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Clinical Science

Arterial clamping leads to stenosis at clamp sites after femoropopliteal bypass surgery

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KEYWORDS:
Vascular clamping; Artery wall damage; Clamp force; Atherosclerosis; Stenosis

Abstract

BACKGROUND: To date, the incidence and clinical relevance of arterial stenosis at clamp sites after femoropopliteal bypass surgery is unknown.

METHODS: Ninety-four patients underwent a femoropopliteal bypass in which the arterial inflow and outflow clamp sites were controlled by the Fogarty-Soft-Inlay clamp and marked with an hemoclip. The number of pre-existing atherosclerotic segments, clamp force, and clamp time were recorded and the occurrence of a stenosis at the clamp site was determined.

RESULTS: After a mean follow-up of 83 months, a significant stenosis was confirmed at 23 of the 178 clamp sites (12.9%; 95% confidence interval 8.4 to 18.8). The mean number of pre-existing atherosclerotic segments (P = .28) and the mean clamp force (P = .55) was similar between the groups with and without a stenosis. There was a significant difference regarding clamp time between the group with and without a stenosis (38 minutes and 26 minutes, P = .001).

CONCLUSION: Arterial clamping, even with the Fogarty-Soft-Inlay clamp, can lead to clamp stenosis and seems to be related to the duration of clamping, but not to pre-existent atherosclerotic burden.

At the time this study started inclusion in ISRCTN was not required.
There were no relevant financial relationships or any sources of support in the form of grants, equipment, or drugs.
The authors declare no conflicts of interest.
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Conducting a vascular reconstructive procedure requires control of the proximal and distal blood flow. To achieve this, occlusion balloon catheters, vessel loops, or vascular clamps can be applied. Although vessel loops can be used with good results for at least distal anastomoses, vascular clamps are most commonly used. Various animal studies show that vascular clamps can lead to moderate or severe intimal damage.1-6 In vitro experiments with human
material also showed that clamping the artery causes
damage to the vessel wall.7–10
To prevent damage specially designed vascular clamps
have been developed. The Fogarty-Soft-Inlay (FSI) clamp
(Applied Medical, Rancho Santa Margarita, CA) may be
less traumatic compared with other methods of clamping
(Fig. 1).3,11,12
To our knowledge, in the current literature no human
in vivo studies of clamp-related arterial damage, including
acceleration of intima hyperplasia and as a consequence
stenosis formation, are available. The effect of clamp-
related vessel wall damage in the form of developing a
stenosis has yet to be defined. The aim of this study was to
determine the development of arterial clamp site stenosis
after femoropopliteal bypass surgery.

Patients and Methods

Between November 1997 and February 2005, a total of
99 consecutive vascular surgery patients were prospectively
evaluated. Patient data were processed and electronically
stored according to the Declaration of Helsinki-Ethical
principles for medical research involving human subjects.
The Institutional Review Board approved the study and
written informed consent was obtained from all patients.
Patients were eligible for inclusion if they were operated on
electively and received a femoropopliteal (above knee [AK]
or below knee [BK]) bypass for atherosclerotic occlusive
disease with at least one crural outflow artery. Patients were
excluded if they failed to undergo pre- or postoperative
imaging (ie, patients with renal dysfunction), if they had a
high probability of nonadherence to the follow-up
requirements, or when an additional endarterectomy was
performed at the level of one of the clamp sites.

All patients received duplex ultrasound examination
preoperatively to determine preoperative stenosis. Patients
could be included twice if a femoropopliteal bypass was
performed on both legs. Preoperative risk assessment
consisted of age, sex, diabetes mellitus, hypertension,
hypercholesterolemia, cardiac disease, cerebrovascular dis-
ease, smoking, chronic limb ischemia classification (CLI)
according to Rutherford,13 and anticoagulant therapy use.
Perioperative outcome parameters included macroscopic
evaluation of atherosclerosis at the clamp site, clamp force
applied (notches), and clamp time (minutes). Both the prox-
imal inflow and distal outflow clamp sites were assessed.

The procedure was carried out as follows. In all cases, a
longitudinal groin incision was used to gain access to the
femoral artery with a medial approach for exposure of the
distal popliteal artery. After administration of 5,000 U of
heparin intravenously, the arterial inflow and outflow were
controlled by the FSI clamp—a “cross-membered”
metallic clamp with jaws cushioned with soft rubber
pads. The arterial inflow was controlled by placing the
FSI clamp anterior to posterior proximal on the common
femoral artery (CFA) just distal of Poupart’s ligament. At
the distal site, the clamp was placed distal on the popliteal
artery. The other sides of arteriotomy were controlled by
regular vascular clamps or vessel loops.

The clamp force was increased until the blood flow was
controlled and the exact position of the clamp was marked

Figure 1  Fogarty-Soft-Inlay clamp used in this study.
with an hemoclip. The distance between the clamp site and the anastomosis was measured (mm) and recorded. Postoperative surveillance for stenosis on the clamp site was determined by duplex ultrasound examination and/or angiography. Visualization of the clip usually proved possible by duplex examination. This in combination with the recorded distance made it possible to identify the exact clamp location and a possible stenosis. If by duplex the clip could not be identified, and the clinical suspicion of a stenosis existed, an additional angiography was performed. The results of the duplex and/or angiography were compared with the preoperative imaging. All manipulation maneuvers and repositioning of the clamps were recorded as possible confounders.

The presence of atherosclerosis at the clamp sites was assessed during surgery by determining the number of pre-existing atherosclerotic segments. For this purpose, the lumen was divided into 8 equal segments (Fig. 2). The surgeon then assessed the number of atherosclerotic segments macroscopically. This macroscopic evaluation was only possible at the proximal clamp site; at the distal clamp site, exposure was insufficient for a reliable determination. Three to 6 months after surgery all patients received duplex ultrasound examination and/or angiography wherein the clamp sites were imaged and the presence and degree of a stenosis were defined. A stenosis was considered significant if duplex ultrasound showed a peak systolic velocity ratio greater than 3.0, accounting for a stenosis of greater than 70%.14 All patients were then checked annually and bypass function was assessed using ankle–brachial index measurement and duplex ultrasound examination and/or angiography.

Figure 2  Visual assessment of pre-existing atherosclerotic segments at the clamp site.

Assessed for eligibility: 99 patients

Included: 94 patients

6 patients included both legs

Analysis: 100 legs

Lost to follow up
- Incomplete follow-up (n=2)
Discontinued follow-up (n=8)
- Deaths (n=6)
- Graft removal (n=2)

Analysis: 90 legs

88 legs proximal and distal clamp site

2 legs distal clamp site

Analysis: 178 clamp sites

Figure 3  Flow chart of participants in the study.
The primary endpoint of this study was the presence or absence of a significant stenosis at one or both clamp sites. If a stenosis occurred, the influence of the number of pre-existing atherosclerotic segments on the proximal clamp site, clamp force, and clamp time and the number of reinterventions were assessed.

Patients only underwent a reintervention in case of a stenosis of greater than 70% and disabling claudication (Rutherford classification 3 to 6). Analyses of the endpoints were performed per limb.

Statistics

Differences between categorical variables were tested with Pearson’s chi-square test (2 variables) or Fisher’s exact test as appropriate. Differences between means were tested with Student 2-tailed test (normally distributed continuous variables) or Mann–Whitney U test (skewed continuous variables). Two-tailed P values were used throughout and significance was set at P value less than .05. Data are presented as means ± standard deviation, unless stated otherwise. All statistical analyses were done with the Statistical Package for the Social Sciences (SPSS version 15.0.1.1; SPSS, Chicago, IL).

Results

A total of 99 patients were found eligible for inclusion in this study. Five patients were excluded, of which 2 did not meet the inclusion criteria, 2 declined to participate,
and 1 patient failed to undergo postoperative imaging. After applying the inclusion and exclusion criteria, 94 patients were included, all treated with a femoropopliteal bypass. In 6 patients, both legs were included in the study allowing 100 legs to be analyzed. In 10 patients, follow-up could not be completed, of which 6 patients have died, in 2 the graft had to be removed because of infection, and 2 patients were lost to follow-up. Eventually 90 bypasses were analyzed.

In 88 limbs, both the proximal and distal clamp sites were included, in 2 limbs only the distal clamp site was included because an endarterectomy was performed at the level of the CFA. As a consequence, 178 clamp sites were properly assessable (Fig. 3).

**Study cohort**

The study population consisted of 76 (76 %) male and 24 (24 %) female patient legs. The mean age was 66.5 ± 10.3 years (range 30 to 87 years).

Baseline characteristics are outlined in Table 1. Preoperative comorbidities such as diabetes mellitus, hypertension,

<table>
<thead>
<tr>
<th>Number of atherosclerotic segments</th>
<th>Significant stenosis at the proximal clamp site</th>
<th>No stenosis at the proximal clamp site</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3</td>
<td>41</td>
<td>44</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>8</strong></td>
<td><strong>80</strong></td>
<td><strong>88</strong></td>
</tr>
</tbody>
</table>

Mean (SD) 3.13 (3.0) 2.43 (2.9) \( P = .52 \)

Median (IQR) 3.0 (0–5.75) 0 (0–4.75) \( P = .47 \)

IQR = interquartile range; SD = standard deviation.
hypercholesterolemia (total cholesterol > 5.0 or low-density lipoprotein > 2.5), cardiac disease, cerebrovascular disease, smoking habits, and CLI classification were equally divided between both groups (Table 1). Distribution in terms of CLI was 58% for grade I (Rutherford classification 1 to 3), 22% for grade II (Rutherford classification 4), and 20% for grade III (Rutherford classification 5). Patients with grade I CLI were only considered for surgery in case of inability to or unsuccessful (supervised) exercise and medical therapy. Forty-seven patients received coumarin derivatives postoperatively, and 42 patients received acetyl salicylic acid. One patient did not receive any anticoagulant therapy (Table 1).

Ninety bypasses were successfully analyzed: 30 autologous bypasses (9 AK and 21 BK) and 60 prosthetic bypasses (51 AK and 9 BK) (Table 1). The prosthetic bypasses consisted of ringed \((n = 8)\) or nonringed \((n = 1)\) polytetrafluoroethylene (PTFE), ringed polyester \((n = 18)\) and nonringed polyester \((n = 33)\) bypasses. In 2 legs with a BK bypass, a venous cuff was used. There was no difference in clamp stenosis between the autologous and prosthetic grafts. There was also an equal distribution with respect to the operated side. Eighty-seven percent of the patients received duplex ultrasound scanning and 69% of the patients an angiography.

One procedure was complicated by an infected seroma and this patient died during hospital admission after 36 days because of myocardial infarction. There was no additional 30-day mortality. After a mean follow-up of 83 months, 47 patients deceased, 1 patient was lost to follow-up, and in 1 patient the graft had to be removed because of infection.

### Clamp stenosis: short-term follow-up

After 6 months at 13 clamp sites a significant stenosis \((>70\%)\) had occurred, including 8 inflow stenoses \((9.1\%)\) and 5 outflow stenoses \((5.5\%)\). The total incidence of clamp stenosis was 7.3% \((95\% \text{ CI } 3.5 \text{ to } 11.1\)\). In 4 patients, this resulted in a reintervention. One patient received an endarterectomy of the CFA, 1 patient a percutaneous transluminal angioplasty (PTA) of the CFA, and 2 patients a PTA of the popliteal artery at the distal clamp site.

### Clamp stenosis: long-term follow-up

After a mean follow-up of 83 months, 23 clamp sites showed a significant stenosis consisting of 16 \((18.2\%)\) inflow stenoses and 7 \((7.8\%)\) outflow stenoses (Fig. 4). This led to a total incidence of 12.9% \((95\% \text{ CI } 8.4 \text{ to } 18.8\)\) clamp stenosis and an additional 17 reinterventions. In 5 cases an endarterectomy of the CFA was performed.

### Table 3 Number of atherosclerotic segments at the clamp site (83 months)

<table>
<thead>
<tr>
<th>Number of atherosclerotic segments</th>
<th>Significant stenosis at the proximal clamp site</th>
<th>No stenosis at the proximal clamp site</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5</td>
<td>39</td>
<td>44</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>72</td>
<td>88</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>3.50 (2.99)</td>
<td>2.26 (2.90)</td>
<td>(P = .13)</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>3.00 (0–5.75)</td>
<td>0 (0–4.0)</td>
<td>(P = .10)</td>
</tr>
</tbody>
</table>

**IQR** = interquartile range; **SD** = standard deviation.

### Table 4 Clamp force at the clamp site (6 months)

<table>
<thead>
<tr>
<th>Clamp force (notches)</th>
<th>Significant stenosis at the clamp site</th>
<th>No stenosis at the clamp site</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>22</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>69</td>
<td>72</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>39</td>
<td>43</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>138</td>
<td>148</td>
</tr>
<tr>
<td>Mean</td>
<td>2.10</td>
<td>2.27</td>
<td>(P = .55)</td>
</tr>
</tbody>
</table>

### Table 5 Clamp force at the clamp site (83 months)

<table>
<thead>
<tr>
<th>Clamp force (notches)</th>
<th>Significant stenosis at the clamp site</th>
<th>No stenosis at the clamp site</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>20</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>63</td>
<td>72</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>37</td>
<td>43</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>127</td>
<td>148</td>
</tr>
<tr>
<td>Mean</td>
<td>2.19</td>
<td>2.28</td>
<td>(P = .76)</td>
</tr>
</tbody>
</table>
in 7 a PTA at the clamp site of the CFA, 4 times a PTA at the clamp site of the popliteal artery, and in 1 patient an additional thrombectomy of the distal popliteal artery was required. The 2 bypasses in which a venous cuff was used did not develop a significant stenosis. In 6 patients with a significant stenosis, we refrained from intervention because of absence of symptoms.

Table 6  Relationship between clamp time and the development of stenosis at 6 months

<table>
<thead>
<tr>
<th>Clamping time (minutes, seconds)</th>
<th>Significant stenosis at the clamp site</th>
<th>No stenosis at the clamp site</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>32.08 (14.31)</td>
<td>28.41 (13.53)</td>
<td>.53</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>28.00 (21:00–42:00)</td>
<td>23.00 (19:00–36:30)</td>
<td>.44</td>
</tr>
<tr>
<td>Distal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>32.40 (04.02)</td>
<td>26.53 (11:42)</td>
<td>.40</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>35.00 (28:00–35:00)</td>
<td>25.00 (19:00–34:00)</td>
<td>.16</td>
</tr>
</tbody>
</table>

IQR = interquartile range; SD = standard deviation.

Table 7  Relationship between clamp time and the development of stenosis at 83 months

<table>
<thead>
<tr>
<th>Clamping time (minutes, seconds)</th>
<th>Significant stenosis at the clamp site</th>
<th>No stenosis at the clamp site</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>38.33 (15:46)</td>
<td>26.25 (12:13)</td>
<td>.001</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>36.30 (23:30–55:00)</td>
<td>21:40 (16:00–34:00)</td>
<td>.004</td>
</tr>
<tr>
<td>Distal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>25.48 (10:08)</td>
<td>27.14 (11:41)</td>
<td>.79</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>28.00 (15:30–35:00)</td>
<td>25.00 (19:00–34:30)</td>
<td>.95</td>
</tr>
</tbody>
</table>

IQR = interquartile range; SD = standard deviation.

Pre-existent atherosclerosis, clamp force, and clamp time

At the proximal clamp site, 44 patients had pre-existing atherosclerosis in a mean of 2.49 segments (range 0 to 8). Forty-four patients had no atherosclerosis according to the perioperative assessment, of which 3 limbs (6.8%) developed a significant stenosis. In the group with pre-existing atherosclerosis, 5 limbs (11.4%) developed a stenosis. The mean number of affected segments in this group was 3.13 compared with 2.43 in the group without stenosis (P = .52) (Table 2). After 83 months, these numbers were 3.50 compared with 2.26 (P = .13) (Table 3).

The clamp force was assessed 148 times. The mean clamp force after 6 months was 2.10 notches in the stenosis group and 2.27 in the group without a stenosis (P = .55) (Table 4).

After 83 months, these numbers were 2.19 in the stenosis group and 2.28 in the group without a stenosis (P = .76) (Table 5).

Clamp time was recorded 145 times. The mean proximal clamp time (at 6 months) was 32 minutes in the stenosis group and 28 minutes in the group without a stenosis (P = .53). The mean distal clamp time was 32 and 26 minutes, respectively (P = .40) (Table 6). After 83 months, the mean proximal clamp time was 38 minutes in the stenosis group compared with 26 minutes in the group without a stenosis (P = .001). The mean distal clamp time was 25 and 27 minutes, respectively (P = .79) (Table 7).

Comments

This study shows that the incidence of arterial clamp site stenosis after femoropopliteal bypass surgery was 7.3% after 6 months of follow-up. After a mean follow-up of 83 months, this number increased to 12.9%. To our knowledge, we present the first clinical study demonstrating the incidence and development of clamp site stenosis. After 6 months, 13 out of 100 legs developed a clamp site stenosis with 4 patients needing a reintervention. After 83 months, 12.9% of patients developed a stenosis of which an additional 17 needed a reintervention. Our bypass surgery results are in accordance with the literature with 5-year primary and secondary patency rates after venous femoropopliteal bypasses of 75.6% and 79.7%, respectively. For PTFE and Dacron bypasses, these numbers were 36%, 51%, 52%, and 70%, respectively. However, to obtain these patency rates various reinterventions were needed.

Multiple factors are involved in determining the level of trauma inflicted on the vessel wall by arterial clamps, including the condition of the vessel, applied clamp force, and the clamp time. Vascular clamping can lead to significant vessel wall injury with microscopical progression.
of atherosclerosis. Pre-existing atherosclerotic vessels are even more sensitive to this pathophysiological process. Nevertheless, using a visual macroscopic assessment, we were unable to demonstrate this relationship. Whether a luminal diameter measurement using either duplex ultrasound examination or angiography would be more specific cannot be determined based on this study. At cellular level pre-existent atherosclerotic vascular disease also seems to play an important role. Experimental studies using scanning electron microscopy and light microscopy data showed a direct relationship between vascular compressive forces (applied pressure) and vessel wall damage. The amount of endothelial injury was significantly less in sheep carotid and femoral arteries in which the closure force was limited to 3 and 4 notches compared with that for 5 and 6 notches.

We found no relationship between the average applied clamp force and the development of a stenosis. We focused on clinical effect and used only a noninvasive test instead of assessing it on endothelial level. Hypothetically, this could have led to an underestimation of the actual effect despite the lack of clinical consequence. We did demonstrate a significant relationship between clamp time and the development of a significant stenosis after 83 months, but only on the proximal clamp site. This outcome has also previously been demonstrated in the literature at a histological level. Despite growing evidence of the adverse effect of arterial clamping at a microscopic level and its negative effect on the mechanical properties of the vessel wall, to date clinical evidence is still missing.

Our study has some shortcomings that need to be addressed. The method of measuring the clamp force has proved very difficult. We decided to express it in notches, which although not an international quantitative unit is repeatedly used in experimental studies. Before the start of the study, we have attempted to measure the clamp force in Newton in collaboration with the Technical University Twente, The Netherlands. In these experiments, elastic loops were fixed to the clamp legs and the resistance was transformed to an electric signal thus allowing to measure the force and express it in Newton. Although the experimental results were reliable and reproducible, when further tested it proved too inconsistent for clinical use. Factors influencing wall compression pressure are multiple and, in most cases, more dependent on the nature of the arterial wall than on the clamp itself. Other contributing factors are temperature, length of the clamp legs, blood pressure, size of the vessel wall, amount of atherosclerosis, moisture of the vessel wall, and compliance of the vessel. These factors are in accordance with a recent publication. The choice for using notches, although a coarse measurement, was supported by the reliable reproducibility and the absence of an interobserver variability.

We only used the atraumatic FSI clamp on the proximal inflow and distal outflow site. We are aware that there are alternatives available. For example, in previous studies a decrease in vessel wall injury was observed when using a pressure controlling clamp. Other alternatives to clamping are an intraluminal balloon, a vessel loop, or vascular occluder gels, each with their own advantages and disadvantages. Intraluminal balloons exert a compressive force directly at the intima, the most vulnerable part of an already diseased artery. Nevertheless, animal studies show that intraluminal balloons resulted in less damage to the vessel wall compared with regular clamps. Similar results were found with the use of vessel loops compared with the FSI clamp. However, balloons and vessel loops in general are considerably less user friendly, can be cumbersome, and cannot be used for larger vessels or when traction on the artery is required. Even though vascular clamps certainly have a downside, to date they are most commonly used. When using them one should be aware of the potential effects and take into account the clamp force and duration of clamping.

Finally, in this study, we focused on the clamp site and only on the potential effects of the FSI clamp with regard to the occurrence of a stenosis. The development of a postbypass stenosis may be dependent on a number of factors such as abnormal flow patterns and compliance mismatches. Although we acknowledge this pathophysiology, we believe that our method of examining the exact clamp position is reliable and the demonstrated relationship therefore real. However, other contributing factors cannot be excluded based on this study. Whether a different clamp would have had a different effect on the stenosis rate cannot be answered based on our data.

Conclusion

Arterial clamping during femoropopliteal bypass surgery can lead to a substantial number of stenoses at the clamp site, even when using the FSI clamp. The occurrence seems related to the duration of clamping, but interestingly not to pre-existent atherosclerotic burden. More research is needed, especially comparison with alternative vessel occlusion techniques, to confirm our results.

References


