Removal of Periocular Veins by Sclerotherapy

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Purpose: Prominent periocular veins, especially of the lower eyelid, are not uncommon and patients often seek their removal. Sclerotherapy is a procedure that has been successfully used to permanently remove varicose and telangiectatic veins of the lower extremity and less frequently at other sites. Although it has been successfully used to remove dilated facial veins, it is seldom performed and often not recommended in the periocular region for fear of complications occurring in adjacent structures. The purpose of this study was to determine whether sclerotherapy could safely and effectively eradicate prominent periocular veins.

Design: Noncomparative case series.

Participants: Fifty adult female patients with prominent periocular veins in the lower eyelid were treated unilaterally.

Patients and Methods: Sclerotherapy was performed with a 0.75% solution of sodium tetradecyl sulfate. All patients were followed for at least 12 months after treatment.

Main Outcome Measures: Complete clinical disappearance of the treated vein was the criterion for success.

Results: All 50 patients were successfully treated with uneventful resorption of their ectatic periocular veins. No patient required a second treatment and there was no evidence of treatment failure at 12 months. No new veins developed at the treated sites and no patient experienced any ophthalmologic or neurologic side effects or complications.

Conclusions: Sclerotherapy appears to be a safe and effective means of permanently eradicating periocular veins. *Ophthalmology 2001;108:442–448 © 2001 by the American Academy of Ophthalmology.*

Removal of asymptomatic facial veins, especially periocular veins, for cosmetic enhancement is a frequent request. In some, these veins are relatively inconspicuous, whereas in others they are noticeable, appearing dilated, tortuous, and protruded. They often become more prominent with aging because of loss of subcutaneous tissue and after chemical and laser resurfacing. Because periocular veins are normal constituents of the skin, they may be more apparent in those with fair complexions. Sclerotherapy has been used for decades to eradicate both venous telangiectases and varicose veins on the lower extremity.[1] It has also been used to eradicate ectatic veins at other cutaneous sites, including the face.[2,3] However, there has been a reluctance by physicians, even those who actively use sclerotherapy for lower extremity veins, to advocate removal of periocular veins by sclerotherapy for fear of inflicting ophthalmologic and neurologic complications. These concerns persist even though there is an absence of any reports of such complications. Fifty consecutive patients requesting removal of periocular veins were treated by sclerotherapy to determine the efficacy and safety of this procedure for these facial veins.

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Patients and Materials

Fifty consecutive patients requesting removal of asymptomatic periocular veins because of their perceived unasthetic appearance were included in this series. All were women, and they ranged in age from of 25 to 62 years. None of the patients had ever sustained trauma at or near their unwanted veins, nor had any received previous treatment. All were in excellent health without any past or present history of ophthalmologic or neurologic disorders. At examination, all had conspicuous periocular veins coursing on the inferior and lateral skin around the eye. These veins were all easily compressible and were nontender.

Patients were initially seen in consultation regarding their unwanted facial veins. During this visit, the procedure was thoroughly explained, including the method of action of the sclerosing agent, expectations from treatment, possible side effects (i.e., bruising, swelling, discomfort), and complications (i.e., linear hyperpigmentation, telangiectatic capillary matting, ulceration, and those oft-repeated but undocumented risks of blindness and stroke). If the patient wished to pursue treatment, a subsequent appointment was scheduled. All 50 of the initial patients wished to pursue treatment, and informed consent was obtained from each.

Treatment was performed by sclerotherapy. This procedure involves cannulating the targeted vein, followed by the infusion of a sclerosant.[4] The sclerosant used was an aqueous solution of sodium tetradecyl sulfate, a Food and Drug Administration-approved sclerosing agent, in a concentration of 0.75%.[5] This is a clear, colorless solution that is free of any particulate matter. The solution was injected through a 30-gauge needle using a 3-ml polypropylene syringe. All needles were used for only one injection. If more than one site was injected along the course of the vein, a new needle was used. Patients were asked to remove all

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makeup, thoroughly wash their skin, and not apply any products to their skin all within 1 hour before their treatment. After thoroughly wiping the skin with isopropyl alcohol, the targeted vein was cannulated. After cannulation, when the needle tip was confirmed to be intraluminal by passive retrograde flow of blood into the needle hub, slow infusion of solution began. Slow infusion is maintained by injecting under minimal pressure—just enough to overcome intraluminal pressure—displacing the blood from the vein. This slow rate of infusion maximizes the duration to deliver a finite volume of sclerosant and, therefore, the duration of contact of the sclerosant with the intraluminal mural layers. The vein was always cannulated such that the needle pointed towards the lateral aspect of the face, as would be the direction of sclerosant flow as it emanated from the needle. Into any one injection site, 1 to 3 ml of sclerosant was infused. One or two injection sites were chosen along the course of the vein, so that a total of 2 to 6 ml was infused. The total volume of solution infused was determined in part by the volume of the targeted vein, which is a function of its width and length. Because the sclerosing solution is colorless, during infusion its course in the vein was apparent as it displaced intraluminal blood. This provided confirmation of the exposure of all intraluminal surfaces to sclerosant. All treatments were administered with the patient in a supine position.

Immediately after infusion of the solution, a gauze pad was placed over the length of the treated vein, and digital pressure was applied to delay the return of blood into the intraluminal channel. After maintaining digital pressure for 60 seconds, the vein was examined; if the clinical endpoint of treatment was achieved, the patient was allowed to stand and leave the office. The clinical endpoint of treatment was the cessation of any blood flow through the targeted vein. Absence of any blood flow was defined clinically as one of two circumstances: either with the appearance of an intraluminal thrombus or the apparent absence of any blood in the vein. In either circumstance, there is usually concomitant linear urticaria, that is, erythema and edema, in the skin along the course of the vein, which appears within minutes of injection and persists for 30 minutes or longer. This probably results from nonspecific histamine release from perivascular mast cells, because it is seen with all sclerosants. If, after treatment, blood could be displaced from the vein by digital pressure but would promptly return on release of pressure then, based on past clinical experience, it was concluded that not enough mural injury was sustained. In that circumstance, the vein was again cannulated and solution was injected. There was no attempt at fabricating a pressure dressing.

Course

After treatment, the patients were permitted to resume all activities with no limitations. Patients were observed at 1 week after the procedure and then monthly until there was complete disappearance of the veins by inspection and palpation. They were then seen at 3-month intervals until at least 12 months after the veins were observed to have disappeared. All patients were available for follow-up. All patients had mild erythema and edema of the skin surrounding the course of the treated vein that persisted for 2 to 7 days. This was more diffuse than the transient linear urticaria that appears immediately after treatment. None had sufficient eyelid swelling to interfere with their field of vision. The skin surrounding the treated vein was asymptomatic or there was minimal pruritus or tenderness, which persisted for a few hours up to several days. No patient reported any significant symptoms during or anytime after treatment, including ophthalmologic or neurologic changes. In six patients there was purpura (6 mm in diameter or less) at the injection site that developed at the time of treatment. In all six patients the purpura was resorbed within 10 days after treatment. Clinically, there was no extravasation of sclerosant at the time of treatment, and no cutaneous changes associated with extravascular sclerosant were observed. No patient, on her return visit in 1 week, had a patent vein in which blood could be displaced. In all patients, the treated vein was either nonpalpable or palpable as a firm cord that represented the denatured, thrombosed vein. It was resorbed, visually and by palpation, in all cases within 2 months after treatment.

Results

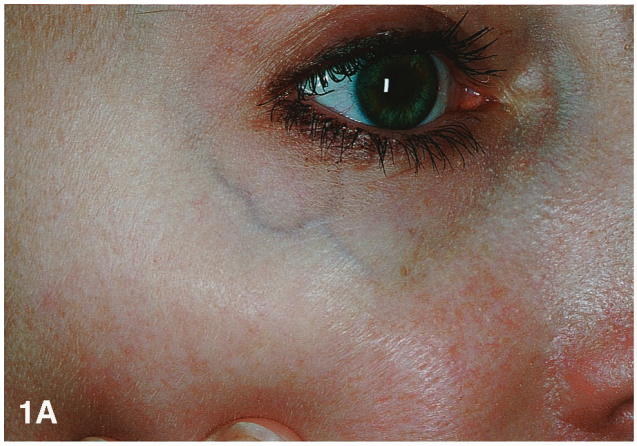
In all 50 patients, resorption of the treated veins was uneventful. No patient had any ophthalmologic or neurologic signs or symptoms either during or after treatment. Six patients experienced localized purpura at an injection site at the time of treatment that resolved within 10 days. No patient experienced purpura subsequent to treatment. During the first 6 weeks after treatment, some of the treated veins were palpable as firm cords. In all patients, there was complete clinical resorption within 2 months after treatment. There was no clinical evidence of treatment failure, that is persistence or reappearance of the vein resulting from reconstitution and recanalization, during the subsequent 12 months nor did any new veins appear (Figs 1–4). No patient required a second treatment. There was no linear hyperpigmentation along the course of the treated veins, as is often observed after sclerotherapy of both venous telangiectases and varicose veins of the lower extremity. None of the patients experienced telangiectatic capillary matting subsequent to treatment, which is also commonly seen after sclerotherapy.

Discussion

This study demonstrated the efficacy and safety of sclerotherapy using sodium tetradecyl sulfate at a concentration of 0.75% for removing unwanted periocular veins. Sclerotherapy, if properly performed, remains a consistently effective method to eradicate any linear segment of a vein.[6] Proper technique requires that an appropriate concentration and volume of sclerosant is infused into the targeted vein and that the sclerosant has the opportunity to bathe the mural layers of the vein, at the intraluminal interface, for an adequate period of time.[7] An appropriate concentration and volume of sclerosant would be the minimal amount necessary to denature the targeted vein without inflicting injury to nontargeted veins or the surrounding tissue.

Mechanism of Sclerotherapy

Because this was not a pilot study, the successful eradication of periocular veins in all treated patients is not unexpected. The author has previously treated periocular veins using different concentrations and volumes of sclerosant. The concentration and volumes used in this study were based on past experience that consistently resulted in the successful removal of similar periocular veins. Lower con-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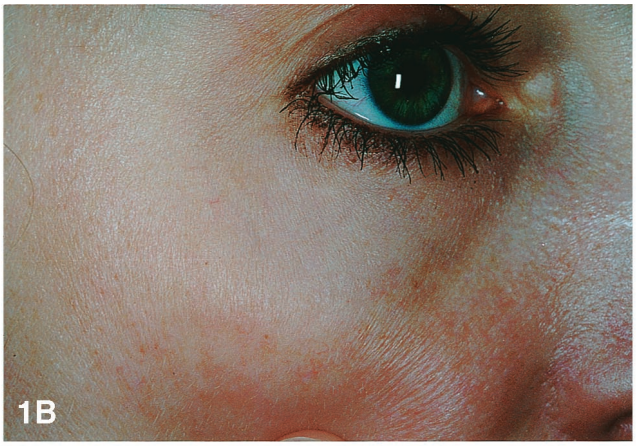
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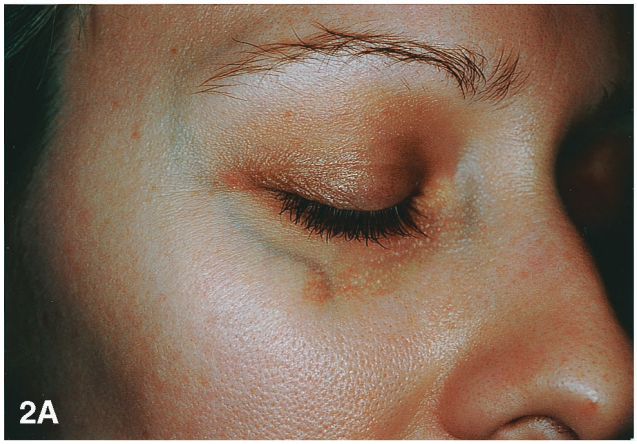
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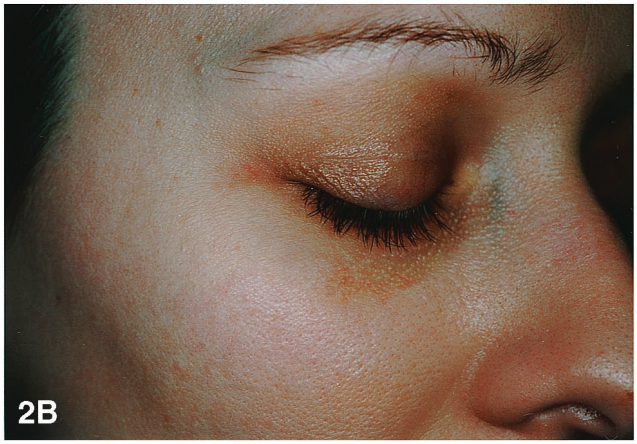
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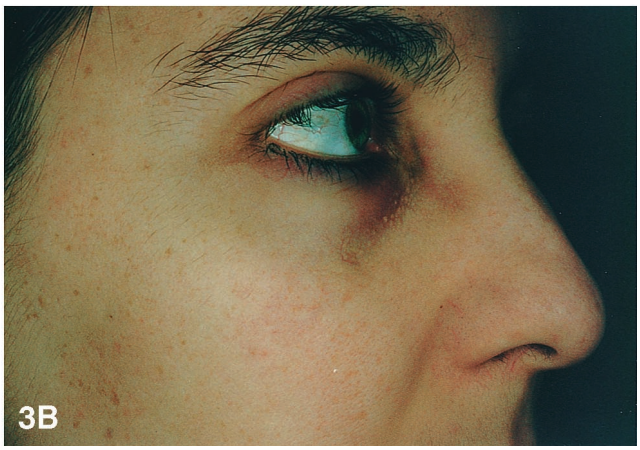
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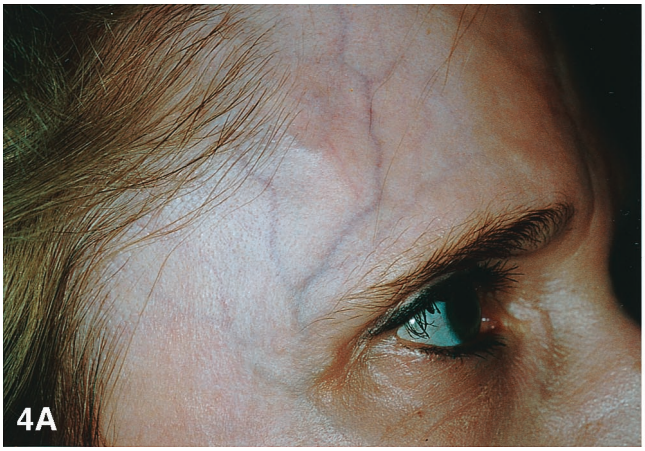
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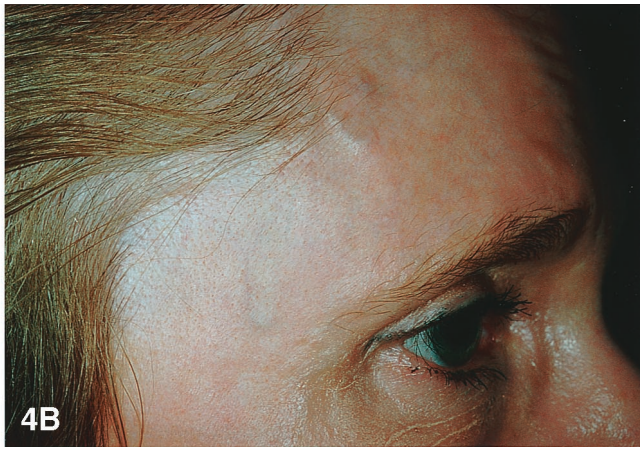
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Figure 1. A, before treatment. **B,** 12 months after sclerotherapy.
Figure 2. A, before treatment. **B,** 12 months after sclerotherapy.
Figure 3. A, before treatment. **B,** 12 months after sclerotherapy.
Figure 4. A, before treatment. **B,** 12 months after sclerotherapy.
←

centrations than 0.75% of sodium tetradecyl sulfate have been associated with treatment failure. That is, the veins persisted because of incomplete mural denaturation. The mechanism of vein persistence with only partial mural denaturation is reconstitution of the mural layers, repopulation of the endothelial cells, and recanalization of the intraluminal channel by the resorption of any formed thrombus.[6] If an adequate concentration and volume of sclerosant is infused to effect full mural denaturation, there will follow resorption of the denatured mural layers and obliteration of the intraluminal channel. From past clinical experience, if there was no recurrence within 6 months, there was no subsequent reappearance. Because the goal of treatment was the visual disappearance of the ectatic vein, duplex ultrasound scanning was not used in this study. However, from past examinations, when the vein was visually and palpably gone after 12 months, it was also absent on duplex ultrasound examination.

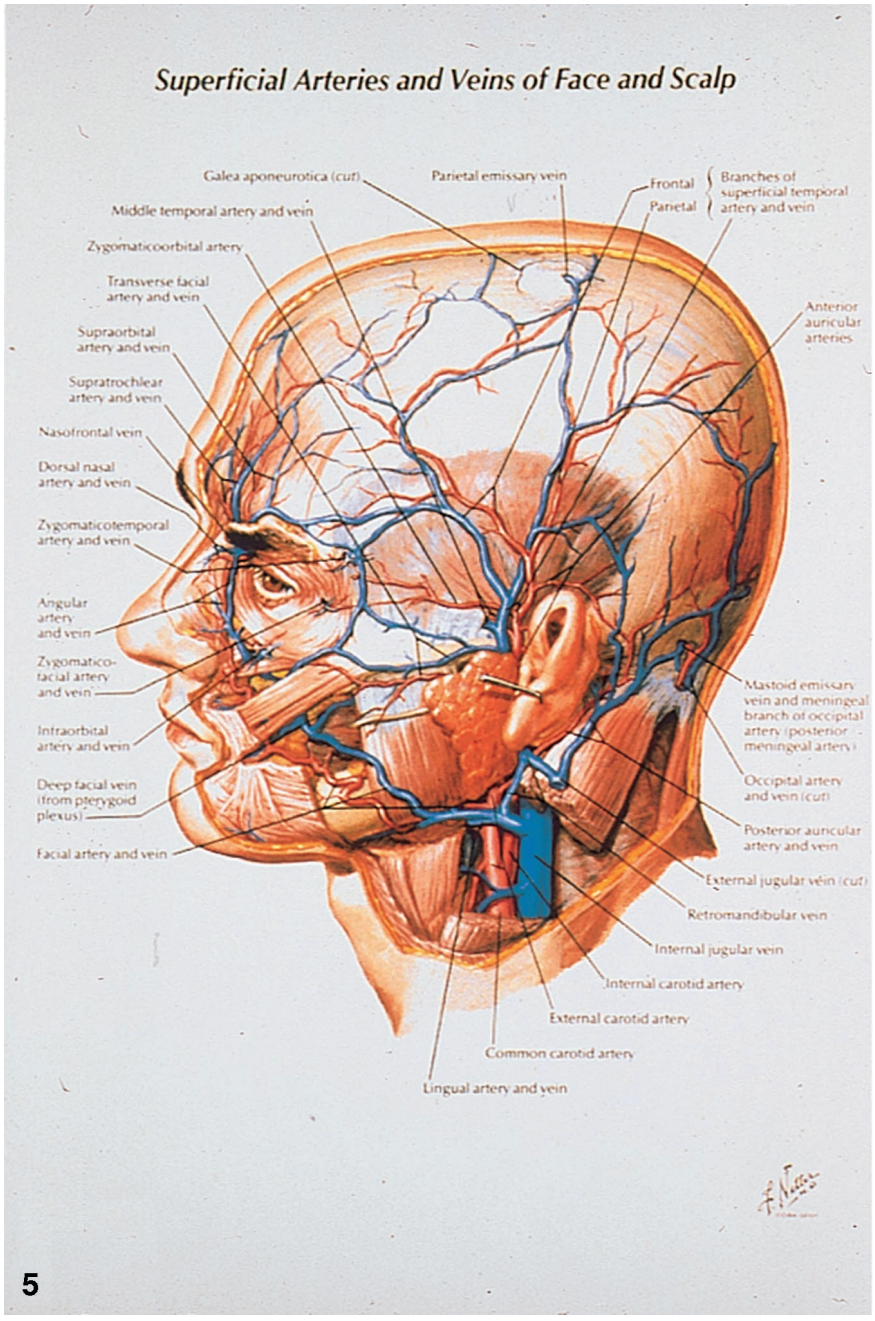

Figure 5. Superficial arteries and veins of the face and scalp. Periocular veins drain into superficial veins of the face, including the superficial temporal vein and the facial vein, which themselves drain into the external jugular vein and internal jugular vein, respectively. Drainage into the orbital and cerebral veins is not the drainage pathway for periocular veins.

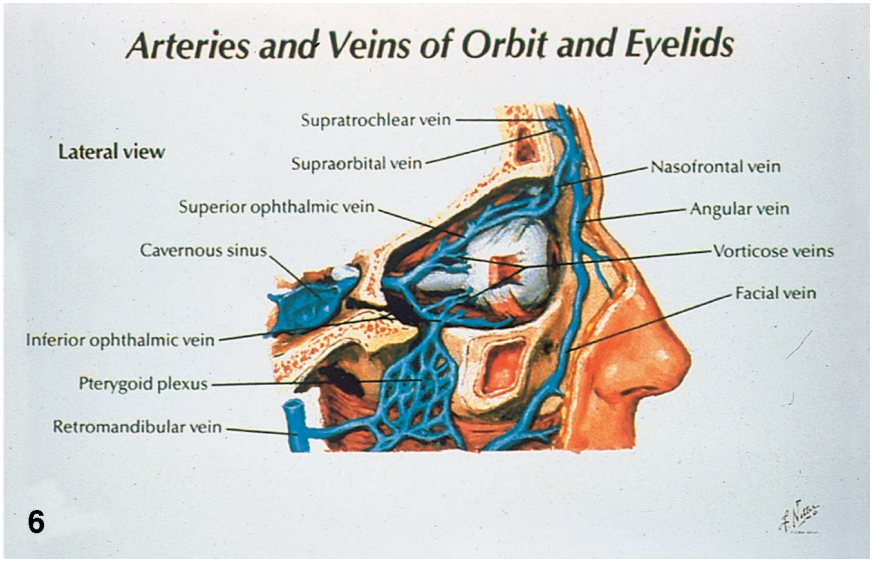

Figure 6. Arteries and veins of the orbit and eyelid. Orbital and cerebral veins are not the usual drainage pathway for periocular veins.

Sclerosants

Sclerosing agents vary widely in their chemical nature and in their ability to denature mural layers of a vein. The most widely used groups of sclerosants include surfactants, such as sodium tetradecyl sulfate used in this study, and hyperosmotic solutions. The hyperosmotic agents, in particular hypertonic saline (which is commercially available in a 23.4% concentration) should not be used for periocular sclerotherapy. This solution is quite painful during infusion and usually produces significant muscle contraction and cramping because of the high sodium concentration. Although uncomfortable in the lower extremity, it is even more disturbing in the periocular region.

Intravascular Thrombosis and Sclerotherapy

Whether an adequate concentration and volume of sclerosant is infused into the vein, an intraluminal thrombus is usually a sequela of sclerotherapy. The thrombus develops from blood that flows into the vein after infusion of sclerosant is terminated. Thrombosis occurs because mural disruption associated with treatment exposes subendothelial collagen.[8] Direct exposure of clotting factors to subendothelial collagen may activate the intrinsic pathway of coagulation. In addition, the intrinsic pathway of coagulation may be activated by platelet-derived phospholipids that are released after recruitment and alteration of platelets stimulated by newly exposed subendothelial collagen. The presence of an intraluminal thrombus does not affect the ultimate fate of the vein. However, minimizing venous return, and thus the volume of the intraluminal thrombus, minimizes the time for complete resorption of the denatured vein. Although postsclerotherapy compression is provided after treating varicose veins of the lower extremity to minimize the volume of the formed thrombus, there was no attempt at fabricating a compression bandage in this study. No such bandaging would have provided adequate compression while being tolerated by the patients. Although it may have taken longer for the vein to be resorbed compared with if compression was applied, it did not adversely affect the outcome of treatment.

On the lower extremities, linear hyperpigmentation paralleling the course of treated veins has been reported in up to 30% of patients, but may be higher.[9] This dermal pigment represents hemosiderin, a metabolically dead-end byproduct of hemoglobin.[10] The greater the quantity of trapped blood within the intraluminal channel, that is the greater the volume of the thrombus, the greater the likelihood of linear hyperpigmentation appearing after treatment. Although it is unclear why linear hyperpigmentation was not observed with the frequency encountered after treating veins of the lower extremity, the finding was not surprising from this author's past experience. Even when linear hyperpigmentation on the lower extremity appears, it almost invariably fades over a period of months to years, only rarely persisting for up to 3 years. Likewise, telangiectatic capillary matting that is observed in up to 16% of cases after sclerotherapy to veins of the lower extremity has not been observed after treating periocular veins.[11]

Hemodynamics within Periocular Veins

Not unexpectedly, none of the patients experienced any adverse ophthalmologic or neurologic disturbances. However, concerns about thrombotic phenomena within ocular, orbital, or cerebral veins associated with periocular vein sclerotherapy have been raised. Such complications would be unlikely to occur and have never been reported. Sclerosant introduced into periocular veins would be expected to flow in an antegrade direction into progressively larger veins. As it does, it is rapidly diluted, rendering it increasingly harmless to the endothelial and mural layers. As noted in "Patients and Methods," injection pressure is deliberately minimal to prolong the duration of contact between the sclerosant and the mural layers of the targeted veins. The

low injection pressure also minimizes the risk of retrograde flow into smaller diameter veins. This is because the flow of a liquid will follow the course of least resistance, which is into larger veins. The resistance to fluid flow in a tube is, theoretically, inversely related to the fourth power of the radius of the tube.[7] This is expressed by Poiseuille's formula:

$$R = \frac{8 \cdot \eta \cdot l}{\pi r^4},$$

where R is the resistance to fluid flow in a tube, η is the viscosity of the solution, l is the length of the tube, and r is the radius of the tube. Even though the sclerosant is under the influence of external pressure (which can far exceed the normal intraluminal pressure), it will, nonetheless, most likely travel along the course of least resistance, which is through larger diameter veins in the antegrade direction, rather than those smaller diameter veins in the retrograde direction. However, even with retrograde flow there is rapid dilution of sclerosant.

Periocular (Eyelid) Vein Anatomy

The veins of the eyelid are subdivided into preseptal and postseptal divisions. The superficial cutaneous vessels of the lower eyelid, of the type described in the study, are preseptal veins. Essentially, their venous drainage is into the superficial temporal vein or the anterior facial vein, which themselves drain into the external jugular vein and the internal jugular vein, respectively (Fig 5).[12–15] They would not normally be expected to drain into the ocular, orbital, or cerebral venous systems. The postseptal veins, which are not visible on inspection of the eyelid nor ever treated by sclerotherapy, more directly join the orbital veins, deep facial branches of the anterior facial vein, and the pterygoid plexus (the venous plexus between the temporalis and pterygoid muscles). These veins do terminate in the cavernous sinus.

Low-pressure infusion of a sclerosant into a prominent vein along the lateral aspect of the lower eyelid is likely to result in sclerosant flowing laterally into the zygomaticofacial vein or a vein that drains into the superficial temporal vein. The superficial temporal vein joins the maxillary vein to become the retromandibular vein (also referred to as the *posterior facial vein*). The posterior ramus of the retromandibular vein joins the posterior auricular vein to become the external jugular vein. The anterior ramus of the retromandibular vein joins the common facial vein and drains into the internal jugular vein. If sclerosant flows medially, it would go toward the angular vein. Direct drainage from the angular vein is caudad through the anterior facial vein, which courses along the cheek to drain into the common facial vein, which flows into the internal jugular vein. The angular vein is formed cephalad by the union of the supraorbital and supratrochlear veins. The supraorbital vein communicates with the superior ophthalmic vein, which courses along the medial aspect of the orbit, ultimately draining into the cavernous sinus. However, flow from the angular vein is less likely to drain through these smaller veins. On the lateral periocular skin and temple, flow would be expected to proceed to the superficial temporal vein, ultimately draining into the external jugular vein.

Any flow of sclerosant into the orbital or ocular veins would have to enter through the superior ophthalmic vein or the inferior ophthalmic vein. Access to the cerebral circulation would have to occur through the cavernous sinus from sclerosant flow that initially enters it from the superior ophthalmic or inferior ophthalmic veins (Fig 6). Direct and significant sclerosant flow into the superior ophthalmic or inferior ophthalmic veins would be highly unlikely from infusion into the preseptal veins. To drain into the superior ophthalmic vein, flow would have to proceed cephalad from the angular vein. This is hemodynamically unlikely because flow would more favorably travel caudad from the angular vein into the larger anterior facial vein. Even if some sclerosant entered the superior ophthalmic vein, it would be significantly diluted along its course, likely rendering it nonirritating to the mural layers. As a precaution against any flow traveling into the superior ophthalmic vein, digital pressure can be placed just above the angular vein during injection to preclude any flow into the supraorbital vein. Sclerosant access to the inferior ophthalmic vein is even more improbable. Flow from the superficial temporal vein would have to flow in a retrograde direction into the maxillary vein, which connects with the pterygoid venous plexus. The anterior facial vein also communicates with the pterygoid venous plexus via the deep facial vein. The inferior ophthalmic vein, which drains into the cavernous sinus, communicates with the pterygoid venous plexus. However, such a retrograde and indirect pathway would not only be improbable but would render the sclerosant harmless because of the dilution it would have sustained.

The risk of intraarterial injection of sclerosant is very small. A targeted vein is clearly visualized, and infusion only commences after retrograde flow of venous blood is confirmed into the needle hub and syringe. During infusion, the slow displacement of blood by sclerosant within the vein is easily visualized.

Inadvertent extravascular infusion as a result of the vein not being properly cannulated or of extravasation from a site of mural disruption during intravascular injection is immediately recognized as a localized area of swelling. If observed, injection should be abruptly discontinued. Because infusion of sclerosant is always deliberately slow, the quantity of sclerosant, which could become extravascular, should be minimal. Nonetheless, sclerosant that is extravascular can produce denaturation of tissue, including ulceration. For this reason, the injection site should always be carefully observed during the infusion of the solution.

Conclusions

Sclerotherapy appears to be a safe and effective means of permanently eradicating periocular veins. There were no ocular, orbital, or cerebral complications of treatment of

superficial cutaneous veins among the 50 patients treated and described in this report.

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