FREQUENTLY ASKED QUESTIONS

GORE PROPATEN® VASCULAR GRAFT

PERFORMANCE
through collaboration

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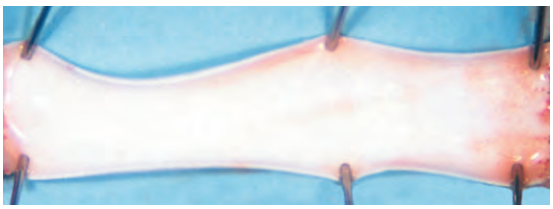
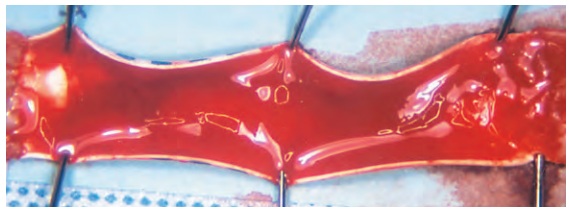
Technology

What is the GORE® PROPATEN® Vascular Graft?

The GORE® PROPATEN® Vascular Graft is an ePTFE vascular graft that has heparin bonded to its luminal surface via a proprietary end-point covalent bonding mechanism which imparts thromboresistant properties to the vascular graft.





The Bioactive luminal surface of a 3 mm diameter GORE® PROPATEN® Vascular Graft (left) remains free of thrombus, while the non-bioactive surface of a control graft (right; 3 mm diameter) is covered with thrombus. Grafts were explanted after two hours in a challenging carotid shunt canine model.

The unique features of the CBAS® Surface of the GORE® PROPATEN® Vascular Graft include:

- A proven thromboresistant surface
- Proprietary end-point covalent bonding
- Sustained bioactivity*

The heparin molecules are covalently bound to the luminal surface through a proprietary end-point attachment mechanism (CARMEDA® BioActive Surface (CBAS® Surface)) which serves to anchor heparin molecules to the luminal surface while still maintaining heparin's intrinsic bioactive properties. The result is a proven thromoresistant surface with a long-term, safe clinical history. There are two decades of clinical use across multiple applications and more than 400 scientific and clinical publications related to the CBAS® Surface.

Who is Carmeda AB?

Carmeda AB, a Swedish company, invented the end-point heparin bonding technology used on the GORE® PROPATEN® Vascular Graft and other medical devices. In October, 2005, Carmeda AB became a wholly owned subsidiary of W. L. Gore & Associates, Inc.

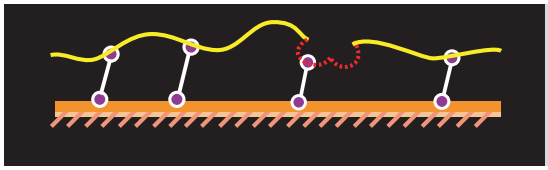
Does Carmeda AB have a history working with heparin technology?

Carmeda AB is recognized as the world leader in heparin technology with a long history of pioneering research in this field since the company was founded in 1984.

How does end-point covalent bonding of heparin differ from conventional covalent bonding of heparin and ionic bonding of heparin?

Conventional Covalent Bonding

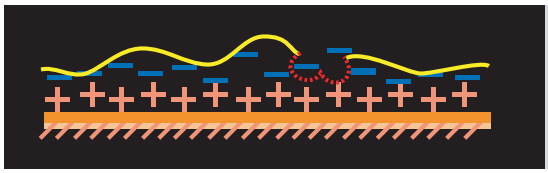
In conventional, multiple point covalent bonding, the anticoagulant properties of heparin are lost due to the fact that the heparin active site is not available to antithrombin.



The heparin molecule (yellow) is bonded (white) at multiple points to the device surface (orange). The heparin active site (red) is unavailable to antithrombin. Without bioactivity, the anticoagulant properties of heparin are lost.

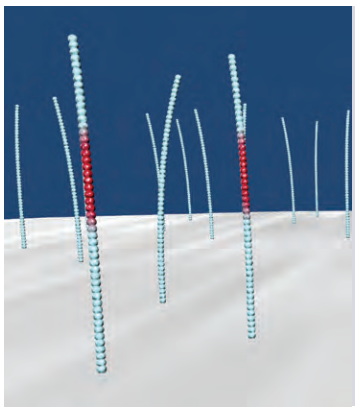
Ionic Bonding

In ionic bonding, the attractive force of negatively charged heparin and positively charged coating can be broken, thus allowing heparin to be released from the surface over time.



The negatively (-) charged heparin molecule (yellow) is attracted to a positively (+) charged coating (orange) on the device surface. This attractive force can be broken, allowing heparin to release from the surface over time. Sustained thromboresistance cannot be achieved.

End-point Covalent Bonding



End-point covalent bonding is a unique concept that allows the anticoagulant properties of heparin to be applied directly on the graft surface. The end of each heparin molecule is bonded to the surface, allowing the heparin active site to freely interact with anti-thrombin. Consequently, heparin is retained on the graft surface in a bioactive form.

What kind of heparin is bonded on the GORE® PROPATEN® Vascular Graft?

CBAS® 2-Heparin, a reduced molecular weight heparin of porcine origin and sourced in North America is used in the construction of all GORE® PROPATEN® Vascular Grafts. This CBAS® 2-Heparin is a component specific for end-point attachment to medical devices.

How long does the heparin bioactivity of the GORE® PROPATEN® Vascular Graft last?

It is unknown how long the heparin bioactivity lasts. Graft explants from an in vivo canine model demonstrated the continued presence of heparin on the graft surface and showed sustained heparin bioactivity over a period of 12 weeks.[1] Four human explants after eight months, three years, four years, and eight years, respectively, have also demonstrated heparin bioactivity. (For more information please refer to the Speakers' Presentation Resource, available on the Gore Medical website.)

How many International Units of heparin are on the surface of a GORE® PROPATEN® Vascular Graft?

Heparin activity in solution is typically measured in terms of International Units (IU). Surface bound heparin, on the other hand, is normally measured in terms of picomoles of antithrombin III (ATIII) uptake. A reasonable estimate of the number of IU on the graft surface can be obtained via theoretical calculations. Based on the mass of heparin on the surface of a large graft, theoretical calculations yield an estimate of < 400 IU per graft. This is very small compared to the typical intraoperative heparin dose of ~5,000 IU.

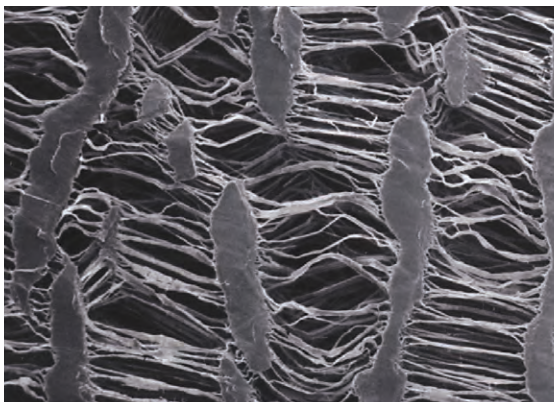
Does heparin release from the surface?

The CBAS® Surface is designed to bind heparin to the graft luminal surface via a stable covalent linkage. In vitro experiments have shown that a small residual amount of unbound CBAS® Heparin, well below the typical endogenous level in humans, is released into solution during the first 24 hours after initiation of flow.[2] The CBAS® Heparin bound to the device surface does not elute over time, and there is no systemic anticoagulant effect.[9]

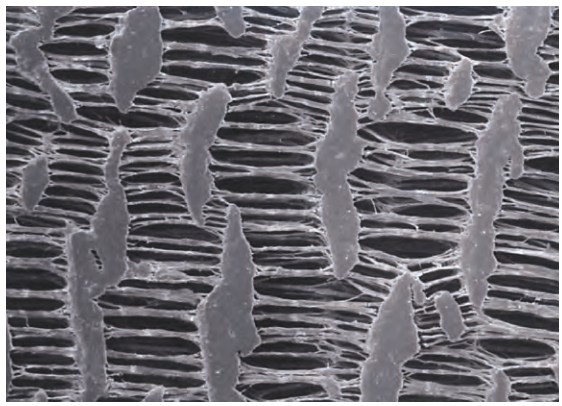
Are the microstructures of GORE-TEX® Stretch Vascular Grafts and GORE® PROPATEN® Vascular Grafts similar?

A GORE® PROPATEN® Vascular Graft and a GORE-TEX® Stretch Vascular Graft have similar microstructure when tensioned.

GORE-TEX® Stretch Vascular Graft



GORE PROPATEN® Vascular Graft



Does the GORE® PROPATEN® Vascular Graft utilize a Stretch technology?

The GORE® PROPATEN® Vascular Graft has similar extensibility, feel and handling characteristics to a GORE-TEX® Stretch Vascular Graft. The GORE® PROPATEN® Vascular Graft should be tensioned like a GORE-TEX® Stretch Vascular Graft using moderate tension per the *Instructions for Use*.

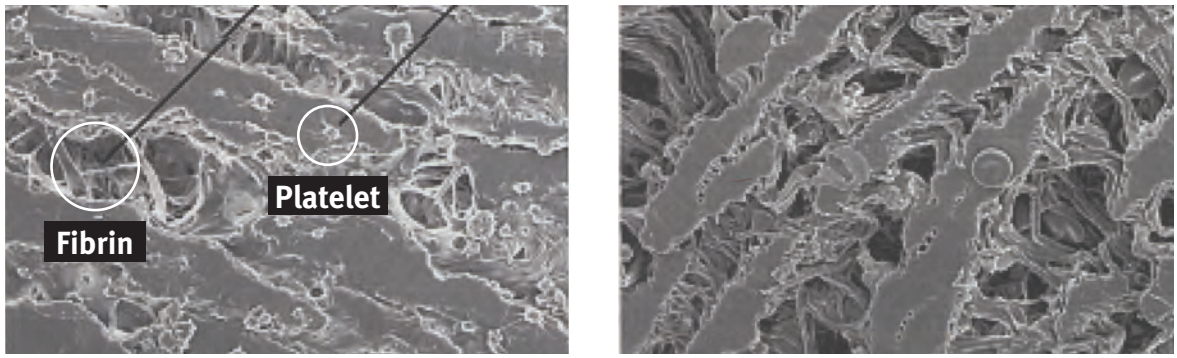
Isn't hyperplasia the real failure mode of vascular grafts? So why do you need this heparin technology?

Hyperplasia is recognized as an important failure mode of vascular grafts. Thrombotic failure is also an important failure mode, especially in the early postoperative months, and it is this failure mode that is targeted with the GORE® PROPATEN® Vascular Graft. Grafts are at greatest risk of failure in the first few postoperative months, so targeting this part of the patency curve makes sense.

There are also animal data to suggest that heparin, and specifically CBAS® heparin, may reduce and / or delay intimal hyperplasia.[4, 5, 6, 7] Small-caliber GORE® PROPATEN® Vascular Grafts have been shown to significantly reduce platelet deposition and anastomotic neointimal hyperplasia and cell proliferation in both the baboon and canine animal models.[4, 5]

Does the GORE® PROPATEN® Vascular Graft inhibit platelet deposition?

Data from a human ex vivo model,[3] a baboon ex vivo model[4] and canine model[5] show that significantly fewer platelets are deposited on a GORE® PROPATEN® Vascular Graft compared to a standard ePTFE graft.



Scanning electron microscopy showing platelet adhesion and fibrin deposition (arrows) on the untreated GORE-TEX® expanded Polytetrafluoroethylene Vascular Graft perfused for six minutes with nonanticoagulated whole blood (A) and no such adhesion or deposition on the GORE-TEX® Graft treated with CARMEDA® BioActive Surface technology (CBAS® Surface) (B). Representative images from a single human volunteer are shown.

Clinical Practice

What is the clinical experience with the GORE® PROPATEN® Vascular Graft?

Since its market introduction in 2002 the GORE® PROPATEN® Vascular Graft has been used in a variety of peripheral applications, including lower extremity revascularization and dialysis access. Published clinical evidence and over 100,000 implants worldwide support the safety and efficacy of the GORE® PROPATEN® Vascular Graft (Table 1).

In which clinical situations is the GORE® PROPATEN® Vascular Graft potentially the most beneficial?

In general, the GORE® PROPATEN® Vascular Graft is designed to improve clinical outcomes in patients with a significant risk of early thrombotic failure. To date, several prospective and retrospective investigations have been reported (Table 1). The greatest clinical benefit has been observed for **below-knee bypasses** and in patients with advanced vascular disease or poor run-off. Encouraging primary patency and limb salvage rates for up to five years post-operatively have been reported for lower extremity revascularization, especially in below-knee bypasses. For more information, please refer to the Clinical Published Literature.

How does the GORE® PROPATEN® Vascular Graft compare to current treatment modalities in below-knee bypasses?

Numerous studies have shown that the GORE® PROPATEN® Vascular Graft achieves one-year primary patencies that approach those of autologous vein grafts. A review of literature reveals one-year primary patency of 79% and 60% for vein and ePTFE bypasses, respectively, for below-knee revascularization (Table 1). The overall weighted average primary patency of published studies on the GORE® PROPATEN® Vascular Graft is 76% for below-knee bypasses.

Table 1

Primary Patency in Below-knee Bypass (1-year)	
Vein (N = 11,956)	79%*
GORE® PROPATEN® Vascular Graft (N = 743)	76%†
ePTFE Vascular Graft (N = 2,660)	60%*
Primary Patency in Below-knee Bypass (2-year)	
Vein (N = 10,458)	77%*
GORE® PROPATEN® Vascular Graft (N = 662)	68%†
ePTFE Vascular Graft (N = 2,339)	47%*
Primary Patency in Below-knee Bypass (3-year)	
Vein (N = 9,867)	75%*
GORE® PROPATEN® Vascular Graft (N = 477)	60%†
ePTFE Vascular Graft (N = 1,982)	40%*

** Data based on an analysis of current literature: several Medline® database searches were performed to identify publications pertaining to ePTFE synthetic vascular graft and vein infragenicular bypasses. Search criteria included (1) articles published from January 2000 to January 2012, (2) key words used were below knee, polytetrafluoroethylene, prosthetic, bypass, patency, (3) articles in the English language, (4) N equal or greater than 30 bypasses, (5) clinical publications, (6) reviews, case reports or meta-analysis articles were excluded, (7) articles containing the key word AV access (including synonyms) were excluded. Articles that did not meet the above criteria were deemed ineligible for this analysis. In studies where 1-year and 3-year patency data were reported, but 2-year patency data were not reported, the 2-year patency rate used in this analysis was interpolated as the average of the 1-year and 3-year patency rates. Data of analysis on file.*

Has there been a randomized clinical study comparing the GORE® PROPATEN® Vascular Graft against vein?

Several non-randomized, prospective and retrospective clinical studies have been undertaken and other studies are currently ongoing. These studies have provided significant insight into the clinical benefits of this bioactive, heparin-bonded graft. There are currently no published randomized studies comparing the GORE® PROPATEN® Vascular Graft to vein.

What kind of antiplatelet and / or anticoagulation regimen is recommended?

The physician should determine the appropriate intraoperative and postoperative antiplatelet and / or anticoagulation therapy based on the pharmacological requirements and medical history of the patient. A prospective, randomized trial has shown that clopidogrel plus acetylsalicylic acid confers benefit in patients receiving prosthetic grafts for below-knee bypass without significantly increasing major bleeding risks.[8] The presence of heparin on the GORE® PROPATEN® Vascular Graft is not intended to serve as an alternative to intraoperative or postoperative anticoagulation. The anticoagulant effect of the CBAS® Surface is limited to the device surface and does not have a systemic anticoagulant effect.[9]

What happens if the GORE® PROPATEN® Vascular Graft is clamped – does it damage the bioactivity of the graft?

Graft clamping, according to recommended procedure, has no effect on the heparin surface. The heparin bonding is very stable and is not easily removed by mechanical methods. As with any prosthetic vascular graft, atraumatic or guarded clamps should be used and repeated, localized clamping of the same graft section should be avoided.

What kind of suture may be used with the GORE® PROPATEN® Vascular Graft?

Either GORE-TEX® Suture (Table 2) or polypropylene suture may be used with the GORE® PROPATEN® Vascular Graft.

Table 2 – Commonly Requested GORE-TEX® Sutures for Lower Extremity Bypass Procedures

Proximal Anastomoses			
Thread Size	Needles	Thread Length (cm)	Catalogue Number
CV-5	PT-13	91	5N08
	TTc-13	91	**5N02**
CV-6	TTC-12	76	**6M10**
	PT-13	76	6M08
	TTC-13	76	6M04
	TTc-9	79	6M02
Distal Anastomoses			
CV-6	PT-9	61	6K06
	TTc-9	76	**6M02**
CV-7	PT-9	76	7M04
	TTc-9	61	7K02
	TTc-9	76	7M02
CV8	TTc-9	76	8M02

Numbers in bold indicate 1:1 needle to thread ratio to minimize suture hole bleeding

Have you seen any difference in anastomotic bleeding compared to other ePTFE grafts?

No significant differences in anastomotic bleeding have been observed with the GORE® PROPATEN® Vascular Graft as compared to other ePTFE grafts.

Is there any difference in perioperative or postoperative bleeding?

Since the GORE® PROPATEN® Vascular Graft is designed to provide anticoagulant activity at the graft surface, systemic anticoagulation remains unaffected. This explains why no differences in perioperative or postoperative bleeding have been reported. Furthermore, a prospective, randomized, multicenter study comparing the GORE® PROPATEN® Vascular Graft to standard ePTFE grafts showed no difference in perioperative bleeding.[10]

What is the effect of Protamine on the GORE® PROPATEN® Vascular Graft?

Although Protamine reverses the anticoagulant activity of heparin, its effect is transient. Protamine can only remain bound to heparin when it is present in sustained excess quantities. Since Protamine is rapidly removed from the circulation, any effect is short-lived.

What is HIT?

Heparin-Induced Thrombocytopenia (HIT) occurs in a relatively small subset of the patient population and is defined as a decrease in platelet count during or shortly following exposure to heparin.[11] There are two distinct types of HIT, each with very different clinical ramifications.

HIT type I is characterized by a mild and transient asymptomatic thrombocytopenia that develops within the first few days of starting heparin treatment and disappears quickly following heparin cessation.[12] This type of HIT is benign and is not associated with an increased risk of thrombosis.

HIT type II is characterized by rapid or delayed-onset thrombocytopenia that is associated with a risk of thrombosis.[12] In the following discussion, HIT type II will be referred to simply as HIT. The mechanism underlying HIT is an immune response as antibodies are formed against a heparin-platelet factor 4 (PF4) complex. The antibody-heparin-PF4 immunocomplex binds to platelets, inducing platelet activation and aggregation. Thrombocytopenia results from the clearance of activated platelets and antibody-coated platelets by the reticulo-endothelial system.[13, 14] Typically, HIT patients receiving heparin for the first time experience the onset of thrombocytopenia five – fourteen days after the administration of heparin; however, the onset can be rapid (< one day) in patients with antibodies from a previous exposure[15, 16, 22] or delayed up to three weeks after heparin therapy has stopped.[17-20]

Can the CBAS® Surface on the GORE® PROPATEN® Vascular Graft cause or contribute to the condition of HIT?

Available data on HIT and GORE® Vascular Devices suggest that the risk of developing HIT due to the covalently immobilized heparin in the CBAS® surface on GORE® Vascular Devices is very low.

In controlled, multi-patient studies, there is no evidence of a link between HIT and the presence of GORE® Vascular Devices with an immobilized heparin surface. *(For more information please refer to the HIT Flyer, available on the Gore Medical website.)*

What treatment protocol should I follow if a GORE® PROPATEN® Vascular Graft patient develops HIT?

The incidence of HIT type II is low in vascular bypass patients receiving systemic heparin over a period of several days. If HIT type II is diagnosed, established procedures for the treatment of this condition, including immediate cessation of systemic heparin administration, should be followed.[20] If symptoms persist, alternative procedures may be considered at the discretion of the attending physician. In some patients with suspected HIT, GORE® PROPATEN® Vascular Grafts have remained implanted without HIT-related clinical sequelae.[21]

Can a GORE® PROPATEN® Vascular Graft be implanted in patients with existing HIT?

The GORE® PROPATEN® Vascular Graft is contraindicated for use in patients with known hypersensitivity to heparin, including those patients who have had a previous (or existing) incident of HIT type II.

Can the GORE® PROPATEN® Vascular Graft be revised?

All standard revision procedures may be performed on the GORE® PROPATEN® Vascular Graft, including lytic therapy and balloon thrombectomy. The heparin surface remains intact even after repeated in vitro pseudo balloon thrombectomy procedures (data on file).

Does a thrombectomy procedure damage the heparin bonding?

In vitro tests have shown that the heparin surface is still intact even after an inflated thrombectomy balloon was pulled through the graft three times (data on file).

Clinical Published Literature

$$\dagger \quad \text{Weighted Average} = \frac{(N_1 \times \text{Primary Patency}_1) + (N_2 \times PP_2) + \ldots + (N_n \times PP_n)}{N_1 + N_2 + \ldots + N_n}$$

(calculated from first seven references: published clinical studies of below-knee bypasses, excluding congress abstracts and duplicate patient populations)

Daenens K, Schepers S, Fourneau I, Houthoofd S, Nevelsteen A. Heparin-bonded ePTFE grafts compared with vein grafts in femoropopliteal and femorocrural bypasses: 1- and 2-year results. *Journal of Vascular Surgery* 2009;49(5):1210-1216.

Hugl B, Nevelsteen A, Daenens K, *et al*; PEPE II Study Group. PEPE II - a multicenter study with an end-point heparin-bonded expanded polytetrafluoroethylene vascular graft for above and below knee bypass surgery: determinants of patency. *Journal of Cardiovascular Surgery* 2009;50(2):195-203.

Kirkwood ML, Wang GJ, Jackson BM, Golden MA, Fairman RM, Woo EY. Lower limb revascularization for PAD using a heparin-coated PTFE conduit. *Vascular & Endovascular Surgery* 2011;45(4):329-334.

Lösel-Sadée H, Alefelder C. Heparin-bonded expanded polytetrafluoroethylene graft for infragenicular bypass: 5-year results. *Journal of Cardiovascular Surgery* 2009;50(3):339-343.

Peeters P, Verbist J, Deloose K, Bosiers M. Will heparin-bonded PTFE replace autologous venous conduits in infrapopliteal bypass? *Italian Journal of Vascular & Endovascular Surgery* 2008;15(3):143-148.

Pulli R, Dorigo W, Castelli P, *et al*; Propaten Italian Registry Group. Midterm results from a multicenter registry on the treatment of infrainguinal critical limb ischemia using a heparin-bonded ePTFE graft. *Journal of Vascular Surgery* 2010;51(5):1167-1177.

Walluscheck KP, Bierkandt S, Brandt M, Cremer J. Infrainguinal ePTFE vascular graft with bioactive surface heparin bonding-first clinical results. *Journal of Cardiovascular Surgery* 2005;46(4):425-430.

Battaglia G, Tringale R, Monaca V. Retrospective comparison of a heparin bonded ePTFE graft and saphenous vein for infragenicular bypass: implications for standard treatment protocol. *Journal of Cardiovascular Surgery* 2006;47(1):41-47.

Bosiers M, Deloose K, Verbist J, *et al*. Heparin-bonded expanded polytetrafluoroethylene vascular graft for femoropopliteal and femorocrural bypass grafting: 1-year results. *Journal of Vascular Surgery* 2006;43(2):313-319.

Dorigo W, Di Carlo F, Troisi N, *et al*. Lower limb revascularization with a new bioactive prosthetic graft: early and late results. *Annals of Vascular Surgery* 2008;22(1):79-87.

Dorigo W, Pulli R, Alessi Innocenti A, *et al*. Lower limb below-knee revascularization with a new bioactive prosthetic graft: a case-control study. *Italian Journal of Vascular & Endovascular Surgery* 2005;12(3):75-81.

Dorigo W, Pulli R, Castelli P, *et al*; Propaten Italian Registry Group. A multicentric comparison between autologous saphenous vein and heparin-bonded expanded polytetrafluoroethylene (ePTFE) graft in the treatment of critical limb ischemia in diabetics. *Journal of Vascular Surgery*. In press

Dorrucci V, Griselli F, Petralia G, Spinamano L, Adornetto R. Heparin-bonded expanded polytetrafluoroethylene grafts for infragenicular bypass in patients with critical limb ischemia: 2 year results. *Journal of Cardiovascular Surgery* 2008;49(2):145-149.

Lindholt JS, Gottschalksen B, Johannesen N, *et al*. The Scandinavian Propaten® Trial – 1-year patency of PTFE vascular prostheses with heparin-bonded luminal surfaces compared to ordinary pure PTFE vascular prostheses – a randomised clinical controlled multi-centre trial. *European Journal of Vascular & Endovascular Surgery* 2011;41(5):668-673

Peeters P, Verbist J, Deloose K, Bosiers M. Results with heparin bonded PTFE grafts for femorodistal bypasses. *Journal of Cardiovascular Surgery* 2006;47(4):407-413.

References

1. Begovac PC, Thomson RC, Fisher JL, Hughson A, Gällhagen A. Improvements in GORE-TEX® Vascular Graft performance by Carmeda® bioactive surface heparin immobilization. *European Journal of Vascular and Endovascular Surgery* 2003;25(5):432-437.
2. Fisher JL, Thomson RC, Moore JW, Begovac PC. Functional parameters of thromboresistant heparinized e-PTFE vascular grafts. *Cardiovascular Pathology* 2002;11(1):42.
3. Heyligers JMM, Lisman T, Weeterings C, *et al.* Heparin immobilization reduces thrombogenicity on small-caliber ePTFE grafts. *Journal of Vascular Surgery* 2006;43:587-591.
4. Lin PH, Chen C, Bush RL, Yao Q, Lumsden AB, Hanson SR. Small-caliber heparin-coated ePTFE grafts reduce platelet deposition and neointimal hyperplasia in a baboon model. *Journal of Vascular Surgery* 2004; 39(6):1322-1328.
5. Lin PH, Bush RL, Yao Q, Lumsden AB, Chen C. Evaluation of platelet deposition and neointimal hyperplasia of heparin-coated small-caliber ePTFE grafts in a canine femoral artery bypass model. *Journal of Surgical Research* 2004;118:45-52.
6. Kocsis JF, Llanos G, Holmer E. Heparin-coated stents. *Journal of Long-Term Effects of Medical Implants* 2000;10(1&2):19-45.
7. Chen C, Lumsden AB, Hanson SR. Local infusion of heparin reduces anastomotic neointimal hyperplasia in aortoiliac expanded polytetrafluoroethylene bypass grafts in baboons. *Journal of Vascular Surgery* 2000;31(2):354-363.
8. Belch JJ, Dormandy J, Biasi GM, *et al*; CASPAR Writing Committee. Results of the randomized, placebo-controlled clopidogrel and acetylsalicylic acid in bypass surgery for peripheral arterial disease (CASPAR) trial. *Journal of Vascular Surgery* 2010;52(4):825-833.
9. Heyligers JMM, Lisman T, Verhagen HJM, Weeterings C, de Groot PG, Moll FL. A heparin-bonded vascular graft generates no systemic effect on markers of hemostasis activation or detectable heparin-induced thrombocytopenia–associated antibodies in humans. *Journal of Vascular Surgery* 2008;47(2):324-329.
10. Lindholt JS, Gottschalksen B, Johannesen N, *et al*. The Scandinavian Propaten® Trial – 1-year patency of PTFE vascular prostheses with heparin-bonded luminal surfaces compared to ordinary pure PTFE vascular prostheses – a randomised clinical controlled multi-centre trial. *European Journal of Vascular & Endovascular Surgery* 2011;41(5):668-673.
11. Warkentin TE. An overview of the heparin-induced thrombocytopenia syndrome. *Seminars in Thrombosis & Hemostasis* 2004;30(3):273-283.
12. Franchini M. Heparin-induced thrombocytopenia: an update. *Thrombosis Journal* 2005;3(1):14-18.
13. Jang IK, Hursting MJ. When heparins promote thrombosis: review of heparin-induced thrombocytopenia. *Circulation* 2005;111(20):2671-2683.
14. Chong BH. Heparin-induced thrombocytopenia. *Journal of Thrombosis & Haemostasis* 2003;1(7):1471-1478.
15. Lubenow N, Kempf R, Eichner A, Eichler P, Carlsson LE, Greinacher A. Heparin-induced thrombocytopenia: temporal pattern of thrombocytopenia in relation to initial use or reexposure to heparin. *Chest* 2002;122(1):37-42.
16. Warkentin TE, Kelton JG. Temporal aspects of heparin-induced thrombocytopenia. *New England Journal of Medicine* 2001;344(17):1286-1292.
17. Jackson MR, Neilson WJ, Lary M, Baay P, Web K, Clagett GP. Delayed-onset heparin-induced thrombocytopenia and thrombosis after intraoperative heparin anticoagulation-four case reports. *Vascular & Endovascular Surgery* 2006;40(1):67-70.
18. Rice L, Attisha WK, Drexler A, Francis JL. Delayed-onset heparin-induced thrombocytopenia. *Annals of Internal Medicine* 2002;136(3):210-215.
19. Smythe MA, Stephens JL, Mattson JC. Delayed-onset heparin-induced thrombocytopenia. *Annals of Emergency Medicine* 2005;45(4):417-419.
20. Warkentin TE, Kelton JG. Delayed-onset heparin-induced thrombocytopenia and thrombosis. *Annals of Internal Medicine* 2001;135(7):502-506.
21. Kasirajan K. Outcomes following heparin-induced thrombocytopenia in patients with heparin bonded vascular grafts. Abstract presented at the 2011 Vascular Annual Meeting; June 16-18, 2011; Chicago, IL. *Journal of Vascular Surgery* 2011;53(6)Supplement 1:17S-18S. Abstract SS4.
22. Warkentin TE, Grienacher A, Koster A, Lincoff AM. Treatment and prevention of heparin-induced thrombocytopenia: American College of Chest Physicians evidence-based clinical practice guidelines (8th edition). *Chest* 2008;133:340S-380S.

*Sustained CBAS® Surface heparin bioactivity has been measured in a controlled three-month animal study.[1]

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