vivo brain mechanics.
Methods In this study cerebral MRE was applied to test the viscoelastic properties of the brain in 20 patients with primary (N=14) and secondary (N=6) NPH prior and after (91±16 days) shunt placement. Viscoelastic parameters were derived from the complex modulus according to the rheological springpot model. This model provided two independent parameters μ and α, related to the inherent rigidity and topology of the mechanical network of brain tissue.
Results The viscoelastic parameters μ and α were found to be decreased with −25% and −10%, respectively, compared to age-matched controls (P<0.001). Interestingly, α increased after shunt placement (P<0.001) to almost normal values whereas μ remained symptomatically low.
Conclusion The results indicate the fundamental role of altered viscoelastic properties of brain tissue during disease progression and tissue repair in NPH. Clinical improvement in NPH is associated with an increasing complexity of the mechanical network whose inherent strength, however, remains degraded.

Keywords Normal pressure hydrocephalus · Shunt · Magnetic resonance elastography (MRE) · Viscoelasticity · Springpot

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Introduction

The pathophysiology of normal pressure hydrocephalus (NPH) is still a matter of controversy and intensive research. It is accepted that cerebrospinal fluid (CSF) flow, CSF resorption, and intracranial cavity pressure are altered in NPH [1–4]. However, the role of parenchymal changes to pathogenesis and symptoms in NPH remains unclear. The complexity of the pathophysiology underlying NPH arises from a cascade of events including metabolic changes, altered blood flow, impaired CSF exchange, and

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