## Common iliac artery aneurysm: imaging-guided pathophysiology

The interesting report by Steenberge et al[1] deserves some insights from pathophysiology. According to Laplace's law, the aneurysm size will increase proportionally to the increase in inner tension. However, because the common iliac artery aneurysm (CIAA) is an almost inelastic structure, other factors can intervene. First, aortic laminar flow with a high Reynolds number will become turbulent. Second, the Bernoulli equation states that as the bloodstream velocity decreases, the parietal tension will increase. Third, the lack of elasticity in the aneurysm walls will cancel the Windkessel effect, normally capable of transferring a part of the kinetic energy from the systolic to the diastolic phase of the sphygmic wave. Fourth, according to the parallelogram of forces, the multiple segments derived from the split main blood flow will collide against the aneurysm rigid walls with different trajectories, or will oppose the original direction of flow (Fig). Overall, the CIAA will have an increased risk of rupture owing to the energy unevenly distributed on its walls, the irregular wall thickness, and the unpredictable degree of residual static strength.

At present, aneurysm blood flow can be outlined using four-dimensional reconstructed magnetic resonance imaging, and computed tomography, and color Doppler ultrasound can demonstrate well the CIAA walls and intraluminal thrombus.[2-7] In CIAAs, the fibrin filaments and entrapped red blood cells, undergoing premature lysis, will promote inflammatory processes, rapidly propagating to the surrounding aneurysm walls, which will be followed by increased contrast enhancement on magnetic resonance imaging. The consequent release of cytokines, mainly elastases and metalloproteinases, will contribute to the destruction of parietal elastic fibers and the dispersal of smooth muscle cells, generating dissection, lamination, and fractures inside the thrombus itself.[8] In the medium to long term, this process can evolve into fibrous sclerosis and calcification, further threatening aneurysm wall strength.[9,10] In addition, the hemodynamics can also be evaluated quantitatively, which will correlate with decreased blood flow in the downstream collateral vessels. The Hagen-Poiseuille equation demonstrates that the blood flow, proceeding through an inelastic CIAA with irregular geometry and a rigid external iliac artery, will reduce its kinetic energy.

In conclusion, the vascular surgeon's diagnostic and decisional armamentarium can be improved, just in the present era of endovascular surgery, in which two or more correlated pathologies can be treated concurrently.

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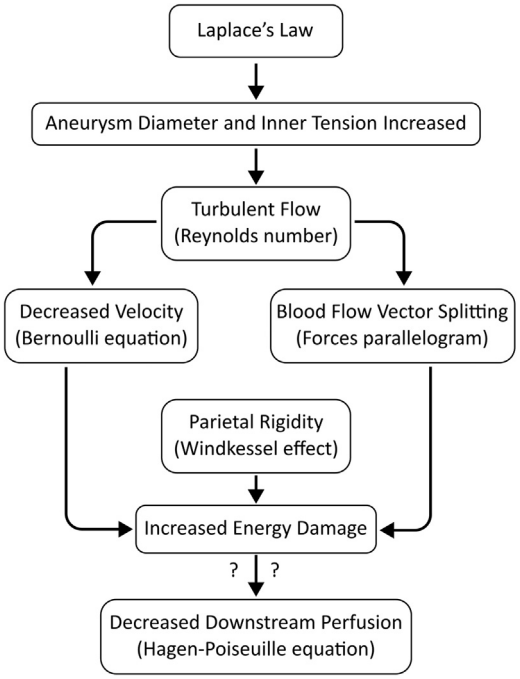


**Fig.** Diagram of different factors affecting volume, inner tension, and inelastic energy damage in common iliac artery aneurysms (CIAAs).

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## Reply

We appreciate the consideration and commentary of our report by Manenti et al and their review of the aneurysmal pathophysiology. However, the question remains regarding the optimal treatment diameter and morphology, or otherwise, of isolated common iliac artery aneurysms, with the exception of our data, which have suggested that such aneurysms had been treated traditionally at diameters posing an uncertain risk/benefit ratio.[1]

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## Poor mid- and long-term outcomes after complex endovascular aortic aneurysm repair procedures call for careful patient selection

In the last few years, endovascular repair of juxta-/suprarenal, paravisceral, and thoracoabdominal aortic aneurysms has become possible with the use of several advanced devices/techniques, such as chimneys, periscopes, and fenestrated stent grafts.[1-4]

The results of a recent, single-center retrospective study reviewing the outcomes of the management of 58 patients with such complex aneurysms revealed some particularly concerning findings.[1] Besides the high reintervention (n = 15 of 58 patients; 25.8%) and complication (n = 21 of 58 patients; 36.2%) rates, survival at 1 year was 61.3% (75% for elective; 37% for urgent/emergent procedures) and 27% at 5 years. This means that nearly 4 of 10 patients with juxta-/suprarenal, paravisceral, and thoracoabdominal aortic aneurysms in this study were not alive 1 year after their complex endovascular procedure.[1]

Another recent study assessing the effects of patient frailty status on outcomes after fenestrated-branched endovascular repair of complex abdominal/thoracoabdominal aortic aneurysms demonstrated similar findings.[4] Patients were grouped into low-, mid-, and high-risk groups based on the presence of several comorbidities (eg, chronic kidney disease, respiratory disease, congestive heart failure, history of myocardial infarction/stroke, etc).[4] The 5-year survival estimates ranged from 60% ± 4% in the low-risk to 32% ± 6% in the high-risk groups ($P < .001$), whereas these rates were even worse (16% ± 7%) in the high-risk patient subgroup with additional chronic kidney disease (Table).[4]

These alarmingly high 1- and 5-year mortality rates raise concerns about the mid- and long-term effectiveness of such complex endovascular techniques/procedures. What is the rationale of offering such advanced endovascular procedures to patients with complex aortic aneurysms if more than a third of these individuals will not survive the first year postprocedurally?[1] What is the overall benefit for these patients if they end up suffering not only high complication and reintervention rates, but also particularly high 1- and 5-year mortality rates?[1,4]

The poor mid- and long-term outcomes after complex endovascular aortic aneurysm repair procedures call for careful patient selection to ensure that advanced endovascular procedures are offered to those individuals most likely to benefit from such techniques.