CHIVA versus ablation

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Abstract

CHIVA is the French acronym for *Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire*, ie, Conservative and Hemodynamic Treatment of Venous Insufficiency in outpatients. Ablation is not conservative, and CHIVA is based on a different hemodynamic approach. It is counterintuitive because it is difficult to imagine that the varicose veins could disappear without ablation either by extraction or by endovenous destruction. This treatment raises scientific questions that require us to revisit our understanding of classical venous pathophysiology in light of what echo-Doppler has contributed to our progressing knowledge of hemodynamics. CHIVA strategy requires more demanding diagnostic procedures than the ablative methods–in particular, a hemodynamic mapping that considers more elaborate hemodynamic data. Results from both methods allow us to evaluate the relevance of their respective pathophysiological basis. Studies have shown that results with CHIVA are often superior, sometimes equivalent, but never inferior to ablation. Such findings support conservative approaches, justified scientifically for hemodynamic reasons and ethically because of the preservation of the venous bypass capital. The effort made to improve knowledge of hemodynamics, making CHIVA possible, is rewarded by a much more in-depth understanding of venous disease, not only superficial disease, but also deep venous insufficiency and venous malformations.

Keywords:

ablation; CHIVA; great saphenous vein sparing; hemodynamics; shunt; varicose veins; venous disease.

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Introduction

Comparing CHIVA (French acronym for *Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire* or Conservative and Hemodynamic Treatment of Venous Insufficiency) proposed in 1988[1,2] with ablation introduced in 1905[3] cannot be reduced to a comparison of two techniques because they are two treatments based on radically different pathophysiological concepts. This explains the differences in instrumental evaluation and interpretation of data (especially ultrasound), which results in different diagnosis, strategy, tactics, and assessment of results. Due to their different concepts, the same signs, such as flow direction, have the same name of reflux−antegrade and retrograde flow−but differ in pathophysiological meaning. Furthermore, the hemodynamic model that explains CHIVA introduces new definitions such as venovenous shunts (open vicarious shunts, closed shunts, open deviated shunts, and mixed shunts) and dynamic fractionation of gravitational hydrostatic pressure (DFGHSP).[4] This new language is of course shocking to those who have been trained in the classical ablative approach. Their discomfort with it is amplified by the perhaps forgotten, though necessary, knowledge of fluid mechanics required to understand venous hemodynamics and to perform appropriate hemodynamic ultrasound examination.

With regard to CHIVA, flow pathology is not dependent on direction. It depends on its origin, destination, and the transmural pressure (TMP) exerted against the veins and capillary walls. TMP is pressure resulting from the opposition of the outer pressure (tissue + atmospheric pressure) and inner pressure (gravitational hydrostatic + residual pressure provided by the microcirculation + valvo-muscular pump pressure). TMP control is the cardinal function of the venous system, ie, tissue drainage, heart preload, and thermoregulation. The venous system consists not only of veins, but also of the venular side of the microcirculation, the cardiac, thoraco-abdominal, and valvo-muscular pumps. Types of venous dysfunction depend on the damage in this system, which can be valve incompetence, occlusions, or low microcirculation resistance responsible for corresponding hemodynamic pathologies such as DFGHSP impairment, shunts, and resistance to flow, which increase TMP.

Another cause of misunderstanding is the difference in instrumental assessment, especially echo-Doppler. The CHIVA strategy requires much more accurate topographic and hemodynamic mapping than ablation, owing to the greater complexity of the pathophysiological concepts involved.

Treatment is also assessed differently. Occlusion of the great saphenous vein (GSV) is considered an ablative success but a CHIVA failure. Persistent flow is a failure for ablation and a success for CHIVA, even if it remains retrograde if it is no longer overloaded.

The CHIVA hemodynamic model is a fresh approach for diagnosis and treatment of venous malformations and deep venous insufficiency, especially in post thrombotic syndrome.[5]

The final but crucial difference we'll mention for these two approaches is CHIVA's sparing of the GSV, not only to avoid impeding venous drainage but also, above all, to preserve the undeniable potential of vitally important arterial bypass.[6-10]

In this regard, CHIVA methods arose out of concern to preserve the GSV because it was too often unusable for vital bypass procedures because of previous ablation for treatment of benign varicose veins. This raises ethical questions, and discussion on informed consent should stress this issue and offer conservative solutions besides ablation.

CHIVA is at least as painless and unrestrictive as noninvasive ablative procedures because it is open, mini-surgery under local anesthesia, and immediate resumption of walking post procedure is advised.[11,12]

It is lower cost than most such procedures because it requires minor surgical equipment.

Like any scientific model, the CHIVA cure has been subjected to experimental proof and compared with ablative methods, of which stripping is the gold standard. Controlled trials have shown CHIVA to be strongly or slightly better vs all other methods, but its results are never inferior in terms of complications and long-term recurrences.[13-17] Surprisingly, the advantage of GSV preservation is not mentioned in the trials despite its relevant value in terms of health with regard to vitally important arterial treatments in the aging population.

Unfortunately, more widespread use of CHIVA is thwarted by the steep learning curve associated with performing this technique: though previously reported to be better than compression and at least equivalent to stripping of varicose veins in preventing ulcer recurrence, in acknowledgment of the complexity of the approach, it has been noted that "a high level of training and experience is needed to attain the results presented" in that publication.[18] Hindrances to more widespread use of CHIVA thus include the lack of teachers for training in this technique, and the popularity of easier, ready-to-go ablative techniques offered by the sponsors of so many congresses which would not otherwise exist.

Indeed, the CHIVA-based hemodynamic model is not yet taught in most universities and not included at most congresses.

This article, though too short for an exhaustive explanation, will suffice to introduce the basis of the CHIVA cure. For more information, an extensive PDF book published in 2021 can be downloaded free of charge.[19]

Pathophysiology

Cause of varicose veins

In 2017, Jacobs et al[20] looked closely at the pathophysiology of varicose veins, which available evidence suggests is complex an influenced by a number of factors, with the inciting factor not known conclusively. For example, they asked the key question of whether venous hypertension and valvular incompetence lead to alterations in the venular wall or whether it's such changes that lead to venous hypertension and valvular incompetence, something not known with certainty.

If vein wall changes precede venous hypertension and valvular incompetence of refluxing veins, ablation could be justified.

On the contrary, CHIVA considers that venous hypertension and valvular incompetence precede and influence the development of vein wall changes. This is proved by the caliber of reduction[21] and remodeling after shunt disconnection and DFGHSP restoration. In his article published in 2019, Delfrate[22] describes results from a study in 22 patients needing hydrostatic column fracturation 1 year after saphenous femoral disconnection:

In 21 of the 22 they found that the histoarchitecture of the 3 general layers of the GSV was maintained, including the following: (i) the endothelial layer, which remained intact; (ii) the medium layer consisting of 3 different smooth muscle layers showing only mild hypertrophy and hyperplasia; and (iii) the adventitial layer, consisting of nerves and vessels with multiple endothelial cells surrounded by smooth muscle cells.

Cause of varicose recurrence

Bradbury[23] describes varicose recurrence as the development of new varicose veins, often in a second saphenous system, after the original operation. They state possible causes as: i) "inadequate assessment at the time of the initial treatment," though they note that this should be less common since full duplex ultrasound mapping is carried out in most before intervention; and ii) "reflux developing at a site that was previously demonstrated to be competent; in other words, true disease progression."

So, performance of the most extensive ablation possible is justified if true that post-ablation recurrence is not a result of the ablation itself but due to incompetent veins left behind or to true disease progression such as when reflux develops at a site that was previously demonstrated to be competent. However, studies do not support this. Even though varicose recurrence after CHIVA can also be due to untreated incompetent veins and recanalization of ligations rather than new "natural evolution of the disease," only ablation produces neo varicose veins. This is clinically obvious in cases of post-ablation "anarchical" new varices and those with "no apparent source" or "uncertain cause" on echo-Doppler. For example, Perrin et al[24] in their study of cases of varicose recurrence after surgery report no apparent source of reflux in 10%, and uncertain or unknown cause in 35%. Such neo varicose veins are not seen after CHIVA. Carandina et al[25] compared stripping and CHIVA in patients with superficial venous incompetence that resulted in chronic venous disease.

With regard to long-term results, they found that after 10 years, the main between-group difference was that the stripping group had 22% neo varicose veins with no detectable reflux point, a recurrence they believed was due to the absence of an all-important drainage by the saphenous system, something they believed key following varicose vein surgery to avoid neoangiogenesis. In support of this, they point out that even for CHIVA (a conservative surgery), if incorrectly performed and GSV thrombosis and occlusion arise after surgery, impeding drainage, the number of recurrences is higher than in draining GSV systems, and they suggest that this could also be relevant in the case of modern endovascular techniques in which the GSV is removed. They also point out that there is no published long-term evidence to consider with regard to the GSV after endovenous laser ablation, radiofrequencey ablation and foam sclerotherapy

Practically, it can be considered that a number of post-ablation recurrences are due to preexisting thin collateral veins that are dilated and forced by the draining flow (residual pressure [RP]) to bypass the ablated paths that impede tissue drainage.

Cause of venous ulcer

The cause of venous ulcer could be sole or multifactorial depending on the pathophysiological explanation.

The perforator underneath the ulcer is usually considered the cause. According to this assumption, these perforators are ablated (via ligation, sclerosis, subfascial endoscopic perforator surgery [SEPS], or Linton operation) and then discarded due to bad results. Yet, most of them are draining (substantial diastolic inward Doppler flow) despite a little systolic outflow and not considered pathologic in the absence of a deep obstacle downstream. Indeed, the CHIVA model considers the TMP excess to be the cause of the venous ulcer. Ulcer-centered perforators rarely indicate deep venous hypertension but usually drive inward the flow of superficial closed shunts submitted to DFGHSP impairment. So, restoring the DFGHSP and disconnecting the closed shunt at its escape point, CHIVA achieves healing. In this case, ablating the reentry would impair the drainage of the ulcer and, simultaneously, ulcer healing. The reason ablation is used so frequently seems to be because of the systolic reflux elicited by the vessel incompetence of large perforators below the knee, although not pathogenic when it precedes a very substantial diastolic inflow.

This is confirmed by the ulcer healing without any ablation of the ulcer-centered perforator. So, excess TMP is corrected by increasing the extravenous pressure with compression and/or reducing the intravenous pressure (via CHIVA). In 2002, CHIVA disconnection was reported to have less recurrence than compression,[17] and in 2021, endovenous ablation showed similar results.[26] Currently, there are no long-term results comparing CHIVA and ablation.

Vein ablation versus conservation

Ablation suppresses the reflux, but at the same time, it also suppresses flow drainage by the microcirculation. This obstacle to flow drainage can lead to skin conditions, such as telangiectasia, matting, and bypassing varicose veins. As a matter of fact, resistance to the draining flow increases the residual pressure, which opens micro shunts, forces and dilates capillaries, venules, and collaterals. Therefore, CHIVA preserves the veins, even if refluxing, so as not to impede drainage. This explains Perrin et al's finding, as mentioned above, in cases of recurrence after surgery that there was no apparent source of reflux in 10%, and uncertain or unknown cause in 35%,[24] and Carandina et al's findings for a "detectable reflux point" in 0% (0/70 patients) treated via CHIVA in their study vs 22% (12/54 patients) treated with stripping.[13]

In addition, CHIVA-preserved GSV shows a reduction in caliber[21] and normal histoarchitecture.[22]

Reflux ablation

Reflux ablation is mandatory for ablative methods according to the concept that any retrograde flow is pathogenic and the vein it flows through is pathological as well. For CHIVA, direction defines neither pathology nor pathogenicity of any flow. The content of the flow (volume, pressure, source, reentry) is more important than its direction. CHIVA consists of gravitational hydrostatic pressure (GHSP) fractioning, disconnection of the escape points (source) of the closed shunts and the open deviated shunts, and drainage preservation at reentry points. It leaves behind a "physiologic" flow, though refluxing, because it is no longer overloaded and complies with the "hierarchy of drainage." This has been called a "shunt 0," or a "no shunt." In fact, a shunt is a conduit that steals part or all of the flow of another vessel. A venovenous shunt is a vein that drains all or part of the flow from another vein, flow it would not normally carry. The N3, N2, N1 venous network anatomy described in 1999[27] was translated from the French R3, R2, R1 network that was previously described via echo-Doppler in 1988, not only anatomically, but also functionally[1]; there is a drainage hierarchy from the suprafascial tributaries (N3) into N2 (GSV and short saphenous vein [SSV] through duplicated fascia) and then into the deep subfascial network N1 or directly from N3 into N1.

A closed shunt is N2 or N3 overloaded by an N1 through an N1>N2 or N1>N3 escape point that drains into N1 through an N3>N1 or N2>N1 reentry point. The N1>N2 or N1>N3 flow is a "true" reflux because contrary to the physiological hierarchical direction through an N1>N2 or N2>N3 escape point (perforator, saphenofemoral junction [SFJ], or saphenopopliteal junction [SPJ]), it is elicited by calf valvo-muscular pump diastole (squeezing relaxation or Paranà maneuver diastole) and thoraco-abdominal pump systole (positive Valsalva) that drives it backward into N1 upstream of the valvo-muscular pumps. For CHIVA, finding and disconnecting the escape points is crucial (eg, perforator, SFJ, SPJ, or pelvic leak points that are perineal, inguinal,[28] clitoral,[29] obturator, superior or inferior gluteal), whereas GSV endovenous ablation is performed below the descending tributaries of the GSV, so finding the SFJ escape point is not involved. Different types of closed shunts are created (eg, Shunt types I, III, IV, V, VI) according to these escape points and the succession of overloaded N2, N3 tracks. It is triggered by the calf valvo-muscular pump diastole (squeezing relaxation or Paranà maneuver diastole) and the thoraco-abdominal pump systole (positive Valsalva).

Open deviated shunts are generated with N3 overloaded by N2 through an escape point "true" reflux N2>N3 because it's contrary to the physiological hierarchical direction N3>N2. It is triggered by the squeezing relaxation or Paranà maneuver diastole like for a closed shunt, but *not* by the Valsalva maneuver, which is crucial for differentiating closed shunts from open deviated shunts.

Shunt 0 shows only N2 or N3 Paranà diastolic reflux and no N2>N3, N1>N2, or N1>N3 escape point.

Notice that both closed shunts and open deviated shunts work only when the valvo-muscular pump is activated, ie, essentially, when walking.

An open vicarious shunt is any vein−deep or superficial− that bypasses an obstacle. It is overloaded by a flow that is upstream of a block in another vein through an escape point and reinjected downstream through a reentry point. It is triggered by the systole of calf squeezing relaxation or Paranà maneuver, but *not* by the Valsalva maneuver. In some cases, these flows are retrograde (for example at the SFJ in spontaneous Palma), and their ablation aggravates the venous insufficiency!

A mixed shunt is made of the combination of a closed shunt and an open vicarious shunt that share the same escape point activated by both systole and diastole of the Paranà or squeezing relaxation maneuvers. Then, they flow through the same track, which splits into 2 tracks. One is activated only by systole and drains the open vicarious shunt into a specific reentry point. The other one is activated only by diastole and drains the closed shunt into its specific reentry point. So, they have the same escape point but different reentry points. A mixed shunt is fed most of the time by a systolic-diastolic reflux of the SPJ due to a constitutional stenosis of the femoral vein at the Hunter hole and by the SFJ in case of iliac vein occlusion.

By preserving the open vicarious shunt and disconnecting only the closed shunt part of the mixed shunt, one avoids impeding the drainage flow and leaves behind a drainage flow called shunt 0 in the specific track of the previous closed shunt.

Reservoir effect and siphon effect

Varicose veins, especially clusters, are sometimes implicated in reflux and worsening due to their alleged aspirative function, related to a so-called “reservoir effect” and/or “siphon effect” of varicose veins, though clusters do not fulfill the physical conditions for exerting a “reservoir effect” or “siphon effect.” In fact, varicose vein reflux is activated only by the diastolic valvo-muscular pump aspiration, regardless of the presence of clusters or dilated incompetent veins. This eliminates the reservoir effect of the clusters. Moreover, the classic physiologic “reservoir effect” is defined by the capability of the venous bed to amortize the pressure variations owing to its compliance. In physics, the siphon effect is an open circuit, with a pipe with one end immersed in a tank, emerging higher than the surface of the liquid, and then bending downward so that the other open end outside the tank is lower than the surface of the liquid. An incompetent GSV cannot be a siphon because it is not open but closed and connected to the deep veins through the escape and reentry points and has no emerging intermediate segment.

Varicogenesis according to upward or downward progression

Whatever the disputed model of varicogenesis direction, it doesn’t change the CHIVA approach. Varicogenesis could be an argument for intervening to prevent proximal varicose extension by ablating distal incompetent tributaries.

Diagnosis and mapping

As ablation-based concepts need to assess refluxing veins only, the resulting mappings are much simpler than those for CHIVA where the various types of shunts must be identified.

Treatments

What is the strategy for CHIVA or ablation?

CHIVA is usually performed as an outpatient procedure under local anesthesia with few incisions: 1 to 7, with an average of 3 incisions. CHIVA involves: (i) gravitational hydrostatic pressure fractionation; (ii) disconnection of closed shunts and open deviated shunts; (iii) no disconnection of open vicarious shunts; (iv) no vein ablation, particularly of GSV.

Ablation procedures vary according to technique, but involves vein ablation, particularly of the GSV.

What tactics are involved in CHIVA and ablation?

For CHIVA: under local anesthesia, GSV crossotomy (SFJ flush division; no absorbable ligation; no arch, tributaries division, or ligation) or triple saphenous flush ligation (TSFL)[30,31] of the SFJ are performed. Shunts are disconnected at the flush escape points, with no stump.

For ablation: methods vary from use of general anesthesia for stripping to no anesthesia for foam sclerotherapy; it involves GSV crossectomy; open air or endovenous ablation.

Results

CHIVA results can depend on the physician's level of expertise in performing the method: it has been described as being better than stripping if carried out by experts, but less so if carried out by non-experts.[32]

Note, there is no randomized controlled trial showing ablation to be better than CHIVA. However, out of 120 studies and trials about CHIVA, 5 randomized controlled trials[13-17] are favorable for CHIVA.

A recent publication in the *Cochrane Database System Review*[11] describes little or no difference in varicose vein recurrence when comparing CHIVA with either stripping or radiofrequency ablation and also no difference in recurrence or side effects when compared with endovenous laser therapy. However, it mentions a possible slight reduction in nerve injury and hematoma in the lower limb with CHIVA vs stripping, as well as the possibility of more bruising vs radiofrequency ablation. It should be noted that all these findings were based on low-certainty evidence, with limitations named as the small number of trials, the high risk of bias because surgery effects could not be hidden, and imprecision of results because of the small number of events.

Guo et al[12] reported on a study including 39 eligible RCTs (a total of 6917 limbs), determining that CHIVA had the best long-term efficacy (the highest successful treatment rate, with a surface under the cumulative ranking [SUCRA] value of 0.37) and was most likely to achieve the lowest long-term recurrence rate (with a SUCRA value of 0.61). Reliability of the main results was analyzed, with most direct comparisons based on moderate- or high-level evidence Thus, CHIVA appears to have superior clinical benefits with regard to long-term efficacy in varicose vein treatment, though further trials to provide more supporting evidence are needed.

New trials comparing CHIVA and ablation should be carried out to ascertain superiority. Nevertheless, GSV ablation versus a better or at least equally conservative method presents an ethical issue when GSV remains the arterial bypass gold standard versus prosthetics.

Conclusion

CHIVA intervention results, short- and long-term, appear to be better or are at least equivalent to ablation. Furthermore, GSV sparing with CHIVA is a crucial difference between these two approaches in terms of the potential need for vital arterial bypass surgery. The approach taken by CHIVA requires a revisiting of classical pathophysiology, in particular hemodynamics, and therefore an intellectual effort for those trained in the dogmas of ablation. Moreover, this new hemodynamic knowledge offers a new diagnosis and a new therapeutic management of venous disease.

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