Pearls & Oy-sters:
Spontaneous venous pulsation and its role in differentiating papilledema from pseudopapilledema

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Spontaneous venous pulsation (SVP) of the optic disc can be used as a surrogate marker of intracranial pressure (ICP) in patients with discs of normal morphology. The presence of SVP can suggest an ICP within normal limits.[1] Based on this premise, SVP is also referred to when differentiating anomalous pseudopapilledematous optic nerves from true papilledema. However, this application may be limited by the fact that anomalous discs are less likely to demonstrate SVP.[2] The case detailed below supports this contention.

CASE REPORT A 48-year-old white woman presented to our hospital complaining of a recent onset of blurred vision. She was known to have migraine with aura and had a past ocular history of unilateral disc drusen (left eye only). On examination, the best-corrected visual acuity was 0.7 in the right and 0.5 in the left. Anterior segment examination was unremarkable in both eyes. Color vision was normal in the right and slightly reduced in the left (10/13 Ishihara) and there was a left relative afferent pupillary defect. Funduscopy revealed left optic nerve drusen with a clearly elevated drusenoid left optic disc and a normal-appearing right optic disc (figure). Both maculae and peripheral fundi were normal. Goldmann visual fields demonstrated a left superior scotoma. Fundus fluorescein angiography confirmed the presence of disc autofluorescence in the left eye without leakage (figure). MRI scanning failed to demonstrate any intracranial pathology. Left optic neuropathy secondary to disc drusen was diagnosed. Interestingly, SVP was noted in the right eye but was absent from the left (videos 1 and 2 on the *Neurology*® Web site at www.neurology.org).

DISCUSSION Spontaneous retinal venous pulsation is the rhythmic change in the caliber of one or more retinal veins at the optic disc caused by variation in the pressure gradient between the intraocular retinal veins and the retrolaminar portion of the central retinal vein, which is subjected to ICP as it passes through the subarachnoid space.[1] Since the variation of intraocular venous pressure between systole and diastole (the ocular pulse pressure) is greater than the CSF pulse pressure, the velocity of venous outflow from the eye increases during systole and decreases during diastole.[3] This leads to the systolic collapse of the retinal vein as it exits the eye characteristic of SVP. The pulse is only manifest at the distal vein over the optic disc since, due to the inherent resistance to fluid flow within the retinal venous system (to which the viscosity of blood is a contributory factor), the pressure difference between intraocular and extraocular veins is only transmitted a short distance into the eye.[3] The outflow theory better explains the etiology of SVP than the classic theory, which suggests that the vein collapses in response to the ocular arterial pulse. In fact, this should not occur since the retinal venous pressure is consistently higher than intraocular pressure throughout the cardiac cycle.[4] The venous outflow theory also explains how SVP and ICP can be related. Elevation of ICP is accompanied by an increase in CSF pulse pressure. When CSF pulse pressure equals retinal venous pulse pressure, the SVP is extinguished.[3]

The absence of SVP, however, is not solely associated with raised ICP. SVP is reported to occur in 90% of healthy individuals and therefore is absent in 10%.[5] Absence of SVP has also been reported in cases with unilateral disc swelling due to anterior ischemic optic neuropathy or optic neuritis in the presence of normal ICP, presumably by local compression of the superficial veins.[6] Another proposed mechanism is the increase in retrolaminar retinal venous pressure as a result of elevated tissue pressure within the optic nerve, which then dampens the variation in the pressure gradient between the intraocular retinal veins and the retrolaminar portion of the central retinal vein, eliminating SVP.[1] Most importantly, cessation of SVP has been noted in individuals with unusual optic disc–vessel configuration only, in the absence of elevated ICP or disc swelling.[2] Disc features associated with failure to show SVP include veins obscured by arteries or glial tissue as they course into

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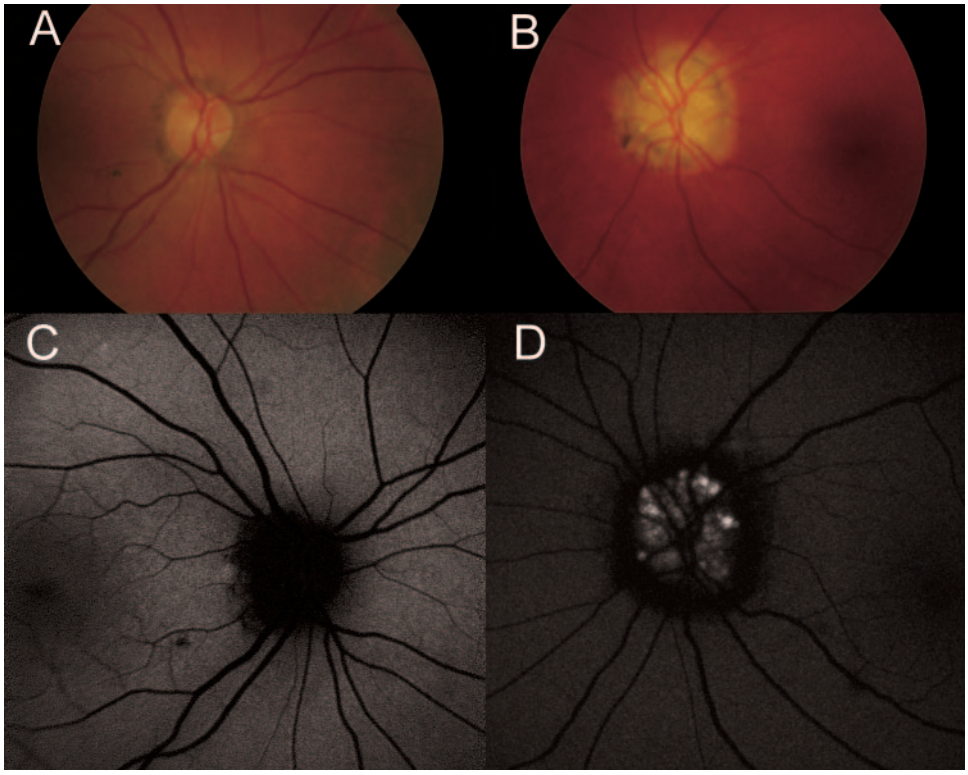
Figure Color fundus and autofluorescent images



Color fundus images showing disc drusen in left eye only (A and B), confirmed with autofluorescence (C and D).

the cup, absent cup with veins appearing flat on the surface of the disc rather than end-on, and congenitally full discs and veins entering the disc peripherally rather than centrally so that the venous branches are not visible in the shallow cup.[2]

Although there are specific examples of patients continuing to demonstrate SVP in conjunction with raised ICP, an association between increasing ICP and SVP cessation is well-established.[7,8] The important caveat is that the association is only to be applied to patients with optic discs of normal morphology. Using SVP to differentiate papilledema from pseudopapilledematous optic nerves (with normal ICP) is therefore less appropriate. Consistent with this, the reported patient with unilateral disc drusen demonstrated SVP in the non-drusen eye only, supporting the premise that SVP can be abolished by isolated optic disc anomaly independent of the ICP. This case therefore demonstrates the problem of using SVP in isolation to differentiate anomalous nerves from papilledematous nerves, since disc anomaly can of itself be associated with an absent SVP.

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