

In vivo viscoelastic properties of the brain in normal pressure hydrocephalus

Kaspar-Josche Streitberger^{a,†}, Edzard Wiener^{b,†}, Jan Hoffmann^{c,d}, Florian Baptist Freimann^e, Dieter Klatt^a, Jürgen Braun^f, Kui Lin^g, Joyce McLaughlin^g, Christian Sprung^e, Randolph Klingebiel^{b,h,*} and Ingolf Sack^{a,**}

Nearly half a century after the first report of normal pressure hydrocephalus (NPH), the pathophysiological cause of the disease still remains unclear. Several theories about the cause and development of NPH emphasize disease-related alterations of the mechanical properties of the brain. MR elastography (MRE) uniquely allows the measurement of viscoelastic constants of the living brain without intervention. In this study, 20 patients (mean age, 69.1 years; nine men, 11 women) with idiopathic ($n = 15$) and secondary ($n = 5$) NPH were examined by cerebral multifrequency MRE and compared with 25 healthy volunteers (mean age, 62.1 years; 10 men, 15 women). Viscoelastic constants related to the stiffness (μ) and micromechanical connectivity (α) of brain tissue were derived from the dynamics of storage and loss moduli within the experimentally achieved frequency range of 25–62.5 Hz. In patients with NPH, both storage and loss moduli decreased, corresponding to a softening of brain tissue of about 20% compared with healthy volunteers ($p < 0.001$). This loss of rigidity was accompanied by a decreasing α parameter (9%, $p < 0.001$), indicating an alteration in the microstructural connectivity of brain tissue during NPH. This disease-related decrease in viscoelastic constants was even more pronounced in the periventricular region of the brain. The results demonstrate distinct tissue degradation associated with NPH. Further studies are required to investigate the source of mechanical tissue damage as a potential cause of NPH-related ventricular expansions and clinical symptoms. Copyright © 2010 John Wiley & Sons, Ltd.

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INTRODUCTION

Normal pressure hydrocephalus (NPH) was first observed and described in 1965 by Hakim and Adams (1), and represents an epidemiologically important disease with an incidence of 5.5 per 100,000 and a prevalence of 21.9 per 100,000 (2). The syndrome is

characterized by a triad of clinical symptoms: a slowly progressive gait disorder (usually the earliest feature of the syndrome), followed by symptomatic dementia and urinary incontinence. Cerebral imaging typically shows an enlargement of all ventricles, without sulcal widening, whereas cerebrospinal fluid (CSF)

* Correspondence to: R. Klingebiel, Neuroradiology and Radiology Institute, Klinik im Park, Seestrasse 220, CH-8027 Zürich, Switzerland.
E-mail: randolf.klingebiel@hirslanden.ch

** Correspondence to: I. Sack, Department of Radiology, Charité – Universitätsmedizin Berlin, Charitéplatz 1, 10117 Berlin, Germany.
E-mail: ingolf.sack@charite.de

a K.-J. Streitberger, D. Klatt, I. Sack
Department of Radiology, Charité – University of Medicine Berlin, Berlin, Germany

b E. Wiener
Department of Neuroradiology, Charité – University of Medicine Berlin, Berlin, Germany

c J. Hoffmann
Department of Neurology, Charité – University of Medicine Berlin, Berlin, Germany

d J. Hoffmann
Department of Neurology, University of California San Francisco, San Francisco, CA, USA

e F. B. Freimann, C. Sprung
Neurosurgical Department, Charité – University of Berlin, Berlin, Germany

f J. Braun
Institute of Medical Informatics, Charité – University of Medicine Berlin, Berlin, Germany

g K. Lin, J. McLaughlin
Mathematics Department, Rensselaer Polytechnic Institute, Troy, NY, USA

h K. Lin, J. McLaughlin
Neuroradiology and Radiology Institute, Klinik im Park, Zürich, Switzerland

† These authors contributed equally to this work.

Abbreviations used: CSF, cerebrospinal fluid; f , ω , driving (vibration) frequency, angular driving frequency; G^* , G' , G'' , $\overline{G^*}$, $\overline{G'}$, $\overline{G''}$, complex modulus, storage modulus, loss modulus and their spatially averaged quantities denoted by an overbar; $\overline{G'}$, $\overline{\alpha}$, storage modulus and springpot power exponent, averaged over the drive frequency; MEG, motion encoding gradient; MRE, MR elastography; MS, multiple sclerosis; NPH, normal pressure hydrocephalus; PV, periventricular; ROI, region of interest; SD, standard deviation; \mathbf{x} , position vector; $\overline{\mu}$, $\overline{\alpha}$, spatially averaged viscoelastic parameters according to the springpot model; $\overline{\mu}$, $\overline{\eta}$, spatial averages of shear elasticity and shear viscosity derived by the springpot model.