Intracranial aneurysm pathophysiology

see also Intracranial aneurysm hemodynamics.

Alterations in the internal elastic membrane (lamina elastica interna) of cerebral arteries are thought to weaken vessel walls and render them less resistant to changes in intraluminal pressure [1].

These changes most frequently develop at sites of vessel bifurcation, where blood flow is most turbulent and shear forces against the arterial wall are greatest [2].

This can be because of acquired disease or hereditary factors. The repeated trauma of blood flow against the vessel wall presses against the point of weakness and causes the aneurysm to enlarge. As described by the Law of Young-Laplace, the increasing area increases tension against the aneurysmal walls, leading to enlargement.

Despite evidence for a potential role of angiotensin in the pathophysiology, angiotensin-receptor blockers have shown little-to-no efficacy in preventing aneurysm formation and growth [3].

Despite technical and diagnostic progress there are still open questions in the understanding of the pathophysiology of intracranial aneurysms.

Assuming a preexisting reduced resistibility of the vessel wall to pressure changes and an area of permanently low wall shear stress WSS, an increase in pressure induces geometrical changes. These cause changes of intravascular flow distribution, lowering the already low WSS in specific locations. This leads to endothelial damage in this area and to a decreasing stability of the vessel wall, causing aneurysm development, growth, and rupture [4].

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