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# Fundamentals of transorbital sonographic evaluation of optic nerve sheath expansion under intracranial hypertension

## II. Patient study

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**Abstract** Up to now, the presence of elevated intracranial pressure (ICP) in acute neurological disorders is suspected by clinical and neuroimaging findings, but its verification depends on invasive techniques. Based on our experimental findings of rapid dilatation of human optic nerve sheaths (ONS), we investigated whether this phenomenon not only happens under chronic, but also under acute conditions of intracranial hypertension. Using optic nerve sonography the ONS was measured at 3 mm behind the papilla in axial transbulbar view. Thirty-nine children admitted to the intensive care unit (ICU) were examined. Of these 24 were being treated for elevated ICP (head trauma, metabolic disorder) and were compared to control patients (outpatients). The ONS diameter (ONSD) found in ICU patients with elevated ICP ranged up to 6.8 mm and was significantly enlarged compared with normal data (Wilcoxon's test, $P = 0.007$). The ONSD of ICU patients without pressure elevation was in the same range as that of control patients (2.7–4.0 mm). Considering the error of measurement (0.35 mm), the ONSD is regarded as definitely enlarged when 5 mm is exceeded in children above age 4. In younger children, smaller ONSD have to be taken into consideration. We conclude that ultrasound studies of the optic nerve may contribute information about the acutely increased ICP in critically ill patients.

## Introduction

In order to estimate intracranial pressure (ICP), invasive methods utilizing epidural transducers or intraventricular catheters have been applied, but these cause additional traumatization and carry the risk of infection [1, 2]. Non-invasive evidence of elevated ICP can be obtained by means of cranial computed tomography (CCT; narrowed basal cisterns, cortical sulcus width), but this demands transport of the usually critically ill, ventilated patients. Other non-invasive methods, such as fontanometry [3], Doppler ultrasound of intracranial vessels [4] and recording of evoked potentials, require high levels of experience [5], manpower and equipment. Besides this, conclusive findings cannot be expected before advanced stages of intracranial hypertension, and the prognosis declines the longer ICP persists [6, 7].

On the basis of experimental results (see companion paper) from postmortem human optic nerve preparations, the present study compares ultrasound findings in control patients and children with elevated ICP in order to determine the clinical relevance of a pressure-induced increase in optic nerve sheath diameter (ONSD).

## Materials and methods

All ultrasound examinations were carried out with a commercially available scanner (Acuson 128, Mountain View, Calif.). As shown in Fig. 1, a 7.5-MHz linear array probe was placed on the upper eyelid after applying transmission gel. Alert patients were asked

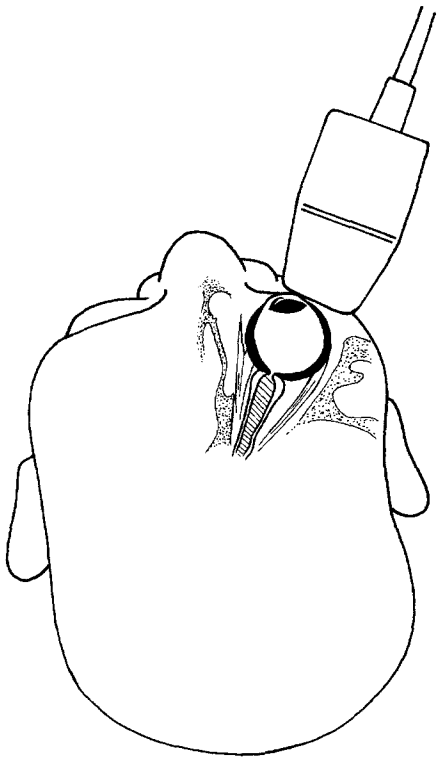

**Fig. 1** Schematic drawing of the sonographic procedure. The sound beam enters the orbit from the temporal eyelid. After traversing the bulbus, the optic nerve is displayed in axial direction

to keep their eyes in mid-position and to suppress eye movements. Similar to the experimental position (described in the accompanying study) the axial sound direction was used to depict the optic nerve entry zone. Behind the eyeball, the optic nerve appeared as a longitudinal echolucent band surrounded by echogenic orbital fat tissue. The optic nerve entry zone was selected as region of interest and magnified by zoom function (Fig. 2). In accordance with our experimental findings, the diameter of the optic nerve sheath (ONS) was measured at 3 mm behind the papilla (Fig. 2). To control the variability of this scanning procedure, repeated measurements ($n = 6$) were carried out for every optic nerve and mean values and standard deviations were calculated.

### Study groups

Three groups were defined for study purposes.

#### *Control subjects*

In 51 patients aged from newborn to 18 years, without optic nerve disease or intracranial pathology, the ONSD was determined bilaterally.

#### *ICU patients without elevated ICP*

These patients ($n = 15$) were examined to obtain control data under intensive care unit (ICU) conditions. In none of them was any evidence of intracranial pathology found.

#### *ICU patients with elevated ICP*

Twenty-four children who fulfilled the following criteria for elevated ICP were examined:
1. Clinical signs of intracranial hypertension (e.g., reduced consciousness level not due to medication, epilepsy or metabolic disorders) and/or signs of uncal herniation (secondary midbrain syndrome, see [8]).
2. Narrowed ventricles as demonstrated by CCT or transcranial ultrasound
3. Institution of aggressive therapy (e.g., analgosedatives plus hyperventilation and/or osmotherapy)

In seven patients, data from invasive monitoring with intracranial transducers was available which verified ICP elevation (above 20 mm Hg). The mean age was 4.8 years (range 2 months to 18 years). None of the children showed evidence of a local optic nerve disorder. For diagnoses, see Fig. 3. Of these children, 16 died due to uncontrollable ICP elevations.

For statistical analysis, the ONSD data obtained at maximum ICP (e.g., before treatment or in advanced stages of disease) were chosen and compared by means of non-parametric tests (Wilcoxon).

## Results

Orbital sonography as described was possible in all cases. The precision of measurement was controlled as follows: by the sum of squares within groups; the error of measurement was calculated using multiple measurements ($n = 6$). Over all age groups from birth to 18 years, the error was 0.11 mm (right ONSD) and 0.09 mm (left ONSD).

### Control subjects

In the first 4 years of life, the ONSD varied between 1.9 and 3.5 mm. Older children of up to 18 years had ONSD between 2.2 and 4.0 mm (Fig. 4). Side differences were mostly (in 93 % of all cases) less than 0.3 mm (maximum 0.5 mm).

### Children without elevated ICP

Because of one case of eye infection, only 29 of 30 possible measurements were performed. The ONSD ranged from 2.2 to 3.9 mm (mean: right 3.0 mm, left 3.1 mm). Mean age was 4.4 years (range 3 days to 15 years).

### Children with elevated ICP

The ONSD values of this group ($n = 24$ patients, 48 ONSD values) ranged between 3.6 and 6.8 mm, with a mean on both right and left sides of 5.3 mm. A typical example is shown in Fig. 5.

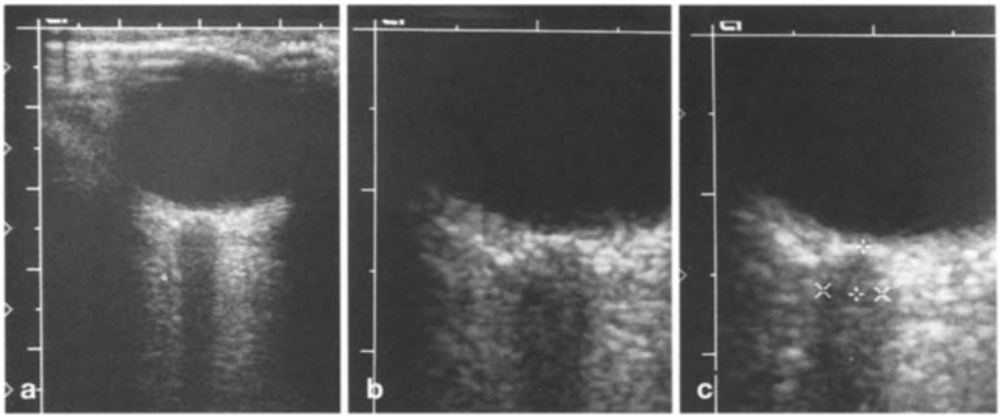


**Fig. 2a–c** This 3-year-old boy complained of headache and periorbital pain. **a** An overview of the right globe is shown. **b** The retrobulbar region is focused. **c** The perfect ONSD measurement (see *marks*) is demonstrated (3.0 mm behind the papilla)

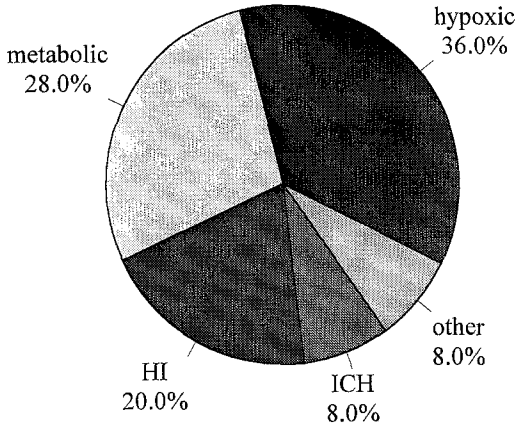


**Fig. 3** Summary of the diagnoses made in the subgroup of 24 children with intracranial hypertension. Note the high proportion of severe hypoxic injury and metabolic coma (*HI* head injury, *ICH* intracranial hemorrhage)

ONSD measurements in children without ICP compared to those with ICP

Non-parametric statistical comparison of the data of children without and those with ICP revealed a highly significant difference (Wilcoxon's rank sum test, $z = 2.60$, $P = 0.007$). The means of the two ICU groups differed by 2.3 mm (Fig. 6).

## Discussion

The main result of this study is the demonstration of a significant ONSD increase in patients with elevated ICP, which shows that dilatation of the ONS also takes place in acute disorders. This was demonstrated by ultrasound investigations of the optic nerve such as are routinely performed in ophthalmology for evaluation of patients with papilledema and intrinsic optic nerve disorders [9, 10]. This forms the basis for a new diagnostic method in which elevated ICP can be indicated by optic nerve sonography findings.

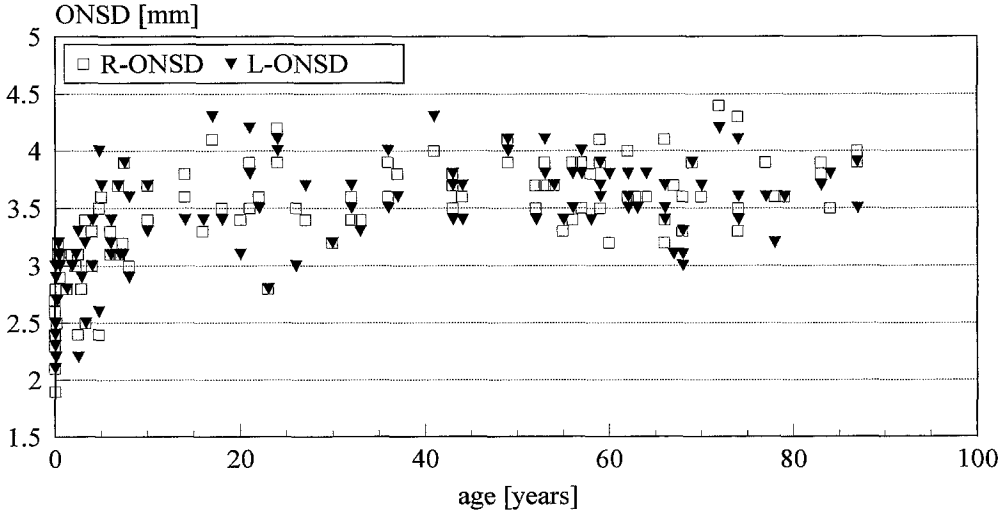


**Fig. 4** Relation between age and ONSD. For each patient right *(R)* and left *(L)* diameters are presented. There is an increase of ONSD during the first 4 years of life, after which the adult range is reached (data from unpublished studies)

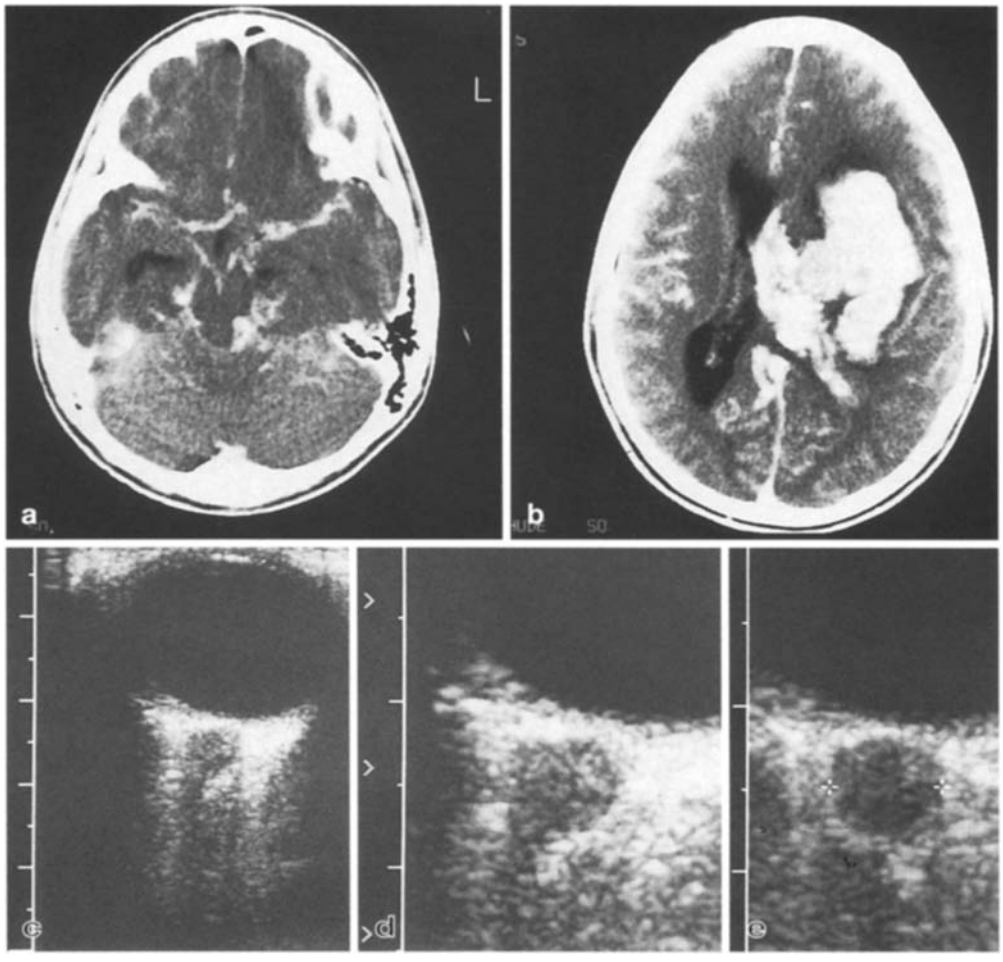


**Fig. 5a–e** A 13-year-old boy with large intracerebral hemorrhage after angioma resection. Cranial computed tomography (CCT) and orbital sonography were performed on the same day. **a, b** CCT. Hemorrhage involves the parenchyma and the subarachnoidal space. There is a midline shift and general cerebral edema. **c–e** Orbital sonography. **c** An overview shows the dilated optic nerve (echolucent rim) in transverse section. **d, e** Details of the optic nerve entry zone are shown. Both optic nerve sheath diameters (ONSD) are clearly enlarged (**d** 6.0 mm; **e** 6.2 mm)

For evaluation of patients using this method, control data were collected and a norm defined. Measurements in 51 patients in the first 18 years of life revealed that ONSD in children under the age of 4 years are lower than values found thereafter. Between the 4th and 5th years of life the ONSD of children reach the adult range. This contrasts with results of Ossoinig [11] and Szabo et al. [12] who found no age-related difference using A-mode examinations. The results of other authors, however, confirm our findings of a postnatal development. Harwood-Nash [13], who presented radiological normative data of the optic canal diameter, showed that its width progressively increases until the age of 4 years. Recently, Lorke and Lauer [14] performed longitudinal anatomical studies on mice and reported an age-related increase after birth, which was associated with postnatal myelinization of the optic nerve.

Normal ONSD values determined in the present study in older children correspond well with findings in adults reported elsewhere [15]. Usually, for adults, the 95 percentile range of the normal ONSD renders a value of 4.1 mm, the median being 3.7 mm. Our results show that, for pediatric clinical practice, lower values are expected only in younger children (between 2.0 to 3.5 mm).

To investigate the practical relevance of ONS evaluation under clinical conditions, age matched-groups were examined in two different situations: as ICU and outpatients. Comparison of the three groups shows that ICP elevation is accompanied by enlargement of ONSD throughout all age groups. Furthermore, it was demonstrated that only the ICU patients with elevated ICP had abnormal ONSD, while ICU patients free from pressure elevation showed ONSD similar to the control group.

This phenomenon corresponds well with the pressure-induced ONS changes found under experimental conditions (in vitro, see accompanying study). Though different age groups were investigated in the in vitro and in vivo studies, the ONS increase of 1–3 mm was similar. Only as a consequence of different sound direction, does the ONSD tend to be larger under in vivo conditions. Therefore, it is essential to compare clinical findings with control data obtained under identical ultrasound conditions.

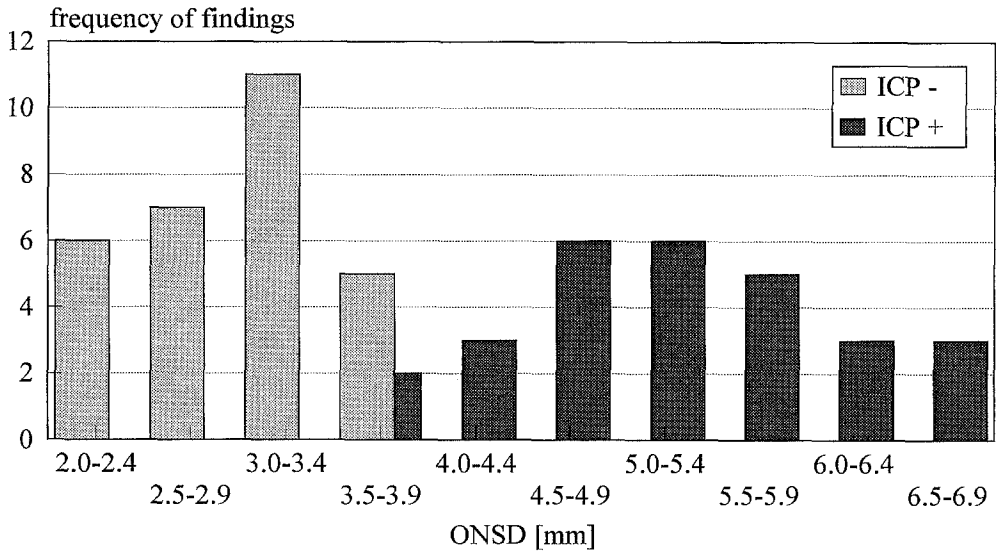


**Fig. 6** Histogram of optic nerve sheath diameter (ONSD) data obtained from children undergoing treatment in the intensive care unit. *Gray bars* represent the subgroup without intracranial hypertension *(ICP-)*, while the *black bars* represent children with elevated ICP *(ICP+)*. There is a marked difference between the two subgroups: most patients with elevated ICP exceeded 4.0 mm ONSD

Clinical interpretations of ONSD findings must take the error of measurement into account: under repeated measurements, the error is a mean of 0.3 mm. Therefore, in older children (above age 4) an ONSD above 4.5 mm may be considered "borderline," while an ONSD exceeding 5.0 mm could be regarded as "definitely" enlarged. In children under the age of 4 an ONSD above 4.0 mm can already be considered "definitely" enlarged. This interpretation is limited to patients without intrinsic optic nerve afflictions that could cause swelling. Pathogenetic certainty in such cases can then be achieved by comparison with the opposite optic nerve.

Although no normal ONSD findings under elevated ICP were encountered in pediatric patients in the present study, this possibility should be kept in mind. We have recently reported [15] that 1 out of 16 adult patients with severe head injury or cerebrovascular disorder and ICP elevation did not reach the ONSD "borderline" level. This may be attributed to blocked optic canals or extremely small optic nerves before onset of elevated ICP. For this reason, optic nerve sonography will not be able to exclude the presence of elevated ICP with absolute diagnostic certainty.

In summary, the present study demonstrates that in children with both acutely and chronically elevated ICP, dilatation of the ONS is regularly present. Maximum changes detected in anterior regions can be depicted sonographically. The phenomenon should be checked for bilaterality; when present, intracranial hypertension can be deduced.

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