



An Up-to-Date of Popliteal Artery Anatomy, Static and Dynamic

17

Antonino Cavallaro

The treatment of popliteal artery (PA) disease with stenting or stent-grafting procedures has renewed the interest in the definition of its anatomic features at rest and, more compelling and intriguing, during knee movements, as the popliteal region is a very dynamic anatomic segment [1]. The main problem related to endovascular devices applied to PA is represented by the close relationship with the knee joint [2–4]. PA is susceptible to simultaneous forces of flexion, extension, and rotation [5]. Ageing and atherosclerosis cause elongation and loss of elasticity of PA, which may consequently kink upon flexion of the knee [6, 7].

The PA is conventionally divided into three segments:

1. From its origin, at the adductor hiatus, to the origin of the superior genicular arteries.
2. From the origin of the superior genicular arteries to the origin of the inferior genicular arteries.
3. From the origin of the inferior genicular arteries to the terminal branching.

However, this classic division may be modified in relation with the response of the artery to the mechanical stress caused by knee move-

ments. In particular, the origin of the popliteal segment of the arterial tree may be placed slightly more cranially, at the take-off of the descending genicular artery (arteria anastomotica magna, the branch of the superficial femoral artery directly contributing to the knee arterial network). This, because the artery, on dynamic imaging studies, looks relatively fixed at the origin of the descending genicular artery and at the origin of the anterior tibial artery [8–10].

Closely referring to the challenges of endografting, Kropman et al. [5] consider the following three segments:

- P1: Between the adductor hiatus and the top of the knee cap
- P2: Until the split of the knee articulation
- P3: Until the division into the crural arteries

These Authors stress the fact that the bending point of the knee is located between segments P1 and P2.

As for stent grafting for aneurysmal disease, Kolvenbach and Pinter [11] make a distinction between above-knee and below-knee aneurysms, as flexing movements of the knee joint appear responsible for early graft thrombosis in most cases.

Normally, the diameter of PA ranges between 5 and 9 mm [12–14].

Relying on US measurements, Wolf et al. [15] report a diameter of 6.0 ± 0.8 mm in women and

A. Cavallaro (✉)

Past Professor of General Surgery, “Sapienza” University, Rome, Italy

of 6.8 ± 0.8 mm in men. They observed a larger diameter in the proximal and middle portions of the artery and a difference of 2 mm or more between the proximal and distal portions. According to these Authors and others [16] the PA diameter is related with age, gender and body surface or body mass index.

Ebaugh et al. [17] determined, through computerized planimetry based on CT scan, the diameter of PA at the adductor hiatus and at the level of femoral condyles (proximal and middle segments) in 104 limbs of 52 patients with abdominal aortic aneurysm (considered at risk for PA aneurysm) and in 18 limbs of nine patients, matched for sex and age, without aortic aneurysm (evaluated for deep vein thrombosis). They found a significantly larger PA diameter in the former group and that, in the subjects at risk, the middle segment was larger than the proximal one; as a consequence, they hypothesized a dilating diathesis in subjects at risk for PA aneurysms, with a predisposition of PA to enlarge in its mid-portion; in contrast with Sandgren et al. [18], who excluded a generalized dilating diathesis in patients with abdominal aortic aneurysms as far as the arteries of lower extremity are concerned.

The first to study *in vivo* the effects of knee flexion on the morphology of PA were Browse et al. [19] from St. Thomas' Hospital, in London, in 1979. They performed experiments on greyhounds, but the more interesting data derived from the arteriographic investigation on 10 male subjects, aged 45–65 years: six had normal vessels, in four the lower limb arteries showed mild signs of atherosclerosis. The popliteal artery was measured, in the lateral projection, with straight leg, between the origins of two identifiable branches (the lower being the anterior tibial artery) and the measurement was repeated with the knee flexed at 55 – 140° (the fully flexed knee implying an angle of 30 – 45°). They observed a shortening of the artery of 0.11 – 0.36% per degree of flexion. For example, in one case, a 90° flexion determined the change of the arterial length from 269 to 227 mm. No kinking or tortuosity was apparent in the anteroposterior projection; as well, no change in the arterial diameter was observed; however, crinkling of the internal sur-

face was observed on full bending. Looking at the arteriographic images in their paper, it is evident that some angles, opened either anteriorly or posteriorly, appear along the artery course when the knee is flexed.

Vernon et al. [20] studied in six limbs, post-mortem, through arteriography, the modifications of the popliteal axis during knee flexion, and observed, in the sagittal plane, marked flexures in the upper part of the artery and a smooth curve in the lower part. These modifications took place between two fixed points: cranially the origin of the descending genicular artery and distally the origin of the anterior tibial artery.

Availability of MR angiography facilitated the study of the problem. Wensing et al. [9] studied 22 healthy volunteers, dividing them into four age groups (20–30 years; 31–45 years; 45–60 years; >61 years). Two basic considerations come from their work:

1. When the arterial axis leaves the adductor canal, the environment changes radically, from the firm support of the muscles of the thigh to the soft fatty tissue of the popliteal fossa.
2. An excess of length of the artery is the consequence of knee flexion; this is compensated in part by the longitudinal elasticity of the artery but contrasted by the hemodynamic force of blood flow, resulting an extra length which produces tortuosity and eventually kinking; these phenomena are enhanced by atherosclerosis, which implies elongation and loss of elasticity of the affected vessel. The Authors used three-dimensional reconstructions to investigate tortuosity (i.e., the series of turns and twists between two points of the artery) and observed, when the knee is flexed, a concertina-like effect both in the adductor canal and in the popliteal fossa. This effect was different in younger and older subjects: in the latter, there was a trend to form more marked and sharper curves and as well to concentrate the compensatory mechanism in the popliteal fossa. This aspect of compensation in the elderly could derive from the fibrosis of perivascular tissue in the adductor canal, due

to ageing [21, 22], which obstacles the easy gliding of the artery; the artery, as a consequence, would appear fixed at the level of the adductor canal. Moreover, in elderly subjects, curves in arterial course following knee flexion persisted after knee extension.

Similar results were reported by Avisse et al. [8], who studied by arteriography and MRI the lower limbs of five non-atherosclerotic volunteers and put into evidence that, with increasing flexion of the knee, the popliteal artery becomes increasingly tortuous in the supra-articular portion while the distal portion assumes an even curvature that retracts the artery from the posterior surface of the tibia.

The movement of the artery away from the tibia during knee flexion was investigated by Shiomi et al. [10] by MRI, in relation to orthopedic procedures: in 15 volunteers, they observed at the level of knee joint and 15 mm below, an increasing distance of the artery from the tibia with progressive knee flexion.

Color flow Doppler sonography was used by Shetty et al. [23] to study the variations of the course of PA during knee flexion, aiming to prevent vascular complications during the procedures of total knee replacement or high tibial osteotomy: the study involved the lower limbs of 25 men and 25 women (aged 21–96 years, mean 56.4 years) and the findings were supported by MR imaging, arteriography and cadaver dissections. The results were consistent with an increase of the distance of the artery from the tibia, when the knee was flexed at 90°, in most but not in all cases; variations in the branching of the artery and in the shape and volume of the popliteus muscle were supposed to be probably the cause of the lack of uniformity of response to the maneuver.

Diaz et al. [24] used dynamic angiography to study the behavior of 63 PAs (in 57 patients with arterial disease) during knee movements: a static phase (knee bent at 100°) was followed by passive full extension of the knee. In all cases but one they observed the formation of one main and most acutely angled curvature hinged on a point (hinge point) that was never located at the level of

the knee joint. The hinge point corresponded to the medial supracondylar tubercle and, in the horizontal plane with the knee extended, to the upper margin of the patella. Accessory flexions were observed in 46 arteries (73%) and were significantly associated with high blood pressure.

An accurate postmortem study on the biomechanical forces involving the femoropopliteal arterial segment was published in 2005 by Smouse et al. [25] aiming to disclose the modifications of the arteries before and after implantation of stents and to explain the eventual adverse event of stent fracture. Fourteen limbs (seven dead bodies) were submitted to angiography in the neutral position (straight leg) and during hip and/or knee flexion to simulate walking, stair climbing and sitting-to-standing. The study was performed on native arteries and after insertion of nitinol stents into the superficial femoral artery and the PA. Knee flexion caused axial compression and bending of the artery in the popliteal fossa, behind the knee; no elongation, tortuosity, or twisting were observed. According to the Authors, longitudinal elasticity accounted for foreshortening of the vessel and, once reached the maximum effect, bending and curves allowed to complete accommodation of the artery to the reduced distance due to flexion; increasing rigidity of the artery would reduce axial compression and enhance bending; rigidity is increased in the stented segments (particularly in zones of stent overlapping) and this may produce several curvatures instead of the smooth C-shaped curve of the normal artery. The different rigidity between stented and non-stented arterial segments may produce kinking at the ends of the former.

Klein et al. [26] studied in vivo the conformational changes of the femoropopliteal artery during movement. Ten arteries of nine patients aged 57 ± 10.2 years (most of them recently submitted to PTA but without occlusive disease) were investigated through a methodology which created three-dimensional models of the popliteal artery based on acquisition of images during routine peripheral angiography; this allowed quantitative assessment of changes in length, curvature, torsion, twist angle, and new flexion angles ($>15^\circ$) when passing from the straight leg position to the

crossed leg position. They observed that the knee movement necessary to cross the leg under evaluation over the contralateral limb implied the following statistically significant changes: shortening of about 30%, increase of the main curvature and torsion, appearance of 1–5 (mean 2.4) new flexion angles, appearance of a twist angle of 3.46 ± 1.61 degrees/cm.

All the above data, albeit partial and retrieved from limited experiences, witness the tremendous mechanical stress to which the PA is submitted by the movements of the knee. Increase of arterial rigidity, due to spontaneous disease such as atherosclerosis or aneurysm or to stenting, intuitively would exaggerate the changes in morphology and course.

A further insight into these phenomena may derive from computer studies on simulation of axial arterial length during locomotion and other daily activities [27].

Shortening, changes in the course and eventual tortuosity of PA during knee movements have been studied with different aims: the localization of occlusive atherosclerosis [19], the prevention of arterial damage during knee surgery [10, 23, 28], the choice of the more convenient stent-grafting technique [17, 25, 26, 29, 30]. The eventual influence on aneurysm formation remains obscure and a matter of speculation; the iterative bending and tortuosity could determine turbulence, which is considered one of the concurrent causes of aneurysmal dilatation [31].

References

- Kudelko PE II, Alfaro-Franco C, Dietrich EB, Krajcer Z. Successful endoluminal repair of a popliteal aneurysm using the Wallgraft endoprosthesis. *J Endovasc Surg.* 1998;5:373–7.
- Henry M, Amor M, Henry J, Klonaris C, Tzvetanov K, Buniet JM, Amicabile C, Drawin T. Percutaneous endovascular treatment of peripheral aneurysms. *J Cardiovasc Surg.* 2000;41:871–83.
- Henke P. Popliteal artery aneurysms: tried, true and new approaches to therapy. *Semin Vasc Surg.* 2005;18:224–30.
- Mousa AY, Beauford RB, Henderson P, Patel P, Faries PL, Flores L, Fogler R. Update on the diagnosis and management of popliteal aneurysm and literature review. *Vascular.* 2006;4:103–8.
- Kropman RHJ, De Vries JPPM, Moll FL. Surgical and endovascular treatment of atherosclerotic popliteal artery aneurysms. *J Cardiovasc Surg.* 2007;48:281–8.
- Ohrlander T, Holst J, Malina M. Emergency intervention for thrombosed popliteal aneurysm: can the limb be salvaged? *J Cardiovasc Surg.* 2007;48:289–97.
- Cina CS, Moore R, Maggisano R, Kucey D, Dueck A, Rapanos T. Endovascular repair of popliteal artery aneurysms with Anaconda limbs: technique and early results. *Catheter Cardiovasc Interv.* 2008;72:716–24.
- Avisse C, Marcus C, Ouedragogo T, Delattre JF, Menanteau B, Flament JB. Anatomic-radiological study of the popliteal artery during knee flexion. *Surg Radiol Anat.* 1995;17:255–62.
- Wensing PJW, Scholten FG, Buijs PC, Hartkamp MJ, Mali WPTM, Hillen B. Arterial tortuosity in the femoropopliteal region during knee flexion: a magnetic resonance angiographic study. *J Anat.* 1995;186:133–9.
- Shiomi J, Takahashi T, Imazato S, Yamamoto H. Flexion of the knee increases the distance between the popliteal artery and the proximal tibia: MRI measurements in 15 volunteers. *Acta Orthop Scand.* 2001;72:626–8.
- Kolvenbach R, Pinter L. Stentgraft exclusion of asymptomatic popliteal aneurysms: medium term results. *Eur J Vasc Endovasc Surg Extra.* 2003;6:29–31.
- Davis RP, Neiman HL, Yao JS, Bergan JJ. Ultrasound scan in diagnosis of peripheral aneurysms. *Arch Surg.* 1977;112:55–8.
- Zierler RE, Zierler BK. Duplex sonography of lower extremity arteries. In: Zwiebel WJ, editor. *Introduction to vascular ultrasonography.* Philadelphia, PA: W.B. Saunders; 1983. p. 237–51.
- Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. Subcommittee on reporting standards for arterial aneurysms Ad hoc Committee on reporting standards Society for Vascular Surgery and North American Chapter of the International Society for Cardiovascular Surgery. *J Vasc Surg.* 1991;13:452–8.
- Wolf YG, Kobzantsev Z, Zelmanovich L. Size of normal and aneurysmal popliteal arteries: a duplex ultrasound study. *J Vasc Surg.* 2006;43:488–92.
- Sandgren T, Sonesson B, Ryden-Ahlgren A, Lanne T. Factors predicting the diameter of the popliteal artery in healthy humans. *J Vasc Surg.* 1998;28:284–9.
- Ebaugh JL, Matsumura JS, Morasch MD, Pearce WH, Nemcek AA, Yao JST. Morphometric analysis of the popliteal artery for endovascular treatment. *Vasc Endovasc Surg.* 2003;37:23–6.
- Sandgren T, Sonesson B, Ryden-Ahlgren A, Lanne T. Arterial dimensions in the lower extremity of patients with abdominal aortic aneurysm – no indication of a generalized dilating diathesis. *J Vasc Surg.* 2001;34:1079–84.
- Browse NL, Young AE, Lea TM. The effect of bending on canine and human arterial walls and blood flow. *Circ Res.* 1979;45:41–7.
- Vernon P, Delattre JF, Johnson EJ, Palot JP, Clément C. Dynamic modifications of the popliteal arterial

- axis in the sagittal plane during flexion of the knee. *Surg Radiol Anat.* 1987;9:37–41.
21. De Souza RR, Ferraz de Carvalho CA, Merluzzi Filho TJ, Andrade Vieira JA. Functional anatomy of the perivascular tissue in the adductor canal. *Gegenbaurs Morphol Jahrb.* 1984;130:733–8.
 22. De Oliveira F, Bragança de Vasconcellos Fontes R, da Silva Baptista J, Paganini Mayer W, de Campos Boldrini S, Aparecido Liberti E. The connective tissue of the adductor canal – a morphologic study in fetal and adult specimens. *J Anat.* 2009;214:388–95.
 23. Shetty AA, Tindall AJ, Qureshi F, Divekar M, Fernando KWK. The effect of knee flexion on the popliteal artery and its surgical significance. *J Bone Joint Surg.* 2003;85-B:218–22.
 24. Diaz JA, Villegas M, Tamashiro G, Micelli MH, Enterrios D, Balestrini A, Tamashiro A. Flexions of the popliteal artery: dynamic angiography. *J Invasive Cardiol.* 2004;16:712–5.
 25. Smouse R, Nikanorov A, LaFlash D. Biomechanical forces in the femoropopliteal arterial segment. *Endovasc Today.* 2005;60–6.
 26. Klein AJ, Chen SJ, Messenger JC, Hansgen AR, Plomondon ME, Carroll JD, Casserly IP. Quantitative assessment of the conformational change in the femoropopliteal artery with leg movement. *Catheter Cardiovasc Interv.* 2009;74:787–98.
 27. Young MD, Streicher MC, Beck RJ, van de Bogert AJ, Tajaddini A, Davis BL. Simulation of lower limb axial arterial length change during locomotion. *J Biomech.* 2012;45:1485–90.
 28. Yoo JH, Chang CB. The location of the popliteal artery in extension and 90 degree knee flexion measured on MRI. *Knee.* 2009;16:143–8.
 29. Jonker FHW, Schlosser FJW, Moll FL, Muhs BE. Dynamic forces in the FSA and popliteal artery during knee flexion. Consequences of stress to consider for stent durability and design. *Endovasc Today.* 2008;53–8.
 30. Tamashiro GA, Tamashiro A, Villegas MO, Dini AE, Mollon AP, Zelaya DA, Soledispa-Suarez CI, Diaz JA. Flexions of the popliteal artery: technical considerations of femoropopliteal stenting. *J Invasive Cardiol.* 2011;23:431–3.
 31. Nichols WV, O'Rourke MF. McDonald's blood flow in arteries. Theoretical, experimental and clinical principles. London: Arnold; 1998. p. 52.