Table. Included studies characteristics and quality of studies scoring

Source	Country	Study design	Name of trial, database, and recruitment period	No. of patients	Surgical intervention	Outcomes of interest	Selection	Comparability	Outcome	Sum
Arya 2018[5]	United States	Retrospective	National veterans' health administration corporate data	152,010	Amputation	Amputation	3	2	3	8
Durham 2010[2]	United States	Retrospective	Single Institution	187	Femoropopliteal revascularization (open and endovascular)	Primary-assisted patency related to the initial treatment modality	3	0	3	6
Ferguson 2010[3]	United Kingdom	Retrospective	OPCS4	163	Lower limb amputation	Lower limb amputation rate	3	0	3	6
Hawkins 2019[6]	United States	Retrospective	VQI (Virginia Quality Initiative)	40,109	Infrainguinal bypass	Major adverse limb events	4	0	3	7
Henry 2011[4]	United States	Retrospective	NIS (Nationwide Inpatient Sample)	475,802	Lower extremity revascularization, major amputation	Major lower extremity amputation	4	1	3	8
Hughes 2019[7]	United States	Retrospective	NIS (National Inpatient Sample)	83,242	Major amputation, revascularization (open or endovascular)	Incidence of amputations and revascularizations, patient comorbid conditions, postoperative complications	3	1	3	7
McGinigle 2014[8]	United States	Retrospective	North Carolina Inpatient Discharge Database	222,920	Amputation	Number of major amputations	4	2	3	9
Minc 2020[9]	United States	Retrospective	West Virginia Health Care Authority Data	458,776	Amputation	Amputation rate	4	1	3	8
Rowe 2010[10]	United States	Retrospective	Nationwide Inpatient Sample	29,768	Bypass graft, amputation, endovascular	Use of amputation, revascularization, or endovascular	3	0	2	5
Tunis 1993[11]	United States	Prospective	Single Institution	7080	Amputation or revascularization	Amputation or revascularization	3	0	3	6
Ultee 2015[1]	Netherlands	Retrospective	Single Institution	324	Bypass, amputation	Amputation, mortality	3	2	3	8

Selection: Adequate case definition, representative of nonexposed cohort, ascertainment of exposure; demonstration of outcome was not present at the start of the study.
Comparability: Comparability of cohort based on the design or analysis controlled for confounders.
Outcome: Assessment of outcome, sufficient, and adequate follow-up.
Sum Mean: Sum of selection, comparability, and outcome score.
Risk of bias was defined as low (Newcastle-Ottawa Score [NOS] score ≥8), moderate (NOS score 6-7), and high (NOS score ≤5).
NOTE: All outcomes reported in these 11 studies were recorded for transparency and completeness. Some outcomes (eg, primary-assisted patency) may not be analyzed because of the insufficient number of studies for meta-analysis.

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Changed pathophysiology of thoracic aorta after aortic arch repair

The interesting paper by Tenorio et al[1] demonstrates as today possible the total endovascular aortic arch repair, although entailing hemodynamic changes in this vessel. In fact, rigid endoprosthesis implants cause complete loss of elasticity of the corresponding vascular, also if residual to a pre-existing pathology, typically atherosclerotic.[2] It follows that a grafted aortic arch becomes unable to accumulate energy during systole and give it back in diastole, through the "windkessel" phenomenon. Reflection waves, generated inside rigid walls, differ from the physiological ones by having a decreased energy and different synchronism with the normal systolic peaks of the aortic blood flow. In particular, their vectors, when incident with an angle >90° on the aortic stream, negatively impact on its dynamics: this typically occurs in the descending part of the aortic arch. Moreover, the new reflection waves miss their diastolic component, occurring after the aortic valve closure, greatly reducing the coronary perfusion. The aortic blood flow, a non-Newtonian fluid, through a high Reynolds number, can change from a laminar to a turbulent flow, wasting more energy and becoming a possible mechanical factor of endoleak, mainly of type 1b.[3] These hemodynamic factors translate in an increased afterload to the left ventricle, in the long-term remodeling through a thickened parietal wall, that can lead to a discrepant coronary

blood inflow.[4,5] Cleary, also an intrinsic aortic pathology can cause similar adverse effects, but often with a slower progression and a delayed incidence. On the contrary, these disadvantages become clinically more evident when added one to another in quick succession, such as after an open- or endovascular procedure, also reducing the rheological advantages obtained by correcting an abnormal morphology of the corresponding aortic tract. Considering all these characteristics, we identified a particular "stiff aorta cardiomyopathy," to be evaluated when planning an extended grafting in the thoracic aorta and requiring a careful intra- and postoperative monitoring and follow-up in each patient. In respect to open surgical procedures, the endovascular treatments avoid the drawbacks of the cardiopulmonary bypass, which would add to those of a suddenly changed aortic arch hemodynamics. In perspective, this newly emergent pathology merits to be further investigated in its multifaceted features, through morphological and functional researches.

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