Starling resistor

The “Starling resistor,” which has been conserved across species, acts at the junction between cortical bridging veins that drain into the superior sagittal sinus, prevents siphoning of venous blood, and maintains ICP as we stand up.

As the collapsible cortical bridging veins are draining into the rigid venous sinuses, they traverse the subarachnoid space and are subject to surrounding external CSF pressure. Essentially, the Starling resistor is a mechanism that maintains constant flow through collapsible tubes (such as cortical draining veins) when they are surrounded by variant ambient pressure (CSF in the subarachnoid space) all contained in a rigid box, such as the skull in the hydraulic model.

The Starling resistor is a site of compression, also known as a “choke point,” at the junction between the bridging vein and the sagittal sinus such that when pressure in the superior sagittal sinus drops during upright posture, the higher CSF pressure will choke the downstream connection to the sagittal sinus and prevent venous overdrainage.

Concurrently pressure in the cerebral veins proximal to the Starling resistor is maintained at a higher level than CSF pressure because of the created back pressure.

Thus the Starling resistor is responsible for maintaining the hierarchy of pressures of the various liquid compartments in the cerebrum, that is, arterial in - flow pressure > cerebral venous pressure > subarachnoid CSF pressure > sagittal sinus pressure. This, in turn, creates a pressure gradient to promote CSF drainage into the sagittal sinus. Various anatomical and MRI studies of the terminal regions of bridging veins are consistent with the idea that the Starling resistor functions as a flow resistor.

There is overwhelming evidence now that the cerebral venous system plays a major role in intracranial pressure (ICP) dynamics especially when one takes into account the effects of postural changes, atmospheric pressure, and gravity on the craniospinal axis as a whole. The CSF and cerebral venous compartments are tightly coupled in two important ways. CSF is resorbed into the venous system, and there is also an evolved mechanism that prevents overdrainage of venous blood with upright positioning known as the Starling resistor. With loss of CSF pressure, this protective mechanism could become nonfunctional which may result in posture-related venous overdrainage through the cranial venous outflow tracts leading to pathologic states [1)].

A investigation was undertaken to determine whether a Starling resistor or venous waterfall effect exists between the sagittal sinus and the cerebral veins such that increases in sagittal sinus pressure (Pss) do not abolish cerebral venous outflow and to examine two possible contributions of extracranial venous valves in regulating outflow. Anesthetized dogs were subjected to positive end-expiratory pressure (PEEP) before and after intracranial pressure (Pic) was elevated by inflation of an epidural balloon. PEEP raised Pss equally in all animals, but Pic and cerebral venous pressure (Pcv) increased less in the presence of intracranial hypertension. When Pss was low, passage of a catheter in the cerebral vein in and out of the sagittal sinus demonstrated an abrupt drop in pressure as the sinus was entered. When Pss was raised and lowered independently of superior vena caval pressure (Psvc) the changes in Pic and Pcv were less when Pss was decreased than when it was increased. Sustained

increases and decreases in Psvc caused increases and decreases in Pcv, Pic, Pss, and external jugular venous pressure (Pejv) regardless of whether external jugular venous valves were present or absent. We conclude that a Starling resistor between the sagittal sinus and the cerebral veins regulates cerebral venous outflow when Pss is increased by PEEP and other maneuvers that raise Psvc. The waterfall maintains Pcv and Pic at normal levels when Psvc and Pss are reduced. Extracranial venous valves are not essential to this mechanism [2)].

The incidence of posttraumatic hydrocephalus (PTH) has been reported to be 0.7-51.4%, and De Bonis et al., have frequently observed the development of PTH in patients undergoing decompressive craniectomy (DC).

They analyzed the area of craniotomy and the distance of the craniotomy from the midline. Results showed that patients who had undergone craniectomies with a superior limit less than 25 mm from the midline had a markedly increased risk of developing hydrocephalus. They hypothesized, based on the Starling resistor concept, that during each cardiac cycle in which extracellular fluid is produced during systole and absorbed in diastole, an imbalance between the production and absorption of extracellular fluid in favor of absorption would cause a decrease in brain parenchyma volume and consequently increase the size of the ventricles [3)].

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