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Experimental determination of the best time and duration for endovenous great saphenous vein electrocoagulation

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Objective: **Endovenous electrocoagulation provokes immediate selective venous wall necrosis. In this study, we aim to determine the best power and time of electrocoagulation necessary to cause intima and media but not adventitia layer damage in great saphenous vein (GSV) insufficiency treatment.**

Methods: **We studied 100 varicose GSV fragments submitted to endovenous electrocoagulation. The power (60, 90, or 120 W) and time (5, 10, or 15 seconds) were randomly assigned. The fragments were submitted to histopathologic examination to analyze the depth of tissue necrosis. Dose-response models for the analysis of binary data were used to identify the best association between power and the time of electrocoagulation necessary to cause intima and media but not adventitia layer necrosis. We also applied a logistic regression model to investigate the impact of body mass index and GSV diameter on the electrocoagulation effects.**

Results: **The time (odds ratio [OR], 1.26; $P = .0009$) was found to be a stronger predictor of the depth of vessel necrosis than the power of electrocoagulation applied (OR, 1.05; $P < .0001$). The power and time that were most likely to cause intima and media but not adventitia layer destruction were 60.4 W × 5 seconds, 58.8 W × 10 seconds, and 8.9 W × 15 seconds. The initial GSV diameter (median, 5.36 mm; minimum, 2.3 mm; maximum, 10 mm; OR, 0.96; $P = .82$) and body index mass (median, 24.7 kg/m^2; minimum, 15.6 kg/m^2; maximum, 36.2 kg/m^2; OR, 1.08; $P = .26$) showed a poor correlation with the depth of histologic vessel destruction.**

Conclusions: **The time of electrocoagulation strongly predicts the depth of GSV wall necrosis more than the amount of power applied. Determination of the best time and power of electrocoagulation ratio may help optimize GSV endovenous electrocoagulation closure rates and decrease the complications index. The GSV diameter and body mass index do not influence endovenous electrocoagulation effects. (J Vasc Surg: Venous and Lym Dis 2014;2:315-9.)**

Clinical Relevance: **The successful treatment of lower extremity chronic venous insufficiency includes the elimination of all sources of venous reflux. Endovenous ablation of varicose veins with radiofrequency ablation and endovenous laser therapy has reported advantages over traditional open surgical treatment but is costly. We have described a simple endovenous electrocoagulation apparatus and technique that provoke immediate selective venous wall damage and use a conventional electrosurgical generator as the energy source. In this study, we aim to determine the best power and the time of electrocoagulation necessary to cause intima and media but not adventitia layer damage in lower limb varicose vein treatment.**

Chronic venous insufficiency affects 20% of the adult population, with varicosities in the great saphenous vein (GSV) distribution being the most common manifestation. The standard treatment has historically been high GSV ligation and stripping. In recent years, many surgeons have adopted endovascular techniques with good results.[1,2]

In our prior publications, we have described an endovenous electrocoagulation apparatus and technique that provoke immediate selective venous wall damage in an animal model.[3] Furthermore, we demonstrated the same effect in human varicose veins, including the fact that the time of electrocoagulation strongly predicts the depth of vessel wall necrosis more than the power of energy applied.[4]

In this study, we aim to determine the best power and the time of electrocoagulation necessary to cause intima and media but not adventitia layer damage in lower limb varicose vein treatment.

METHODS

The study was conducted according to the Helsinki Declaration. The experimental protocol and informed consent were approved by the Institutional Review Board. All the study subjects gave informed consent with local ethical committee approval (IDPC-FMUSP/CEP 3904/2010).

We studied 100 varicose vein fragments obtained from 78 patients with clinical, etiologic, anatomic, and pathologic classes 3 to 6; GSV insufficiency with venous diameters between 2.3 and 10 mm (mean, 5.36 mm) was documented by ultrasound examination. Subjects with GSV diameter >12 mm and <2 mm, acute or previous phlebitis, previous surgery or sclerotherapy in the study leg, previous or current

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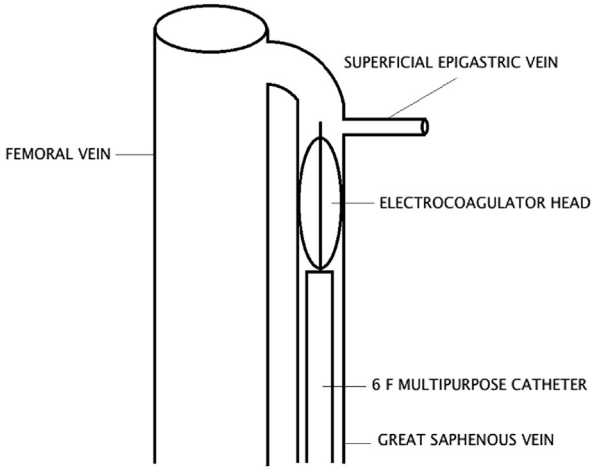

Fig 1. Endovenous electrocoagulation apparatus positioned at the proximal portion of the great saphenous vein (GSV).

deep venous thrombosis, previous coagulopathy history (congenital or acquired thrombophilia or a prothrombotic state), arterial occlusive disease, active malignant disease, pregnancy, multiple saphenous aneurysms (segmental varicose vein dilatations, two times the adjacent GSV diameters), or congenital malformations were excluded.

Patients were submitted to standard surgical groin and ankle GSV dissection. Just before high ligation and stripping of the GSV, an endovenous electrocoagulation apparatus was positioned immediately beneath the superficial epigastric vein (Fig 1). The diameter of this segment was measured in millimeters and submitted to endovenous electrocoagulation with use of the Valleylab FX Electrosurgical Generator (Covidien, Mansfield, Mass) as the energy source. The energy intensity, power, and time of electrocoagulation were determined according to a randomization table. In a previous study, we observed that electrocoagulation with 120 W for 15 seconds could provoke macroscopic GSV shrinkage, induration, and inside carbonization. We fixed this as the highest dose and randomly studied the histologic effects of 60, 90, and 120 W per 5, 10, and 15 seconds[4] (Table I). Immediately after the procedure, the temperature adjacent to the vessel was measured (TD-100 thermometer; ICEL, Manaus, Brazil).

The venous fragments submitted to electrocoagulation (20 mm) were extracted and fixed by 75% alcohol; the paraffin inclusions were stained with hematoxylin and eosin, cross-sectioned, and submitted to light microscopy. Presence of vacuolization, delamination, coagulation, loss of tissue, perforation, nuclear rarefaction and pyknosis with disappearance of the cellular membrane, and cytoplasm fusion due to the coagulation process were investigated and considered signs of electrocoagulation effects.[5,6] The damage of the venous wall was then classified according to the depth of appearance of these effects: group A, intima and media necrosis; and group B, intima, media, and adventitia necrosis. In the postoperative period, occurrence of deep venous thrombosis was investigated by duplex scanning done immediately before the patient's hospital discharge and 30 days after the procedure.

For safety reasons, we checked the best association between power (watts) and the time (seconds) of electrocoagulation necessary to cause intima and media (group A) but not adventitia layer necrosis (group B). Dose-response models for the analysis of binary data were used for this investigation and tolerated the presence of vessel perforation in 10% of the cases.

A logistic regression model was applied to investigate the impact of body mass index, saphenous vein initial diameter, and temperature on the depth of vessel wall necrosis caused by the electrocoagulation. The test was considered statistically significant when $P < .05$.

RESULTS

The histologic evaluation of the studied fragments showed damage to the intima in all specimens, full-thickness vessel injury in 53 specimens (53%), and perforation in 1 (1%) (Table II). Samplings of the vessel wall circumference damages caused by the electrocoagulation are shown in Fig 2. The temperature reached at the tissue adjacent to the electrocoagulation (median, 51.6°C; minimum, 32°C; maximum, 82.2°C; $P = .0006$) and the depth of vessel destruction ($P < .0005$) were correlated to the energy of electrocoagulation applied.

The initial GSV diameter (median, 5.36 mm; minimum, 2.3 mm; maximum, 10 mm; odds ratio [OR], 0.96; $P = .82$) and body index mass (median, 24.7 kg/m^2; minimum, 15.6 kg/m^2; maximum, 36.2 kg/m^2; OR, 1.08; $P = .26$) showed a poor correlation with the depth of histologic vessel destruction. The time of electrocoagulation (OR, 1.26; $P = .0009$) was found to be a stronger predictor of this phenomenon than the power used (OR, 1.05; $P < .0001$) (Table III).

We fixed the electrocoagulation time and measured what would be the power that minimizes the chances of adventitia layer necrosis and considered that this fact would be tolerable in 10% of the cases. We found that the best power × time ratio would be 60.4 W × 5 seconds, 58.8 W × 10 seconds, and 8.9 W × 15 seconds (Fig 3).

DISCUSSION

Laser and radiofrequency energy causes thermal ablation of the inner layers of the vessel and promotes their

Table I. Endovenous electrocoagulation randomization table

Group	*Energy (J)*	*Power (W)*	*Time, seconds*
I	0	0	15
II	300	60	5
III	600	60	10
IV	900	60	15
V	450	90	5
VI	900	90	10
VII	1350	90	15
VIII	600	120	5
IX	1200	120	10
X	1800	120	15