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.

To understand the significance of SVPs, one must first understand the physical principles behind them. Coccius 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 Elliot but was challenged by Duke-Elder, 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.

Boiseuille'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. Baumann 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, Attarivala et al subsequently observed in cats by direct measurements that the intraocular RVP was consistently higher than IOP regardless of how high the IOP was raised.

Levine explained the physics of SVPs by using a comprehensive mathematical model. As stated above, the intraocular RVP exceeds the IOP throughout the cardiac cycle. 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. 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), 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
Intraocular venous pressure increases during systole (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 pulsatility is dampened by the physical properties of the vein, blood, and surrounding structures.

Why are SVPs important to the clinician? SVPs are present in 81% of all eyes and 90% of normal subjects,


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 pulsatility is dampened by the physical properties of the vein, blood, and surrounding structures.

Why are SVPs important to the clinician? SVPs are present in 81% of all eyes and 90% of normal subjects,


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 pulsatility is dampened by the physical properties of the vein, blood, and surrounding structures.

Why are SVPs important to the clinician? SVPs are present in 81% of all eyes and 90% of normal subjects,


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 pulsatility is dampened by the physical properties of the vein, blood, and surrounding structures.

Why are SVPs important to the clinician? SVPs are present in 81% of all eyes and 90% of normal subjects,
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 Archiv 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 directed by Otto Binswanger. There he re-directed by Otto Binswanger. There he re-

In 1929 Berger cited Caton's valuable earlier work. His paper entitled Ueber die Anwendung des Augen-Spiegels, nebst Angabe eines neues Instrumentes. Leipzig: Muller, 1853:3–23. discusses cerebral potential change evoked by sensory stimulation. 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 stomps. 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.