

Increased Blood Pressure and Increased Optic Nerve Sheath Diameter: Intracranial Involvement or a Nonspecific Marker?

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Short Editorial related to the article: Ultrasound Measurement of Optic Nerve Sheath Diameter in Acute Hypertensive States For Detection of Intracranial Involvement: A Meta-Analysis

Effects of increased blood pressure extend beyond the systemic arteries

A hypertensive emergency is defined by presence of acute target-organ damage, not merely by an increased blood pressure reading. In brain, however, identifying such involvement at bedside remains challenging. Headache, nausea, visual disturbances, seizures, and altered consciousness are clinically important but nonspecific manifestations. Although fundoscopy has traditionally played an important role in clinical assessment, its reliability depends on adequate training, appropriate technical conditions, and patient cooperation. Moreover, papilledema may be absent during initial hours of increased intracranial pressure (ICP).¹ Within this diagnostic window, ultrasonographic measurement of optic nerve sheath diameter (ONSD) represents an appealing option: it is rapid, portable, and repeatable and capitalizes on anatomical continuity between intracranial subarachnoid space and perioptic compartment.^{2,3}

Article accompanying this short editorial provides most focused synthesis to date of ONSD findings in acute hypertensive states. Its results are consistent with those of previous studies involving acute hypertension, severe preeclampsia, and eclampsia.⁴⁻⁶ Review included 14 studies comprising 995 participants, predominantly patients with preeclampsia or eclampsia, together with a considerably smaller group of nonpregnant patients with hypertension. Across nine studies with continuous data suitable for pooling comprising 426 hypertensive patients and 283 controls, ONSD was significantly greater in hypertensive group, with a large standardized mean difference (Hedges'g: 1.55; 95% CI: 1.06-2.04; $p < 0.001$). This is not a trivial finding: it suggests that association between acute hypertension and optic nerve sheath distension is sufficiently consistent that it cannot readily be dismissed as an isolated artifact.

Palavras-chave

Arterial Pressure; Optic Nerve; Intracranial Pressure; Ultrasonography

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DOI: <https://doi.org/10.36660/abcimg.20260107i>

A robust effect does not establish a validated diagnosis

Large effect size, however, coexisted with substantial heterogeneity ($I^2 = 87\%$). Eight of nine studies reported large and directionally consistent effects, whereas one yielded a discordant result, with numerically greater ONSD values among controls. Excluding this study increased the pooled effect estimate to 1.77 (95% CI: 1.50-2.04) and reduced I^2 to 51.8%, which suggests that much of inconsistency was attributable to a single study. Nevertheless, remaining heterogeneity should not be overlooked: study populations, control groups, acquisition techniques, measurement boundaries, insonation planes, and ultrasound equipment varied across studies.

Dichotomous findings pointed in same direction but warrant similar caution. In five studies reporting observed event counts, hypertensive patients had greater odds of an ONSD exceeding the threshold adopted in each study (odds ratio [OR]: 11.58; 95% CI: 1.45-92.85; $I^2 = 73.7\%$). Because of the extremely wide confidence interval, frequent zero-event cells, and reliance on a continuity correction, this estimate is more informative about the direction of the association than about its precise magnitude. After exclusion of discordant study, OR increased to 29.54 and heterogeneity fell to zero. This apparent improvement in precision, however, does not overcome the limitations of the underlying studies.

The principal evidence gap is more fundamental: none of 14 studies compared ONSD with invasive ICP measurements or cerebrospinal fluid opening pressure. Meta-analysis therefore demonstrates that on average acutely hypertensive patients have a larger ONSD than controls. It does not demonstrate that all such patients have intracranial hypertension nor does it establish the diagnostic accuracy of ONSD for detecting this condition. Receiver operating characteristic curves reported in individual studies assessed the ability of ONSD to distinguish preeclampsia from normotension, a distinction already identifiable from blood pressure measurements and the obstetric context, rather than its ability to detect increased ICP. Conflating these diagnostic targets would transform a phenotypic association into an unwarranted claim of pathophysiological equivalence.

These limitations do not negate the diagnostic performance of ONSD measurement in other populations. In neurocritically ill patients without traumatic brain injury, a systematic review of 18 studies comprising 1,484

participants reported a pooled sensitivity of 92%, specificity of 90%, and area under the curve of 0.925 for detecting increased ICP.⁷ These findings support use of ONSD as a screening tool and reinforce its biological plausibility. However, they cannot be extrapolated automatically to acute hypertensive states, which differ in their underlying etiologies, measurement techniques, and reference standards.

Autoregulation and Monro-Kellie 4.0: the mechanism matters

Cerebral autoregulation provides the principal mechanistic link among mean arterial pressure (MAP), ONSD, and ICP. When autoregulation is intact, an increase in MAP triggers arteriolar vasoconstriction, thereby limiting the transmission of systemic pressure to the cerebral microvasculature. When the upper limit of autoregulation is exceeded or cerebrovascular reactivity is impaired, pressure is transmitted more passively, promoting hyperperfusion, increased cerebral blood volume, and vasogenic edema.^{8,9}

The Monro-Kellie 4.0 model further posits that this response is governed by intracranial compliance and compensatory reserve, both of which vary dynamically across intracranial regions. Interactions among the blood, cerebrospinal fluid, and interstitial compartments, compounded by venous congestion or focal lesions, may create pressure gradients and exhaust regional compensatory reserve before overt global deterioration becomes apparent.⁹

Within this framework, ONSD should be viewed as a marker of pressure within the perioptic compartment rather than as a standalone measure of global intracranial dynamics. When measured bilaterally approximately 3 mm behind the globe using a standardized technique,^{1-3,7} ONSD may reflect rapid changes in pressure, although measurements remain susceptible to individual anatomical variation and technical factors. This sensitivity is both its principal strength and a major source of interpretive uncertainty.

A single cutoff may provide false reassurance

Accompanying article shows that available evidence does not support the use of a universal ONSD threshold. Reported cutoffs varied widely across studies and were derived for different clinical conditions. The 5.8-mm threshold frequently cited in obstetric research was extrapolated from neurocritical care populations and has not been specifically validated in this clinical setting. Even seemingly minor methodological differences (including the choice of the inner or outer dural border, measurement axis, and imaging plane) can alter ONSD values by approximately 0.5-1.0 mm. Such variation is clinically meaningful when proposed thresholds fall within the narrow range of 5-6 mm.

Moreover, the available evidence is overwhelmingly derived from patients with hypertensive disorders of pregnancy. Only one of the nine studies included in the

continuous-data meta-analysis evaluated nonpregnant patients with hypertension. Although that study also reported a large effect, its findings alone are insufficient to justify extrapolation to nonobstetric hypertensive emergencies. Studies using paired measurements found reductions in ONSD after magnesium sulfate administration or delivery, suggesting that ONSD reflects a dynamic and potentially reversible process. However, without a concurrent reference standard, these changes cannot be attributed solely to changes in ICP.

Positioning statement: context, trends, and multimodal concordance

Available evidence suggests that increased ONSD in acute hypertensive states should be regarded as a marker of possible intracranial involvement — modulated by cerebral autoregulation and compensatory reserve — rather than as a stand-alone diagnosis of intracranial hypertension. In isolation, an increased ONSD should not prompt aggressive blood pressure reduction or ICP-directed therapy. Conversely, a normal ONSD does not exclude severe cerebral disease, a focal intracranial lesion, or evolving intracranial hypertension. An excessively rapid reduction in MAP, particularly in patients with chronic hypertension and a rightward shift of the autoregulatory curve, may convert suspected cerebral hyperperfusion into iatrogenic cerebral hypoperfusion.

We therefore advocate a four-layered approach to interpretation. First, establish the clinical syndrome and confirm the presence of acute target-organ damage. Second, interpret MAP in the context of the patient's usual blood pressure, the rate of blood pressure increase, and the probable rightward shift of the autoregulatory curve associated with chronic hypertension. Third, integrate ONSD findings with the neurological examination, fundoscopy when feasible, and computed tomography or magnetic resonance imaging. Fourth, whenever possible, obtain an early baseline ONSD measurement and prioritize serial assessments performed using a standardized technique, ideally by the same operator. In the absence of a validated universal cutoff, intraindividual changes — interpreted alongside the clinical course, other neuromonitoring modalities, and controlled changes in MAP — may be more informative than a single measurement and may help identify improvement or deterioration over time.

The B-ICONIC consensus was developed for patients with traumatic brain injury, the population in which its supporting evidence is concentrated. Extrapolation to other populations, including patients with acute hypertensive states, is therefore based on low-certainty evidence and should not be presented as a validated protocol.¹⁰ Nevertheless, the underlying physiological principle of multimodal concordance remains relevant: when invasive ICP monitoring is unavailable, no noninvasive modality should be interpreted in isolation. Concordant findings across at least two modalities, such as ONSD measurement, transcranial Doppler or duplex ultrasonography, automated pupillometry, and noninvasive ICP waveform analysis,

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when integrated with clinical and neuroimaging findings, may provide a prudent approach to diagnostic triangulation in these patients.

Accordingly, an increased ONSD should prompt further corroboration rather than be regarded as a diagnostic endpoint. Its clinical relevance increases when it is concordant with abnormal transcranial Doppler findings, altered pupillometry, impaired noninvasive ICP waveform morphology, neurological deterioration, or radiological abnormalities. When findings are discordant, the acquisition technique should be reviewed, the measurement repeated, the possibility of intracranial compartmentalization considered, and the diagnostic evaluation expanded. Brzan Simenc et al., for example, identified discrepancies among ocular ultrasonographic markers in patients with severe preeclampsia, underscoring that indirect markers may respond differently to the same pathophysiological process and should not be regarded as interchangeable.⁵

The next step in this field should not be merely to identify another cutoff. Prospective studies should simultaneously assess MAP, ONSD, invasive ICP when monitoring is clinically indicated or cerebrospinal fluid opening pressure

when available, PaCO₂, patient position, neuroimaging findings, and markers of cerebral autoregulation. They should also standardize the ultrasound equipment, acquisition plane, and measurement boundaries and evaluate preeclampsia/eclampsia, hypertensive encephalopathy, posterior reversible encephalopathy syndrome, intracranial hemorrhage, and nonobstetric acute hypertension as distinct clinical entities. Individual participant data and serial measurements will be necessary to establish technique- and population-specific thresholds and to determine whether ONSD provides incremental prognostic information or meaningfully guides treatment.

The article accompanying this short editorial strengthens the evidence for an association between acute hypertension and increased ONSD while clearly delineating the boundaries of current knowledge. Figure 1 displays the proposed relationship among MAP, autoregulatory integrity, and the response of the perioptic compartment. Measurement is valuable; interpretation is indispensable. Clinical utility of ONSD lies not in treating it as an absolute manometer, but in using it as a dynamic window into a complex intracranial system as part of a concordant, multimodal, physiologically informed assessment.

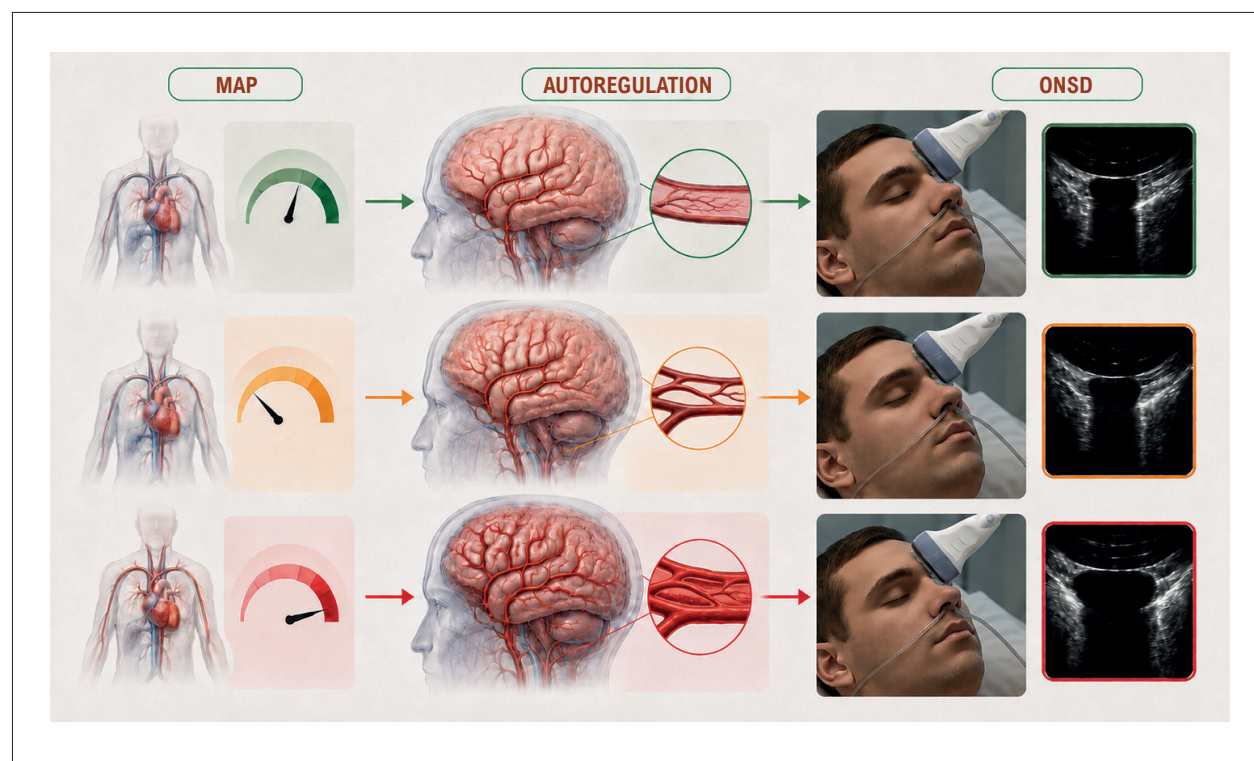


Figure 1 – Proposed physiological model of the relationship among MAP, cerebral autoregulation, and ONSD. In upper panel, preserved autoregulation induces arteriolar vasoconstriction, limiting the transmission of increased systemic pressure to the intracranial circulation and preventing a clinically relevant increase in ONSD. In middle panel, impaired autoregulation allows passive vasodilation and an increase in cerebral blood volume, potentially resulting in ONSD enlargement. In lower panel, more severe autoregulatory failure is associated with cerebral hyperemia, greater intracranial involvement, and more pronounced optic nerve sheath distension. This figure presents a conceptual model and should not be interpreted as a stand-alone diagnostic test. MAP: mean arterial pressure; ONSD: optic nerve sheath diameter.

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