

PHYSICAL SIGNS

Spontaneous retinal venous pulsation: aetiology and significance

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Spontaneous retinal venous pulsation is seen as a subtle variation in the calibre of the retinal vein(s) as they cross the optic disc. The physical principles behind the venous pulsations has been the point of much debate. Initial theories suggested that the pulsation occurred because of the rise in intraocular pressure in the eye with the pulse pressure. This article presents an argument that this is not the case. The pulsations are in fact caused by variation in the pressure gradient along the retinal vein as it traverses the lamina cribrosa. The pressure gradient varies because of the difference in the pulse pressure between the intraocular space and the cerebrospinal fluid. The importance of this is that as the intracranial pressure rises the intracranial pulse pressure rises to equal the intraocular pulse pressure and the spontaneous venous pulsations cease. Thus it is shown that cessation of the spontaneous venous pulsation is a sensitive marker of raised intracranial pressure. The article discusses the specificity of the absence of spontaneous venous pulsation and describes how the patient should be examined to best elicit this important sign.

Spontaneous retinal venous pulsations (SVPs) are rhythmic variations in the calibre of one or more of the retinal veins as they cross the optic disc. SVPs may be subtle and are often limited to a small segment of only one vein. Whether they are obvious or difficult to identify, their appearance is that of a rhythmic movement of the vessel wall in time with the cardiac cycle—narrowing with systole and more rapid dilation with diastole.¹

To understand the significance of SVPs, one must first understand the physical principles behind them. Coccus² first described SVPs in 1853. He concluded that during systole the influx of blood into the eye causes a rise in the intraocular pressure (IOP), thus compressing the vein. This theory was supported by Elliot³ but was challenged by Duke-Elder,⁴ who punctured a retinal vein and showed that blood leaked from the puncture site into the vitreous cavity even after the IOP was raised by intraocular injection of fluid. Duke-Elder thus argued that retinal venous pressure (RVP) is always greater than IOP. In addition, Elliot's hypothesis could not explain why the pulsations occurred only at the optic disc and not along the whole venous system.

Poiseuille's law states that blood flows within a vessel from point A to point B if there is an intravascular pressure gradient between the two points. For example, because retinal capillary pressure is greater than intraocular RVP, blood flows from retinal capillaries to retinal veins. The RVP at the point at which the central retinal vein (CRV) exits the eye is called the outflow pressure, and this is determined by the pressure in the retrolaminar portion of the CRV within the optic nerve. For blood to flow out of the eye this must be less than the intraocular RVP. Baurmann⁵ constructed a model of the retinal venous system and observed pulsations at the point of venous outflow when the IOP was greater than the outflow pressure; however, he noted that the IOP did not have to be greater than the intraocular RVP to induce pulsation. Indeed, Attariwala *et al*⁶ subsequently observed in cats by direct measurements that the intraocular RVP was consistently higher than IOP regardless of how high the IOP was raised.

Levine⁷ explained the physics of SVPs by using a comprehensive mathematical model. As stated above, the intraocular RVP exceeds the IOP throughout the cardiac cycle.^{4,6} The walls of the intraocular retinal veins lack rigidity; thus, fluctuations in IOP are transmitted into the intraocular retinal vessels and the pressure gradient from the vitreous to the blood across the wall of the intraocular retinal vein never reverses. For example, during systole, IOP rises by 1.5 mm Hg and intraocular RVP rises by the same amount (the pulse pressure). Thus, blood flow within the retinal veins does not alter during the cardiac cycle because changes in IOP are transmitted immediately to the retinal veins and capillaries, keeping the flow within these vessels constant.

However, when the CRV exits the optic nerve 10 mm behind the globe, it passes through the subarachnoid space. This segment of vessel is thus subjected to intracranial pressure.^{8,9} Because cerebrospinal fluid (CSF) pressure rises by 0.5 mm Hg during systole and falls by 0.5 mm Hg during diastole (the CSF pulse pressure),¹⁰ the pressure in the retrolaminar portion of the CRV also increases by 0.5 mm Hg during systole and decreases by 0.5 mm Hg during diastole. Thus, the intraocular pulse pressure is 1 mm Hg higher than the retrolaminar venous pulse pressure during systole (1.5 mm rise in intraocular RVP versus 0.5 mm rise in retrolaminar venous pressure) and

Abbreviations: CRV, central retinal vein; CSF, cerebrospinal fluid; IOP, intraocular pressure; RVP, retinal venous pressure; SVP, spontaneous retinal venous pulsation

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Intraocular venous pressure

Intraocular retinal venous pressure rises 1 mm Hg more than retrolaminar pressure with systole. This results in increased flow of blood from the eye. With a constant inflow to the venous system this increased outflow results in partial collapse of the retinal vein.

Venous pressure gradient across lamina cribrosa rises and central retinal vein collapses

Retrolaminar venous pressure

Retrolaminar venous pressure rises 1 mm Hg less than intraocular venous pressure with systole, outflow of blood from the eye increases with systole causing the vein to collapse.

Intraocular venous pressure

Intraocular retinal venous pressure falls 1 mm Hg more than retrolaminar pressure with diastole. This results in decreased flow of blood from the eye with diastole. As venous outflow is constant this causes the retinal vein to expand.

Venous pressure gradient falls and central retinal vein expands

Retrolaminar venous pressure

Retrolaminar venous pressure falls 1 mm Hg less than intraocular pressure with diastole, outflow of blood from the eye reduces causing the vein to expand.

Figure 1 Relation between intraocular and retrolaminar retinal venous pressure, explaining the origin of intraocular retinal venous pulsations. Intraocular pulse pressure is 3 mm Hg and retrolaminar pulse pressure is 1 mm Hg. With raised intracranial pressure the retrolaminar pulse pressure rises to equal the intraocular pulse pressure. As the intraocular and retrolaminar retinal venous pressure vary by the same amount with the cardiac cycle, there is no longer a variation in the pressure gradient in the retinal vein across the lamina cribrosa, flow of blood from the eye does not vary with the cardiac cycle, and retinal venous pulsations cease.

1 mm Hg lower during diastole. Blood flow from the eye therefore increases during systole and decreases during diastole (blood flow is dependent on the pressure gradient according to Poiseuille's law). As the flow into the venous system from the retinal capillaries is constant, the increased flow at the point of venous outflow during systole decreases the volume of blood in that segment of vein, causing it to collapse. In diastole, the flow at the point of venous outflow decreases, blood volume increases, and the vein expands (fig 1). The length of venous segment that pulsates is small because the pulsation is dampened by the physical properties of the vein, blood, and surrounding structures.⁷

Why are SVPs important to the clinician? SVPs are present in 81% of all eyes and 90% of normal subjects^{11,12}; thus, 10% of otherwise normal persons do not have SVPs. This is important for two reasons. Firstly, it is clear from the above discussion that CSF pressure is a major determinant of blood flow within the CRV and, hence, SVPs. Indeed, Levin¹² showed that SVPs occurred only in patients with CSF pressures below 190 mm H₂O. Patients with optic disc swelling, regardless of the cause, generally have no SVPs. The reason for this phenomenon is not fully understood. Presumably, increased tissue pressure within the optic nerve results in increased retrolaminar RVP and this dampens the variations in the pulse pressure gradient from the intraocular to the retrolaminar retinal vein, eliminating SVPs. Thus, in a patient in whom it is unclear whether one or both discs are swollen, the presence of SVPs indicates that the disc is not swollen. Secondly, as the intracranial pressure increases, CSF pulse pressure rises,¹⁰ eventually equalling the intraocular pulse pressure. When this occurs, there is no longer a fluctuating intravascular pressure gradient between the intraocular retinal veins and the retrolaminar retinal vein, venous blood flow becomes constant, and SVPs cease. Thus, if SVPs are present in a patient suspected of having increased intracranial pressure (with or without papilloedema), the CSF pressure must be normal (that is, the intracranial pressure must be \leq 190 mm H₂O at that moment).

It must be emphasised that neither the presence nor absence of SVPs is sufficient to state with certainty that the intracranial pressure is normal. For example, in patients with an intracranial tumour that produces increased intracranial pressure, CSF pressure tends to be consistently increased. Thus, CSF pressure does not fluctuate and SVPs are consistently absent. However, in idiopathic intracranial hypertension (pseudotumour cerebri), intracranial pressure often fluctuates and may even be normal at times.¹³ Thus, SVPs may be present at times. This does not mean that the patient does

not have idiopathic intracranial hypertension but only that at the moment the fundus is being observed, the patient's intracranial pressure is less than 190 mm H₂O.

The anatomy of the optic disc may affect the ease with which SVPs can be detected and, indeed, whether SVPs are present.¹⁴ The length of the venous segment that collapses depends in part on the optic disc-vessel configuration. In addition, the veins may be obscured by arteries or glial tissue.

As with all examination skills, the correct technique must be used in assessing the presence or absence of SVPs. It is generally much easier to observe SVPs through a dilated pupil.^{15,16} Thus, it is best to dilate the patient's pupil with a short acting mydriatic agent, such as tropicamide. It is also important when assessing the presence or absence of SVPs to have adequate magnification. The best instrument to use for observing SVPs is the direct ophthalmoscope because it provides a significant degree of magnification (about 15 times depending on the refractive error of the patient),¹⁵ although a 78 or 60 dioptre lens can be used with a slit lamp biomicroscope. SVPs cannot be detected with an indirect ophthalmoscope because of the lack of adequate magnification provided by this instrument.¹⁵ In patients in whom SVPs do not appear to be present, a useful technique is the use of digital pressure on the globe through the eyelid to induce venous pulsations.¹ Once venous pulsations are observed in a particular location, one stops the pressure and continues observing the area to see whether there are still venous pulsations that simply were not detected initially. Because SVPs occur only with an intracranial pressure below 190 mm H₂O, one might be tempted to try to assess intracranial pressure indirectly by pressing on the globe with a finger and seeing how much digital pressure was required to induce venous pulsations. In fact, because there is no way to know how much pressure one is placing on the globe and what effect this has on retrolaminar venous pressure, we do not consider this technique to be reliable and would hesitate to estimate the level of intracranial pressure based on its use.

In conclusion, SVPs are an important clinical sign caused by a fluctuating intravascular pressure gradient between the intraocular retinal veins and the retrolaminar portion of the CRV. The pulsations are observed as a subtle narrowing and expansion of one or more retinal veins on the optic disc and are present in 90% of normal persons. The examination technique requires adequate visualisation and magnification. The presence of SVPs allows the examiner to conclude that the patient does not have optic disc swelling and that the patient's CSF pressure at that time is $<$ 190 mm H₂O. Finally, this clinical sign needs to be interpreted in the light of the history and other clinical findings.

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NEUROLOGICAL STAMP.....

Hans Berger (1873–1941), Richard Caton (1842–1926), and electroencephalography

Hans Berger recorded the first human electroencephalograms (EEGs) in 1924. He obtained his medical degree from the University of Jena, Germany, in 1897 and then joined the university psychiatric clinic directed by Otto Binswanger. There he remained until retirement in 1938. Berger succeeded Binswanger as director of the clinic and became Professor of Neurology and Psychiatry at the University of Jena in 1919. In his early work Berger had hoped to discover the physiological basis of psychic phenomena. The results were disappointing and Berger turned to investigating electrical activity of the brain. He characterised the wave patterns including α and β waves and coined the term "electroencephalogram". Berger's paper *Über das Elektrenkephalogramm des Menschen* (On the EEG in humans), published in 1929 in the *Archive für Psychiatrie und Nervenkrankheiten*, was the first of 23 publications on the subject. He described or touched upon a large number of normal and abnormal EEG phenomena, for example EEG changes associated with attention and mental effort, and alterations in the EEG associated with cerebral injury. His reports, at first disbelieved, were even derided by some until Adrian and Matthews confirmed his basic observations in 1934. In the mid 1930s, Alfred Loomis (1887–1975) showed that in humans EEG patterns changed dramatically during a night's sleep. Unrelated to EEG, in 1920 Berger also described intellectual changes after prefrontal cortex injuries, and in 1923 his was one of the first good descriptions of perseveration after damage to the frontal lobes.



In 1929 Berger cited Caton's valuable earlier contribution to the field. Caton reported his initial findings to the British Medical Association in 1875. In 1877 these were reported more fully in a supplement to the *BMJ*, and again in 1887 to the Ninth International Medical Congress in Washington DC. Caton placed unipolar electrodes on the surface of both hemispheres or one electrode on the cerebral cortex or on the grey matter and the other on the surface of the skull. Currents were measured by optical magnification of the meniscus in his Thompson's galvanometer. Currents were found to increase with sleep and variations in the baseline unrelated to cardiac or respiratory rhythms were observed. These currents were vulnerable to anoxia and anaesthesia, and were abolished by the animal's death. Caton also found that strong current variations occurred when light was shone into the eyes. He also discovered cerebral potential change evoked by sensory stimulation. Caton is better remembered as

becoming Lord Mayor of Liverpool in 1907. His work received no attention among English speaking electrophysiologists. The *Lancet*, in its obituary column, did not mention Caton's contribution to electrophysiology. The *BMJ* noted only that he did some work on the localisation of movements in the 1870s. Of interest, Berger was also not honoured in his own country. This was in part owing to his opposition to the Nazis. Berger became increasingly depressed after retirement in 1938, and died by suicide in 1941.

EEG has been illustrated on a number of stamps. An Italian stamp of 1988 shows a pictorial representation of an EEG and St Valentine (Stanley Gibbons no. 1989, Scott no. 1743). St Valentine was the first bishop of Temi in Umbria. Some of the mythology is not entirely clear, but St Valentine was probably a physician who was martyred by the Romans on February 14, 273. He is patron saint of both lovers and epilepsy. There are also other patron saints of epilepsy. Legend has it that St Valentine miraculously cured a young fiancée, Serapia, afflicted with a mysterious illness, thought now to be epilepsy. Sites where St Valentine was thought to have lived or visited became pilgrimage destinations for cure of the disorder. These destinations included Rome and Temi in Italy, Ruffach in France (where a hospital for epilepsy was later built), Poppel in Belgium, and Passau in Germany. Soon after Valentine's death young lovers started making pilgrimages to Temi to be blessed by the Bishop on the 14th hour of every month for eternal love.

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