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Table S1: Comparison of aneurysm epidemiology, morphology, clinical treatments, intervention criteria, simulation considerations, and types of clinical studies. Abbreviations: D, Diameter; KD, Kawasaki disease

location	**epidemiology and morphology**	**intervention criteria and current treatments**	**simulation considerations and types of clinical studies**
all locations	connective tissue disorder, atherosclerosis [1–10]	diameter, diameter-based z-score, or rate of increasing diameter[2–4,11–14]	subject-specific boundary conditions and geometry are needed[15]
cerebral aneurysms (IAs)	**risk factors**: hypertension, smoking, atherosclerosis, family history of subarachnoid hemorrhage, polycystic kidney disease, connective tissue disorders, and female sex[1,16] **prevalence**: 3.2% (95% confidence interval 1.9% - 5.2%[1]) **morbidity/mortality**: 60% mortality rate within the first 6 months of a subarachnoid hemorrhage[16] **morphology**: saccular (90%, 85% of which are at the Circle of Willis), fusiform, dissecting, and micotic[17]	**concern**: subarachnoid hemorrhage[1] **treatment**: endovascular coiling, neurosurgical clipping, and endovascular stents[1] **surgical intervention**: based on clinical history, family history, location, and size (D ≥ 7 - 10 mm)[1,11,16,18]; however, the most recent consensus guidelines do not define a diameter cutoff[1]	• proper location of vessel truncation[19] and inclusion of side arterial branches[20] improves accuracy • small vessel size reduces Newtonian blood flow assumption[21], segmentation[22], and velocity measurement[23] accuracy • small aneurysm size limits boundary condition and geometry sources (reduced accuracy with 4D flow MRI[24]; limited use with ultrasound) • rigid wall assumption can alter WSS values and increase flow instability[25,26] ➢ **case series surgical planning[27,28], studies comparing rupture cases to unruptured controls[29,30], large studies comparing rupture cases to control[31–34], prospective study[35]**
thoracic aortic aneurysms (TAAs)	**risk factors**: connective tissue disorder (e.g. Marfan syndrome), aortic valvular disease (bicuspid valve), hypertension, smoking, and infection (syphilis)[2,6] **prevalence**: 0.16 - 0.34%[36,37]	**concern**: dissection, rupture[2] **treatment**: open aneurysm repair or endovascular aneurysm repair[2] **surgical intervention**: D ≥ 55 mm[2,40] or a growth rate of ≥ 5	• pulsatile helical flow observed[41,42] • deformable walls can account for the influence of vessel wall motion on hemodynamics[43]

location	epidemiology and morphology	intervention criteria and current treatments	simulation considerations and types of clinical studies
	morbidity/mortality: acute dissection 27.4% and rupture is fatal for 59% before reaching the hospital[38] **morphology**: fusiform > saccular[6], the majority occur in the root or ascending aorta (~60%)[39]	mm/year with considerations for genetic syndromes or bicuspid valve and other factors[2,6]	• dissection or thrombus may alter geometries [44,45] ➢ **case study**[45], **case compared to control**[46], **treatment options**[47–49]
abdominal aortic aneurysms (AAAs)	**risk factors**: smoking, connective tissue disorder, atherosclerosis, hypertension, and male sex (but rupture is associated with female sex)[2–4] **prevalence**: 1.4%[4] **morbidity/mortality**: > 80% mortality with rupture[3] **morphology**: fusiform more common[3]	**concern**: rupture[3] **treatment**: open aneurysm repair, endovascular aneurysm repair[3] **surgical intervention**: $D \geq 50$ - 55 mm or 10 mm/year[2,3,4]	• pulsatile simulations capture transient formation and disappearance of vortices[50–53] • inclusion of material properties and aortic dynamics can mimic vessel wall behavior[51], but rigid wall sufficiently quantifies blood flow dynamics[54] • can couple with growth and remodeling studies to assess AAAs dynamics[51] • inclusion of an intraluminal thrombus and major neighboring branching vessels improves accuracy[51,55,56] ➢ **case study**[57] **and treatment options**[58–61]
peripheral artery aneurysms (PAAs)	**carotid, axillary, brachial, femoral, and popliteal arteries** **risk factors**: hypertension, atherosclerosis[9] smoking[10], trauma (femoral artery pseudoaneurysm), and male sex is associated with iliac, femoral, and popliteal artery aneurysms[62–64] **prevalence**: not well known and varies across locations (popliteal artery aneurysm are found in 1% men over 65)[12,62]	**concern**: thrombosis and rupture[12] **treatment**: open aneurysm repair, or endovascular aneurysm repair[12] **surgical intervention**: if $D \geq 20$-40 mm, and depends on location, reviewed in[12,13]	• low prevalence[12,62] limits data sets[65] • variation in anatomical location[65] complicates models ➢ **case study in an idealized geometry**[66], **comparisons between ruptured and unruptured aneurysm**[65]

location	epidemiology and morphology	intervention criteria and current treatments	simulation considerations and types of clinical studies
	morbidity/mortality: thromboembolic complications for popliteal artery aneurysm 35% (8 - 100%)[9] **morphology**: saccular, fusiform[9]		
visceral artery aneurysms (VAAs)	**splenic, celiac, superior and inferior mesenteric, renal, and hepatic arteries** **risk factor**: connective tissue disorders, atherosclerosis, fibromuscular dysplasia, trauma, vasculitis, hypertension, pregnancy (splenic), splenic and renal artery aneurysm associated with female sex, and hepatic artery aneurysm associated with male sex[7,8] **incidence**: 1% (range 0.098% - 10.4%)[8] 0.01 - 0.2%[67] **morbidity/mortality**: 25 - 70% mortality rate depending on location[8,67] **morphology**: saccular or fusiform depends on location[8]	**concern**: rupture[12] **treatment**: open aneurysm surgical repair or endovascular aneurysm repair[13] **surgical intervention**: if $D \geq 20$ mm[12,13]	• low prevalence[8,67] limits data sets • variation in geometries and anatomical location of the aneurysm[68] complicate models ➢ **case study**[69], **case study for surgical planning**[70]
coronary artery aneurysms (CAAs)	**risk factors**: Kawasaki disease (KD) and other vasculitides, atherosclerosis, trauma, connective tissue disorders, and male sex[5] **incidence**: 1.65% (range 0.3 - 5.3%)[5] **morbidity/mortality**: 5% of patients with myocardial infarct less than 40 years old have CAA with a history of KD[71] **morphology**: complex in shape, saccular, fusiform, or appear as a string of pearls[72,73]	**concern**: thrombosis[14] **treatment**: antiplatelet and anticoagulation therapies[14] **medical intervention**: antiplatelet therapies (z-score ≥ 2) and antiplatelet therapies and anticoagulation treatment if $D \geq 8$ mm or a z-score ≥ 10[14]	• coronary arteries translate with cardiac motion[74] • blood flow occurs during diastole[74] ➢ **case studies and series**[74,75], **cases compared to controls without aneurysm**[73] **or without sequala**[76,77]

Table S2: Modeling parameters and inputs commonly used in computational modeling of aneurysms[51]

common modeling parameters	common inputs	how commonly obtained
arterial geometry	3D model created from segmentation of medical images; common segmentation software[51] includes MIMICS, VMTK[78], ITK-SNAP, etc.	magnetic resonance imaging, computed tomography, ultrasound, etc.
numerical settings	software/numerical scheme dependent; common solvers include SimVascular[79], Crimson, ANSYS Fluent[80,81], ANSYS CFX[82], STAR CCM+[83], OpenFOAM[84], Oasis, FEBio[85], etc.	based on experience, from literature, derived during verification studies
inlet boundary condition(s)	flow waveform and profile[86,87]	literature, *in vivo* flow measurements[78], eg. phase contrast magnetic resonance imaging, catheter-based probes, ultrasound
outlet boundary condition(s)	pressure[51,86,87]	literature, *in vivo* flow measurements, eg. catheter-based probes[51]
	lumped parameter models[86,87] (e.g. Windkessel)	literature, morphometric relations (e.g. generalized Murray's law), *in vivo* flow measurements, iterative tuning
FSI material properties	wall thickness[51,88,89,90], elasticity[51]	literature, histology from resected tissue for wall thickness, biomechanical tests from resected tissue for elasticity
fluid properties	Newtonian, incompressibility, density, viscosity[91]	literature

Table S3: Hemodynamic parameters commonly used in computational modeling of aneurysm[92]

hemodynamic parameter	nomenclature/equation	common units	hemodynamic considerations
wall shear stress (WSS)	$\overrightarrow{\tau_w} = \mu \frac{\partial v}{\partial r}$, evaluated at the wall	Dynes/cm^2, Pa	measure of hemodynamic stress on vessel wall - related to thrombosis risk and endothelial function; low levels associated with proinflammatory and prothrombotic and high levels associated with vascular disease pathogenesis[93,94]
time-averaged wall shear stress (TAWSS)	$TAWSS = \frac{1}{t}\int_0^t \lvert WSS \rvert \, dt$	Dynes/cm^2, Pa	
wall shear stress gradient (WSSG)	$WSSG = \sqrt{\left(\left\lvert\frac{\partial \vec{\tau}_w}{\partial x}\right\rvert\right)^2 + \left(\left\lvert\frac{\partial \vec{\tau}_w}{\partial y}\right\rvert\right)^2 + \left(\left\lvert\frac{\partial \vec{\tau}_w}{\partial z}\right\rvert\right)^2}$	Dynes/cm^3, Pa/mm	magnitude of spatial gradient of WSS, positive WSSG (i.e. accelerating flow) associated with cerebral aneurysm growth and remodeling[93]
oscillatory shear index (OSI)	$OSI = 0.5\left(1 - \frac{\lvert \int_0^t \overrightarrow{WSS} dt \rvert}{\int_0^t \lvert \overrightarrow{WSS} \rvert dt}\right)$	dimensionless	measure of flow directionality and disturbed flow, associated with proinflammatory changes[93] minimum OSI = 0: unidirectional flow; maximum OSI = 0.5: equal flow in both directions[92]
residence time (RT) measures	quantifiable via Lagrangian or Eulerian methods[95]	s, 1/Pa, dimensionless	measure of flow stagnation, related to thrombosis and wall inflammation[95,96] there is no globally appropriate RT method; approach should be selected based on the context of the simulation and quantities of interest[95]

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