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Regarding "Stroke after varicose vein foam injection sclerotherapy"

We read with great interest Forlee et al's report of a patient who experienced an ischemic stroke moments after undergoing foam injection sclerotherapy for treatment of varicose veins.[1] The patient was later determined to have a patent foramen ovale. We commend the authors for having the presence of mind to perform a carotid duplex scan immediately to reveal what no doubt were intracarotid bubbles resulting from the foam injection. We note that the patient received a total of 20 mL of polidocanol foam prepared by the double syringe method using room air.

We previously demonstrated, using an in vivo model of arteriolar embolization after polidocanol microfoam sclerosant administration, that two prominent—and potentially controllable—features of foam manufacture contribute to the number and size of bubbles present as well as the resultant duration of blood flow obstruction that is caused by the intravascular gas load.[2] We found that foam made with room air, rather than a gas admixture comprised of carbon dioxide and oxygen, was directly associated with increased bubble number and size and caused the longest obstruction of blood flow. We attribute this to the difference in nitrogen gas content, as nitrogen is far less soluble and diffusible in tissues than are metabolic gases. We also found that bubbles made by the double syringe technique were larger than those created using mechanisms specifically engineered to dispense microfoams having a highly controlled bubble size distribution.

We are relieved that the patient recovered significantly, but we are not surprised by this report of a patient with a patent foramen ovale experiencing a stroke after foam injection sclerotherapy. Although we do not think that more careful attention to patient cardiac anatomy through echocardiographic screening is an effective means of improving patient safety for this treatment, we do believe that our previous findings regarding gas content and foam formation and this case report illustrate the need to change clinical practice regarding what is injected, and not into whom, to assure procedural safety.

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Reply

We would like to thank Dr Morrison and Dr Eckmann and their colleagues for the interest they have shown and the important points they raise in response to our recent case report.[1]

The great saphenous vein was cannulated first in order to minimize the delay between foam production and injection. Foam was produced using the Tessari method, with a 4:1 air-to-liquid ratio. Half the volume of foam was injected with the leg on the level, while compressing the saphenofemoral junction. The leg was then elevated and the rest of the foam was injected and massaged into the targeted veins using ultrasound guidance. Compression stockings were applied with the leg elevated.

With regard to the volume of foam used, we note that in Dr Morrison's study using different volumes of foam, our patient would have been classified in the "low volume" group. Juan Cabrera, the creator of the patented polidocanol foam, writes that volumes of 20 mL to 100 mL of foam can be safely used.[2] There is, however, a lack of consensus regarding the optimal volume, and the European Consensus statement recommends limiting volumes to 8 mL per treatment using the Tessari method.[3]

Given the high prevalence of a patent foramen ovale in the general population, it is surprising that more events have not been reported. This would imply that most are small and hemodynamically insignificant. We agree that screening before foam injection would be impractical and probably unnecessary.

Carbon dioxide is absorbed faster than air in the body and has been shown to produce a foam that degrades quicker.[4] The transient visual symptoms reported in the literature[5] are possibly due to small amounts of air embolism. An argument could thus be made for carbon dioxide to be used as the carrier gas.

We agree with Drs Eckmann and Kobayashi that the quality of foam produced is very important, not only with regard to safety but also efficacy of the procedure. For maximum stability, the size of the bubbles in the microfoam should ideally be $<100\mu m$, spherical, and of uniform size.[6] With lack of uniformity in the size of the bubbles, La Place's Law ($t = p/r$) dictates that the smaller bubbles will empty into the bigger bubbles, resulting in larger bubbles with an increased potential for the air-block effect. Although the Tessari and other methods have been shown to be effective in producing a foam that meets these criteria, it is difficult to accurately regulate or measure bubble size and quality of foam in the clinical setting. This may be a strong argument for the use of the standardized, commercially produced microfoam preparation when treating varicose veins.

Foam sclerotherapy has been shown to be safe and efficacious. Our report describes a rare, but potentially life threatening complication of this treatment.

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Regarding "Preservation of infected and exposed vascular grafts using vacuum assisted closure without muscle flap coverage"

We congratulate Dosluoglu et al for their excellent results using vacuum-assisted closure (VAC) therapy for the preservation of infected and exposed bypass grafts (J Vasc Surg 2005;42:989-92). We have used VAC therapy for >5 years with highly satisfactory results in similar cases.

However, we recently experienced anastomotic disruption and bleeding in a 73-year-old patient with an infected and exposed silver-coated polyester (InterGard Silver, Datascope, Montvale, NJ) axillary unifemoral bypass graft in the right groin. Central vascular reconstruction was not possible because of high cardiac risks, and the left groin was unaccesible because of a low-grade infection after reconstruction for a pseudoaneurysm that followed iliacofemoral prosthetic surgery.

The patient presented 10 days after axillofemoral bypass with a deep infection of the right groin. *Staphylococcus aureus* was cultured and treated with intravenous antibiotics. No systemic sign of sepsis existed. We performed a wound débridement and started VAC therapy that same day with constant suction of 75 mm Hg. A polyvinylalcohol sponge was used because of its less ingrowth characteristics compared with polyurethane sponges.

On the third day progressive swelling developed in the patient's right groin and the vacuum system was occluded after collecting 1 liter of blood. An emergency operation revealed disruption of the distal anastomosis. We removed the bypass, used venous patches to close the proximal and distal anastomosis, and in a second operation, we had to perform an above-knee amputation for severe ischemia of the leg.

We describe this case to alert readers that while VAC therapy is often successful, it does not always control infection and can be associated with anastomotic disruption.

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Reply

We would like to thank Dr. Brehm for his kind remarks about our paper regarding the use of vacuum-assisted closure (VAC) for graft preservation in patients with exposed grafts. His letter addresses the possibility of arterial "blow-out" associated with use of VAC, a complication we have (fortunately) not seen. Understanding this concern, we will emphasize a number of points that may reduce the risk of this life-threatening complication.

1. The importance of controlling infection with radical débridement, culture-directed antibiotics, and serial wound explorations cannot be overemphasized, as this is a prerequisite for graft preservation. This is not only true for VAC systems but also for those in whom muscle flap is contemplated. Graft preservation (with VAC or muscle flaps) cannot be accomplished unless the bacterial load is minimized. Case selection for graft preservation is extremely important, and graft removal should be considered when eradication of infection is not achieved.
2. We do not advocate starting VAC therapy early after the initial débridement. In our series, VAC was begun on the third day after débridement in those with an exposed anastomosis. Before VAC placement, we use silver-containing gel for dressing changes. This allows daily inspection of the wound to assess the adequacy of débridement and ensure lack of a significant infectious process. The presence of VAC in the wound does not allow close monitoring of the wounds in this early period.
3. Once the VAC is placed, we use nonadhering dressings around the anastomosis and personally inspect the wound during each VAC change. The nonadherent dressing is used until the anastomosis is no longer visible in the wound. This practice stems from our hypothetical concern about direct trauma to the anastomosis from the VAC device. However, we believe that the most likely cause of arterial disruption associated with groin wound infections is arterial infection. As Dr Brehm concludes that the arterial blow-out in his patient was "possibly . . . due to the suction of the VAC system," use of a nonadherent dressing around the anastomosis may reduce the risk of direct arterial trauma and subsequent bleeding.

We agree with Dr Brehm's conclusion that the VAC should be used in highly selected patients with infected and exposed grafts. Because anastomotic blowout is a possible complication of attempted graft preservation with exposed anastomosis, these patients should be carefully monitored in the acute care setting and discharged only after the anastomosis is fully covered with granulation tissue with no evidence of infection.

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Regarding "Current results of open surgical repair of descending thoracic aortic aneurysms"

I read with great interest the review article on the surgical treatment of descending thoracic aortic aneurysms (TAAs) by Black and Cambria[1] describing the advantages of the various surgical strategies on the surgical treatment of TAAs. However, I was surprised not to see included and cited the technique that offered the best results on the prevention of spinal cord ischemia.

In 1999, Biglioli et al[2] proposed the "quick, simple clamping technique" for the replacement of descending aorta to prevent spinal cord injury. In 49 consecutive patients, they had 0 paraplegia; however, in the groups of "selective atriodistal bypass" (66 patients) and "simple clamping technique" (28 patients), they had 4.5% and 14.3% incidence of paraplegia, respectively. They also demonstrated that the aortic cross-clamping time is the most important factor correlated to spinal cord injury. Indeed, the proposed technique aims in the reduction of aortic clamping under the crucial 25 minutes.

Many other authors[3] found that spinal cord ischemic time >30 minutes has been considered a critical event, regardless of the spinal cord protective techniques employed, and all spinal-protective approaches, including deep hypothermia and circulatory arrest, intercostals arteries reimplantation, cerebrospinal fluid drainage, distal circulatory support, left-heart by-pass, could not completely eliminate the occurrence of paraplegia. In the article by