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Medical Compression Stockings Significantly Increase Local Tissue Factor Levels in Advanced Chronic Venous Insufficiency Patients and Healthy Volunteers

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Objective: Evidence that medical compression stockings (MCS) prevent deep venous thrombosis (DVT) is weak. Furthermore, the body position that predisposes to DVT is not fully known. It is assumed that standing is protective through involuntary leg muscle contractions and that lying stationary may provoke DVT. Previous work using ultrasound has shown the presence of venous sludge in the popliteal veins, in both positions. The aim was to investigate the effect of standing, lying, and compression on thrombogenicity. This was achieved by taking local venous blood samples and measuring an array of factors considered relevant in thrombogenesis.

Methods: Patients with advanced chronic venous insufficiency awaiting endothermal ablation (C4a, C4b) and healthy volunteers (n = 14 legs in each group, 1 leg per patient) had local leg blood samples taken after 1 hour of standing, lying, and standing with compression on separate days. Knee-length MCS of 23 to 32 mm Hg were administered. Platelet-poor plasma samples were analyzed for procoagulant phospholipids, tissue factor, D-dimer, fibrin monomer, and factor VIIa-antithrombin complexes. This was in addition to a thrombin generation test using the PPP-reagent, which measured lag time, endogenous thrombin potential, peak, time to peak, and mean rate index.

Results: The most responsive was tissue factor, with significant increases after an MCS was worn in standing compared with lying and standing with compression in both volunteers and patients (Fig 1). Standing and compression made no difference to D-dimer or factor VIIa-antithrombin levels in either group (Table). Thrombin generation testing revealed no differences in the volunteer group, but in the patients, compression appeared to have a favorable significant effect in four of five measurements compared with lying (Table).

Conclusions: Local tissue factor concentrations were elevated significantly with MCS. This was on a background of unaffected D-dimer and factor VIIa-antithrombin levels, thereby questioning the thrombogenic significance of the elevated tissue factor. However, compression reduced thrombin generation parameters, but only in patients with chronic venous insufficiency. Patients also demonstrated significantly reduced thrombin generation when standing compared with lying. This research supports our hypothesis that standing and compression may offer additional protection from thrombosis, but only in patients.

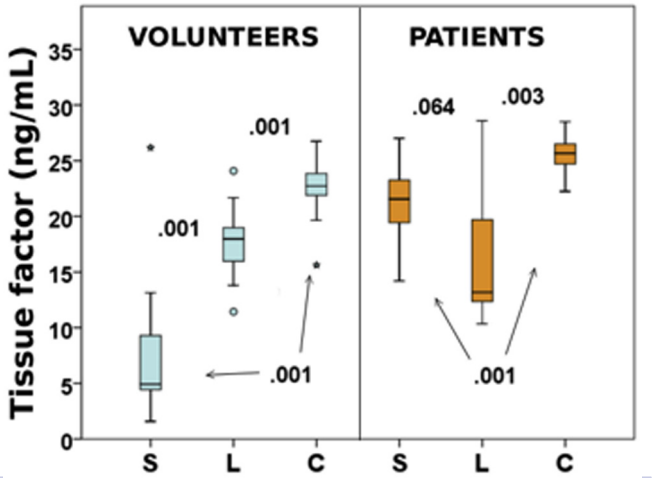

Fig. Local tissue factor concentrations in response to standing (*S*), lying (*L*), and compression (*C*) in volunteers and patients. Significance levels are shown (Wilcoxon).

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Effectiveness of the PowerWire Radiofrequency Guidewire in Recanalizing Chronically Occluded Iliac Venous Stents

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Objective: Iliac venous stenting is a commonly performed procedure in treating post-thrombotic and nonthrombotic iliac venous disease. Whereas overall stent patency rates are high, stent occlusion does occur. Recanalizing chronically occluded stents is technically challenging and often impossible with conventional guidewires and catheters. The PowerWire (Baylis Medical, Burlington, Mass) radiofrequency guidewire is a 0.035-inch guidewire that delivers radiofrequency energy at the end of the guidewire.

Methods: A retrospective chart review was conducted of patients with chronically occluded iliac venous stents who underwent an iliac venous recanalization attempt using the PowerWire from March 2015 to June 2018. A total of 15 patients underwent a recanalization attempt with the PowerWire. All patients had initial unsuccessful attempts at recanalizing the occluded iliac vein stents with conventional guidewires.

Table. Median (interquartile range) values in three different laboratory situations for 1 hour

	Volunteers			Patients (C4)		
Parameters	Standing	Lying	Compression	Standing	Lying	Compression
Procoagulant phospholipids, $	**67.7 (63.5-80)**[a]	76.7 (66.6-92.6)	71.5 (67-80.7)	82.8 (74.7-91.2)	89.5 (76.3-96.5)	**77.1 (67.6-81.8)**[a]
Tissue factor, ng/mL	**4.94 (4.03-10)**[a]	17 (15.5-19.1)	**22.7 (21.7-23.9)**[b]	21.6 (18.7-23.3)	13.2 (12.2-20.4)	**25.7 (24.6-26.7)**[b]
D-dimer, µg/mL	0.27 (0.27-0.34)	0.27 (0.27-0.32)	0.27 (0.27-0.45)	0.35 (0.27-0.9)	0.34 (0.28-0.63)	0.29 (0.27-0.53)
Fibrin monomer, µg/mL	5 (5-5.8)	5 (5-60.3)	5 (5-50.4)	5 (5-7.32)	5 (5-5.07)	5 (5-5)
Factor VIIa, units/mL	74.7 (55.2-300)	71.9 (54.3-54.5)	**17.3 (10.6-241)**[a]	26.7 (19.7-55.5)	28.1 (19.9-55.9)	32.5 (24.6-67.5)
Lag time, minutes	6.74 (5.49-8.92)	5.91 (4.74-7.16)	5.67 (4.67-6.93)	6.75 (5.04-9.13)	6.46 (5.06-7.42)	7.25 (5.33-8.88)
ETP, µM/min	1.1 (0.75-1.25)	1.1 (0.72-1.72)	1 (0.77-1.54)	0.94 (0.84-1.08)	1.08 (0.68-1.38)	0.96 (0.43-1.11)[a]
Peak, nM	150 (95.5-194)	1.69 (95.2-349)	136 (97.3-291)	150 (101-182)	**175 (108-214)**[a]	**150 (62.8-185)**[a]
ttPeak, minutes	10.8 (9-12.3)	8.83 (7.87-11.2)	9.83 (7.55-10.8)	10.8 (8.51-13.7)	**10 (7.9-11.9)**[a]	**10.9 (8.67-12.9)**[a]
MRI	40.8 (21.7-61.1)	48.6 (24.3-150)	36.7 (24.1-107)	44.9 (21.3-56.6)	**51.3 (31.3-69.4)**[a]	**41.9 (18.8-56.7)**[a]
Factor VIIa-antithrombin, ρM	102 (82.5-179)	115 (57.4-227)	82.9 (53.6-194)	118 (91.4-161)	111 (97-133)	118 (101-146]

ETP, Endogenous thrombin potential; *MRI,* mean rate index; *ttPeak,* time to peak.
[a]$P < .05$ vs standing.
[b]$P < .05$ vs lying.