

**VARICOSE DISEASE (VARICOSE VEINS): A COMPREHENSIVE OVERVIEW
OF DIAGNOSIS AND TREATMENT**

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Abstract: Varicose veins, often referred to as varicosities, are a prevalent manifestation of chronic venous disease (CVD). They are dilated, elongated, and tortuous subcutaneous veins typically found in the lower extremities, resulting from valvular incompetence, venous hypertension, and subsequent venous wall remodeling. While many view varicose veins primarily as a cosmetic concern, they can significantly impair quality of life and progress to severe complications, including skin changes and ulcerations if left untreated. This comprehensive article aims to provide a thorough overview of varicose veins, including their historical context, epidemiology, anatomy, pathophysiology, risk factors, classification, clinical presentation, diagnostic modalities, and the various conservative, minimally invasive, and surgical treatments available. The article also addresses the importance of prevention, emerging therapeutic options, complications associated with the condition, and future directions in research and management.

1. Introduction

Varicose veins are dilated subcutaneous veins commonly found in the lower extremities. They result primarily from venous valvular incompetence and elevated venous pressures, which lead to progressive remodeling of the venous wall. This condition, often viewed through a cosmetic lens, has far-reaching implications for patient health and well-being. Chronic venous disease (CVD), of which varicose veins are a significant component, may contribute to pain, leg heaviness, skin changes, and the potential formation of venous ulcers.

Historically, varicose veins have been recognized and documented for centuries, with numerous descriptions and therapeutic measures evolving throughout time. Over the past few decades, dramatic changes in diagnostic and treatment modalities have revolutionized patient care. Minimally invasive procedures—such as endovenous laser ablation (EVLA), radiofrequency ablation (RFA), and ultrasound-guided foam sclerotherapy—have supplanted traditional surgical vein stripping in many cases. These modern interventions offer lower morbidity, reduced pain, and faster recovery, reflecting a paradigm shift in managing varicose veins.

Despite the surge in advanced management options, varicose veins remain a common clinical concern worldwide. Factors such as advancing age, sedentary lifestyles, and obesity can exacerbate venous insufficiency, increasing the disease burden. As our understanding of the condition's pathophysiology expands, clinical guidelines and therapeutic strategies continue to evolve, offering improved outcomes and reduced complication rates for patients.

2. Historical Perspective

The recognition and management of varicose veins have ancient roots:

- **Ancient Civilizations:** Evidence of varicose veins is found in Egyptian mummies, suggesting that this condition has been present for millennia. The Ebers Papyrus (circa 1550 BCE) may allude to venous disorders consistent with varicose veins.
- **Hippocrates (460–377 BCE):** Often regarded as the “Father of Medicine,” Hippocrates described treatments for varicose veins, advocating compression techniques and cautery. His foundational approaches highlight the long-held belief that external pressure can mitigate vein distension.
- **Galen (130–210 CE):** A prominent physician in ancient Rome, Galen contributed to the understanding of venous anatomy and noted the prevalence of varicosities. His anatomical insights laid foundational knowledge for subsequent venous disease research.
- **19th Century:** This era saw growing interest in surgical approaches. Sir Benjamin Brodie and Friedrich Trendelenburg both worked on describing ligation of the great saphenous vein. Trendelenburg’s test, used to assess venous reflux, remains part of the historical clinical examination.
- **20th Century:** Vein stripping, introduced in the early 1900s, became the mainstay of surgical treatment for many decades. Over time, specialized vascular surgery, innovative endovenous techniques, and improved diagnostic imaging transformed varicose vein management into a more precise field.

Today, advances in ultrasound technology, the introduction of endovenous thermal ablation, and the evolution of sclerotherapy demonstrate a relentless pursuit of less invasive, more effective, and safer treatments for varicose veins.

3. Epidemiology

Varicose veins constitute one of the most common vascular disorders worldwide. The prevalence varies based on geographical region, diagnostic criteria, and population demographics:

- **Prevalence:** Estimates suggest that 20–40% of adults have some form of varicose veins, with higher rates in developed countries. Women are generally more affected than men, potentially due to hormonal influences that can weaken venous walls.
- **Age Distribution:** While varicose veins can appear at any age, prevalence increases with advancing age. Hormonal fluctuations during adolescence and childbearing years can also predispose younger women to earlier onset.
- **Socioeconomic Impact:** Varicose veins and associated chronic venous insufficiency (CVI) can result in lost work productivity, healthcare expenditures, and impaired quality of life. Treatments can vary significantly in cost, with newer endovenous procedures sometimes imposing higher initial outlays but often reducing long-term expenses and complications.
- **Geographic Factors:** Lifestyle, diet, occupational activities, and genetic predispositions contribute to variations in prevalence among different regions. Populations that engage in prolonged standing or exhibit higher rates of obesity may have increased susceptibility to venous insufficiency.

4. Anatomy and Physiology of the Venous System

A thorough understanding of normal venous anatomy and physiology is critical for diagnosing and treating varicose veins:

1. Venous Anatomy of the Lower Extremities

- **Deep Venous System:** Located deep within the leg muscles, the deep veins (e.g., femoral, popliteal, anterior, and posterior tibial veins) carry the majority of venous return (approximately 80–90%).

- **Superficial Venous System:** This system, which includes the great saphenous vein (GSV) and the small saphenous vein (SSV), is closer to the skin surface. The GSV runs from the medial aspect of the foot to the groin, while the SSV traverses the lateral aspect of the foot up the posterior calf.

- **Perforator Veins:** These veins connect superficial veins to deep veins, ensuring blood flow from the superficial to the deep system under normal conditions.

2. Venous Valves

- Valves are designed to allow one-way blood flow toward the heart, preventing retrograde flow due to gravity. Incompetent or absent valves in the superficial veins or perforators are a primary pathological basis for varicosities.

3. Venous Physiology

- **Muscle Pump:** The calf muscle pump is essential for venous return. As the calf muscles contract, they compress the deep venous system, propelling blood upward. Competent valves prevent backflow.

- **Respiratory Pump:** Changes in intrathoracic and intra-abdominal pressure during respiration also facilitate venous return, although this factor is less impactful than the muscle pump in the lower limbs.

Disruption of this system—whether via valve incompetence or venous outflow obstruction—leads to elevated venous pressure (venous hypertension), which contributes to the formation of varicose veins.

5. Pathophysiology of Varicose Veins

Varicose veins arise due to a convergence of factors causing valvular incompetence and weakening of the venous wall:

1. Valvular Incompetence:

- Vein valves may become incompetent because of structural changes (e.g., stretching and thinning) in the vein wall or direct valvular damage.

- Once valves fail, venous pressure in the superficial system rises (venous hypertension), causing further dilation of the vein and additional valvular dysfunction in a vicious cycle.

2. Venous Hypertension:

- Chronic elevation of venous pressure leads to venous stasis (reduced blood flow), perpetuating endothelial damage and a pro-inflammatory state in the vein wall.

- Over time, vessel dilation worsens, exacerbating reflux and promoting the progression from mild varicosities to more significant chronic venous insufficiency (CVI).

3. Vein Wall Remodeling:

- Chronic inflammation triggers proteolytic enzymes (e.g., matrix metalloproteinases), which degrade collagen and elastin.

○ This process weakens the structural integrity of the vessel wall, causing further distension and tortuosity.

4. **Genetic Influences:**

○ Family history is a major risk factor, suggesting a significant hereditary component. Certain connective tissue disorders also predispose to vein wall weakness.

Thus, the interplay between genetic predisposition, hormonal changes, mechanical factors (e.g., prolonged standing), and inflammation leads to the typical manifestation of varicose veins in susceptible individuals.

6. Risk Factors

Multiple risk factors contribute to the development and progression of varicose veins:

1. **Age:** The prevalence and severity of varicose veins increase with age due to cumulative valvular wear and tear.

2. **Sex and Hormonal Factors:**

○ Women have a higher incidence of varicose veins, possibly linked to progesterone and estrogen, which reduce venous tone and elasticity.

○ Pregnancy is a notable risk factor because of increased venous pressure from a gravid uterus and the hormonal milieu.

3. **Genetics:**

○ A positive family history of varicose veins suggests a hereditary predisposition.

○ Specific genetic markers remain an active area of research.

4. **Lifestyle and Occupational Factors:**

○ Prolonged standing or sitting (e.g., in healthcare or service industries) can heighten venous pressure in the legs.

○ Sedentary behavior and obesity contribute to venous stasis.

5. **Obesity:**

○ Excess body weight increases intra-abdominal pressure and overall strain on venous valves.

6. **Pregnancy:**

○ Increased blood volume, hormonal changes, and uterine compression of pelvic veins cumulatively raise venous pressure in the lower extremities.

7. **Physical Inactivity:**

○ A lack of calf muscle pump activation leads to reduced venous return, exacerbating venous hypertension.

8. **Smoking:**

○ Tobacco use impairs vascular function and contributes to systemic inflammation, possibly aggravating varicose vein formation.

9. **Previous Vein Disease or Trauma:**

○ Deep vein thrombosis (DVT) or leg trauma can damage valves, predisposing the development of varicose veins.

Recognizing these risk factors guides prevention strategies, screening, and patient counseling regarding lifestyle modifications.

7. Classification Systems

Several classification systems describe the severity and etiology of chronic venous disorders, including varicose veins. The most widely used is the CEAP classification:

1. **C (Clinical):**
 - C0: No visible or palpable signs of venous disease
 - C1: Telangiectasias or reticular veins
 - C2: Varicose veins
 - C3: Edema
 - C4: Skin changes (C4a: pigmentation or eczema; C4b: lipodermatosclerosis or atrophie blanche)
 - C5: Healed venous ulcer
 - C6: Active venous ulcer
2. **E (Etiology):**
 - Ec: Congenital
 - Ep: Primary
 - Es: Secondary (e.g., post-thrombotic)
3. **A (Anatomy):**
 - As: Superficial veins
 - Ap: Perforator veins
 - Ad: Deep veins
4. **P (Pathophysiology):**
 - Pr: Reflux
 - Po: Obstruction
 - Pr,o: Both reflux and obstruction

The CEAP classification provides a standardized framework for assessing disease progression and guiding therapeutic decisions. In addition to CEAP, other clinical tests—such as the Trendelenburg test and Perthes test—have historical significance in determining superficial or deep venous reflux. Nevertheless, duplex ultrasound remains the primary diagnostic standard for accurately classifying venous disease.

8. Clinical Presentation and Symptoms

Varicose veins can present with a range of clinical findings:

1. **Visible Signs:**
 - Dilated, tortuous superficial veins, most easily observed when a patient stands.
 - In early stages, patients may only exhibit telangiectasias (“spider veins”) or reticular veins, which can progress to larger varicosities.
2. **Subjective Symptoms:**
 - Leg heaviness, aching, throbbing, or burning.
 - Swelling around the ankles and calves, typically worse after prolonged standing or sitting.
 - Night cramps or restless legs.
3. **Skin Changes:**

- Hyperpigmentation (usually brownish discoloration) due to hemosiderin deposition from venous stasis.
 - Eczematous dermatitis, pruritus, and dryness.
 - Lipodermatosclerosis (fibrotic changes in the skin and subcutaneous tissue).
 - Atrophie blanche (white, scar-like areas on the skin).
4. **Ulceration:**
- Chronic venous insufficiency can culminate in venous ulcerations, commonly located around the medial malleolus.
 - Ulcers may be painful, recurrent, and challenging to heal without addressing underlying venous hypertension.

Patients often seek medical attention for cosmetic reasons, discomfort, or complications like edema and skin changes. Prompt identification of varicose veins and appropriate treatment can prevent progression to more severe forms of chronic venous disease.

9. Diagnostic Approaches

9.1 Clinical Evaluation

A thorough clinical history and physical examination guide the initial assessment:

- **History:** Elicit information on duration of symptoms, aggravating or relieving factors (e.g., compression stockings, elevation), and any personal or family history of venous disease.
- **Inspection and Palpation:** Examine the lower limbs in a standing position to visualize varicosities. Palpate for tenderness, cords (superficial thrombophlebitis), and measure edema.
- **Special Tests:** Historically, the Trendelenburg test helped differentiate between superficial and deep venous incompetence. Though largely supplanted by duplex ultrasound, it can still provide basic clinical insight.

9.2 Duplex Ultrasound

Duplex ultrasound is the primary diagnostic tool for varicose veins. This noninvasive imaging modality combines B-mode ultrasound (for structural imaging) with Doppler (for flow evaluation):

- **B-Mode:** Allows visualization of vein diameter, wall characteristics, presence of thrombi, and anatomical variations.
- **Color and Spectral Doppler:** Assesses blood flow direction and velocity, identifying valvular reflux in superficial, deep, or perforator veins.
- **Mapping:** Duplex ultrasound can generate a detailed “venous map,” indicating the precise location and severity of reflux. This information is critical for planning treatment, especially if endovenous interventions are being considered.

9.3 Additional Diagnostic Techniques

- **Continuous-Wave Doppler:** Provides a simpler, less detailed assessment of venous flow and reflux. It has largely been replaced by duplex ultrasound.
- **Venography:** Once the gold standard, contrast venography is now reserved for complex or unclear cases where ultrasound findings are inconclusive.
- **Magnetic Resonance Venography (MRV) / Computed Tomographic Venography (CTV):** Useful for pelvic vein evaluations or atypical venous disease. These modalities help identify potential extrinsic venous compression or complex anomalies (e.g., May-Thurner syndrome).

Following diagnostic confirmation, the physician and patient can discuss the most appropriate management strategy tailored to the location, extent, and severity of varicose veins.

10. Treatment Modalities

Management of varicose veins aims to alleviate symptoms, prevent complications, and improve cosmetic appearance. A tailored approach considers disease severity, patient preference, comorbidities, and anatomical specifics as determined by duplex ultrasound.

10.1 Conservative Management

10.1.1 Compression Therapy

- **Compression Stockings:** Graduated compression stockings (20–30 mmHg or higher) help counteract venous hypertension by compressing the superficial veins. This external support promotes venous return, reducing edema, pain, and the risk of ulceration.
- **Impact on Long-Term Outcomes:** While compression does not eliminate varicose veins, it can significantly alleviate symptoms and slow disease progression, making it particularly valuable for patients with contraindications to invasive treatment.

10.1.2 Lifestyle Modifications

- **Exercise:** Regular activities like walking or cycling activate the calf muscle pump, improving venous return.
- **Weight Management:** Losing excess weight decreases venous pressure and symptom severity.
- **Leg Elevation:** Periodic elevation of the legs can temporarily reduce edema and venous pressure.
- **Avoiding Prolonged Standing or Sitting:** Frequent breaks and walking can mitigate stasis in occupational settings.

10.1.3 Pharmacological Agents

- **Venoactive Drugs:** Medications such as micronized purified flavonoid fraction (MPFF), rutosides, and horse chestnut seed extract may reduce symptoms (e.g., pain, swelling) by improving venous tone and decreasing inflammatory processes.
- **Analgesics or Anti-inflammatory Drugs:** Over-the-counter NSAIDs may help manage pain or superficial phlebitis but do not directly correct underlying venous pathology.

Conservative measures are often the first line in mild cases or when patients are not suitable candidates for interventional or surgical procedures.

10.2 Endovenous Ablation Techniques

Endovenous procedures have revolutionized the treatment of varicose veins, offering highly effective, minimally invasive options with lower complication rates and faster recovery compared to traditional surgery.

10.2.1 Endovenous Laser Ablation (EVLA)

- **Mechanism:** A laser fiber is inserted into the incompetent vein under ultrasound guidance. Thermal energy delivered by the laser causes vein wall collapse and occlusion.
- **Procedure:** Often performed under local anesthesia with or without sedation. Tumescence anesthesia is injected around the target vein, providing a heat sink and protection for surrounding tissues.
- **Advantages:** High success rates (over 90% vein closure), minimal postoperative pain, and rapid return to daily activities.
- **Complications:** Possible bruising, local discomfort, transient paresthesia, and very rarely deep vein thrombosis (DVT) or thermal skin injury.

10.2.2 Radiofrequency Ablation (RFA)

- **Mechanism:** Similar to EVLA, RFA delivers thermal energy (via radiofrequency waves) to the vein's interior, causing controlled damage and closure.
- **Procedure:** Under ultrasound guidance, a specialized catheter is placed in the vein, and radiofrequency energy is applied segment by segment.
- **Outcomes:** Comparable efficacy to EVLA, with similarly high closure rates and low complication risk.
- **Differences from EVLA:** RFA catheters typically achieve uniform heating along short segments, potentially reducing post-procedural pain, but clinical outcomes between EVLA and RFA are largely similar.

10.2.3 Mechanochemical Ablation (MOCA)

- **Mechanism:** This newer technique uses a rotating wire (mechanical agitation) along with the injection of a sclerosant solution to destroy the endothelium and occlude the vein.
- **Advantages:** No thermal energy is used, reducing the need for extensive tumescence anesthesia.
- **Limitations:** More long-term data are needed, but early results are promising in terms of efficacy and reduced discomfort.

Endovenous ablation has become a mainstay for treating incompetent saphenous veins, and numerous studies demonstrate efficacy superior to or comparable with traditional surgical ligation and stripping.

10.3 Sclerotherapy

Sclerotherapy involves injecting a chemical irritant (sclerosant) into the vein to induce inflammation and fibrosis, ultimately obliterating the vein lumen:

1. Liquid Sclerotherapy

- **Indication:** Commonly used for smaller varicose veins (reticular veins) and telangiectasias (spider veins).
- **Agents:** Polidocanol, sodium tetradecyl sulfate (STS), and glycerin-based solutions.
- **Procedure:** Typically performed in an outpatient setting, where multiple small injections are administered. Patients are advised to wear compression stockings post-procedure to enhance efficacy and reduce side effects.

2. Foam Sclerotherapy

- **Technique:** A sclerosant solution (e.g., polidocanol or STS) is mixed with air or CO₂ to create a foam, which displaces blood in the vein, allowing better contact with the vessel wall.
- **Ultrasound Guidance:** Foam is often injected under ultrasound guidance for better visualization, especially when treating larger varicose veins or perforator incompetence.
- **Efficacy:** Foam has been shown to achieve higher obliteration rates in larger veins compared to liquid sclerotherapy.
- **Complications:** Generally mild, including localized pain, hyperpigmentation, or matting of new superficial veins around treated areas. Rare complications include transient neurological symptoms (e.g., visual disturbances) or, extremely rarely, strokes in predisposed individuals.

Sclerotherapy can be repeated to achieve optimal results. It is often used in combination with endovenous ablation or surgery for residual or recurrent varicosities.

10.4 Surgical Interventions

Although endovenous techniques are favored for their minimally invasive nature, surgical interventions remain relevant, especially in cases with extensive varicose veins, large venous trunks, or certain anatomical variations unsuitable for ablation.

10.4.1 High Ligation and Stripping

- **Traditional Procedure:** The great saphenous vein (GSV) is ligated at the saphenofemoral junction, and the vein is stripped from the thigh or calf using a stripping device.
- **Advantages:** Proven long-term success rates for treating saphenous vein insufficiency.
- **Disadvantages:** General or spinal anesthesia is often necessary, postoperative bruising and pain can be significant, and recovery time is longer compared to endovenous methods.

10.4.2 Phlebectomy (Ambulatory or Microphlebectomy)

- **Indication:** Ideal for removing isolated superficial varicosities.

- **Procedure:** Performed through tiny incisions (1–2 mm), using hooks to extract the varicose vein segments.
- **Anesthesia:** Typically local anesthesia or tumescent infiltration suffices.
- **Outcomes:** Provides excellent cosmetic results with minimal scarring and a relatively quick recovery.

Surgery is often combined with sclerotherapy or endovenous ablation to address both the main trunk incompetence (GSV or SSV) and residual branch varicosities.

10.5 Adjunctive Therapies

10.5.1 Ultrasound-Guided Perforator Vein Ablation

- In cases with incompetent perforator veins contributing to venous stasis ulcers, targeted ablation of these perforators via endovenous approaches can facilitate ulcer healing and prevent recurrence.

10.5.2 Skin Care and Wound Management

- **Topical Emollients:** Alleviate dryness and eczema associated with venous stasis.
- **Compression Wrapping:** Particularly important for managing venous ulcers.
- **Advanced Wound Dressings:** If an ulcer is present, modern dressings that maintain a moist environment and support optimal wound healing may be necessary.

10.5.3 Treatment of Superficial Thrombophlebitis

- Inflammation or thrombosis of superficial veins (superficial thrombophlebitis) can occur in varicose veins. Management may include NSAIDs, low-molecular-weight heparin in more extensive cases, and possibly sclerotherapy or surgery if chronic and recurrent.

11. Complications of Varicose Veins and Their Management

Varicose veins, if unaddressed, can progress to more severe complications:

1. **Chronic Venous Insufficiency (CVI):** Characterized by edema, skin changes (pigmentation, eczema, lipodermatosclerosis), and potential ulceration. Managing CVI involves compression therapy, endovenous or surgical correction of reflux, and meticulous skin care.
2. **Venous Ulcers:** Open sores typically around the medial malleolus. They can be painful, prone to infection, and slow to heal without proper venous pressure reduction and wound management. Comprehensive treatment includes compression, local wound care, and sometimes advanced interventions (e.g., perforator vein ablation).
3. **Bleeding:** Superficial varicose veins may rupture from minimal trauma, leading to bleeding. Direct compression, elevation, and definitive treatment of the bleeding varicosity (e.g., sclerotherapy or phlebectomy) are required.
4. **Superficial Thrombophlebitis:** Inflammation and clot formation within a superficial vein can cause redness, pain, and swelling along the vein's course. Management typically includes NSAIDs, compression, and mobilization. In extensive cases, low-

molecular-weight heparin or direct oral anticoagulants may be considered to prevent progression to deep vein thrombosis.

5. **Pigmentation and Matting:** Hyperpigmentation can persist after sclerotherapy or spontaneously occur in chronic venous insufficiency. Matting refers to the formation of fine telangiectasias near treated veins. Laser therapy or repeated sclerotherapy may mitigate these cosmetic concerns.

Recognizing early warning signs and employing comprehensive treatment strategies can prevent or minimize these complications.

12. Prevention Strategies

Primary and secondary prevention of varicose veins emphasizes reducing venous hypertension:

1. Lifestyle Measures:

- Maintain a healthy weight and engage in regular exercise (e.g., walking, cycling).
- Avoid prolonged periods of immobility; incorporate breaks to walk or stretch.
- Elevate the legs when possible to reduce edema.

2. Compression Stockings:

- Especially beneficial for individuals at high risk (e.g., prolonged standing at work, pregnancy).
- Support circulation post-procedure to reduce recurrence.

3. Occupational Adjustments:

- Provide employees with ergonomic options, such as adjustable desks for standing or sitting, and encourage short, frequent walks to activate the calf pump.

4. Pharmacological:

- While venoactive medications cannot fully prevent varicose veins, they may alleviate symptoms and slow progression in high-risk groups.

5. Patient Education:

- Raising awareness of early symptoms and risk factors facilitates timely intervention and may reduce disease severity.

Prevention is especially critical for individuals with a strong family history or other predisposing factors. Early intervention can reduce disease burden and improve quality of life.

13. Emerging Treatments and Future Directions

Research and innovation in the field of varicose vein treatment continue to expand:

1. New Sclerosants:

- Ongoing development of safer and more efficacious sclerosant agents aims to reduce complications such as hyperpigmentation or systemic effects.

2. Advanced Ablation Devices:

- Improvements in catheter design for thermal ablation may further diminish post-procedural pain and complications.

○ Non-thermal, non-tumescent (NTNT) methods like mechanochemical ablation (MOCA) and cyanoacrylate vein closure (vein “superglue”) could simplify procedures and increase patient comfort.

3. **Bioactive Materials for Ulcer Management:**

○ Novel wound dressings integrated with growth factors or bioactive compounds may enhance venous ulcer healing.

4. **Gene Therapy and Molecular Targets:**

○ Researchers are investigating genetic and molecular pathways linked to venous wall remodeling and valve incompetence. Targeted therapies could one day slow or reverse the pathological changes underlying varicose veins.

5. **Wearable Technology:**

○ Innovations in wearable devices could continuously monitor venous pressure or leg volume, facilitating early intervention and personalized care.

As the pathophysiological underpinnings of varicose veins become clearer, individualized treatment strategies will likely incorporate advanced diagnostics, biologic interventions, and further refinement of endovenous techniques.

14. Conclusion

Varicose veins represent a multifactorial, progressive manifestation of chronic venous disease. Although often dismissed as a cosmetic problem, they can lead to debilitating pain, edema, and ulcerations when untreated. Accurate diagnosis begins with a detailed history, physical examination, and duplex ultrasound to assess the extent of venous reflux and map the affected veins.

Therapeutic strategies have evolved substantially, shifting from traditional surgical vein stripping to more patient-friendly, minimally invasive treatments such as endovenous laser ablation, radiofrequency ablation, sclerotherapy, and mechanochemical ablation. These methods boast high success rates, reduced post-procedural pain, and quick recovery times. Surgical interventions remain pertinent for particular cases with extensive disease or unsuitable anatomy.

Prevention and early intervention are pivotal in reducing disease burden. Compression therapy, lifestyle modifications, and meticulous patient education can help slow progression and avert complications. Moreover, research into novel therapies, advanced ablation techniques, and gene-related interventions heralds further improvements in patient outcomes.

Ultimately, patient-centered management—recognizing individual risk factors, comorbidities, and treatment preferences—ensures optimal care for varicose veins. By integrating evidence-based practices, the medical community continues to refine and expand the repertoire of diagnostic and therapeutic approaches, improving the lives of patients worldwide.

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