CSF pressure assessed by lumbar puncture agrees with intracranial pressure

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Abstract—The accuracy of estimating intracranial pressure in brain tissue (ICP<sub>BT</sub>) via lumbar space was investigated using preset pressure levels in the interval 0 to 600 mm H<sub>2</sub>O in patients with communicating hydrocephalus. Lumbar space ICP correlated excellently to ICP<sub>BT</sub>, demonstrated by a measured mean difference of 10 mm H<sub>2</sub>O (0.75 mm Hg) and a regression coefficient of 0.98. The concurrence supports the lumbar puncture as an accurate technique to determine ICP in patients with communicating CSF systems.

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There are several neurologic conditions where assessment of intracranial pressure (ICP) is clinically important. In contrast to intensive care, where ICP is measured directly inside the cranium, ICP is under these circumstances often estimated indirectly via lumbar space, a procedure starting with a lumbar puncture.

The lumbar puncture technique was introduced in 1891 and resulted in new options to investigate the intrathecal environment. Although not explicitly proven, the technique is believed to accurately estimate ICP when CSF circulates freely, a supposition that laid the foundation of the development of lumbar infusion tests to assess, it is assumed, intracranial hydrodynamics and pressure.

Unfortunately, the definition of ICP is ambiguous, regarding both where and how it is measured. Hitherto, ventricular CSF pressure has been considered the gold standard. However, in state-of-the-art neurosurgery and intensive care, brain tissue microprobes are used when determining ICP, redefining the ICP standard to brain tissue pressure.

This challenges the lumbar puncture technique: Is it applicable when measuring brain tissue pressure as well? We present a study comparing the two over a pressure range of 0 to 600 mm H<sub>2</sub>O using our unique computerized lumbar infusion apparatus, aiming to establish their absolute and relative relationship.

Methods. Clinical material. The study was based on 10 patients with idiopathic normal pressure hydrocephalus (INPH). Their mean age was 72.4 years, and they had symptoms of gait disturbance, memory deficiency, and urinary incontinence. MRI showed communicating hydrocephalus without stenosis and no significant ischemic or white matter lesions. All patients later received a CSF shunt, and at a follow-up all patients but one had improved. Informed consent was obtained from each patient, and the local ethics committee approved the study.

ICP measurement and data sampling. The set-up and execution of the parallel ICP measurement are shown in figure 1. An intraparenchymal catheter tip sensor (Codman MicroSensor™ Johnson & Johnson Professional, Raynham, MA) inserted into the roof of the right ventricle measured the direct ICP (ICP<sub>BT</sub>). The patients were then subjected to overnight ICP registrations.

The indirect ICP (ICP<sub>LS</sub>) measurement and infusion test via lumbar space have previously been described. In short, with the brain tissue sensor still present, two 1.2-mm needles were inserted into the lumbar subarachnoid space, and the patient was placed in supine position. One needle was used for CSF pressure measurement by a transducer (Becton Dickinson, Franklin Lakes, NJ) and the other for pressure control by CSF volume alteration.

The vertical separation distance (D<sub>sep</sub>) between the sampling positions was measured on CT scans and translated to its corresponding pressure difference (P<sub>sep</sub>) according to the formula

\[ P_{\text{sep}} = \rho \cdot g \cdot D_{\text{sep}} \]

where \( \rho \) = water density and \( g \) = gravitational constant (table).

Pressure data were sampled at 100 Hz using an acquisition card (MIO16X50; National Instrument, Austin, TX) and recorded on a computer. The pressure investigation was divided into three stages depending on the pump direction, and each stage was sliced into 1-minute intervals, of which the average pressures were calculated. Together, 1,273 1-minute intervals were included in the analysis.

Statistics. The difference of each 1-minute sample pair was calculated, and the patients’ individual and total mean differences and SDs were estimated. Additionally, the differences were correlated to pressure.

The regression coefficient \( k \) (slope) between ICP<sub>LS</sub> and ICP<sub>BT</sub> was determined by applying the following general linear model (GLM) to the data:

\[ \text{ICP}_{\text{LS}} - P_{\text{sep}} = k \cdot \text{ICP}_{\text{BT}} + m_{\text{Pump}} + m_{\text{Patient}} \]
**Results.** The total mean and SD of the measured differences between ICP_{LS} and ICP_{BT} are \(-10 \pm 29\) mm H\(_2\)O (\(-0.75 \pm 2.10\) mm Hg). The corresponding individual means are similar (table), except for two cases falling below \(-40\) mm H\(_2\)O, but the SDs are smaller.

The measured differences are homogeneously distributed (figure 2A), and the correlation to increasing pressure is minute (\(r = 0.16\), slope = \(-0.02\), \(p < 0.001\)). The extremes are \(-90\) and +84 mm H\(_2\)O, and 95% of the observations fall in the interval \(-69\) to +35 mm H\(_2\)O (\(-5.1\) to +2.6 mm Hg).

The GLM determination coefficient is 0.996, and all parameters contribute to determine ICP_{LS} (\(p < 0.001\) for ICP_{BT} and m_{Patient}; \(p < 0.01\) for m_{Pump}). The slope is 0.98 (CI: 0.97 to 0.99), and the pump factors are m_{Infusion} = 3 mm H\(_2\)O and m_{Drain} = \(-6\) mm H\(_2\)O, the latter significant in relation to m_{Halt} = 0. The patient-dependent factors (table) are significant, except in two cases.

**Discussion.** This is the first systematic and simultaneous comparison of ICP measured via lumbar space (ICP_{LS}) to ICP in brain tissue (ICP_{BT}), and the results demonstrate the accuracy of the lumbar puncture technique to determine ICP in both absolute and relative terms (figure 2). Instead of analyzing trauma patients with spontaneous ICP variation, we have analyzed patients with communicating CSF systems over a vast pressure interval utilizing our unique pressure control technique. The rising of ICP above physiologic range is standard procedure when diagnosing INPH patients\(^6\) and was well tolerated by the patients.

The total mean of the measured differences, \(-10\) mm H\(_2\)O, is clinically irrelevant, and their unbiased distribution (figure 2A), diminutive correlation to pressure, and 95% observation interval of (\(-69\), +35) mm H\(_2\)O further support the measurability of ICP via lumbar space. The individual SDs (table) were smaller than the total, indicating that the main contributor to the magnitude of the 95% observation interval was the varying individual means, not a common large variation.

The GLM-analysis showed that ICP_{BT}, pump factors, and patient-dependent factors all significantly contributed to explain the variation in ICP_{LS}, although the pump effect was marginal. Importantly, CSF infusion did not cause misreading of ICP_{LS}, a prerequisite when determining CSF outflow resistance via lumbar space in the course of diagnosing INPH\(^6\) and evaluating shunts.\(^7\) The determination coefficient of 0.996 and slope of 0.98 suggest that changes in brain tissue pressure are equally well recognized via lumbar space, an obligation for viable ICP detection by lumbar puncture.
where ICP measurement is considered, for in-

Paraplegia caused by invasive spinal aspergillosis

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A 40-year-old woman with a history of multiple sclerosis (MS), asthma, and Crohn disease suddenly developed paraparesis, which was attributed to MS. She had received infliximab for Crohn disease 14 days prior to the onset of paraparesis, as well as methotrexate and corticosteroids in the more distant past for asthma. Eight months later, she developed progressive upper extremity weakness and sensory changes. Physical examination revealed flaccid paralysis in both lower extremities, and sensation was absent below T3. MRI revealed an edematous spinal cord from C5 through the conus and multiple enhancing extramedullary masses (figure, A). Spinal cord biopsy revealed a caseating granuloma with fungal hyphae morphologically consistent with Aspergillus species (figure, B), although cultures were negative. Chest and abdomen CT performed within 3 days of hospital admission revealed no source of aspergillosis. Despite 7 months of therapy with voriconazole and caspofungin for presumed spinal cord aspergillosis, gradual progression of the spinal cord lesions was noted on MRI. Nine months after switching to posaconazole, she remains clinically stable with no further radiographic deterioration by serial MRI scans. There is a risk of granulomatous infections following tumor necrosis factor antagonist therapy, which may be caused by invasive fungi and may present with severe neurologic manifestations (figure).

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Figure. (A) MRI of the spine 8 months after the onset of paraparesis, indicating multiple enhancing intradural, extramedullary masses (arrows). (B) High-power view of spinal cord biopsy, demonstrating fungal hyphae consistent with Aspergillus species. Gomori methenamine silver stain.
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