## So as to avoid any misunderstanding about Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire (CHIVA)

The article 'Venous haemodynamics: What we know and don't know' is very interesting. But, as Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire (CHIVA) creators and researchers for the last two decades, we must bring some comments in order to avoid any misunderstandings and add some important information. CHIVA is a peculiar venous insufficiency (VI) treatment. Its strategy is designed to improve the drainage of the tissues by the way of excessive transmural pressure (TMP) reduction and draining venous network conservation. So, CHIVA is at the same time conservative and haemodynamic. Its mini-invasive surgical implementation makes it ambulatory.<sup>2</sup>

So, CHIVA doesn't treat only the great saphenous vein reflux due to sapheno-femoral junction (SFJ) incompetence. Actually, the basic CHIVA strategy consists of three principles. Firstly, to interrupt the overloading flows and pressures, by fractioning the column of gravitational hydrostatic pressure and disconnecting ANY shunt (closed or deviating) at the precise escape point (can be any perforator, pelvic escape point, SFJ, etc.).3 Secondly, to preserve the incompetent venous segment (varicose or not, saphenous or not) in order to avoid recurrences (due to substitute veins dilation forced by residual pressure). Thirdly, to check and preserve the re-entry perforators that permit an efficient drainage into the deep venous network. So, contrary to the article statement, venovenous shunts are not interrupted at the second-order tributaries level but at the precise escape points.

CHIVA model is not a reduction to rigid tubes because it involves the passive and active viscoelastic compliance of the venous wall. CHIVA is neither a reduction of the venous physiopathology to haemodynamics, but a comprehensive implementation of the hydrodynamic laws to improve the understanding, the diagnosis and treatment of the VI. On this basis, CHIVA states that VI disorders are due to an excessive TMP responsible for venous dilation and tissue drainage impairment (oedema, hypodermitis, ulcer). The haemodynamic causes are various. They are responsible for TMP excess effects but through different processes. The obstacles to the flow (post-thrombotic and post-therapeutic ablation) are responsible for vicarious varices. The valve incompetence (agenesis,

post-thrombotic, rupture) is responsible for varicose shunts. Obviously, the effects of the excessive TMP are more or less important in proportion to the biological status (venous wall and valve constitutional or secondary weakness, capillary permeability level, tissue adaptation level to drainage impairment). But the haemodynamic factors are necessary, even if not sufficient, to be responsible for VI. So, the biologic factors cannot cause VI if TMP is lower than its pathogenic threshold. As CHIVA consists in identifying and correcting the haemodynamic disorder, it demands a sufficient knowledge of its pathophysiological background and rigorous haemodynamic duplex mapping, tailored strategy and surgical management. Various studies and trials that report clinical and instrumental (APG, AVP, LRR) improvements are not cited in this article.<sup>4–8</sup>

In addition, being conservative, CHIVA doesn't destroy the venous capital that may be necessary in future as arterial bypass in case of coronary or peripheral arterial disease that grow with the continuously ageing population.

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