8 Definition, Prevalence, and Etiopathogenesis

Antonino Cavallaro

According to the almost universally accepted suggestion by the Subcommittee on Reporting Standards for Arterial Aneurysms (from the Society for Vascular Surgery and the North American Chapter of the International Society for Cardiovascular Surgery [1]), an aneurysm is a "permanent localized (i.e. focal) dilation of an artery having at least a 50% increase in diameter compared to the expected normal diameter of the artery in question." The subcommittee accepted as normal value for popliteal artery (PA) a diameter of 0.90 ± 0.20 cm (measurement through B-mode ultrasound by Davis et al. [2]). Another proposed definition, more versatile, is "given the assumption that the arterial diameter proximal to dilation is normal, by common convention, an increase in diameter greater than 50% has been considered evidence of an aneurysm."

In the practical setting, many authors continue to follow what proposed by Szilagyi et al. [3], considering aneurysmatic a PA with a diameter ≥2 cm. McSweeney et al. [4], from Charing Cross Medical School, stated that "a ratio of ≥1.5 maximum popliteal fossa diameter/supragenicular diameter is likely to represent a true dilation of the artery in the popliteal fossa." Dawson et al. [5] consider a PA aneurysmatic when its external diameter exceeds 2 cm or 1.5 times the size of the normal artery.

8.1 A Rare Disease?

Textbooks of Pathology, in the early years of the twentieth century [6, 7], asserted that with the exception of the aortic aneurysms, popliteal artery aneurysms (PAAs) are found more frequently than those of any other artery.

In 1949, Linton [8] stated that the true incidence of atherosclerotic PAAs is probably not given in any available statistic. This assessment was repeatedly confirmed [9–11].

The causes of this defective knowledge depend on several factors:

- A number of PAAs are fully asymptomatic.
- Postmortem investigations rarely involve the popliteal space unless there are precise indications on the basis of symptomatic lesions or dedicated studies.
- Palpation of the popliteal fossa is not properly ease, and often, it is not performed as a routine. Theis [12], in 1937, wrote "as a rule, the popliteal space is carefully examined only when serious complications are produced by advanced stage of the aneurysm." This may be a little surprising because any hospital admission or first visit should include the appreciation of peripheral pulses; however, very

A. Cavallaro (✉)
Past Professor of General Surgery,
"Sapienza" University, Rome, Italy

A. Cavallaro (ed.), *Aneurysms of the popliteal artery*,
https://doi.org/10.1007/978-3-030-49687-6_8

frequently, if not constantly, at the lower limb level, only the femoral pulse and eventually the very distal pulses are investigated. This should not apply to angiologists and vascular surgeons, but the fact remains that palpation of the popliteal artery, even if correctly performed (Fig. 8.1), may be difficult and misleading, particularly in the obese patient.

An idea about the prevalence of PAAs, at least of those clinically relevant, may derive from the comparison with other more easily detectable aneurysms. In 1959, Crawford et al. [13] reported, during a 5-year period, 650 cases of aortic aneurysms and 54 cases of peripheral aneurysms: of these, 30 (55%) were popliteal. Flamand et al. [14] observed, during 11 years, 131 aortoiliac aneurysms and 41 peripheral aneurysms (in 31 patients), of which 28 (68.2%) were popliteal. MacSweeney et al. [4] registered, during the same period, 232 abdominal aortic aneurysms (AAAs) and 24 PAAs (of which only 11 were detected clinically). Bacciu et al. [15] observed, from 1976 through 1986, 206 AAAs and 24 atherosclerotic PAAs (in 15 patients). In men aged 65–79 years, the prevalence of AAA is reported in 5–10% [16]; in a similar age category, PAA is observed in 1% [17]. These and other observations led to the approximative evaluation [18] that PAAs represent 6–8% with respect to AAAs. Shortell et al. [19] report that, during a 25-year period, PAAs represented almost 7% of all aneurysms submitted to repair. Szilagyi et al. [3] report, in the period 1964–1971, a ratio 15:1 of AAAs vs PAAs; the ratio was 13:1 in the experience of Farina et al. [20] for the period 1972–1988.

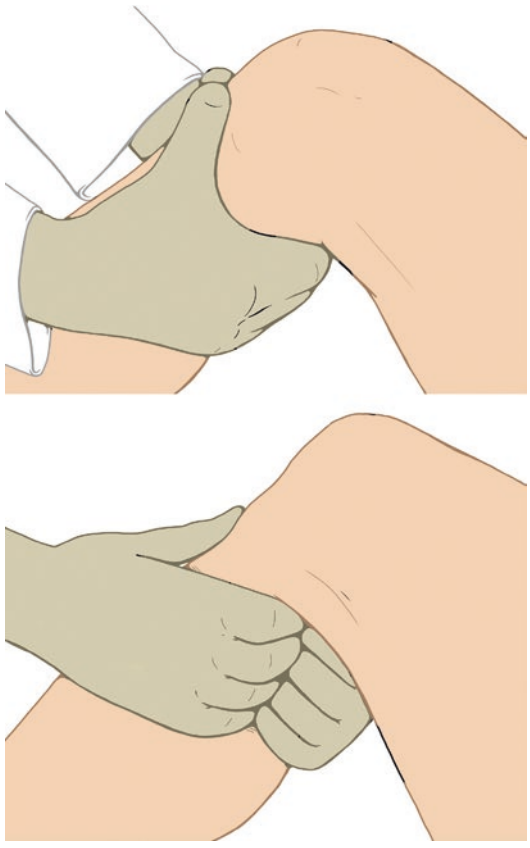

Fig. 8.1 Palpation of the popliteal pulse

The introduction of B-mode ultrasound, shortly after the introduction of duplex scan, would allow a more realistic evaluation of the problem. In 1981, Hirsch et al. [21] reported on 100 consecutive patients, suspected to have aortic or peripheral aneurysm, studied through B-mode ultrasound: they found 53 AAAs and 12 PAAs, of which nine were associated with aortic or femoral aneurysm. Batt et al. [22] found a yearly incidence of five to seven PAAs vs 20–25 AAAs.

As a consequence, the suggestion by Buxton et al. [23] that PAA would occur more frequently than generally thought was confirmed.

A further and deeper insight could derive, always approximatively, by some studies relying on series of patients investigated by duplex scan. Diwan et al. [24] studied 313 consecutive patients affected with AAA: of these, 24 (7.6%) presented also a PAA (for a total of 39 lesions); the authors specified that most of these aneurysms were not detectable clinically. Trickett et al. [17] screened 1076 male subjects aged 75–80 years, finding 11 patients (about 1%) with PAA. Morris-Stiff et al. [25], screening 449 subjects for AAA, extended duplex-scan evaluation to lower limb arteries: they did not find any PAA, but 39/898 (4.3%) popliteal arteries were dilated, with a diameter >1 cm. This finding could be significant: Magee et al.

[26], following up for 3.1 years 67 patients with mono- or bilateral ectasia of the PA, observed that within 2 years, seven of them progressed to aneurysm (diameter >2 cm); these cases presented a statistically significant association with extra-popliteal aneurysms.

In spite of a growing mass of information, the true prevalence of PAA remains speculative.

More than 50 years ago, Hunter et al. [10] concluded that apparently PAAs are less common than aneurysms of aortic bifurcation and more common than aneurysms within the chest and of the major arteries of limbs or neck.

The latter assessment was confirmed by several experiences. Abelleyra et al. [27] reported 31 peripheral aneurysms, of which 18 (58%) were popliteal. Hands and Collin [28] observed 25 femoral and 34 popliteal (57.6% of the total) aneurysms. Agrifoglio et al. [29] stated that, on the basis of the available literature, PAAs represent 62% of peripheral aneurysms. In contrast with this general consensus, Whitehouse et al. [30] reported, during a 40-year period, 88 PAAs and 172 femoral artery aneurysms, considering however that many PAAs are small and asymptomatic and that palpation of the groin is much easier and more reliable than that of the popliteal fossa.

Trying to define, if not the prevalence, at least the eventual increase in diagnosis (and consequently the improvement in treatment), we tried to tabulate the data from several series, applying a dividing watershed represented by the year 1985, when duplex-scan apparatuses became widely available. This was a milestone event, as awareness of the importance of the disease, due to the ominous sequelae observed in undiagnosed/misdiagnosed cases, was already established following the reports of Gifford et al. [31], Wychulis et al. [32], and Bouhoutsos and Martin [33].

In the first review (Table 8.1), with the fair exception of the Mayo Clinic (reporting a mean of more than 25 cases/year), the single experiences relied on 1.5–9.8 cases/year, with only two series approaching ten cases/year. In the second

Table 8.1 Series of atherosclerotic popliteal aneurysms collected (or largely collected) before 1985

Author, year	Study period	Patients	Aneurysms	Pts/year	Ans/year
Linton [8], 1949	1942–1947	14	15	2.8	3.0
Janes [34], 1951[a]	1940–1949	42	63	4.2	6.3
Gifford [31], 1953[a]	1913–1951	64	95	1.6	2.4
Lord [35], 1957	7 years	10	13	1.4	1.9
Crawford [13], 1959	5 years		30		6.0
Friesen [36], 1962[a,b]	1950–1960	64	100	5.8	9.1
Edmunds [37], 1965	1948–1963	80	96	5.0	6.0
Baird [38], 1966	1938–1964	36	51	1.3	1.9
Crichlow [39], 1966	1953–1965	42	60	3.2	4.6
Wychulis [32], 1970[a]	1961–1968	150	231	18.8	28.9
Bouhoutsos [33], 1974	1958–1972	71	102	4.7	6.8
Buda [40], 1974	1951–1972	59	81	2.7	3.7
Gaylis [41], 1974	15 years	38	49	2.5	3.3
Hardy [42], 1975	18 years	21	29	1.2	1.6
Buxton [23], 1975	1963–1974	23	34	1.9	2.8
Evans [43], 1976[c]	15 years	52	86	3.5	5.7
Towne [44], 1976	21 years	80	119	3.8	5.7
Tompkins [45], 1977	1968–1976	18	26	2.0	2.9
Chitwood [46], 1978	10 years	26	35	2.6	3.5
Inahara [47], 1978	1963–1977	30	44	2.0	2.9
Guvendik [18], 1980	1969–1976	20	27	2.5	3.4
Szilagyi [3], 1981	1964–1979	61	86	3.8	5.4

(continued)

Table 8.1 (continued)

Author, year	Study period	Patients	Aneurysms	Pts/year	Ans/year
Vermilion [48], 1981[c]	1960–1980	87	147	4.1	7.0
Laskar [49], 1982	1970–1981	27	32	2.3	2.7
Reilly [50], 1983	1958–1982	159	244	6.4	9.8
Whitehouse [30], 1983	1943–1982	61	88	1.5	2.2
Takolander [51], 1984	1971–1982	13	18	1.1	1.5
Downing [11], 1985	1960–1983	39	61	1.6	2.5
Salo [52], 1986	1960–1980	19	21	0.9	1.1
Mellière [53], 1986	1970–1983	50	73	3.6	5.2
Anton [54], 1986	1952–1974	56	73	2.4	3.2
Flamand [14], 1971	1975–1984	54	87	5.4	8.7
Raptis [55], 1986	1972–1983	36	61	3.0	5.1
Schellack [56], 1987	1965–1985	60	95	2.9	4.5
Englund [57], 1987	1968–1985	75	103	4.2	5.7
Lilly [58], 1988	1978–1987	35	59	3.5	5.9
Bacciu [15], 1988	1976–1986	15	24	1.4	2.2
Farina [20], 1989	1972–1988	33	47	1.9	2.8
Cole [59], 1989	1976–1987	38	59	3.2	4.9
Halliday [60], 1991	1982–1989	33	51	4.1	6.4
Shortell [19], 1991	1964–1990	39	51	1.4	1.9
Dawson [61], 1991	1958–1985	50	77	1.8	2.7
Roggo [62], 1993	1965–1991	162	247	6.0	9.1
Lowell [63], 1994[a]	1980–1985	106	161	17.6	26.8
Vettorello [64], 1996	1970–1994	26	37	1.0	1.5
Sarcina [65], 1997	1974–1994	58	69	2.9	3.4
Davidovic [66], 1998	36 years	53	64	1.5	1.8

In this table, as in the following one, we tried to give an idea about the experience of different authors in the field of atherosclerotic aneurysms of the popliteal artery, with exclusion of aneurysms of different etiology

Only atherosclerotic PAAs are dealt with in papers at refs. 8, 13, 15, 23, 30, 34, 38, 39, 43–47, 49, 51, 52, 55, 57, 61, and 63

Papers at refs. 3, 11, 18–20, 31–33, 35–37, 40–42, 53, 60, 62, and 66 include also a small number of non-atherosclerotic PAAs. However, it was easy to extract data pertaining only to atherosclerotic PAAs

Papers at refs. 48, 50, 54, 56, 58, 59, 64, and 65: not stated if all PAAs are atherosclerotic

[a]From the Mayo Clinic, Rochester, Minnesota: overlapping of study period of ref. 31 vs ref. 34; partial overlapping of ref. 36 vs ref. 31

[b]In effects, Friesen et al. report having seen 110 patients (i.e., 10 pts/year), but details are given only for patients receiving surgical treatment

[c]It is not clear if data of report at ref. 48 include data from report at ref. 43

review (Table 8.2), the Mayo Clinic remains the leading center, with more than 30 cases/year; however, eight centers reported an annual mean approaching or trespassing ten cases/year (one approaching 30 cases/year).

Given the characteristics of the various experiences, essentially clinical, the impression is that PAAs are diagnosed with increasing frequency, but this is the consequence of an increased attention in the search of contralateral asymptomatic PAA or of PAA in patients with AAA. Large screenings are still lacking, and therefore, the true prevalence of PAA remains unknown. Perhaps PAA is not really a rare disease, and, in any case, it has been a companion of surgeons during the centuries, so as to justify and share the statement by Laskar et al. [49]: "Il n'est pas étonnant que les premiers balbutiements de la chirurgie vasculaire se soient concentrés autour du diagnostic et du traitement des anévrismes poplités en raison de la fréquence de ces lésions."

Table 8.2 Series of atherosclerotic popliteal aneurysms collected (or largely collected) after 1985

Author, year	Study period	Patients	Aneurysms	Pts/year	Ans/year
Dawson [67], 1994[a]	1985–1992	27	41	3.4	5.1
Carpenter [68], 1994	1979–1992	33	54	2.4	3.9
Gawenda [69], 1995	1981–1994	39	58	2.9	4.3
Duffy [70], 1998	1987–1997	25	42	1.9	3.2
Taurino [71], 1998	1980–1995	23	28	1.5	1.9
Dijkstra [72], 1998	1984–1996	17	23	1.3	1.8
Locati [73], 1999	1982–1998		63		3,7
Gouny [74], 2000	1992–1997	35	52	7.0	10,4
Irace [75], 2001	1990–1999	45	75	4.5	7.5
Stiegler [76], 2002	1995–2000	46	65	7.7	10.8
Kauffman [77], 2002	1968–2000	112	175	3.4	5.3
Dorigo [78], 2002	1990–2000	89	109	8.1	9.9
Ascher [79], 2003	4 years	25	34	6.2	8.5
Bowrey [80], 2003	1988–2000	46	67	3.5	5.1
Harder [81], 2003	1997–2000	24	36	6.0	9.0
Laxdal [82], 2004	1974–2000	49	70	1.8	2.6
Aulivola [83], 2004	1992–2002	38	63	3.5	5.7
Martelli [84], 2004	1985–2000	38	56	2.4	3.5
Galland [85], 2005	1988–2004	73	116	4.3	6.8
Stone [86], 2005	1995–2004	46	55	4.6	5.5
Pulli [87], 2006	1984–2004	137	159	6.5	7.6
Beseth [88], 2006	1981–2003	35	43	1.5	1.9
Huang [89], 2007[b]	1985–2004	494	651	24.7	32.6
Davies [90], 2007	1988–2006	57	72	3.0	3.8
Curi [91], 2007	2000–2006	43	56	6.1	8.0
Lichtenfels [92], 2008	2000–2004	40	60	8.0	12.0
Dzieuciuchowicz [93], 2009	1995–2005	61	82	5.5	7.4
Zimmermann [94], 2010	2000–2007	46	63	5.6	7.9
Zaraca [95], 2010	1991–2009	35	49	1.8	2.6
Pulli [96], 2012	2005–2010	59	81	9.8	13.5
Kropman [97], 2014	1993–2011	218	368	11.5	19.4
Huang [98], 2014[b]	2005–2012	217	271	28.9	36.1
Serrano-Hernando [99], 2015	1993–2013	142	211	6.8	10.0
Mazzaccaro [100], 2015	1998–2011	65	94	4.6	6.7
Ronchey [101], 2015	2000–2013	67	101	4.8	7.2
Wagenhauser [102], 2015	1996–2013	30	50	1.7	2.8
Leake [103], 2016	2006–2014	156	247	17.3	27.4
Personal series	1981–2005	58	82	2.3	3.3

Papers at refs. 67, 69, 70, 72, 74, 77, and 80 deal only with atherosclerotic PAAs. In addition, personal series includes only atherosclerotic PAAs

Non-atherosclerotic PAAs are included in papers at ref. 73 (two cases of PA entrapment syndrome) and at ref. 83 (one case of Marfan syndrome); however, it was easy to extract data regarding only atherosclerotic PAAs

Paper at ref. 89: not all atherosclerotic PAAs. Out of 236 pathology findings: atherosclerosis 233; two fibromuscular dysplasia; one thromboangiitis obliterans

Papers at refs. 68, 71, 75, 76, 78, 79, 81, 82, 84–88, and 90–103: not stated if all PAAs are atherosclerotic

Papers at refs. 71–73, 78, 86–88, 90, 91, and 95 deal only with cases submitted to repair (either surgical or endovascular)

[a]Cases previously reported [61] are not included

[b]From the Mayo Clinic, Rochester, Minnesota

8.2 Etiopathogenesis

In the past, several factors were the object of speculation as responsible for PAA formation. Broca [104] took into consideration the abuse of alcohol and the prolonged use of mercurial drugs, but he thought that trauma and syphilis were the more frequent causes of the disease. Trauma could be acute, under the form of a contusion, but more often iterative and, in some way, inherent to daily activities. Home [105] observed a high incidence of PAA in coachmen, postilions, and horse-riding men (as cavalry soldiers), attributing the cause of chronic trauma not only to repeated strenuous movements but also to the superior border of rigid leather boots. Delbet [106] favored the traumatic etiology, observing that PAAs are more frequent in the laboring class, and also the predominant involvement of men was explained with the harder physical activity proper of male individuals. Syphilis was recognized as a frequent cause of aneurysms, but as for PAA, several doubts arose about its true role. Broca [104] observed that in colonized countries, syphilis was a diffuse disease but aneurysms were rare. Delbet [106] reported a similar and important incidence of syphilis in soldiers and sailors, but PAAs were more frequent in soldiers; contrarily, Erichsen [107] observed a particularly high incidence of PAA in sailors.

Shortly before World War II, Wells et al. [108] stated that atherosclerosis is frequently the only demonstrable disease of the aneurysm wall and that trauma or strain may precipitate the dilatation and, in particular, give rise to symptoms. Linton [8] reported on 42 patients with PAA observed at the Massachusetts General Hospital in Boston during the period 1908–1947: in 35 of them, atherosclerosis was identified as the etiological factor (even if four patients were luetic); moreover, he stressed the fact that syphilis was never recognized as the etiological factor in the 25 aneurysms collected from 1938. The facts are that atherosclerotic lesions are observed also in post-stenotic aneurysms [20] and that atherosclerotic involvement per se cannot explain the predilection of aneurysmal disease for the popliteal artery.

The particular anatomic situation of the popliteal artery is considered relevant to the formation of aneurysm in this site. In 1957, Lord [35] textually wrote: "the popliteal artery is adaptable, in that it may change from a straight line course to one forming an angle of 45 degrees when the leg is fully flexed on the thigh. This change is no trick for the young, healthy, flexible vessel, but it is less readily tolerated by an artery that is more rigid and less elastic due to the presence of atheromatous plaques and deposits of Calcium in the wall...Unquestionably, frequent bending is a factor favoring the development of aneurysms in this particular vessel…."

The importance of repeated trauma from flexion/extension of the knee had been already stressed by Boyd et al. [109] and by Lindbom [110].

An attractive etiopathogenetic theory is that PAA may represent a post-stenotic arterial dilatation. A clinical support to this hypothesis relies on the not rarely observed post-stenotic aneurysms in subjects affected with popliteal artery entrapment. The phenomenon is largely recognized both clinically and experimentally. Subclavian artery aneurysms due to anatomical variants in the thoracic outlet were known in the nineteenth century [111]; in 1916, Halsted and Reid [112] produced a circumscribed dilation of an artery downstream a partially occluding band. In 1954, Holman [113] thoroughly studied in the laboratory the phenomenon of post-stenotic dilatation (Fig. 8.2) and concluded:

> "a mass of fluid ejected through a narrow and limited constriction under high velocity strikes against a more slowly moving mass of fluid distal to the stenosis, resulting, first, in the conversion of high kinetic energy into high potential energy or lateral pressure and, second, in the lateral deflection of the rapid stream and even in a complete reversal in the direction of flow, thus producing eddies of alternating high and low pressure whose repeated impacts over prolonged periods against an elastic wall are capable of inducing structural fatigue and distention of the wall, resulting eventually and inevitably in the phenomenon of poststenotic dilatation."

Parietal vibrations of post-stenotic aneurysms were later studied by Simkins and Stehbens [114].

Several zones of possible narrowing of the arterial line, able to produce a post-stenotic dila-

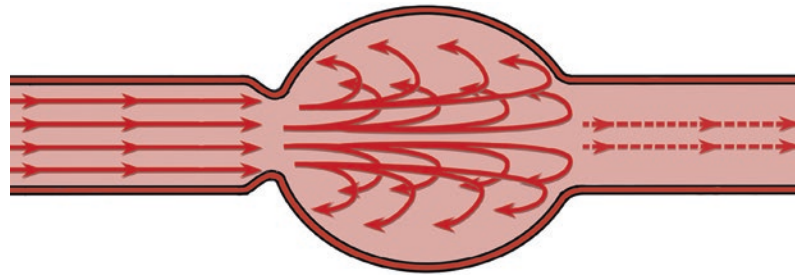

Fig. 8.2 The post-stenotic dilation. Orthograde flow is not represented within the dilated segment, to enhance the effect of the lateral and retrograde deflection of stream lines. (From Holman [113], with permission)

tion of the popliteal artery at different levels, have been described (outside the anatomical variants giving rise to popliteal artery entrapment). The adductor hiatus is probably the more largely known. At this site, which is rather rigid, continuing microtraumas due to the systolic expansion of the vessel wall could be responsible for atherosclerotic lesions [115], giving rise to anatomical and functional narrowing of the vessel. Already in 1946, Lilly [116] had stressed the concept of arterial narrowing by atheromata at the distal end of Hunter's canal; Friesen et al. [36] suggested that PAAs soon distal to the adductor hiatus may well be post-stenotic in origin. But the entire adductor canal, where the superficial femoral artery is pressed against the femur by the repeated action of the adductor muscles, may represent a zone of functional stenosis.

In 1949, Boyd et al. [109] described a fibrous tunnel derived from the fascia of the deep surface of the gastrocnemius; the popliteal artery enters this tunnel after coursing freely mobile through the loose fatty tissue of the popliteal space. The fascial covering of the deep surface of the gastrocnemius would form a definite band, broad 0.25–0.50 in., attached to the capsule of the knee joint and crossing posteriorly the popliteal artery.

In 1961, Gedge et al. [117] highlighted the importance of the arcuate popliteal ligament (Fig. 8.3) in the genesis of aneurysms of the distal popliteal artery: the ligament arches upward from the head of the fibula and the lateral side of the popliteus muscle, crossing this and blending into the posterior ligaments of the knee joint. This fibrous structure, which crosses anteriorly the popliteal artery, becomes particularly sharp and prominent when the leg is fully extended.

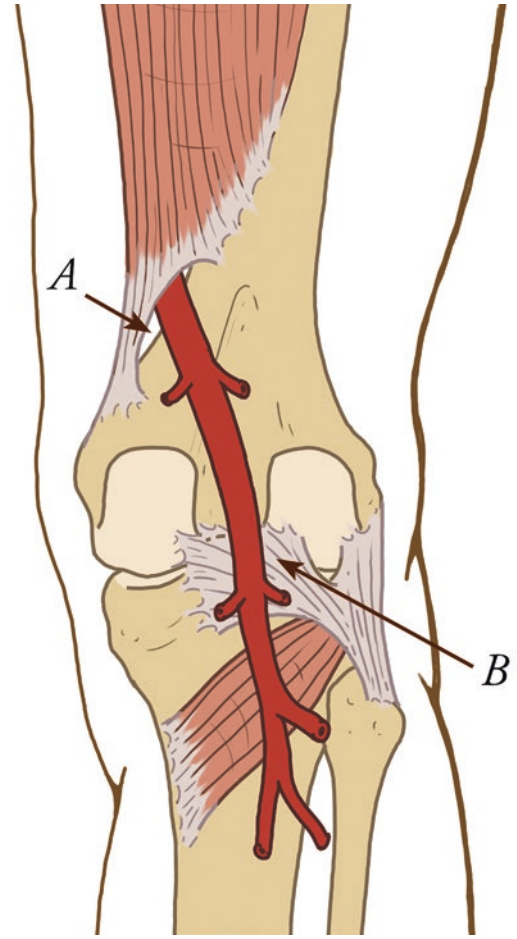

Fig. 8.3 Schematic representation of two potential sites of PA stenosis. (A) The adductor hiatus (dilation of the proximal PA). (B) The arcuate popliteal ligament (dilation of the distal PA). (From Gedge et al. [117], modified, with permission)

Stenosis may play a particular role when localized at a bifurcation of the arterial tree. Guvendik et al. [18] observed that both abdominal aortic

and popliteal aneurysms occur upstream a bifurcation; if atherosclerosis causes narrowing of one or both limbs, this will greatly enhance the reflection of pressure waves from the bifurcation, and considerable fluctuations will occur from the meeting between orthograde and reflected waves.

One of the characteristics of PAAs is the frequent association with aneurysms in other sites, leading to the observation that "popliteal aneurysms caused by atherosclerosis are only single manifestations of a generalized progressive disease." This formal assessment, made by Friesen et al. [36] in 1962, was stressed as a basic concept by Bouhoutsos and Martin [33] in 1974. Towne et al. [44] observed that patients with PAA have an inherent tendency for aneurysmal degeneration. Dawson et al. [61] followed up for 15 years a group of 50 patients with PAA: 16 (32%) developed 23 new aneurysms (six thoracoabdominal, 11 femoral, six popliteal contralateral). Cole et al. [59] asserted that the presence of a popliteal aneurysm indicates high probability of another aneurysm either in the contralateral limb or elsewhere.

The exceptional progress in the basic sciences during the last decades has generated a series of cooperative studies between clinicians and scientists aiming to define the origin of arterial aneurysms and particularly of those which are more frequent and more frequently associated, i.e., abdominal aortic and popliteal aneurysms. The research is still ongoing, but probably at the end (if any), the role of atherosclerosis will be redefined. Lindeman et al. [118], in 2008, could assert that abdominal aortic aneurysm (AAA) is a general inflammatory condition characterized by enhanced expression and activation of pro-inflammatory transcription factors accompanied by IL-6 and IL-8 hyperexpression and exaggerated downstream cellular responses (differently from atherosclerosis).

Pioneer papers appeared in the literature between 1980 and 1990: Busuttil et al. [119] and Menashi et al. [120] observed the increase of elastase and collagenase activities in the wall of AAA; other authors [121–123] stressed the importance of genetic factors, given the preponderance of involvement of the male sex, particularly striking for PAA. The hypothesis of Ward [124] was that of a systemic abnormality.

MacSweeney et al. [4] studied a series of 232 patients with AAA, 24 of them being also affected with PAA. Genotyping for apolipoprotein β and type III collagen did not yield any characterization for patients having also PAA. Fibrillin-1 genotyping yielded a significant difference between 128 patients with AAA and 24 patients with AAA and PAA.

An important research was performed by Sandgren et al. [125] aimed to ascertain if there is a dilating diathesis involving the peripheral arteries of patients with AAA, starting from the hypothesis that AAA is not only a localized vascular disease but also is associated with altered mechanical properties and dilatation of distant arteries [126] and with modifications of arteriolar resistance [127]. The study group comprised 183 consecutive patients waiting for AAA repair. The common femoral artery was measured in 175 subjects (151 male) and the PA in 109 subjects (95 male). Aneurysmal disease was found in eight common femoral and four popliteal arteries. Excluding these 12 patients, no dilating diathesis could be demonstrated in the examined arteries.

Starting from PAAs, the condition of generalized dilating diathesis, as hypothesized by several authors, was confirmed by Widmer et al. [128]: 33 patients undergoing repair of a PAA during the period 1996–2000 were submitted to ultrasound measurement of infrarenal abdominal aorta and common iliac, common femoral, and contralateral popliteal arteries, finding respectively the following:

- Dilation: 45.5%; 51.5%; 81.2%; 21.2%
- Aneurysm: 34.2%; 34.8%; 10.6%; 45.5%

Moreover, patients with multiple aneurysms showed also significantly larger diameters of the brachial and external iliac arteries.

Jacobs et al. [129–131]stressed the importance of apoptosis in the wall of PAAs, emphasizing the role of inflammation; they observed a large number of cells, predominantly T lymphocytes, expressing death-promoting molecules. Loss of vascular smooth muscle cells and disruption of

elastic lamellae were the more evident signs of architectural derangement in the wall of PAAs and of other peripheral aneurysms.

Abdul-Hussien et al. [132] demonstrated, both in AAA and PAA: marked activation of nuclear factor kβ and activated protein 1 proinflammatory transcription factors; hyperexpression of IL-6 and IL-8 on the cellular level; profuse infiltration of macrophages, neutrophils, and T-helper cells; and increased matrix metalloproteinases 8 and 9. According to their findings, genetic and epidemiologic association between AAA and PAA suggest a common origin.

That PA may behave as AA has been suggested in 2004 by Debasso et al. [133], who studied the mechanical properties of the walls of PA in healthy subjects; they observed, with increasing age, the following modifications, particularly evident in male subjects: increase in diameter, increase in stiffness, increase of intima-media thickness, and decrease of distensibility. All these findings suggested a behavior typical of a central elastic artery and not of a true muscular artery, implying susceptibility to dilation and aneurysm formation.

Recently, Hurks et al. [134], from Utrecht, published a detailed study of 38 PAAs (in 36 patients) and 198 AAAs. They found similar wall degradation for elastin disruption and smooth muscle cell decrease; however, the focus of inflammation was the intima in PAAs and the adventitia in AAAs. Cholesterol core presence was more pronounced in AAAs, while iron deposits were more frequent in PAAs, suggesting previous intramural hemorrhages possibly attributable to trauma. Similar levels of MMP-9 were observed in the two types of aneurysm, while PAAs were characterized by higher levels of MMP-2, TNF-α, TNF-β, and interferon-γ. The conclusion was that AAAs have a pathophysiologic mechanism more closely related to atherosclerosis than PAAs.

A still ongoing research at the 1st Dept. of Surgery of Rome University "Sapienza" [135] is focusing attention on the levels of metalloproteinases MMP-2 and MMP-9 and of their specific inhibitors (TIMP-1 and TIMP-2) in the wall of resected PAAs, both by reverse transcription-polymerase chain reaction (RT-PCR) method and immunohistochemistry. Up to now, specimens have been divided into three groups:

- 11 isolated atherosclerotic aneurysms
- Eight atherosclerotic aneurysms associated with AAA
- Six post-entrapment aneurysms

In group 2, levels of MMPs were significantly higher, and levels of TIMPs were significantly lower. No difference in MMP and TIMP gene expression was observed between groups 1 and 3. Immunohistochemistry confirmed the results of RT-PCR. These preliminary results would suggest that single PAAs and post-entrapment PAAs may share the same origin, different from the one triggering the formation of multiple aneurysms.

The studies aiming to elucidate the origin of PAAs and of arterial aneurysm in general are currently flourishing, but we are still in the dawn of this fascinating research.

Finally, it must not be forgotten that sites of arterial fusion during embryogenesis could present some kind of structural weakness, which could be prone to aneurysm formation [136], and this could be true in some cases of PAA [137].

References

1. Johnston KW, Rutherford RB, Tilson D, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. J Vasc Surg. 1991;13:444–50.
2. Davis RP, Neiman HL, Yao JST, Bergan JJ. Ultrasound scan in diagnosis of peripheral aneurysms. Arch Surg. 1977;112:55–8.
3. Szilagyi DE, Schwartz RL, Reddy JD. Popliteal arterial aneurysms. Their natural history and management. Arch Surg. 1981;116:724–8.
4. MacSweeney STR, Skidmore C, Turner RJ, Sian M, Brown L, Henney AM, Greenhalgh RM, Powell JT. Unravelling the familial tendency to aneurysmal disease: popliteal aneurysms, hypertension and fibrillin genotype. Eur J Vasc Surg. 1996;12:162–6.
5. Dawson I, Sie RB, van Bockel JH. Atherosclerotic popliteal aneurysm. Br J Surg. 1997;84:293–9.
6. Adami JC, Nicholls RC. Principles of pathology, vol. 2. Philadelphia: Lea & Febiger; 1909. p. 200.
7. Kaufmann E. Pathology, vol. 1. Philadelphia: P. Blackington's Sons & Co.; 1929. p. 32.

8. Linton RR. The arteriosclerotic popliteal aneurysm: a report of fourteen patients treated by a preliminary lumbar sympathetic ganglionectomy and aneurysmectomy. Surgery. 1949;25:41–58.
9. Julian OC, Dye WS, Javid H, Grove WG. The use of vessel grafts in the treatment of popliteal aneurysms. Surgery. 1955;38:970–80.
10. Hunter JA, Julian OC, Javid H, Dye WS. Arteriosclerotic aneurysms of the popliteal artery. J Cardiovasc Surg. 1962;2:404–13.
11. Downing R, Ashton F, Grimley RP, Slaney G. Problems in diagnosis of popliteal aneurysms. J R Soc Med. 1985;78:440–4.
12. Theis FV. Popliteal aneurysms as a cause of peripheral circulatory disease: with special study of oscillomographs as an aid to diagnosis. Surgery. 1937;2:327–42.
13. Crawford ES, DeBakey ME. Surgical considerations of peripheral arterial aneurysms. Arch Surg. 1959;78:226–38.
14. Flamand JP, Goldstein M, Belenger J, van der Stricht J. Les anévrismes artériels périphériques. Acta Chir Belg. 1971;70:463–71.
15. Bacciu PP, Chiroini S, Noya G, Marongiu G, Gherli T, Cossu ML, Guazzaroni M, Dettori G. L'aneurisma dell'arteria poplitea. Diagnosi e terapia. Minerva Chir. 1988;43:1549–54.
16. Cosford PA, Leng GC, Thomas J. Screening for abdominal aortic aneurysm. Cochrane Database Syst Rev. 2007;2:CD002945.
17. Trickett JP, Scott RA, Tilney HS. Screening and management of asymptomatic popliteal aneurysms. J Med Screen. 2002;9:92–3.
18. Guvendik L, Bloor K, Charlesworth D. Popliteal aneurysm: sinister harbinger of sudden catastrophe. Br J Surg. 1980;67:294–6.
19. Shortell CK, De Weese JA, Ouriel K, Green RM. Popliteal artery aneurysms: a 25-year surgical experience. J Vasc Surg. 1991;14:771–9.
20. Farina C, Cavallaro A, Schultz RD, Feldhaus RJ, di Marzo L. Popliteal aneurysms. Surg Gynec Obstet. 1989;169:7–13.
21. Hirsch JH, Thiele BL, Carter SS, Colacurcio C. Aortic and lower extremity arterial aneurysms. J Clin Ultrasound. 1981;9:29–31.
22. Batt M, Scotti L, Gagliardi JM, Cassar JP, Porcher G, Le Bas P. Les anévrysmes poplités. Notre expérience à propos de 119 cas. J Chir. 1985;132:319–25.
23. Buxton B, Morris P, Johnson N, Royle J. The management of popliteal aneurysms. Med J Aust. 1975;2:82–5.
24. Diwan A, Sarkar R, Stanley JC, Zelenock JB, Wakefield TW. Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. J Vasc Surg. 2000;31:863–9.
25. Morris-Stiff G, Haynes M, Ogunbiyi S, Townsend E, Shetty S, Winter RK, Lewis MH. Is assessment of popliteal artery diameter in patients undergoing screening for abdominal aortic aneurysms a worthwhile procedure. Eur J Vasc Endovasc Surg. 2005;30:71–4.
26. Magee R, Quigley F, McCann M, Buttner P, Golledge J. Growth and risk factors for expansion of dilated popliteal arteries. Eur J Vasc Endovasc Surg. 2010;39:606–11.
27. Abelleyra J, Oglietti J, Solian J, Muzzio S. Resultados del tratamiento quirurgico de los aneurismas arteriales periféricos. Pren Méd Argent. 1976;63:286–90.
28. Hands LJ, Collin J. Infrainguinal aneurysms: outcome for patient and limb. Br J Surg. 1991;78:996–8.
29. Agrifoglio G, Papacharalambus D. Gli aneurismi arteriosi periferici. Minerva Cardioangiol. 1976;24:342–51.
30. Whitehouse WM, Wakefield TW, Graham LM, Kazmers A, Zelenock GB, Dent TL, Lindenauer SM, Stanley JC. Limb threatening potential of arteriosclerotic popliteal artery aneurysms. Surgery. 1983;93:694–9.
31. Gifford RW Jr, Hines EA Jr, Janes JM. An analysis and follow-up of 100 popliteal aneurysms. Surgery. 1953;33:284–93.
32. Wychulis AR, Spittell JA, Wallace RB. Popliteal aneurysms. Surgery. 1970;68:942–52.
33. Bouhoutsos J, Martin P. Popliteal aneurysms: a review of 116 cases. Br J Surg. 1974;61:469–75.
34. Janes JM, Ivins JC. A method of dealing with arteriosclerotic popliteal aneurysms. Surgery. 1951;29:398–406.
35. Lord JW. Clinical behaviour and operative management of popliteal aneurysms. JAMA. 1957;163:1102–6.
36. Friesen G, Ivins JC, Janes JM. Popliteal aneurysms. Surgery. 1962;51:90–8.
37. Edmunds LH, Darling RC, Linton RR. Surgical management of popliteal aneurysms. Circulation. 1965;32:517–23.
38. Baird RJ, Sivasankar R, Hayward R, Wilson DR. Popliteal aneurysms: a review and analysis of 61 cases. Surgery. 1966;59:911–7.
39. Crichlow RW, Roberts B. Treatment of popliteal aneurysms by restoration of continuity: review of 48 cases. Ann Surg. 1966;163:417–26.
40. Buda JA, Weber CJ, Mc Allister FF, Vorhees AB Jr. The results of treatment of popliteal aneurysms. A follow-up study of 86 aneurysms. J Cardiovasc Surg. 1974;15:615–9.
41. Gaylis H. Popliteal arterial aneurysm. A review and analysis of 55 cases. S A Med J. 1974;48:75–81.
42. Hardy JD, Tompkins WC Jr, Hatten LE, Chavez CM. Aneurysms of the popliteal artery. Surg Gynecol Obstet. 1975;140:401–4.
43. Evans WE, Turnipseed WD. Popliteal aneurysms. Vasc Surg. 1976;10:86–91.
44. Towne JB, Thompson JE, Patman DD, Persson AV. Progression of popliteal aneurysmal disease following popliteal aneurysm resection with graft: a twenty year experience. Surgery. 1976;80:426–32.
45. Tompkins WC, Smith AD, Wren HB, Bransford RM. The atherosclerotic popliteal aneurysm. Report

of diagnosis and treatment in twenty six cases. Am J Surg. 1977;134:813–6.
46. Chitwood WR, Stocks LH, Wolfe WG. Popliteal artery aneurysms: past and present. Arch Surg. 1978;113:1078–82.
47. Inahara T, Toledo AC. Complications and treatment of popliteal aneurysms. Surgery. 1978;84:775–83.
48. Vermilion BD, Kimmins SA, Pace WG, Evans WE. A review of one hundred forty seven popliteal aneurysms with long-term follow-up. Surgery. 1981;90:1009–14.
49. Laskar M, Christides C, Kim M. Anévrismes poplités athéromateux. Angéiologie. 1982;34:113–21.
50. Reilly MK, Abbott WM, Darling RC. Aggressive surgical management of popliteal artery aneurysms. Am J Surg. 1983;145:498–502.
51. Takolander RJ, Bergqvist D, Bergentz S-E, Ericsson BF, Sigurjonsson S, Jonsson K. Aneurysms of the popliteal artery. Acta Chir Scand. 1984;150:135–40.
52. Salo JA, Ala-Kuliju K, Ketonen P, Perhoniemi V, Meurala H, Harjola P-T. Reconstructive surgery of popliteal aneurysms, vol. 15. Vasa; 1986. p. 170–3.
53. Mellière D, Veit R, Becquemin J-P, Etienne G. Should all spontaneous popliteal aneurysms be operated on? J Cardiovasc Surg. 1986;27:273–7.
54. Anton GE, Hertzer NR, Beven EG, O'Hara PJ, Krajewski LP. Surgical management of popliteal aneurysms—trends in presentation, treatment and results from 1952 to 1984. J Vasc Surg. 1986;3:125–34.
55. Raptis S, Ferguson L, Miller JH. The significance of tibial artery disease in the management of popliteal aneurysms. J Cardiovasc Surg. 1986;27:703–8.
56. Schellack J, Smith RB III, Perdue GD. Nonoperative management of selected popliteal aneurysms. Arch Surg. 1987;122:372–5.
57. Englund R, Schache D, Magee HR. Atherosclerotic popliteal aneurysms with particular regard to the contralateral side. Aust N Z J Surg. 1987;57:387–90.
58. Lilly MP, Flinn WR, McCarthy WJIII, Courtney DF, Yao JST, Bergan JJ. The effect of distal arterial anatomy on the success of popliteal aneurysm repair. J Vasc Surg. 1988;7:653–60.
59. Cole CW, Thijssen AM, Barber GG, McPhall WV, Scobie TK. Popliteal aneurysm: an index of generalized vascular disease. Can J Surg. 1989;32:65–8.
60. Halliday AV, Taylor PR, Wolfe JH, Mansfield AO. The management of popliteal aneurysms: the importance of early surgical repair. Ann Roy Coll Surg Engl. 1991;73:253–7.
61. Dawson I, van Bockel JH, Brand R, Terpstra JL. Popliteal artery aneurysms: long-term follow-up of aneurysmal disease and results of surgical treatment. J Vasc Surg. 1991;13:398–407.
62. Roggo A, Brunner U, Ottinger LW, Largiader F. The continuing challenge of aneurysms of the popliteal artery. Surg Gynecol Obstet. 1993;177:565–72.
63. Lowell RC, Gloviczki P, Hallett JW Jr, Naessens JM, Maus TP, Cherry KJ Jr, Bower TC, Pairolero PC. Popliteal aneurysm: the risk of nonoperative management. Ann Vasc Surg. 1994;8:14–23.
64. Vettorello G, Rocca T, Taddia MC, Occhionorelli S, Santini M, Mari F, Mascoli F, Donini I. Gli aneurismi dell'arteria poplitea. Nostra esperienza a proposito di 37 casi. Minerva Cardioangiol. 1996;44:437–42.
65. Sarcina A, Bellosta R, Luzzani G, Agrifoglio G. Surgical treatment of popliteal artery aneurysms. A 20 year experience. J Cardiovasc Surg. 1997;38:347–54.
66. Davidovic LB, Lotina SL, Kostic DM, Cinara IS, Cveltkovic SD, Markovic DM, Vojnovic BR. Popliteal artery aneurysms. World J Surg. 1998;22:812–7.
67. Dawson I, Sie R, van Baalen JM, van Bockel JH. Asymptomatic popliteal aneurysm: elective operation versus conservative follow-up. Br J Surg. 1994;81:1504–7.
68. Carpenter JP, Barker CF, Roberts B, Berkowitz HD, Lusk EJ, Perloff LJ. Popliteal artery aneurysms: current management and outcome. J Vasc Surg. 1994;19:65–73.
69. Gawenda M, Sorgatz S, Müller U, Walter M, Erasmi H. The thrombosed popliteal aneurysm with distal arterial occlusion—successful therapy by interdisciplinary management. Thorac Cardiovasc Surg. 1995;43:112–6.
70. Duffy ST, Colgan MP, Sultan S, Moore DJ, Shanik GD. Popliteal aneurysms: a 10-year experience. Eur J Vasc Endovasc Surg. 1998;16:218–22.
71. Taurino M, Calisti A, Grossi R, Maggiore C, Speziale F, Fiorani P. Outcome after early treatment of popliteal artery aneurysms. Intern Angiol. 1998;17:28–31.
72. Dijkstra B, Fleisch J, Knight D. Management and outcome of popliteal artery aneurysms in a New Zealand Provincial Centre. Aust N Z J Surg. 1998;68:255–7.
73. Locati P, Socrate AM, Costantini E, Campanati B. Popliteal aneurysms: current management and outcome. Minerva Cardioangiol. 1999;47:145–55.
74. Gouny P, Bertrand P, Duedal V, Cheynel-Hocquet C, Lancelin C, Escourolle F, Nussaume O, Vayssairat M. Limb salvage and popliteal aneurysms: advantages of preventive surgery. Eur J Vasc Endovasc Surg. 2000;19:496–500.
75. Irace L, Gattuso R, Faccenna F, Cappello F, Siani B, Stumpo R, Boiceff S, Benedetti-Valentini F. Trattamento chirurgico degli aneurismi poplitei in elezione e in urgenza. Minerva Cardioangiol. 2001;49:251–6.
76. Stiegler H, Medler G, Baumann G. Prospective study of 36 patients with 46 popliteal aneurysms with non-surgical treatment. Vasa. 2002;31:43–6.
77. Kauffman P, Puech-Leao P. Surgical treatment of popliteal artery aneurysm: a 32-year experience. J Vasc Bras. 2002;1:5–14.
78. Dorigo W, Pulli R, Turini F, Pratesi G, Credi G, Alessi Innocenti A, Pratesi C. Acute leg ischemia from thrombosed popliteal artery aneurysms: role

of preoperative thrombolysis. Eur J Vasc Endovasc Surg. 2002;23:251–4.
79. Ascher E, Markevich N, Schutzer RW, Kallakuri S, Jacob T, Hingorani AP. Small popliteal artery aneurysms: are they clinically significant? J Vasc Surg. 2003;37:55–60.
80. Bowrey DJ, Osman H, Gibbons CP, Blackett RL. Atherosclerotic popliteal aneurysms: management and outcome in forty-six patients. Eur J Vasc Endovasc Surg. 2003;25:79–83.
81. Harder Y, Notter H, Nussbaumer P, Leiser A, Canova C, Furrer M. Popliteal aneurysm: diagnostic workup and results of surgical treatment. World J Surg. 2003;27:788–92.
82. Laxdal E, Amundsen SR, Dregelid E, Pedersen G, Aune S. Surgical treatment of popliteal artery aneurysms. Scand J Surg. 2004;93:57–60.
83. Aulivola B, Hamdan AD, Hile CN, Sheahan MG, Skillman JJ, Campbell DR, Scovell SD, LoGerfo FW, Pomposelli FB Jr. Popliteal artery aneurysms: a comparison of outcomes in elective versus emergent repair. J Vasc Surg. 2004;39:1171–7.
84. Martelli E, Ippoliti A, Ventoruzzo G, De Vivo G, Ascoli Marchetti A, Pistolese GR. Popliteal artery aneurysms. Factors associated with thromboembolism and graft failure. Intern Angiol. 2004;23:54–65.
85. Galland RB, Magee TR. Popliteal aneurysms: distortion and size related to symptoms. Eur J Vasc Endovasc Surg. 2005;30:534–8.
86. Stone PA, Armstrong PA, Bandyk DF, Keeling WB, Flaherty SK, Shames ML, Johnson BL, Back MR. The value of duplex surveillance after open or endovascular popliteal aneurysm repair. J Vasc Surg. 2005;41:936–41.
87. Pulli R, Dorigo W, Troisi N, Alessi Innocenti A, Pratesi G, Azas L, Pratesi C. Surgical management of popliteal artery aneurysms: which factors affect outcomes? J Vasc Surg. 2006;43:481–7.
88. Beseth BD, Moore WS. The posterior approach for repair of popliteal artery aneurysms. J Vasc Surg. 2006;43:940–5.
89. Huang Y, Gloviczki P, Noel AA, Sullivan TM, Kalra M, Gullerud RE, Hoskin TL, Bower TC. Early complications and long-term outcome after open surgical treatment of popliteal artery aneurysms: is exclusion with saphenous vein bypass still the gold standard? J Vasc Surg. 2007;45:706–15.
90. Davies RSM, Wall M, Simms MH, Vohra RK, Bradbury AW, Adam DJ. Long-term results of surgical repair of popliteal artery aneurysm. Eur J Vasc Endovasc Surg. 2007;34:714–8.
91. Curi MA, Geraghty PJ, Merino OA, Veeraswamy RK, Rubin BG, Sanchez LA, Choi ET, Sicard GA. Mid-term outcomes of endovascular popliteal artery aneurysm repair. J Vasc Surg. 2007;45:505–10.
92. Lichtenfels E, Delduque Frankini A, Bonamigo TP, Cardozo MA, Schulte AA. Popliteal artery aneurysm surgery: the role of emergency setting. Vasc Endovasc Surg. 2008;42:159–64.
93. Dzieuciuchowicz L, Lukaszuk M, Figiel J, Klimczak K, Krasinski Z, Majewski W. Factors influencing the clinical course of popliteal artery aneurysm. Med Sci Monit. 2009;15:CR231–r235.
94. Zimmermann A, Schoenberger T, Saeckl J, Reeps C, Wendorff H, Kuehnl A, Eckstein H-H. Eligibility for endovascular technique and results of the surgical approach to popliteal artery aneurysms at a single center. Ann Vasc Surg. 2010;24:342–8.
95. Zaraca F, Ponzoni A, Stringari C, Ebner JA, Giovannetti R, Ebner H. The posterior approach in the treatment of popliteal artery aneurysm: feasibility and analysis of outcome. Ann Vasc Surg. 2010;24:863–70.
96. Pulli R, Dorigo W, Fargion A, Pratesi G, Alessi Innocenti A, Angiletta D, Pratesi C. Comparison of early and midterm results of open and endovascular treatment of popliteal artery aneurysms. Ann Vasc Surg. 2012;26:809–18.
97. Kropman RHJ, van Meurs A, Fioole B, van Santvoort HC, van Sambeek M, Moll FL, de Vries J-PPM. Association of sex with long-term outcomes after popliteal artery aneurysm repair. Ann Vasc Surg. 2014;28:338–44.
98. Huang Y, Gloviczki P, Oderich GS, Duncan AA, Kalra M, Fleming MD, Harmsen WS, Bower TC. Outcomes of endovascular and contemporary open surgical repairs of popliteal artery aneurysm. J Vasc Surg. 2014;60:631–8.
99. Serrano-Hernando FJ, Martinez López I, Hernández Mateo MM, Rydings MH, Sanchez Hervás L, Rial Horcajo R, Moñuz Ducaju G, Conejero AM. Comparison of popliteal artery aneurysm therapies. J Vasc Surg. 2015;61:655–61.
100. Mazzaccaro D, Carmo M, Dellatana R, Settembrini AM, Barbetta I, Tassinari L, Roveri S, Settembrini PG. Comparison of posterior and medial approaches for popliteal artery aneurysms. J Vasc Surg. 2015;62:1512–20.
101. Ronchey S, Pecoraro F, Alberti V, Serrao E, Orrico M, Lachat M, Mangialardi N. Popliteal artery aneurysm repair in the endovascular era. Fourteen-years single center experience. Medicine. 2015;94:e1130.
102. Wagenhauser MU, Herma KB, Saghan TA, Dueppers P, Schelzig H, Duran M. Long-term results of open repair of popliteal artery aneurysm. Ann Med Surg. 2015;4:58–63.
103. Leake AE, Avgerinos ED, Chaer RA, Singh MJ, Makaroun MS, Marone LK. Contemporary outcome of open and endovascular popliteal artery aneurysm repair. J Vasc Surg. 2016;63:70–6.
104. Broca P. Des anévrysmes et de leur traitement. Paris: Labé; 1856. p. 1–48.
105. Home E. An account of Mr. Hunter's method of performing the operation for the cure of popliteal aneurism. In: Palmer JE, editor. The works of John Hunter. London: Longman & Co; 1837. p. 594–612.
106. Delbet P, Mocquod T. Affections chirurgicales des artères. In: Le Dentu P, Delbet P, editors. Nouveau traité de médecine clinique et opératoire. Paris: J.B. Ballière & Fils; 1911. p. 167–84.
107. Erichsen JH. Science and art of surgery. London: Longman, Green & Co.; 1872. p. 14–55.

108. Wells AH, Coburn CE, Walker MA. Popliteal aneurysm, with report of a case. JAMA. 1936;106:1264–6.
109. Boyd AM, Ratcliffe AH, Jepson RP, James GWH. Intermittent claudication: a clinical study. J Bone Joint Surg. 1949;31-B:325–55.
110. Lindbom A. Arteriosclerosis and arterial thrombosis in lower limb: a roentgenological study. Acta Radiol Suppl. 1950;80:1–80.
111. Poland A. On a case of fusiform and tubular aneurysm of the subclavian artery, and its successful treatment by indirect digital compression. Med Chir Trans. 1869;52:277–307.
112. Halsted WS, Reid MR. An experimental study of circumscribed dilation of an artery immediately distal to a partially occluding band and its bearing on the dilation of the subclavian artery observed in certain cases of cervical rib. J Exp Med. 1916;24:271–86.
113. Holman E. On circumscribed dilation of an artery immediately distal to a partially occluding band: poststenotic dilatation. Surgery. 1954;36:3–24.
114. Simkins TE, Stehbens WE. Vibrations recorded from the adventitial surface of experimental aneurysms and arteriovenous fistulas. Vasc Surg. 1974;8:153–65.
115. Palma EC. Stenosed arteriopathy of Hunter canal and loop of the adductor magnus. Am J Surg. 1952;83:723–33.
116. Lilly GD. The management of aneurysms of the lower extremities. Ann Surg. 1946;123:601–6.
117. Gedge SW, Spittel JA Jr, Ivins JC. Aneurysm of the distal popliteal artery and its relationship to the arcuate popliteal ligament. Circulation. 1961;24:270–3.
118. Lindeman JH, Abdul-Hussien H, Schaapherder AF, van Bockel JH, van der Thusen JH, Roelen DL, Kleemann R. Enhanced expression and activation of proinflammatory transcription factors distinguish aneurysmal from atherosclerotic aorta: IL-6 and IL-8-dominated inflammatory responses prevail in the human aneurysm. Clin Sci. 2008;114:687–97.
119. Busuttil RW, Abou-Zamzam AM, Machleder HI. Collagenase activity of the human aorta: a comparison of patients with and without abdominal aortic aneurysms. Arch Surg. 1980;115:1373–8.
120. Menashi S, Campa JS, Greenhalgh RM, Powell JT. Collagen in abdominal aortic aneurysm: typing, content, and degradation. J Vasc Surg. 1987;5:578–82.
121. Tilson MD, Seashore MR. Human genetics of the abdominal aortic aneurysm. Surg Gynecol Obstet. 1984;158:129–32.
122. Joahnsen K, Koepsell T. Familial tendency for abdominal aortic aneurysms. JAMA. 1986;256:1934–6.
123. Darling RC, Brewster DC, La Muraglia MG, Moncure AC, Cambria RP, Abbott WM. Are familial abdominal aortic aneurysms different? J Vasc Surg. 1989;10:39–43.
124. Ward AS. Aortic aneurysmal disease. A generalized dilating diathesis. Arch Surg. 1992;127:990–1.
125. Sandgren T, Sonesson B, Ryden-Ahlgren A, Lanne T. Arterial dimensions in the lower extremities of patients with abdominal aortic aneurysm—no indication of a generalized dilating diathesis. J Vasc Surg. 2001;34:1079–84.
126. Makita S, Ohira A, Tachieda R, Itoh S, Moriai Y, Niinuma H, Nakamura M, Hiramori K. Dilation and reduced distensibility of carotid artery in patients with abdominal aortic aneurysms. Am Heart J. 2000;140:297–302.
127. Midttun M, Sejrsen P, Paaske WP. Is non-specific aneurysmal disease of the infrarenal aorta also a peripheral microvascular disease? Eur J Vasc Endovasc Surg. 2000;19:625–9.
128. Widmer MK, Blatter S, Schmidli J, Baumgartner I, Gagl B, Carrel T, Savolainen H, Diehm N. Generalized dilating diathesis in patients with popliteal artery aneurysms. Vasa. 2008;37:157–63.
129. Jacob T, Ascher E, Hingorani A, Gunduz Y, Kallakuri S. Initial steps in the unifying theory of the pathogenesis of arterial aneurysms. J Surg Res. 2001;101:37–43.
130. Jacob T, Hingorani A, Ascher E. Examination of the apoptotic pathway and proteolysis in the pathogenesis of popliteal artery aneurysms. Eur J Vasc Endovasc Surg. 2001;22:77–85.
131. Jacob T, Schutzer R, Hingorani A, Ascher E. Differential expression of YAMA/CPP-32 by T lymphocytes in popliteal artery aneurysms. J Surg Res. 2003;112:111–6.
132. Abdul-Hussien H, Hanemaaijer R, Kleemann R, Verhaaren BF, van Bockel JH, Lindeman JH. The pathophysiology of abdominal aortic aneurysm growth: corresponding and discordant inflammatory and proteolytic processes in abdominal aortic and popliteal artery aneurysms. J Vasc Surg. 2010;51:1479–87.
133. Debasso R, Astrand H, Bjarnegard N, RydénAhlgren A, Sandgren T, Lanne T. The popliteal artery, an unusual muscular artery with wall properties similar to the aorta: implications for susceptibility to aneurysm formation? J Vasc Surg. 2004;39:836–42.
134. Hurks R, Kropman RHJ, Pennekamp CWA, Hoefer IE, de Vries J-PPM, Pasterkamp J, Vink A, Moll FL. Popliteal artery aneurysms differ from abdominal aortic aneurysms in cellular topography and inflammatory markers. J Vasc Surg. 2014;60:1514–9.
135. Mosiello G, Sapienza P, Fuso A, Sterpetti A, Cucina A, Di Gioia C, Coluccia P, di Marzo L. Popliteal artery aneurysm formation: a pathogenetic dilemma. Personal commun.
136. Norman PE, Powell JS. Site specificity of aneurysmal disease. Circulation. 2010;121:560–8.
137. Mellière D, Cron J, Lange F, Qvarfordt P, Desgranges J, Becquemin J-P, Cavillon A. Some popliteal aneurysms are congenital. Cardiovasc Surg. 1998;6:42–9.